A PORTFOLIO OF STUDY, PRACTICE AND RESEARCH

Submitted for the
Doctor of Psychology (Psych.D.)
in Clinical Psychology

CONVERSION PROGRAMME

Fiona Barber
1998

Department of Psychology
University of Surrey
ACKNOWLEDGEMENTS

I would like to thank Dr. Christos Pantelis and Dr. Paul Maruff for their helpful advice and comments in the design of this project. Thanks also to Matt O’Brien for his help in recruiting patients and controls.

I am very grateful to Dr Sarah Wilson for her invaluable supervision and, of course to the patients and staff at Royal Park Hospital for their co-operation.

I would also like to thank my parents for their financial and moral support throughout my PsychD.
PORTFOLIO CONTENTS

ACADEMIC AUDIT

Critical Review One
Critical Review two

CLINICAL AUDIT

Clinical Dossier

RESEARCH AUDIT

M.Sc. Thesis

Psych.D Thesis

Copyright (C) Fiona Barber 1998
Intellectual Functioning in Schizophrenia: Are there deficits which are unique to the disorder?

The subject of general intellectual impairment in schizophrenia has long been a focus of interest for neuropsychological research. While it has been established that patients with schizophrenia display greater cognitive impairment than normal/matched controls, a number of questions have been raised with respect to the characteristics of these deficits and their relevance to the aetiology and course of the illness. Specifically, research has been concerned with establishing the extent of cognitive impairment and identifying patterns of performance which are unique to schizophrenic populations. It is hoped that establishing specific patterns of deficit will have diagnostic and prognostic implications for the disease.

The question of specificity is of particular interest to research which is concerned with finding markers of the disease prior to the onset of schizophrenic symptomatology and there have been a number of longitudinal studies which have sought to establish whether there are specific cognitive precursors to the disease. In addition, developmental studies have looked at the prognostic implications of low premorbid IQ.

In this review, the extent to which decline in intellectual functioning is characteristic of schizophrenia as compared to other psychiatric groups or patients with other forms of organic cerebral pathology, for example, dementia and multiple sclerosis, will be discussed. In addition, the issue of whether these deficits are present prior to the onset of the disease is addressed. Methodological issues in the assessment of intelligence are discussed with specific reference to patients with schizophrenia.
The Assessment of Intelligence

Throughout the course of neuropsychological research into schizophrenia there has been overwhelming evidence that patients suffering from the disease display deficits on IQ measures. While these results have been robust and consistent, their veracity is based on the premise that the tests used are valid and reliable indices and that the IQ scores obtained accurately reflect the individual's level of global intellectual functioning or "intelligence". In recent years the problem of accurate estimation of intellectual functioning in patients with schizophrenia has been widely discussed and a number of measures have been employed to assess both current and premorbid functioning. As with all populations, IQ scores for patients with schizophrenia may be affected by a number of confounding variables, such as education and social class, which are intricately linked to the notion of "intelligence" and are difficult to disentangle from it. In addition to these factors, IQ scores for patients with schizophrenia may be affected by symptomatological changes as well as medications and other therapeutic interventions.

In essence, the job of the researcher is to devise a means of assessment which takes all these variables into account and provides as accurate an estimate as possible of the individual's true "intelligence". In the case of premorbid IQ measurement, the test is not only required to account for confounding variables but also to be unaffected by any post onset intellectual deterioration. Thus, the measurement of IQ in schizophrenia is fraught with complications and is highly susceptible to differences in the types of measures used. Some of the methodological problems in assessing cognitive functioning in patients with schizophrenia and practical ways to address them, have been reviewed recently (Nelson, 1994).

Comparison with normal populations, matched controls and other psychiatric patients

As is noted by Aylward et al (1984), the majority of studies examining intellectual
functioning in schizophrenia have been directed towards identifying patterns of cognitive impairment rather than the exploration of global IQ deficits. Despite this lack of interest, a review of the existing literature suggests that patients with schizophrenia display significant impairment on tests of global intellectual functioning when compared to normal controls matched for a number of demographic variables (Pollack et al, 1970; Lehman et al, 1979).

The principle sources of information relating to this issue are the earliest studies of schizophrenia taken before the development of sophisticated cognitive theories and tests. Prior to 1944, the most commonly used scale was the Stanford Binet which provides an age related IQ score. While many early investigators reported low IQ scores for patients with schizophrenia (Roe & Shakow, 1942) on this scale, very few were able to make effective comparisons to normal subjects as the test is not standardised for adults. Despite these limitations, Hunt and Cofer (1944) reviewed the best-controlled studies and found an average intellectual deficit of 20 months for patients with schizophrenia.

Since the introduction of the Weschler Bellevue Scale (1944), the focus of cognitive research has shifted towards analysis of patterns of impairment as measured by subtests within the overall scale. The few studies investigating global IQ deficits in schizophrenia have consistently reported performance deficits in patients with schizophrenia (Payne, 1960). More recent studies in this area using normal controls matched for socio-economic status (SES) and age have supported the conclusion that patients with schizophrenia display performance deficits on measures of overall intellectual functioning such as the WAIS and WAIS-R.

There have been relatively few studies investigating the extent of global intellectual impairment in schizophrenia as compared to patients with other psychiatric diagnoses. In a review of the existing literature Payne (1960) concludes that adult patients with schizophrenia show greater IQ deficits than patients with neurotic disorders or alcoholics and that patients suffering from personality disorders or other psychotic
disorders display similar IQ deficits to those of patients with schizophrenia.

Similar results were reported by Cullari (1985) in a study of patients with schizophrenic disorders, affective disorders and 'other' nonpsychotic psychiatric disorders. Patients were matched for years of education although there was no mention of matching for age or length of illness. The patients with schizophrenic disorders had significantly lower full scale IQ scores than the affective group but not the 'other' group. In addition, only the patients with schizophrenia revealed significant differences between scores on the verbal and performance sections of the test.

A more recent study by Goldberg et al (1993) compared patients with schizophrenia to those with unipolar depression and bipolar disorder. Patients with schizophrenia displayed significantly lower full scale IQ scores than either of the other groups, as measured by the Weschler Adult Intelligence Scale-Revised (WAIS-R; Weschler 1981).

Thus, there is little doubt that patients with schizophrenia have lowered IQ scores compared to normal controls. It would appear that these deficits are not present in other, unipolar psychiatric populations although evidence suggests that other psychotic disorders are also associated with IQ deficits. It is evident that the majority of the research in this area is somewhat outdated and as such, suffers from a number of methodological problems in comparison with more recent studies. The majority of the research was conducted prior to the development of sophisticated cognitive theories of intellectual functioning and thus the concept of global IQ is poorly defined. The heterogeneous nature of the disease is not addressed by these studies and it is only recently that symptom rating scales and separation of clinical subsyndromes has been conducted. In addition, other factors such as ECT treatments, medication and length of hospitalization are not controlled for.

Comparison between Patients with Schizophrenia and Neurological Patients

Fiona Barber
Critical Review 1
PsychD.
In addition to comparisons with other psychiatric populations, investigators have sought to establish whether patients with schizophrenia may be distinguished from those with organic brain damage on the basis of qualitative or quantitative differences in cognitive functioning. Research in this area has examined global intellectual impairments as well as patterns of performance on batteries of tests.

Studies using the Weschler IQ scales have yielded inconsistent results with respect to this issue. Watson (1965) compared patients with schizophrenia to two groups of patients with cerebral lesions categorised as high and low hospitalisation. Organic patients included those with trauma injuries, syphilis, arteriosclerosis and cerebrovascular accidents. Subjects were matched for age, length of hospitalisation and education. The results showed that both groups were characterised by higher Verbal IQ scores than Performance IQ scores. This discrepancy is not found in healthy controls nor affective disorder groups (Cullari, 1985). In addition, Watson (1965) reported that scores on the digit-span subtest were significantly higher for the group of patients with chronic schizophrenia than for the long-term patients with brain damage.

Similar findings were reported by DeWolfe et al (1971) who divided both groups of patients according to their age. Using the WAIS the authors found that the patients with schizophrenia performed significantly better on the digit-span subtest but more poorly on the comprehension test. In the group of older patients, the group with schizophrenia displayed significant deficits on the picture completion as compared to brain-damaged patients.

In contrast to these results, Chelune et al (1979) failed to find any significant differences between patients with schizophrenia and those with diffuse brain damage on the WAIS subtests. As in Watson’s study, organic patients were divided into acute and chronic groups with the former including patients with trauma, hydrocephalus and anoxia, and the latter chronic group including patients with chronic seizure disorder and dementia. The results showed that the mean level of performance of patients with schizophrenia was significantly higher than that of brain damaged
patients. Pattern analysis of the subtest scores, however, revealed no significant differences between the two groups. Other authors have also failed to replicate DeWolfe (1971) and Watson's findings (Davis et al, 1972).

Investigations using alternative test batteries have yielded more useful results in terms of their power to discriminate between the two groups. Purisch et al (1978, as cited in Goldstein 1986) used the Luria-Nebraska neuropsychological test battery (LNNB) to assess patients with chronic schizophrenia and those with brain injury. The tests fell into 10 categories including memory, motor, expressive and impressive speech and intellectual. Patients with schizophrenia performed significantly better on all but four measures of the test. The authors concluded that chronic schizophrenia produced deficits on more complex cognitive tasks, which they defined as memory, intellectual processes, impressive speech and acoustico-motor whilst simpler, less demanding tasks such as reading and writing remained unaffected. In the case of brain injured patients, however, the ability to do all tasks was impaired. In a replication of the Purisch study, Shelly and Goldstein (1983) produced similar results although the level of discrimination between the two groups was not as robust. The authors commented that this may have been due to different levels of impairment in the brain damaged patients tested.

Results from the above studies provide a number of interesting conclusions: Patients with schizophrenia display many neuropsychological similarities with brain damaged groups and show no specific pattern of WAIS subtest scores that can effectively discriminate them from an organic group. While the LNNB provides more evidence for distinctions between the two groups it is not yet clear whether the pattern of scores is specific to patients with schizophrenia or is of any diagnostic significance. One of the major difficulties of all these studies is their use of patients with a wide variety of organic aetiologies. It is likely that there are substantial differences within the groups of ‘neurological’ patients used which would be reflected in their overall IQ scores. For example, a seizure disorder may have a different neuropsychological profile to a dementia. In essence, there is an assumption that diffuse cerebral damage will
inevitably lead to a drop in IQ level and that this drop would be roughly equivalent regardless of the aetiology of the damage. While some studies have attempted to overcome these differences by dividing groups into chronic versus acute patients, this distinction does not take aetiology into account. It would be more helpful to compare patients with schizophrenia to circumscribed groups of brain damaged patients with similar aetiology and degree of severity. In this way, more precise definitions of brain damage and the differences between groups would be obtained.

Goldstein (1986) attributes the failure of neuropsychological tests to discriminate between schizophrenic and brain damaged groups to the fact that there are two subtypes of schizophrenia one of which is associated with a dementia like organic illness while the other is more functional and transient in nature. Thus, impairment displayed in neuropsychological tests may be associated with structural, organic damage or with attitudinal, attentional and motivational factors. As yet, there has been no research which has attempted to look at IQ differences between different subtypes of patients with schizophrenia.

An important consideration when discussing these studies is that of appropriate matching of the brain damaged and schizophrenia groups. In terms of levels of functioning, groups may be matched on premorbid IQ scores. This method reflects their optimum levels of functioning but is unrepresentative of the impairments existing at the time of testing. The alternative strategy would be to match groups on current global levels of functioning, however, it is unclear as to whether it is appropriate to accept these scores at face value when both groups may have suffered considerable decline in global intellectual functioning for potentially different reasons. Future studies need to take account of these issues if the nature of the neuropsychological deficits in schizophrenia are to be clarified.

**The Relationship of Overall Intellectual Ability to Specific Cognitive Deficits**

While the study of global intellectual abilities in schizophrenia has been largely
eclipsed by studies of specific cognitive functioning the former plays a vital role in determining the true nature of the specific deficits. In essence, researchers must establish whether a specific impairment found occurs over and above any general intellectual decline present. The standard approach to this question has been to administer large neuropsychological test batteries to patients in the hope that one specific function will be selectively impaired above and beyond a generalised deficit. This research paradigm has been subject to criticism on the basis that tests used are not matched for difficulty nor normed on the same populations. In addition, variables such as age, medication, side-effects and symptomatology may also affect results.

Many of these confounding factors are mentioned by Braff and colleagues (1991), in their study of chronic outpatients with schizophrenia. By comparison with healthy controls the patients with schizophrenia displayed deficits on a wide range of tests including conceptual reasoning, psychomotor speed and motor/sensory perceptual abilities. In contrast to many previous studies performance on the Wisconsin Card Sorting Test (WCST) was relatively intact, providing little evidence for a specific frontal type deficit. The authors commented that medication, symptom differences and the alternative forms of the WCST may account for discrepant results between studies. Saykin et al (1991) attempted to avoid the confounding affects of medication by testing unmedicated patients with schizophrenia on a battery of neuropsychological measures including many WAIS-R subtests, tests of learning and memory, attention and abstraction. Results showed that patients displayed a generalised impairment as compared to normal controls as well as specific deficits in learning and memory. These results have been challenged by Blanchard and Neale (1994) who question the statistical design of the study with relation to their use of standardised residualised scores. These authors cite Chapman et al (1989) who states that this method should be interpreted with caution due to psychometric artifacts.

In contrast to Saykin et al's findings, Blanchard and Neale (1994) found no evidence for specific cognitive deficits. The study involved the administration of a comprehensive battery of tasks to 28 unmedicated patients and normal controls...
matched for age, education and handedness. The study analysed the results using four different methods: First, the reliability of the tests used was computed and the missing data examined. Second, a series of ANOVAs and a MANOVA were performed to measure the differences between patients with schizophrenia and controls on individual tests. Composite scores were then created which were thought to reflect global cognitive functions, for example, semantic memory, abstraction and motor ability. Finally, the clinical significance of the patients' impairments was evaluated by classifying each score obtained within either the impaired or non-impaired range for that test. The percentage of patients performing within the impaired range was calculated for each task. The patients with schizophrenia showed impairment on all the tests of both right and left hemisphere functioning regardless of the method of analysis used. The authors concluded that the results refuted claims of differential cognitive deficits in schizophrenia.

An alternative strategy to assess the significance of global deficits for specific abilities is to measure the correlations between full scale IQ scores and performance on neuropsychological tests. Warner et al (1987) divided patients with schizophrenia into five groups on the basis of their IQ as measured by the WAIS or WAIS-R. Patients then completed the Halstead-Reitan Battery, the WMS and the WRAT (wide range achievement test). The results showed that IQ was strongly correlated with scores on tasks of problem solving, memory measures, tactual imperceptions and academic achievement. Full scale IQ scores were less related to motor functioning and sensory suppressions. From the results presented above it is possible to infer that tasks requiring problem-solving or memory may be particularly affected by deficits in global intellectual functioning and that subjects with low FSIQ scores will perform more poorly on these tasks than those with high IQs. Thus, the discovery of specific deficits in these areas may merely be a function of a general learning disability and not represent an isolated impairment. As such, the above study emphasises the need for accurate matching of healthy controls and patients on the basis of IQ prior to undertaking neuropsychological tests.
An interesting study also addressing this issue is that of Smith (1964) cited earlier. In his longitudinal study, Smith found evidence for improvements in IQ scores over an eight year period. The study also tested patients on the Weigl Sorting test which is used to investigate set shifting ability in frontal lobe lesions. At first assessment 10 of 48 subjects could not complete the necessary shift in sorting category. At eight year follow-up only 16 patients were still able to complete the shift. The authors conclude that global IQ measures are insensitive to deterioration in chronic patients and that more specific deficits are developing despite stable IQ scores.

It is evident that the literature in this area is plagued by statistical and psychometric difficulties. Whilst it is unclear as to whether specific deficits exist over and above global intellectual deficits, the evidence would suggest that the use of large batteries of neuropsychological tests to achieve this end is an inadequate and error prone procedure. Alternative procedures such as that used by Warner et al may be more useful in determining the precise effects that global intellectual impairments have on neuropsychological test scores.

**Premorbid intellectual impairment in schizophrenia**

While the findings from studies of IQ in schizophrenia have been relatively robust, it is interesting to look at the deficits in the context of research looking at premorbid functioning. In this respect it is interesting to establish whether any deficits found are specific to patients who go on to develop schizophrenia or if they are present in individuals who go on to develop other psychiatric disorders. In addition, the question of whether specific deficits are present over and above generalised IQ deficits in preschizophrenic children is of interest to research.

In order to examine the role of intellectual impairments in schizophrenia it is necessary to investigate whether deficits are evident prior to the onset of symptoms or if they emerge simultaneously with the acute phase of the illness. There is growing evidence that a significant number of adult patients with schizophrenia exhibited
cognitive deficits as children (Offord, 1974). Studies in this area have used a variety of control groups including peers matched for age, sex and SES as well as non-schizophrenic siblings.

In a series of retrospective studies Offord and Cross (1971; Offord, 1974) examined the school performance of adult patients with schizophrenia by comparison with both their siblings and matched age mates. The authors reported that pre-schizophrenic children were significantly more likely than their siblings to have repeated a grade in elementary school (that is, failed to progress to the next grade due to scholastic failure) and to score lower on childhood IQ tests than both siblings and peers. A number of other interesting issues arose from their analysis: within the group of pre-schizophrenic children, those with lower IQs show an earlier onset of the disease and remain institutionalised significantly longer. This result was supported by Pollack et al (1968) who found that low IQ score at hospitalisation was related to poor adjustment after discharge. In addition, within-group analysis revealed that the school performance of pre-schizophrenic children is consistently poorer than their siblings only when the subject is male and from a low IQ sibship (that is, when both children have an IQ below 80 points). The authors conclude that within a family which is predisposed to schizophrenia, the child with the lowest IQ is the most vulnerable to the disease. Thus, it is proposed that high IQ acts as a protective factor, preventing predisposed individuals from becoming overtly schizophrenic. In contrast, low IQ gives little protection and the child with a low IQ will break down earlier and/or more often than a child with a high IQ score.

In support of these findings, Grimes and Walker (1994) examined the relationship between pre-schizophrenic subjects' emotional expressions (from home movies), educational level and age of illness onset. The authors found a significant correlation between educational level and age of illness onset.

Goodman (1987) examined a sample of young children (0-5 years) whose mothers were suffering from either schizophrenia or a depressive illness. Children were tested
on three occasions at one year intervals using the Bayley scales, the McCarthy scales and the Mental Development Index. The children whose mothers suffered from schizophrenia scored significantly lower on IQ measures at first testing (<1 year) and at subsequent testing they were "overrepresented in the lowest scoring group". In addition, mothers with lower IQ scores tended to have children with low IQ scores.

Overall, results from studies of pre-schizophrenic and high risk children would suggest that cognitive deficits are evident prior to the onset of psychotic symptoms. However, a number of these studies have noted that the deficits found were not specific to pre-schizophrenic offspring; children with behavioural disturbance also displayed lower IQ scores than their peers. In addition, there is a consistent sex difference with pre-schizophrenic males scoring less well than their female counterparts.

Longitudinal Studies

In order to evaluate the significance of premorbid IQ deficits with respect to the course and onset of schizophrenia many authors have undertaken longitudinal studies of children at high risk of developing schizophrenia, that is, children who have one or both of their parents diagnosed with schizophrenia. The majority of this research has been concerned with identifying specific areas of deficit, such as attention or memory, in an attempt to find characteristic patterns of impairment which predict disease onset and course. Overall IQ level has also been recorded in most of these studies.

Interest in neurodevelopmental aspects of schizophrenia has only recently become a focus of research and many of the subjects involved in prospective studies have not yet entered the major risk period for schizophrenia. The New York High Risk Project (Erlenmeyer-Kimling & Cornblatt, 1987) is a longitudinal, prospective study comparing high risk children with offspring of parents with other psychiatric diagnoses and children whose parents had no mental disorder. Testing commenced in 1971 when the children were between 7-12 years old and has continued at two to three
year intervals. Assessment on the Weschler IQ scale (WISC) was conducted at entry to the study and approximately 6 years later. Erlenmeyer-Kimling & Cornblatt (1987) list high IQ score as a possible protective factor for children at high risk of schizophrenia. Results from the study have shown that the first five high risk children to be hospitalised had a mean IQ of 11 points below the mean for the group as a whole. Thus, the authors suggest that low IQ may be related to early onset of psychiatric illness.

The results of a British Population Survey have also provided information relating to general intellectual functioning in childhood and later onset of schizophrenia. Jones et al (1994) reported the results of educational tests taken at ages 8, 11 and 15 years. Tests included reading ability, vocabulary, arithmetic, verbal reasoning and non-verbal skills. In all test categories, children who went on to develop schizophrenia in adulthood performed more poorly than controls. The effect became more robust with increasing age. The association between test scores and schizophrenia showed a linear trend thus predicting a higher frequency of the disease in those children with educational difficulties.

The most advanced longitudinal study to date is the Israeli High Risk project which has produced data at 25 year follow-up (Mirsky et al, 1995). While full IQ tests were not performed in this study, other neuropsychological measures were employed which included subtests from the WAIS. The authors reported that children who went on to develop schizophrenia showed a significantly lower level of arithmetic proficiency. In addition, their performance on other tests of cognitive functioning such as the Trail Making Test, the Stroop and the Wisconsin Card Sorting Test was lower than relatives and control populations. At age 26, the high risk group scored worse than controls on all 6 WAIS subtests used. The most striking finding was that of a consistent attentional deficit, identified in children at age 11, which was specific to high risk subjects.

In essence, studies of pre-schizophrenic and high risk children have provided some
evidence that IQ deficits exist prior to the onset of overt symptoms. In order to evaluate the significance of this finding it is necessary to undertake longitudinal studies which assess the importance of premorbid IQ deficits with respect to disease onset and course. Whilst results from these studies are interesting it is unlikely that overall IQ scores will ever be used as a predictor of disease vulnerability. In isolation, an overall IQ deficit may be indicative of a number of disorders and is found in children at risk of other psychiatric illnesses as well as those with behavioural disturbance. At best, low childhood IQ may become one of a number of predictive factors to be considered when assessing susceptibility to schizophrenia. Thus, more recent research has focused on the identification of patterns of cognitive impairment, for example in attention or memory, that are specific to pre-schizophrenic subjects.

With respect to the neurodevelopmental hypothesis of schizophrenia, it has become evident that behavioural dysfunction and cognitive impairments precede the onset of overt symptomatology by a number of years. Walker et al (1994) for example, found that neuromotor deficits occur in pre-schizophrenic children at as early as 2 years. In addition, specific deficits in attention have been found to occur in high risk children who later develop the disease (Erlenmeyer-Kimling & Cornblatt, 1987). Walker (1994), cites these findings as evidence that schizophrenia originates in a congenital central nervous system impairment which is expressed behaviourally in different ways as the CNS matures. Thus, a neurodevelopmental process underlies the different expressions of the disorder at different life stages. In this way, the onset of psychotic symptoms in early adulthood is one point in the expression of the developmentally mediated neuropathology in the same way that neuromotor disturbances a age 2 years are a prior manifestation of the same neuropathological process.

**Summary and Conclusions**

The above review has attempted to answer the question as to whether there are deficits in global intellectual functioning that are specific to patients with schizophrenia. That cognitive impairment exists in schizophrenia appears to be undisputed and the vast
majority of studies have shown that patients display significantly lower scores on IQ tests than healthy control subjects. However, when compared to other groups of patients with organic pathology it would appear that there are very few indications that these deficits are specific to the disorder. Patients with other psychotic illnesses and those with diffuse cerebral pathology also display IQ deficits as compared to normals.

One potential source of further interest was the finding that patients with schizophrenia show a greater verbal IQ/ performance IQ discrepancy than patients with affective or other non-psychotic psychiatric disorders. The Verbal/performance IQ discrepancy was also evident in patients with other forms of organic brain damage and thus may provide a means of distinguishing patients with schizophrenia from those with other, non-organic forms of psychiatric illness.

In terms of childhood markers of the disease, studies have shown that both high risk and pre-schizophrenic children display lowered educational levels when compared to their siblings and peers. In addition, evidence has indicated that lowered IQ scores in childhood are correlated with earlier onset of overt symptoms of the disease. While lowered IQ scores are not specific to pre-schizophrenic children it has been considered that they may be one of a number of risk factors which, in combination, increase the likelihood of developing the disease early in life. Thus, global IQ deficits may have some prognostic value despite not being specific to schizophrenia.

Research in this area is plagued by difficulties and attempts to resolve research issues have been complicated by the presence of a number of confounding variables such as medication and institutionalisation, all of which may contribute to lowered intellectual functioning. Further, the difficulties of finding appropriate control groups is evident. Studies comparing patients with schizophrenia to those with other forms of cerebral pathology have used groups which include an array of different disorders which, while associated with lowered IQ are also characterised by some specific cognitive deficits which adds confusion to the interpretation of the results. In addition, the heterogeneity of symptoms associated with the term schizophrenia may each be
associated with different patterns of cognitive impairment (Liddle, 1987). It is not surprising therefore that the question of whether there are deficits which are specific to schizophrenia remains largely unanswered and that many studies have yielded inconclusive or contradictory results.

More importantly, the study of global intellectual deficits in schizophrenia has largely been replaced by more sophisticated investigations of isolated cognitive deficits which may be specific to the disease. Research in this area has produced significant advances in our understanding of the symptoms and brain areas associated with schizophrenia in a way that global IQ scores are unable to do. Nevertheless, there are some interesting findings and areas which may be of value to research further, for example, childhood markers and verbal versus performance IQ deficits. While these deficits may not be specific to schizophrenia they may provide means of distinguishing patients with the disease from other psychiatric populations.

References


Offord, D.R. & Cross, L.A. (1971) Adult schizophrenia with scholastic failure or low


The perception of schizophrenia as a degenerative disorder has been evident from the initial descriptions by Kraepelin of a dementia praecox (Kraepelin, 1913). Evidence to support this opinion has been sought from studies of intellectual functioning with the expectation that patients with chronic symptoms of the disease will show a progressive deterioration in IQ across the lifespan. Such a pattern has not been consistently identified and alternative theories have been proffered; for example, some recent descriptions of the disease observe that deficits remain relatively stable, analogous to a 'static encephalopathy' (Goldberg et al, 1993). Work in this area is, as yet, inconclusive and a number of different research paradigms have been employed to establish if and when intellectual deterioration is occurring.

Studies examining intellectual functioning across the course of schizophrenia have sought not only to establish whether there is a significant decline in IQ scores following onset of overt symptomatology but also whether deterioration is a continuing process or if a stable deficit state is reached. In addition, investigators have attempted to determine whether any apparent loss in functioning is regained as psychotic symptoms subside. The literature relating to these issues is discussed.

**Premorbid - Postmorbid Intellectual Changes**

**Within Patient comparisons**

A useful strategy in assessing the degree of intellectual deterioration after the onset of illness has been to compare current IQ level with estimates of premorbid IQ. Nelson and colleagues (1990) identified intellectual deficits in a group of 62 patients with...
DSM-III-R (APA, 1987) schizophrenia who had been chronically hospitalised. Using the National Adult Reading Test (NART; Nelson, 1982) and the Schonell Graded Word Reading Test (Schonell, 1942) estimates of premorbid IQ were compared with current IQ as measured by the Weschler Adult Intelligence Scale-Revised (WAIS-R; Weschler, 1981). These patients were found to have a significant drop of about 13 IQ points from their premorbid estimates. In another recent study of a chronically hospitalised group of patients, most with a diagnosis of schizophrenia, Dunkley & Rogers (1994) used similar measures to estimate current and premorbid IQ. They found that 67% of patients completing these assessments showed a significant deterioration from estimated premorbid IQ, while a total of 65 patients were noted to have cognitive deterioration when other measures were also used to provide a cognitive impairment score (CIS). These studies suggest that those patients with chronic schizophrenia suffer a decline in general intellectual functioning from premorbid levels.

There is also evidence to suggest that patients with schizophrenia suffer a greater decline in intellectual functioning than other psychiatric groups. Goldberg et al (1993, as cited earlier) used the Wide Range Achievement Test- Revised (WRAT-R) reading test as a measure of premorbid IQ to assess cognitive functioning in patients suffering from schizophrenia, unipolar depression and bipolar disorder. The results showed that all groups had equivalent scores on the reading test indicating that premorbid intelligence was similar for all three groups. On testing with the WAIS-R it was evident that only the patients with schizophrenia showed a deterioration from premorbid levels.

While the use of within-patient comparisons may be a convenient means of assessing deterioration in functioning it is important to note that the use of the NART and other verbal tests have not been fully validated in patients with schizophrenia. The justification for the use of the NART as an estimate of premorbid functioning is that verbal abilities are better preserved in dementia than are other intellectual functions and as such, will provide the best estimate of how an individual was functioning prior
to the onset of the intellectual deterioration. However, it has been disputed that this preservation is apparent in patients with schizophrenia, that is, reading ability may deteriorate as intellectual functioning declines. Thus, verbally based tests of premorbid ability may not provide an accurate assessment of IQ prior to disease onset. However, if reading abilities are affected by the pathological process underlying schizophrenic illness then the use of the NART will lead to an underestimation of premorbid IQ and a consequently more conservative estimation of the extent of deterioration. In essence, studies reporting deterioration in IQ based on reading ability tasks may be underestimating the level of deterioration but will not be overestimating it. As such these studies provide stronger evidence for postmorbid deterioration in IQ levels.

**Follow up studies from pre-morbid state**

A more reliable source of information regarding post-onset decline in IQ is that derived from longitudinal follow-up studies. One of the earliest studies which compared premorbid and post-onset scores was that of Rappaport and Webb (1950), who used a group of ten subjects who had been tested during high school prior to any hospital admissions. In contrast to most studies of patients with schizophrenia, the majority of those tested were female (90%). Those subjects who had been admitted to hospital with a diagnosis of schizophrenia were reassessed using the same battery of tests as those used in school as well as subtests of the Weschler Bellevue scale. They found that in nine cases there had been a significant decline in IQ with the mean premorbid IQ being 97.6, while the mean present IQ was 63.9. The mean period of time that these patients had been in hospital was 22.3 months. The authors attributed the deterioration to an "operational loss" related more to attitudinal factors than an essential organic loss.

A commonly used source of information for premorbid IQ data has been the army which routinely assesses all new recruits on entry to the forces. Lubin et al (1962) used the Army Classification Battery, to assess army recruits who had later been hospitalised for schizophrenia and compared the results with assessments taken at the
time of recruitment. Patients showed a drop of one sixth to one third of a standard deviation in their scores on four of the subtests administered: Reading and Vocabulary, Army Clerical Speed, Arithmetic Reasoning Test and Mechanical Aptitudes Test. While these results are consistent with those of Rappaport and Webb the levels of deterioration were significantly greater in the earlier study. Lubin and colleagues attributed this discrepancy to the different lengths of hospitalisation between the two groups. Patients tested by Rappaport and Webb had been hospitalised for periods of up to 81 months, whereas all of the patients in Lubin et al's study were tested within 3 months of admission. It was suggested that patients with chronic schizophrenia with long hospitalisation showed more deterioration than acute, recently hospitalised patients. In addition, all patients tested by Lubin and colleagues were male whilst Rappaport and Webb's sample consisted almost entirely of women.

Further evidence of deterioration in cognitive ability following the onset of schizophrenia was reported in a study of conscripts to the Canadian Army, by Schwartzman and Douglas (1962). They retested 80 male veterans with the Canadian Revised Examination M, who had been given the exam on recruitment in 1943. The test comprises 3 parts: non-verbal, mechanical and verbal. A perfect score on the test gives a M score of 211 and the mean score is 127 (standard deviation 32.2).

By 1952, 50 of these men had been hospitalised with a diagnosis of schizophrenia. At the time of testing thirty of these patients with schizophrenia had been in hospital for a period of six months or more and the other twenty ("ex patients") had been discharged for over a year. The remaining thirty subjects served as normal controls. All subjects were well matched in terms of intelligence at the time of enlistment, age, educational level, occupational status and time interval between the two test administrations. The patients and ex-patients were matched for total number of years hospitalised (mean 2.4 years). The control group scored significantly more on retesting from their original M scores (mean difference was 11.8 points approx equivalent to 6 IQ points) while both the patient and ex-patient groups scored significantly less than on first testing (mean difference -15.2 and -5.1, respectively). Interestingly, when the patient groups were
subdivided into chronic and acute groups significant differences were found between the extent of intellectual deterioration found. The chronic group lost an average of 10.5 points more than matched acute patients.

It is interesting to compare studies using premorbid IQ data from adulthood with those which assessed subjects in childhood. Albee et al (1963) extended their study of preschizophrenic children, cited earlier, who had been tested in 2nd and 6th grades using the Stanford Binet, Kuhlmann-Anderson and Cleveland Classification Test. As noted previously, the authors found that pre-schizophrenic children had lower IQs than their siblings and that these children showed a significant decline in IQ between first and second assessments. Subjects who had become schizophrenic were tested in adulthood using the Weschler Bellevue scale. No significant differences were found between childhood and adult IQ scores and the authors hypothesised that intellectual deterioration associated with schizophrenia begins in childhood and is not initiated by the onset of symptoms.

Some inconsistencies in the above results would seem inevitable considering the widely differing methodologies used. Each of the studies used different assessment procedures and tested subjects at different ages without specifying the different length of illness of the patients. In addition, the majority of the reported data was recorded 30 to 40 years ago when the neuropsychological study of schizophrenia was in its infancy. Since that time many sophisticated theories of cognitive functioning in schizophrenia have emerged and the diagnostic criteria used are more refined. In addition to concerns regarding diagnostic differences in subject selection, authors have questioned the comparability of results between studies which have used different testing procedures. The study by Albee and colleagues (1963) used different assessment batteries in adulthood to those used premorbidly. In addition, the tests they used were substantially different to those administered in the Army Batteries, specifically with regard to the number of speeded subtests. Albee et al proposed that these tests would be particularly susceptible to changes in motivation and attention that are integral to schizophrenic symptomatology. To counter these criticisms
Schwartzman and Douglas (1962) controlled for the speed factor in their tests and found that although the patients with schizophrenia continued to manifest a deterioration from premorbid levels by comparison with controls, this decrement was reduced.

The type of assessment used by researchers provides a vital clue to determining the extent and, possibly, the causes of post-onset intellectual deterioration. Controlling for the demands of speeded tasks has shown that large IQ decrements in patients may be accounted for by symptom factors such as decreased attention or avolition. Rappaport and Webb (1950) attempted to delineate the role of attitudinal factors in their study by using the Elgin Test Reaction Scale, an observational measure of motivation, self confidence, effort and attention. They concluded that the significant intellectual decrement found in their sample was "closely related to attention, concentration, negativism preoccupation and apathy". As such it is difficult to say with any certainty whether post-onset deterioration in intellectual functioning is a manifestation of organic brain changes or merely a functional deficit which is reversible following the remittance of overt symptomology. The most effective way of resolving these issues is to look at studies which have examined changes in postmorbid intellectual functioning.

**Post-Onset Intellectual Functioning**

Despite the evidence for a post-onset deterioration in intellectual functioning the assumption that schizophrenia inevitably involves progressive social and intellectual decline as well as worsening symptoms is true for only a proportion of patients. Even Kraepelin (1913), the originator of the term "dementia praecox" revised his initial description when 13% of his own patients showed complete recovery despite long term hospitalisation. In terms of intellectual functioning, it would be inconsistent with the notion of a dementing illness to find that impairments on tests of intellectual functioning improved over time when symptom severity reduced. Such a result would suggest that the cognitive impairments found were not reflective of a dementing process but were related to more functional factors such as motivation and attention.
Alternatively, actual organic damage may be present but that some functional recovery has been possible. Either way, any improvement on tests of intellectual functioning would counter-indicate a dementing process underlying the disease. The literature presented in this section offers two outcome possibilities: (i) that intellectual deterioration in patients with schizophrenia is mediated by symptom severity rather than length of illness, and (ii) that patients reach a stable deficit state after which no further intellectual decline occurs. There is a large body of literature in this area which uses both cross-sectional and longitudinal paradigms. While the former has the potential to span all decades of life, the latter is generally perceived to provide greater accuracy due to its implicit, within-subject design.

Evidence to suggest that intellectual functioning is mediated by symptom severity has come mainly from longitudinal studies. One of the most often cited, short term, longitudinal studies reported is that by Payne (1960) who tested patients on admission to hospital and again 4-13 months later when the patients were still hospitalised. The average IQ fell by 7.6 points on the Weschler scales. In a continuation of their Canadian Army study, cited earlier, Schwartzman et al (1962) conducted a second follow up of 23 patients who had shown an intellectual decrement from premorbid scores. The authors tested these subjects in 1960, a total of 17 years since their original army entrance. The ten patients who had remained in hospital for that time had suffered further intellectual decline which was equivalent to the loss they had sustained between baseline and the first assessment (that is a total of 30 M score points or approx 15 IQ points). The authors also noted that the range of impairments, as measured by individual subtests, increased across the time interval. Of the 13 patients who had been discharged from hospital, their assessed IQ level had improved between the second and third assessments and was not significantly different from their premorbid IQ level (that is an improvement of between 5-15 M points). Thus, for those patients who had remained in hospital there had been a general decline over a 17 year period but for those subjects who had displayed symptomatic remission, functioning was restored to premorbid levels. This study would suggest that the initial loss in intellectual functioning suffered by patients with schizophrenia was not necessarily irreversible. In addition, the study raised questions concerning the possible
effect of chronic hospitalisation and the effects of treatment on intellectual functioning.

More recent research has investigated the specific relationship between IQ and symptomatology. Addington et al (1991) reported significant associations between impairment on tests of general intellectual ability (WAIS) and negative symptoms. The authors also noted that improvement in positive symptoms was related to increased IQ scores while negative symptom improvements were unrelated to cognitive functioning.

Support for non-progression of intellectual decline has also been found in early, longitudinal studies. Smith (1964) assessed two groups of hospitalised patients with schizophrenia: a young group of 11 patients (mean age 34 years, mean length of hospitalisation eight years) and an older group of 13 patients (mean age 53 years, mean length of hospitalisation nine years). Patients were assessed using the Weschler Bellevue scales at an 8.4 year interval. Each group showed some increase in full scale IQ score with the younger group improving by a mean of 6 IQ points and the older group showing a lesser improvement of 2 points. This was taken as evidence against the concept of progressive deterioration in chronic schizophrenia although the authors warned that their sample was atypical (the older group were not diagnosed until after age 40 years) and their results should not be generalised across schizophrenia as a whole.

Even stronger evidence for the assertion of stability in intellectual functioning is provided by Hamlin (1969). He retested Smith's subjects after a further six years and found no evidence for deterioration of intellectual function. In support of Schwartzman et al (1962), Hamlin also found that in cases where the psychosis had improved so had the intellectual functioning. Hamlin stated that "the psychosis of schizophrenia would seem to be not a process that involved inevitable and continued deterioration, but rather a condition that waxes and wanes in severity". Further evidence to support an overall improvement in IQ scores over time was found by

Fiona Barber  Critical Review 2  PsychD.
Klonoff et al (1970) in an eight year follow up study of World War II veterans who had been diagnosed as suffering from schizophrenia. Subjects were assessed using the WAIS as well as symptom rating scales and a neuropsychological test battery. Full scale IQ scores improved significantly, by around 7 points, as did the psychiatric status of the group.

A more recent longitudinal study by the Scottish Schizophrenia Research Group has supported these findings. The group followed-up 49 patients after their first admission to hospital, 45% of whom had suffered no relapse and had no schizophrenic symptoms 12 months later. No evidence of intellectual decline was found on the Ravens's standard progressive matrices, block design and digit copying tests (Gibson & Kendrick, 1979).

In a retrospective study, Buhrich et al (1988) examined patients with chronic schizophrenia either with or without temporal disorientation (that is, inaccurate estimation of their own age). In a previous study (Liddle & Crow, 1984) the matched groups had been given a series of cognitive tests and those with age disorientation were severely impaired on tasks of general orientation, knowledge, Raven's matrices, the digit-symbol test and the mental test. In the current investigation, school attainment and past physical treatments were recorded for all subjects in order to determine whether temporally disorientated patients displayed learning difficulties prior to the onset of schizophrenia. The results showed that there were no differences between the groups based on their school records. However, as both groups had been treated with equivalent amounts of ECT, insulin coma therapy and neuroleptic drugs, it was concluded that cognitive impairment occurs early in the disease process and is not due to physical treatments given. The authors also concluded that premorbid intellectual dysfunction does not inevitably lead to gross cognitive deterioration following disease onset.

While these studies suggest that cognitive decline is non-progressive some studies have suggested that some patients may continue to deteriorate after illness onset. In
the recent study by Dunkley and Rogers (1994), already cited above, these authors used serial measurements of IQ in the subgroup of the patients where these measures were available. They found that the cognitive deterioration apparent in this chronic group of hospitalised patients occurred early after onset of illness with little evidence for cognitive decline after the first five years of illness in those patients completing the IQ measures. However, when the other patients were assessed using the cognitive impairment scale (CIS), a smaller subgroup of patients demonstrated significant deterioration with increasing age and length of illness. The authors suggested the possibility of an interaction effect between the disease process and the decline due to ageing in a subgroup of patients.

Support for the notion of a subgroup of deteriorating patients was also found by Heinrichs and Awad (1993). The authors used four key neuropsychological tasks to identify subtypes of chronic schizophrenia on the basis of different patterns of cognitive functioning. Five clusters of patients were identified, one of which corresponded to a dementia-like multi-focal disturbance. As no differences in SES or education were identified in these patients it was proposed that their global impairments were related to a deteriorative condition. The subtype consisted of 24% of a sample of chronic patients.

Cross Sectional Studies
Cross-sectional research has also challenged the notion of progressive intellectual decline. Hyde et al (1994) conducted a study of five age derived cohorts (18-29, 30-39, 40-49, 50-59 and 60-69 years of age) looking specifically at the evidence for dementia-like cognitive deterioration. Subjects were matched for age of onset and educational level. The authors found no differences across the groups on the MMSE, the Dementia Rating Scale, List Learning and Semantic Fluency tasks. However, all scores were in the impaired range. It was concluded that no progressive decline in cognitive functioning occurs in chronic schizophrenia other than that which would be expected from normal ageing. Citing physiological evidence they suggest that schizophrenia should be classified as a "static encephalopathy" and not a progressive
dementia. The authors define encephalopathy as “an acute or subacute process that diminishes cognitive function resulting in a relatively stable deficit state”. It must be noted, however, that the tests used were not intellectual assessment batteries but merely short-form screening tests which are not standardised against normative populations.

The study by Hyde and colleagues raised an important question regarding the effects of ageing on levels of cognitive functioning. In their study, performance on the Boston Naming test significantly deteriorated with increasing age. This was attributed entirely to the age of subjects and not to the duration of their illness. Heaton et al (1994) conducted a similar cross-sectional study using a more comprehensive neuropsychological test battery. They concluded that deficits are unrelated to age, age of onset and duration of illness and that they are essentially non progressive in nature.

**Summary**

The implications of the above findings are interesting with respect to the effects of the schizophrenic illness on global intellectual functioning. The discrepant findings of no change as opposed to variable improvement may be attributed to the different methodologies used by the research groups. Studies have employed either cross-sectional and retrospective longitudinal designs, which have advantages and disadvantages in terms of reliability and validity.

Cross-sectional research paradigms have yielded results supporting the notion of a static encephalopathy; results provide no evidence of a dementia-like deterioration in intellectual functioning. Findings also show that any decrement in performance is related to the effects of normal ageing and not to the duration of illness. However, this type of study presents a number of methodological difficulties principally because the same subjects cannot be used across each testing stage. While it is possible to overcome this problem with appropriate matching, this process is more error prone
when subjects are taken across different age ranges. Potential criticisms of Hyde et al's study, in terms of the tests used have already been discussed. Another important factor which may affect the reliability of cross-sectional data is possible selection bias with regard to the older subjects. Subjects in this group are invariably chosen from a group of hospitalised, chronic patients while those individuals who suffer only one, or intermittent periods of psychosis are overlooked due to their higher functional capacity. In view of this bias, the 'static encephalopathy' pronounced by authors such as Hyde and colleagues may be challenged. The fact that functioning across all cohorts was in the impaired range would suggest that a more appropriate conclusion is that low intellectual functioning is predictive of poor prognosis. Thus, it is not clear from the studies in their present form whether those patients who do not remain in hospital for extended periods show improvement in global IQ scores in accordance with symptom alleviation.

A similar difficulty is evident in longitudinal follow up studies in that subjects may be "lost to follow up" if they are too high functioning to remain in hospital for extended periods. While this criticism must be noted, it is evident that longitudinal designs present the most effective means of addressing the issue of intellectual deterioration in schizophrenia. Not only do the same patients complete all tests across each age range but the same tests may be given at each encounter. As such, longitudinal studies following children at high risk for developing schizophrenia present one of the most reliable means of assessment. The New York high risk project (Erlenmeyer-Kimling & Cornblatt, 1987) and other High risk follow-up studies have not yet provided enough follow up data to provide information about long term deterioration. However, retrospective studies such as Schwartzman et al, do have a longitudinal perspective and, at present, are the most reliable sources of information. The majority of these studies provide evidence to suggest that deficits are not necessarily stable but may fluctuate depending on the severity of the present symptoms. Thus, it would seem logical to propose that any post-onset deterioration in scores on IQ tests may occur as a result of symptom factors such as poor attention and low motivation. This proposal is supported by the finding that scores may be restored to their premorbid levels.
following symptom remission in some patients. It is important to note, however, that the studies also provide evidence that there is a subgroup of patients with schizophrenia who have consistently low cognitive functioning in conjunction with persistent psychotic symptoms.

An important factor which has been highlighted in these studies is that of institutionalisation and its possible effects on intellectual functioning over time. Schwartzman et al comment that discharge from hospital is not solely reliant on symptom remission, as one might hope, but is often influenced by the availability of alternative accommodation. As such, the authors propose that the cognitive decline observed in patients who remain in hospital may not be related to the severity of their illness but to environmental factors. However, the authors note that decrements in IQ scores were related to the patients status at the time of testing and not to the length of time spent in hospital.

Further to this work, there have been a number of more recent studies specifically investigating the effects of long term hospitalisation on cognitive functioning. Johnstone et al (1981) compared a group of 120 patients who had been discharged 5-9 years earlier with a group of 510 inpatients with schizophrenia. It was found that the inpatients performed less well on the cognitive tests (Withers and Hinton tests) than their out patient counterparts and had higher levels of negative symptoms. However, significant differences were found in age and duration of illness. When these factors were controlled, the authors found that differences in terms of negative symptomatology disappeared but, the difference in level of cognitive impairment remained. However, the authors found evidence from school records that the inpatient group had poorer premorbid functioning and that, in both subject groups, scores on the cognitive tests were related to past academic records. The authors proposed that those patients who display cognitive impairment at younger ages would be less likely to be discharged. As such it is possible that pre existing cognitive impairment has developed throughout the illness and led to long term hospitalisation as opposed to hospitalisation being the primary causative factor.
In support of this work, Goldstein et al (1991) conducted a correlational study looking at hospitalisation, age, education and intellectual functioning using the Halstead Reitan Battery. They found records for 245 patients meeting criteria for schizophrenia who had been tested on the battery. While they found evidence for an apparent decline in cognitive abilities associated with longer periods of hospitalisation, they concluded that this relationship was not significant when age and education were taken into account. However, it must be noted that this is a correlational study and as such, the patients were not placed into discrete comparison groups.

In summary, it would appear that many patients who have been hospitalised for long periods show cognitive deficits by comparison with patients who have spent longer in the community. However, these results may be accounted for by age differences between the groups and the fact that inpatients had pre existing cognitive deficits which may have led to their continued hospitalisation.

Another important factor to consider in the context of cognitive decline is that of medication effects. A comprehensive review of this area is provided by King (1996). In general, findings would suggest that medication tends to improve performance on tests which are affected by symptoms "such as poor concentration, and motivation" (King, 1990). In addition, long term medication may improve performance on tasks requiring "sustained attention and visuomotor problem solving skills" (Cassens et al 1990).

**Conclusions**

The existing literature presents an inconsistent picture of the course of intellectual impairment following disease onset. It would appear that patients show a marked deterioration in IQ scores following the onset of psychotic symptoms but that this condition remains stable or may improve following symptom remission. As such it is suggested that many of the tasks involved in standard IQ tasks are influenced by
symptom variables such as poor motivation and inattention.

As is evident from the outset of this review, research pertaining to global IQ deficits is often out of date and suffers from a number of methodological problems in comparison with more recent studies. Often the heterogeneous nature of the disease is ignored and other issues such as medication effects and ECT treatments are not recorded and as such, the conclusions must be treated with caution.

From the information recorded in this review it is evident that global deficits do exist in patients with schizophrenia and as such, must be considered as one of the characteristics of the disease. While it is apparent that this overall deficit is not specific to schizophrenia, it is still important to discover the possible causes of such an impairment in this population and its implications in terms of the progress of cognitive deficits over time. The identification of global deficits early in the course of the illness or premorbidly may become useful predictors of poor outcomes and continued hospitalisation.
References


CLINICAL DOSSIER

FIONA BARBER

SUBMITTED IN PART FULFILMENT OF THE REQUIREMENTS FOR THE PSYCHD. CONVERSION COURSE

UNIVERSITY OF SURREY

1996
Ms F Barber
Ground Floor Flat
9 Marlborough Road
Chiswick
London
W4 4EU

27 February 1997

LR/ADJ

Dear Ms Barber,

The Committee for Scrutiny of Individual Clinical Qualifications has recently considered the Evaluation of Clinical Competence form submitted in relation to your third year of training in clinical psychology.

The Committee agreed that, following the successful completion of a two-year clinical psychology training course, you had satisfactorily completed a further period of 12 months' supervised practice. You are, therefore, now eligible to register as a Chartered Clinical Psychologist.

Should you wish to register as a Chartered Clinical Psychologist it will be necessary for you to formally apply to the Society for registration, and the appropriate application forms are available from the Society on request. You must not describe yourself as a Chartered Psychologist until you have received notification that such an application is successful. If you also wish to use the adjectival title 'clinical' then you must join the Division of Clinical Psychology. The enclosed booklet 'Information on the Register of Chartered Psychologists' may be of interest to you.

May I take this opportunity to congratulate you on the successful completion of your period of supervised practice.

Yours sincerely,

[Signature]

LOUISE RIGBY (Miss)
Administrative Officer

Enclosures
CLINICAL REPORT OF ASSESSMENT AND INTERVENTION FOR
A CLIENT WITH BULIMIA NERVOSA

Name: Jessica Target
Age: 16 years
Location: Psychiatric Ward, Concord General Hospital, NSW, Australia
Date of Treatment: 20/3/96 to 24/4/96
Number of sessions: 10 (Length of inpatient stay)

All names are fictitious to maintain client confidentiality

Reason for Referral

Miss Target was referred to the department of psychology by Dr. Sarah Morten, the registrar on ward 34 (psychiatry). The referral letter stated that Miss Target had been admitted to hospital on 12th March 1996 with a diagnosis of Bulimia Nervosa. She had previously been seeing a private psychiatrist and was intelligent and motivated to overcome her eating disorder. The referral requested an assessment for suitability for CBT and assistance with Miss Target's management on the ward.

Initial Assessment

Miss Target presented as a rather sulky 16 year old who was prepared to answer questions but did not initiate conversation or give more information than was necessary. At this time she weighed approximately 45 kgs and her BMI was 17. She stated that her eating disorder had started at the age of 12 years at a time when she was slightly overweight. A male friend of hers had told her that comments had been made about her having 'fat legs' and other derogatory comments about her figure. She stated that she had immediately decided to lose weight and had started going to the gym and purging her food. She reported that she had not eaten 'normally' since that time.
Prior to her admission to hospital Miss Target stated that she had been binge eating and purging at least once per day. She stated that she would eat a large lunch, consisting of a main course and dessert, at school which she would then vomit about half an hour later. At home, she stated that she binges on chocolate and ice-cream and other sweet foods. This bingeing usually takes place in the pantry when her family are out of the house.

Miss Target stated that she had used laxatives in the past but that they had not helped her to lose weight and she had stopped using them. She reported that she does not weigh herself at home but uses her clothes as an index of her current weight. She stated that her preferred weight would be between 40 and 50 kgs and that she feels that her thighs and buttocks are fat.

At the time of the initial assessment, Miss Target had been in hospital for one week and had vomited many of the meals she had been given. Her medical file indicated that the ward staff were aware of this behaviour and were monitoring her closely.

Miss Target had been persuaded to be admitted to hospital by her father and they had chosen Concord Hospital as it was the only place with an empty bed. Miss Target commented that the admission had occurred very quickly and she appeared rather confused and overwhelmed by the treatment she was receiving. She stated that she was not really sure what she hoped to achieve from the admission nor how it was to occur. She commented that her eating behaviour means that she can eat all the tasty foods that she wants without putting on weight and that she could not imagine eating normally again without losing these advantages. She could not isolate particular events or triggers for bingeing behaviour other than being in the house on her own with lots of food. She stated that she would often binge when she was nervous, for example, before a test or exam, but that any emotion, such as boredom or depression, could trigger a binge episode.
Family Background

Miss Target currently lives with her father and two elder brothers. She has been living with them for approx. 6 months having previously lived with her mother and stepfather since the break-up of her parent's marriage 10 years ago. Her father and brothers know that she has an eating disorder and she stated that they find it difficult to understand her behaviour. She commented that her eldest brother becomes angry with her, particularly when she eats all the food in the cupboards. When living with her mother, Miss Target stated that she had not been able to be honest about her eating but her mother had known and had followed her to the bathroom and listened at the door.

She described having a good relationship with her father stating that he was very fair allowing her to smoke at home and go out late but disciplining her when necessary.

School and Social Life

Prior to admission, Miss Target was attending high school in year 11. She stated that she had done surprisingly well in her school work and that she was being encouraged to go to university. Socially, she stated that she had a lot of good friends who she spent time with at weekends. Some of these friends know about her eating disorder and are supportive to her. She currently has a boyfriend who also knows about her eating disorder and frequently tells her that he thinks she is beautiful the way she is.

Previous Treatments

Miss Target has been seeing a private psychiatrist for the past 2 months. The psychiatrist's report noted that Miss Target was a very intelligent young lady and felt that her current difficulties were a consequence of her disrupted family situation. Miss Target stated that she did not like her psychiatrist and felt that they had never addressed her eating behaviour in the sessions. She stated that the psychiatrist had prescribed her Prozac but that she had only ever taken one tablet and had lied about the rest.
Miss Target reported that she had seen a psychologist in the past but that she had not found it beneficial. She stated that she had been asked to complete diaries of her food intake and vomiting but had lied to the therapist who had discharged her, believing her to have improved.

In addition to individual therapy, Miss Target was involved in a ward based behavioural programme which rewards clients for weight gain and absence of bingeing. The reward system moves from bed restrictions to ward restrictions and eventually clients are allowed to chose their own menus. Clients are encouraged to finish their meals and are placed on supervised bed rest for an hour after each meal. Three main meals are given each day with three snacks at mid morning, afternoon and supper.

**Initial Formulation and Treatment Plan**

It was evident from the initial assessment that Miss Target has a long standing history of bulimia nervosa. She binges and vomits at least once per day and has used laxatives as an aid to weight loss in the past. She presented as being reticent about discussing her feelings towards her family and friends and, contrary to the referral letter, did not appear highly motivated to improve her condition and work cooperatively in treatment.

Miss Target's remarks concerning previous therapy indicated that she may not necessarily tell the truth and indicated that she may be somewhat manipulative, attempting to establish control of the therapy sessions. Literature regarding Bulimia Nervosa would indicate that cognitive-behavioural therapy would be the preferred method of treatment (Fairburn, 1985) as it has a high success rate (Kirkley et al, 1985) and addresses both the behavioural aspects of disturbed eating patterns as well as distorted cognitions about shape and weight. It was evident from the initial assessment that Miss Target had overvalued ideas about her weight and defined her self-worth almost exclusively in terms of her weight and thus, cognitive therapy would be
appropriate. However, her descriptions of previous therapy would indicate that CBT has been attempted without success. She did not complete homework assignments and did not find them helpful as she lied about her eating patterns. There was evidence that Miss Target did not have a clear idea about her goals for treatment and that she had not yet accepted that her eating behaviours needed to change.

The initial intervention plan was to supplement the behavioural programme on the ward with some introduction of the principles of cognitive therapy and the notion that she needed to change her thinking patterns. One of the aims for therapy was to establish some treatment goals and to use motivational interviewing to encourage Miss Target to change her eating behaviours. As Miss Target appeared unenthusiastic about therapy it was important to establish rapport and gain some trust. Thus, it was important to persevere with therapy, even if sessions were initially difficult.

**Intervention: Phase 1, sessions 3-6**

In order to establish rapport with Miss Target and to find out more about her self image I used a method based on Kelly's personal construct theory (Ewen, 1993). The method involves asking the client to think of significant people in their life including their parents, family, employers, someone they admire and someone they dislike. For each person, the client is asked to think of their positive and negative characteristics and then compare them to themselves and to other people on the list. Miss Target was able to engage in this exercise and was able to think of personal attributes that were not merely superficial or appearance oriented. It was evident that she particularly admired people who were able to stand up for themselves and liked people to be genuine in their motives. When asked to describe her own strengths she had great difficulty in coming up with anything other than that she had 'nice eyes'. She stated that she disliked the rest of her appearance and felt that she was selfish and temperamental. When asked how
other people related to her she stated that they liked her because she was funny and easy
to talk to.

I went through the cognitive model of Bulimia Nervosa with Miss Target which
proposes that low self esteem causes a focus on body shape which then results in
extreme dieting, hunger and consequent binge eating. She appeared to relate to the
model and stated that it described her own pattern of behaviour very closely. We then
discussed her beliefs about her own self esteem and how this could have led to her
eating disorder. In essence, the fact that the need to define herself in terms of her
body/thinness had led her to become overconcerned with eating and the binge eating
may be a means of punishing herself for being a 'bad' person. Miss Target appeared to
accept these ideas but did not venture any further information or expand on her own
personal experience.

At a following appointment Miss Target became very distressed due to a number of
events. She disclosed that she had become involved with one of the other patients on
the ward and that he had left on the previous day. She stated that he had been one of the
only people who she felt had understood her and that she felt that she had helped him in
some way to get over his depression.

Miss Target then went on to disclose more about her family background. She stated that
she had been given no privacy from her mother and commented that her mother had
read her letters and listened in to her phone calls. She reported that her mother had been
very manipulative, refusing to pick Miss Target up from the hospital if she did not go
and stay with her. Miss Target stated that her mother wanted her to move back into
home and that she felt very guilty about not doing so. In addition, Miss Target reported
that her stepfather had been physically abusive to her brothers and had emotionally
abused her and her little sister.
At this appointment Miss Target admitted to bingeing and purging while in hospital. Miss Target cried openly throughout the session and commented that she felt things were hopeless and that she could not see a way for them to improve.

**Secondary Formulation**

As more information came to light about Miss Target's family it was evident that her eating behaviours are related to her feelings about her family. It appears that her mother is very controlling and over involved in Miss Target's life and becomes punitive when this control is challenged. As a consequence Miss Target has feelings of depression and hopelessness as she is unable to control her environment or feel autonomous in any way.

Miss Target commented that her Mother tries to make her feel guilty for moving out of home and also blames her for the stress that she has put them through because of her disorder. It was also evident that Miss Target's mother has high expectations of her daughter both academically and behaviourally. These high expectations and blaming have led Miss Target to feel that she has failed in some way to meet the standards required of her and that she is inadequate. She also feels guilty about the problems she has caused her mother and this guilt serves as a further cause for feelings of depression and worthlessness. It is probable that Miss Target uses bingeing to alleviate these feelings of distress and that it is a way of punishing herself for being a 'failure' and not meeting the standards expected of her. Thus, Miss' Target's binge eating and focus on body image is a consequence of her low self esteem which has occurred as a result of feelings of guilt, inadequacy and lack of autonomy.

**Further Intervention: Phase 2, sessions 7-10**

I presented the above formulation to Miss Target and she was receptive to the ideas. She commented that she did think that her bingeing became worse when she was depressed or anxious and that it was possible that she had misinterpreted these feelings as hunger, or a craving to binge rather than acknowledged them as emotional states. We discussed self-esteem and the way that her family experience may have contributed to
her feelings of inadequacy and worthlessness. Although she was somewhat reticent about these issues she did request that we write down the formulations and she took it away to think about.

Miss Target's mood improved over the next sessions and she informed me that the change had occurred as she had started to accept that the regime on the ward and her visits to psychology were helpful to her and that she was now listening to the advice she was being given. She stated that she had not binged or purged for 10 days which was the longest time since admission.

At this point in therapy an incident occurred on the ward where a male nurse gave Miss Target his address and told her that he had taken hers from her file and would be contacting her while she was on weekend leave. She stated that he had made allusions to marriage and had made it evident to her that he was attracted to her. Initially, this approach did not cause any distress and Miss Target found it amusing and had discussed it with other girls on the ward. The nurse then made further suggestions and was following Miss Target around the ward, listening to phone calls. I informed Miss Target that this behaviour on the part of the nurse was unacceptable and that she and I had an obligation to inform the ward manager. She stated that she did not want to inform anyone at this time as she was concerned that she would be thought of as a trouble maker. We contracted that she would tell the manager or one of the doctors in the next week. I informed other members of the department about the issue and it was agreed that I should persuade Miss Target to tell the ward herself but if this was not achieved then I should tell them myself and inform Miss Target of my intention to do so. Miss Target's parents were aware of the problem and were also trying to persuade her to take some action.

The incident appeared to significantly affect Miss Target's condition and she stated that she had a bad weekend and had binged and purged on three occasions. She stated that she was very angry with her mother for threatening to ring the ward without her consent and felt that she did not want any action to be taken until she left the hospital. In
addition, she commented that she would feel guilty if she affected the nurse's career and did not want to be responsible for him not becoming a registered nurse. We discussed these issues at some length using ideas from literature concerning sexual harassment and the effects on victims. I attempted to normalise her feelings and felt that it was common for women to experience guilt after such events but to be clear that the responsibility lay with the nurse who had violated his professional codes of conduct. We also discussed her mother's reaction and, while acknowledging that it was normal for a parent to feel protective, her reaction had taken control from Miss Target resulting in her feeling agitated and more upset about the incident.

In her final session Miss Target stated that she had told the ward about the nurse and that another female patient had also lodged a complaint.

In this session I went through steps she could take to avoid bingeing. She has taken up writing poetry as an outlet for her emotions and has found that this stops her urge to binge. Alternative behaviours were to go outside or smoke. In addition we decided that she should try to ask herself a series of questions to ascertain why she was bingeing, these included: am I hungry?, am I anxious?, am I depressed?, am I angry? etc.

Miss Target's self esteem appeared to have improved significantly. She commented that she felt that she was able to be more positive about herself and felt that she looked good. Her final comment was that she would like to go and buy herself some new, tighter clothes so that she could show off her figure more.

Miss Target lives some distance from the hospital and therefore decided to find a psychologist closer to home for continuing therapy. No follow-up was necessary and she has not been re admitted to the hospital since going home.

**Discussion**
There were a number of factors that affected the therapy, some specific to the case and others that were concerned with professional or organisational issues.

The primary issue that affected therapy was the difficulty in building rapport with a 16 year old girl who was resistant to therapy. Her attitude during the first five sessions was rather hostile and she would sometimes reply to questions with sarcasm or with the retort, ‘what do you think?’ There was a sense that she expected the therapist to somehow magically make her better without any contribution on her part. Her resistance could be interpreted as a means of gaining a sense of control over the sessions and her silences were extremely powerful, leaving it up to the therapist to do all the talking.

The breakthrough after session six when she expressed her feelings in therapy was her first concession that she was extremely vulnerable. As such, it was very important not to destroy this trust and to develop it into increased cooperation.

From an organisational standpoint, there were some advantages and disadvantages to the lack of communication between the psychiatry ward and the psychology department. Due to long standing conflicts between the departments, psychologists were not invited to team meetings or case conferences on the ward and the only communication was by hospital file notes and phone calls. This situation had obvious drawbacks in that the responsible medical officer had no psychology input and made decisions about the cases without knowledge of the psychological intervention. Thus, patients could be discharged without the psychology department being informed and information about possible discharge dates was conferred by the patient themselves. This made planning therapy difficult as there was no guarantee of how many sessions could be planned.

However, the situation had some advantages in that the perception of objectivity in the therapist was enhanced and the patients felt free to discuss ward based problems without worry that they would be fed back to the ward staff unless absolutely necessary.

Fiona Barber
Clinical Report
PsychD.
The final aspect of the case that raised a number of issues was that of the student nurse making sexual comments to the patients. While it was of paramount importance that the person in question be reported to the appropriate authorities, the wishes of Miss Target also had to be considered. She did not want the nurse to be reported while she was at the hospital and certainly did not want anyone else to take over and report the incidents on her behalf. This latter desire was difficult for both her parents and therapist to respect but necessary if she was to maintain feelings of control and self worth. In the circumstances, the therapist took the option of discussing the issues and attempting to persuade Miss Target to report the incident herself, thus enhancing her self esteem and providing emotional support. The incident highlighted the complicated nature of confidentiality and the importance of peer review and support from other psychology department staff.

References


Word Count: 3598
AN INVESTIGATION OF
THE NEUROPSYCHOLOGICAL CORRELATES OF
EXTREME VIOLENCE IN SCHIZOPHRENIC PATIENTS.

FIONA BARBER

Submitted in part fulfilment of the requirements for the Masters Degree in Clinical Psychology

University of Surrey
1994
The aim of the present study was to investigate the neuropsychological correlates of extreme violence in patients with schizophrenia. It was hypothesised that the most violent patients would show significantly greater impairment on tests sensitive to frontal lobe function than their non-violent counterparts.

The study involved twenty-eight patients overall. Fourteen of these were selected from Broadmoor Hospital where they had been incarcerated following an index offence of murder/manslaughter. The control group was a selection of fourteen outpatients with a diagnosis of schizophrenia from Queen Mary’s Hospital, Roehampton. Subjects completed a battery of neuropsychological tests and symptom severity ratings for the two groups were performed by Senior Registrars at the Hospitals. Subjects were matched for age, IQ and length of illness.

The findings revealed that there were significant differences on only two of the neuropsychological tests with the non-violent sample showing greater impairments than the violent offenders. Significant differences were found on the symptom rating scales for both positive and negative symptoms. Again, the non-violent group displayed greater symptom severity than the violent sample.

The results refuted the primary hypothesis of the study and are discussed in relation to the need for a more detailed analysis of the nature of the murder committed. Consideration of the "protective" environment of Broadmoor is also mentioned in terms of the effects of compliance with medication and adequate nutrition on schizophrenic illness.
ACKNOWLEDGEMENTS

I would like to thank Dr. Sarah Wilson and Dr. Mary Hill for their invaluable supervision of this research and Mr. David Murphy for his guidance with the statistical analysis of the data.

I would also like to thank Dr. Eileen Joyce for her support and advice and all the Day Hospital staff at Queen Mary’s Hospital for their help in finding subjects.

Thank you to Dr. Tim Exworthy for his contribution to the project data and, most importantly, to all those who took part in the study.

Finally, I would like to thank Mr. & Mrs A. Barber and Mr. Simon Collinson for their continual support and encouragement throughout my clinical training.

(c) Fiona Barber 1994
CONTENTS

ABSTRACT

ACKNOWLEDGEMENTS

CONTENTS

LIST OF TABLES AND GRAPHS

SECTION 1 - INTRODUCTION

1.1 The Neuropsychology of the Frontal Lobes
   1.1.1 Anatomy of the Frontal Lobes
   1.1.2 Dorsolateral Prefrontal Cortex
   1.1.3 Orbital Prefrontal Cortex
   1.1.4 Medial Prefrontal Cortex

1.2 Schizophrenia

1.3 The Neuropsychology of Schizophrenia

1.4 Violence - The Problem of Classification

1.5 Mental Illness and Violence

1.6 Violence and its Cognitive Correlates

1.7 The Present Study

1.8 Hypotheses

SECTION 2 - METHODOLOGY

2.1 Design

2.2 Subjects
   2.2.1 Experimental Group
   2.2.2 Control Group

2.3 Procedure

2.4 Neuropsychological Tests Used.
SECTION 3 RESULTS

3.1 Matching Data

3.2 Neuropsychological Test Scores

3.2.1 Means and Standard Deviations

3.2.2 t-Tests

3.2.3 Discriminant Function Analysis

3.2.4 Correlations

3.3 Symptom Rating Scales

3.3.1 Means and Standard Deviations

3.3.2 t-Tests

3.4 Correlations

SECTION 4 DISCUSSION

SECTION 5 REFERENCES

SECTION 6 APPENDICES

Appendix 1 - Letter to Responsible Medical Officer Informing them about the Research.

Appendix 2 - Consent Form for RMOs

Appendix 3 - Debrief Letter for Subjects at Broadmoor

Appendix 4 - Debrief Letter for Subjects at Queen Mary’s Hospital

Appendix 5 - Normative Data Available

Appendix 6 - Data Sheets
LIST OF TABLES AND GRAPHS

TABLES

3.1 Means and Standard Deviations for Matching Variables
3.2 Means and Standard Deviations for Neuropsychological Variables
3.3 Discriminant Function Analysis
3.4 Significant Correlations Between Neuropsychological Test Scores.
3.5 Means and Standard Deviations for Symptom Rating Scales Including T-values and Significance.
3.6 Significant Correlations Between Symptom Scale Ratings and Neuropsychological Test Scores.
A-6 Normative Data Available.

GRAPHS

1. Graph to show Mean scores for the Manchester Scale Ratings for the Two Groups.
2. Graph to show Mean scores for the SANS Scale for the Two Groups.
SECTION 1 - INTRODUCTION

Interest in the neuropsychology of violent behaviour has led to a number of studies seeking to establish a relationship between specific neurological deficits and antisocial/criminal acts. The majority of this work has focused on the possible influence of the frontal lobes due to their association with 'higher' functions such as modulation of behaviour, planning and intelligence.

Impairment of frontal lobe function has also been implicated in recent literature studying the neuropsychology of schizophrenia. Studies have shown that schizophrenic patients perform poorly on tests of frontal function and research is continuing to try to specify the exact nature of these difficulties. Despite these seeming commonalities between the neuropsychological profiles of schizophrenic patients and violent offenders it is clear that not all schizophrenic patients become violent.

The purpose of the proposed study is to look specifically at the neuropsychological profiles of schizophrenic patients who have committed murder/manslaughter. The research will look specifically at impairments of frontal lobe functioning as these have been implicated in both schizophrenia and violent behaviour. In order to provide a background to the study it is necessary to discuss the role of the frontal lobes in man. In addition an overview of the literature concerning the neuropsychology of schizophrenia will be presented along with studies directly concerned with schizophrenia and violent behaviour.
1.1 The neuropsychology of the frontal lobes

The effects of lesions to the frontal lobe structures in man have been widely studied over the past century. The frontal lobes comprise one third of the total mass of the brain. In evolutionary terms they are the most recently developed structures and are larger in humans than in other mammals. As a consequence, some authors have assigned them "higher" functions such as intellect and ethical reasoning. However, research into the clinical presentation of patients with frontal lobe damage has produced a complex mixture of symptoms ranging from disorders of affect and motor control, to language, problem solving and memory difficulties. In recent years there has also been a body of literature implicating frontal lobe dysfunction in the pathogenesis of disorders such as schizophrenia and Parkinsonism. The specific contribution of the frontal lobes to violent behaviour and schizophrenia will be discussed in later sections.

From a neuropsychological perspective, damage to the frontal lobes may result in impaired performance on numerous tasks. The various deficits described were originally termed the "frontal lobe syndrome" (Teuber 1964) and it was supposed that a cluster of different "frontal" symptoms would co-exist, varying broadly along a continuum of severity. Thus, the numerous behavioural manifestations of frontal lesions were thought to all have evolved from a single core deficit.

In order to discuss the neuropsychological aspects of frontal lobe function it is necessary to look briefly at their anatomy and the various deficits associated with damage to a particular region.
1.1.1 Anatomy of the frontal lobes.

The frontal lobes of the human brain comprise all the tissue in front of the central sulcus. They may be subdivided into 4 regions on the basis of anatomical and functional distinctions: 1/ the primary motor cortex, 2/ the premotor area, 3/ the prefrontal cortex and 4/ the frontal eye field. It is the prefrontal region which is of particular importance to psychologists due to its association with the intellectual control of action. The majority of neuropsychological literature focuses solely on this area primarily because it is a common site of damage with closed head injury. In addition, the effects of damage may be very disruptive and can impede rehabilitation programmes.

The prefrontal cortex is divided into 3 parts: orbital, medial and dorsolateral. The first of these is connected to the limbic system, ascending reticular formation and the entorhinal area. As a consequence it appears to be related to the emotional control of behaviour. The medial frontal cortex acts as a supplementary motor area and is responsible for initiating motor behaviours and sustaining them at an appropriate level. The dorsolateral area serves as an integrating station from sensory and motor association areas in the temporal and parietal lobes. As such, it is concerned with the preparation and execution of action.

In essence, the prefrontal region determines the generation of "adaptive/ effective" behaviours, that is behaviour which is goal oriented, motivated and monitored effectively whilst being produced. Luria (1973) describes human thinking and problem solving as a process involving "intention, formation of plans/programmes, regulation of
behaviour and monitoring in order to confirm the fulfilment of the original intentions."

The complexity of these processes is evident and inevitably involves a number of brain regions.

It is important to be aware that when describing the impairments associated with frontal damage, both anatomical and functional divisions are in some sense arbitrary as few patients have suffered isolated damage to a particular region.

1.1.2 The Dorsolateral Cortex

The dorsolateral prefrontal area is responsible for the preparation and execution of action. In order to produce an "effective" action it is essential that some kind of programme for action has been developed, usually involving information from memory stores and from the external environment (via the visual and auditory cortices). As early as 1848, clinical observations of patients with frontal lesions described impairments in planning and problem solving.

The first clinical test used to investigate planning behaviour was the Porteus maze (1959). As with standard maze tasks, patients were expected to find a pathway from the centre of the maze to the exit without crossing any lines or backtracking. Porteus described the skill involved as "pre-rehearsal" and affirmed that his maze test showed consistent loss with frontal lobe damage. Maze tests are still in use by clinicians as a test of frontal function. More sophisticated types of maze have been produced, for example the Austin maze which uses more abstract concepts and is considered to be discriminatory amongst patients with a high premorbid IQ.
Further clinical tests have been found to reflect patients' abilities to plan or pre-rehearse action. Visuo-constructive tasks, for example the Rey figure and Block design (WAIS), have been used extensively with brain damaged subjects, most commonly those with posterior damage, in order to assess difficulties in spatial organisation (constructional apraxia). It has been shown that frontal patients also fail these tasks. However, in these instances it has been evident that the inaccuracies have arisen because of disruption of the various aspects of planning the design reproduction.

The Rey-Osterrieth Complex Figure Test involves asking the subject to copy a complex design as accurately as possible. The test may be scored by looking at the level of organisation used by the subject when copying, for example whether they started with the central rectangle or with a peripheral detail. The subject is then asked to recall the design from memory at a later time. It has been shown that patients with frontal lesions are less likely to use a coherent strategy when copying the figure. As a consequence of this impairment, their ability to remember the figure is also affected. Further experiments have highlighted the differences between frontal and posterior patients by providing them with a serial plan of how to draw the figure. Patients began by drawing the basic rectangle, then had to copy more and more complete figures until the full drawing was completed. Thus, they had experienced a sequential programme for the drawing on a number of occasions. The frontal patients recall was much improved in comparison to posterior lesion patients following the completion of this practice. In addition, it was found that extra practice alone (that is, without provision of a plan) did not lead to improvement in recall performance.
The problems with recall experienced by frontal patients on the Rey figure task highlight a further difficulty associated with impairments of planning ability. The term "Frontal Amnesia" was coined by Barbizet (1970) to describe the problems in learning and memory experienced by frontal patients. These difficulties were thought to be qualitatively different from those experienced by patients with temporal lobe lesions in that there was no deficit on simple verbal and visual tasks. Frontal amnesia is described as "a difficulty in the intellectual organisation of material for the process of committing it to memory" (Walsh 1985). Thus, the deficit is most apparent when novel material is presented which is lengthy or complex. As with the Rey figure experiments, frontal patients may be helped to overcome these memory difficulties by being provided with external organisation for the material.

Observations of subjects on planning tasks reveal a number of related difficulties which affect test performance. Many clinicians have noted that patients with frontal lesions are uncritical of their performance despite being aware of their errors and are unable to correct their performance on the basis of this knowledge. Konow and Pilbram (1970) termed this ability "error utilisation" and noted its distinction from the skills of error evaluation and error recognition. A consequence of this deficit is the phenomena of "imperfect learning". Clinicians noted that on the Austin maze tests, performance improves over the first few trials to produce an errorless performance. Frontal patients, however, may continue to make errors on subsequent trials even after reaching errorless performance. Such deficits become most apparent in situations where new learning or generalisation of existing programs to new situations is required. It is important to be
aware of "imperfect learning", particularly in rehabilitation programmes when a clinician may be deceived into believing that a skill has been learnt on the basis of one perfect trial.

A further test of organisational behaviour in patients with frontal lesions is that of arithmetical problem solving. Despite being able to complete standard addition and subtraction, patients with frontal lesions have difficulty solving arithmetic problems requiring two or three step solutions. For example, "A son is 5 years old, in 15 years his father will be twice as old as he. How old is the father now?" Christensen (1975) summarises the behaviour of frontal patients on this task as "grasping only one particular fragment of the problem". Thus the solution generated will relate to only one of the calculations involved which may be taken out of the context of the problem, for example, replying "10 years old" (i.e. twice as old as 5 years) to the above question. It would appear that frontal patients have difficulties in mental manipulation of the information and are unable to devise a coherent strategy or order in which to calculate the sums. In addition, there is no integration of the environmental/contextual aspects of the problem resulting in ridiculous answers.

This failure of frontal patients to successfully integrate complex information is also shown on tests such as Picture arrangement (WAIS subtest) and the Reitan word finding test. In the first of these tests, patients are presented with a series of cards, initially placed in a random order. The task requires the rearrangement of the picture sequence so that it tells a logical story. Frontal patients tend to describe each card separately, rather than as a part of a story and are less likely than any other group of brain damaged
subjects to change the order of the cards from that originally presented. It would appear that the frontal patients are unable to abstract information from each of the pictures and integrate it into a coherent story. This ability is even more pronounced in the Reitan test in which subjects are given a set of five sentences, each containing a nonsense word. The patient is required to discern the meaning of the nonsense word using the context of the sentences as a guide. Integration of information from 2 or more sentences is required and the subject must be able to abstract the relevant information from a number of sources.

The ability of frontal patients to adopt an "abstract attitude" has been widely discussed in the literature and is closely connected with the controversy over whether the prefrontal region is the site of higher intellectual activity unique to mankind. Reitan (1955) and Goldstein (1944) each took an opposing stance on this issue, debating the presence of quantitative or qualitative changes in abstract thinking of frontal patients. It would appear that whilst there is some impairment on tasks such as The Halstead Category Test, frontal patients show no qualitative differences in abstract thinking than other patients with temporal lesions. In general, research into this area is unsatisfactory as it is extremely difficult to isolate the exact ability that a particular test is measuring; failure may be due to a number of factors independent of abstract thinking.

1.1.3 Orbital Prefrontal Cortex

As previously mentioned, the orbitofrontal region of the human brain appears to be responsible for excitatory, inhibitory and emotional control of behaviour. As such, the hallmark of orbital frontal damage is the loss of control over inhibition of inappropriate
responding. Changes occur in many areas of functioning including personality, behaviour and cognitive skill.

A test which has been commonly used as a measure of orbitofrontal function is the Wisconsin Card Sort task (Grant and Berg 1948; Milner 1963). It acts as a good example of a test which may be interpreted as reflecting a number of different abilities. The subject is presented with 4 stimulus cards, these have designs on them which differ in terms of the colour of the design, the shapes used and the number of shapes on the card. For example, a set of stimulus cards may consist of a red triangle, two blue stars, three green crosses and four yellow circles. The subject is then asked to sort out a set of cards (up to 100) into piles in front of the stimulus cards choosing a logical means of categorisation.

In this case, the three means of sorting are by colour, shape and number of elements depicted on the card. The testing clinician can only inform the subject as to whether they are correct or incorrect in the strategy they have chosen. The correct solution is first colour, then shape and finally number of elements. Once it is evident that the subject has correctly identified the first correct solution (colour) then, without warning, the tester changes the correct solution to shape. The tester makes a number of shifts throughout the procedure and the subject must be able to alter their response set in accordance with the new rule. It has long been recorded that frontal patients fail this test and that their performance is consistently worse than that of patients with posterior lesions. The reasons for their difficulties may be numerous and may be attributed to damage to both the dorsolateral and orbital cortices.
The WCST is primarily seen as a measure of cognitive and behavioural flexibility. Frontal patients characteristically fail to shift their response set continuing to sort on the basis of the original rule (i.e. colour) throughout the entire test procedure. It is interesting that these patients are able to verbalise that they know colour is no longer correct and may be able to propose a new system of categorisation, like shape or form. However, patients continue to perseverate with the original, now incorrect, solution and are unable to use their verbalisations as a guide to action. The fundamental deficit appears to be an inability to inhibit customary modes of responding. Thus, in a situation which requires a decision between a number of competing responses it is necessary to select the appropriate one and suppress those responses which are inappropriate. Patients with orbital frontal damage can no longer monitor this process effectively and the system will perseverate with habituated modes of responding rather than change to fit new environmental demands.

Further clinical tests of disinhibition include the Stroop test (Stroop, 1935) and the Reitan Trail Making Task Part B (Reitan, 1958). In the Stroop test patients are asked to read out a list of colour words which are printed in a colour which is different to the word, for example, the word "red" would be printed in brown. In the second part of the test, the patient must read out the colour the word is printed in rather than the word itself. Frontal patients make more errors on this task than posterior patients and this is attributed to their inability to suppress the intuitive response of reading the word. In the Trail Making test Part B, the subject is expected to switch between two well established series of numbers and letters to make a trail of 1-A-2-B-3-C .... For each item the subject has to suppress the next item of one series whilst selecting the correct one of the
other. Frontal patients' performance tends to be far slower than that of controls and in serious cases the patient fails to inhibit effectively, for example, 1-A-2-B-C-D.

Changes in personality resulting from orbitofrontal damage may also be described as "disinhibitory". The classic case is that of Phineas Gage (1848), who suffered severe frontal damage when a tamping iron was blown through his brain. Relatives and friends described him following the accident as more impatient, irreverent, obstinate and using profane language when he had not done so previously (Kimble, 1963). Such behaviour creates a psychosocial deficit which makes their care more difficult, especially for family. In addition, a lack of social inhibition may lead to excessive alcohol consumption or other substance abuse.

In general, it is difficult to study the effects of frontal damage on personality in any systematic way. All patients start off with very different premorbid characteristics and there will inevitably be large individual variation in a patient's reaction to their disabilities. A fundamental difficulty of research into post-damage personality is to establish what effects are due to irreparable brain damage and those which are emotional reactions to accident or illness.

1.1.4 Medial Prefrontal Cortex

The medial area of the frontal cortex is rarely damaged in isolation due to its protected position in the brain. As a consequence, its functions are less well known and the effects of damage to this region are frequently subsumed by the symptoms of lesions in other regions. The medial region is thought to be responsible for initiating motor
behaviour and sustaining it at an appropriate level. In relatively pure cases of medial damage, patients display a seeming apathy or passivity termed "adynamia". Clinicians have observed that whilst these patients are able to respond appropriately to external stimulation, they are unable to sustain activity when the external prompt is removed. It is apparent that difficulties of this sort present a serious difficulty for rehabilitation programmes and may easily be misunderstood as laziness or depression.

A general lack of behavioural spontaneity has been reported in frontal patients. This may be evident in speech, facial expressions and movement. Some patients have been labelled as "pseudodepressed" on the basis of these observations.

The theories of cognitive neuropsychology have made a contribution an increased understanding of frontal lobe function. Research into frontal lobe functioning has focused on patients with head injury where the site of the damage has been clearly identified by MRI or CT scans. In 1988 Shallice et al. proposed a cognitive model to account for the types of neuropsychological dysfunction which they had found consistently in frontal lobe injury. Shallice et al. proposed that the system for selecting of a particular action out of a choice of possible responses is controlled by a "Supervisory Attentional System" (where an action is a goal directed response usually involving movement).

The model is best represented by a diagram (fig. 1). The lower part of the model represents the number actions which may be triggered by the perception of an environmental stimulus. The contention scheduling process is that by which the most
highly selected action is chosen and the others are suppressed by a process of mutual inhibition. Thus, this system is concerned solely with stimulus driven action; in the absence of an environmental cue the system will either do nothing or perseverate with the last action chosen. In essence, contention scheduling allows the execution of complex, routine actions in response to familiar environmental signals.

Figure 1. Shallice's System for the Control of Action (Frith 1992)
The upper half of fig 1 represents the role of the Supervisory Attentional System (SAS) which is, in a sense, the 'override' mechanism. Thus, the system for choice of action may be controlled in the absence of environmental stimuli and generate new behaviours in novel situations. In addition perseverative behaviour may be halted by the SAS.

Shallice proposes that the SAS is fundamental to the functioning of the frontal lobes, in that patients with lesions in this area suffer from problems in long term planning, the modification of action in response to feedback from the environment and general disorganisation in the execution of effective action. These deficits occur in the absence of any lowering of general IQ. With relation to schizophrenic symptomology, failure of the SAS may be responsible for behaviours such as perseverative and stereotyped activity and inappropriate responses to stimuli (Frith 1992).

A further aspect of the recent literature on the frontal lobes concerns the concept of the "frontal lobe syndrome". A cluster of frontal signs which are expected to co-exist in each patient varying along a continuum of severity. Evidence for the existence of such a syndrome is no longer conclusive and it has been suggested that only some of the processes subserved by the frontal lobes are "system wide" whilst others have their own distinct pathology and may appear in isolation. Studies cited by Shallice and Burgess (1993) indicate that certain frontal tests such as the Stroop task and Verbal Fluency tend to show similar impairments in frontal patients, that is, that patients who perform poorly on one are most likely to achieve a poor score on the other (Perret 1974). One explanation for this result is that both tests make similar cognitive demands (i.e. they are testing the same thing) or, alternatively, there are two separate cognitive processes
involved (response suppression and initiation) which are both effected by frontal
damage.

Shallice et al. (1993) continue in their paper to show that many tests of frontal function
may be double dissociated, for example that deficits on the Hayling sentence
completion task may show on either part A (initiation) or part B (inhibition) but not
necessarily on both.

In essence the study highlights the complexity of the frontal lobe processes and
indicates that different types of frontal deficit may appear in isolation. The study also
looks at the difficulty in finding a "pure" frontal test and emphasises the need to look
carefully at all the possible areas to which a test may be sensitive.
1.2 Schizophrenia

Schizophrenia is a devastating mental illness which effects approximately 1 in 100 people at some time during their life.

The term 'Schizophrenia' encompasses a wide variety of symptoms and the illness is so varied in its manifestations and course that some psychologists (Boyle 1990) have questioned its existence as a single disease entity. Modern classification schemes have, however, produced increasingly reliable and consistent criteria by which to diagnose the disorder and it is now possible to specify a group of patients who may be labelled as schizophrenic. The most widely used diagnostic criteria are those described in the Diagnostic Statistical Manual (DSM III-R; American Psychiatric Association, 1987):

The patient must have

A. Characteristic psychotic symptoms for at least one week.
B. social functioning below previous levels during the disturbance
C. no major changes in mood (depression of elation)
D. continuous signs of the disturbance for at least 6 months
E. No evidence of organic factors (e.g. drugs)

characteristic psychotic symptoms must include

1. two of the following
   a. Delusions
   b. prominent hallucinations
   c. incoherence
   d. catatonic behaviour
   e. flat or grossly inappropriate affect

OR 2. bizarre delusions (e.g. thought broadcasting)

OR 3. prominent hallucinations of a voice with content unrelated to mood.
Thus, in order to be diagnosed as schizophrenic the patient must report bizarre experiences or beliefs such as delusions and hallucinations. Examples of these experiences include hearing voices which may talk to the person directly or comment on their actions. Patients may also have visual, tactile or olfactory hallucinations although these experiences are less common. Bizarre beliefs commonly held by patients include the experience that others are reading their thoughts, delusions of control (that their actions are controlled by an outside force), paranoid delusions (patients believe that other people are trying to harm them) or delusions of reference (patients experience the actions of strangers to be especially relevant to themselves). Such symptoms may only be diagnosed from the patients’ self report rather than objective observation.

The DSM III-R criteria also mention observable symptoms such as catatonic behaviour and flat or inappropriate affect. Further examples of these symptoms include a general reduction in spontaneous behaviour resulting in poverty of speech, alogia, apathy and social withdrawal. These symptoms become more apparent in the later stages of the disease with 30-50% of cases progressing to a chronic deteriorated state within 2-5 years of their first psychotic episode.

Whilst the DSM III-R criteria for diagnosis have been widely used by psychiatrists it is evident that diagnosis may be very difficult for the clinician due to the lack of an independent marker, such as a blood test, which is characteristic of the disease.

Due to the wide range of symptoms encompassed by the term schizophrenia there has been a move to focus research into the disease on specific symptoms or clusters of
symptoms. The first attempts to categorise subtypes of schizophrenic symptoms were produced by Crow (1980) who created the positive and negative symptom dichotomy. Positive symptoms were those characterised by their presence (delusions, hallucinations, thought disorder) whilst negative symptoms were those which signified the absence of a behaviour (affective flattening, alogia, apathy, ahedonia). Crow hypothesised that the two types of symptom were associated with different aetiological factors.

More recently Liddle (1987a) has produced a number of studies showing that the symptoms of chronic schizophrenia separate into three syndromes: Psychomotor poverty, disorganisation and reality distortion. The correlation between symptoms within each syndrome is very high and separate syndromes may co-exist in individual patients. Neuropsychological studies associated with the three syndrome model (Liddle 1987b) have shown specific types of neurological dysfunction associated with different symptoms.

There has been a wide body of research investigating various hypotheses relating to the fundamental causes of schizophrenia. Whilst the primary cause is still unknown it has been generally assumed that schizophrenia has an organic basis. There is strong evidence for a genetic component to the disease and increased risk following birth injury and viral infection during pregnancy.

In terms of the organic pathology of the illness there are two lines of evidence to support a biological explanation of the disease. The first of these was the discovery of
antipsychotic medication. The second was the finding of structural differences in the brains of schizophrenic patients.

Antipsychotic medication, such as chlorpromazine, was first discovered in the 1950s when it was found that the drugs led to a dramatic decrease in the severity of psychotic symptoms. Research has since shown that the medication acts on the receptors of the neurotransmitter Dopamine, blocking its uptake by the cells. In its simplest form the theory suggests that dopamine receptors in schizophrenic patients are supersensitive leading to normal amounts of the transmitter leading to excessive effects.

In terms of the brain mechanisms associated with dopamine, it has been found that the pathways have a role in the control of movement. Treatment with antipsychotic drugs may lead to side effects such as tremor, stiffness and gait disturbances. In addition, tardive dyskinesia (involuntary movements, especially of the mouth, tongue and jaw) may be observed in chronic schizophrenic patients regardless of the medication they have received.

The dopamine theory does not fully explain the causes of schizophrenia and there are many aspects of the theory which are incomplete. Dopamine-receptor blocking drugs do not work in all cases and are in no way a 'cure' for the disease. Approximately half of schizophrenic patients relapse after 2 years on the drugs and there is evidence that they do very little to alleviate negative symptoms. Thus, the dopamine hypothesis may provide an explanation for certain aspects of schizophrenia but it is inadequate to account for the entire range of symptoms.
The second line of evidence for a biological basis to the disease followed the invention of computerised tomography (CT scans) which enabled researchers to look at the brains of live patients. It has been demonstrated that the lateral ventricles of schizophrenic patients are significantly enlarged as compared with normal subjects. The scans do not, however, suggest that it is a neurodegenerative disorder in that the ventricles do not become progressively larger as the disease progresses. Patients were often found to have had enlarged ventricles prior to the onset of symptoms. It must be noted that not all schizophrenic subjects have enlarged ventricles and that they are most commonly found in patients with negative symptoms. The presence of enlarged ventricles is not unique to schizophrenic patients.

It is hypothesised that enlarged ventricles reflect a reduction in size of a nearby structure. Research has suggested that the enlargement is more marked in the area of the temporal lobe (Crow et al. 1989). In addition, the hippocampus may be reduced in size with thinner hippocampal gyri. An interesting finding by Kovelman and Schiebel (1984) looked at post mortem brains of schizophrenic patients and found that the orientation of cells in the hippocampi was haphazard rather than a normal parallel orientation. This pathology was found to be unique to schizophrenia and was not found in other pathologies of the hippocampus, for example Alzheimer’s, Temporal Lobe Epilepsy and Huntington’s Chorea. The hippocampal area is associated with memory and is thought to control the conversion of immediate memories into long-term memories.
Thus, there is sufficient evidence to suggest that schizophrenia is a disease of the brain and that it has an organic basis. The exact nature of the disease and the damaged areas are not yet known and there has been a large amount of research attempting to localise the sites of the pathology. The role of neuropsychology in this research has been to compare the performance of schizophrenic patients, head injured patients and healthy controls on tests of cognitive functioning. In essence, the studies have been aimed at finding whether schizophrenic patients have the same patterns of cognitive deficits as head injured patients where the site of the damage has been localised. The main features of this research are discussed below.

1.3 The Neuropsychology of Schizophrenia

Decades of psychological research has consistently shown that patients suffering from schizophrenia perform more poorly than do healthy controls on a wide array of cognitive tasks. The results of these findings has led to a general acknowledgement that schizophrenia is fundamentally a disorder of the brain and that impaired cognitive performance is a manifestation of the neuropathology of the illness.

As a rule, patients with schizophrenia demonstrate a generalised deficit, that is, they perform at lower levels than controls across a wide variety of tasks. Braff et al. (1991) administered an extended version of the Halstead Reitan Battery and found that patients performed significantly worse than controls on two-thirds of the individual measures tested. Further investigations have sought to discover whether certain cognitive functions are differentially impaired (that is, more impaired than might be expected.
given the patients' general deficit). The cognitive functions which have shown evidence of this differentiation are: Attention, executive function, affect and memory. Attentional deficits in schizophrenic subjects include impairments in the ability to attend to relevant information whilst ignoring unimportant information and the ability to sustain attention. In a review of neurobehavioral studies in schizophrenia Levin (1984a) proposed that there is a dysfunction of the frontal lobe mechanisms that mediate attention and motor behaviour (particularly those involving the mesocortical dopamine projections to the dorsolateral frontal cortex). She proposed that aspects of attention such as distractibility, inability to focus attention, slowness of non-reflexive responses and perseveration were the basis for poor performance by schizophrenics and that these impairments were frequently associated with poor arousal and decreased motivation. In addition there is a phenomenological similarity between the deficits associated with schizophrenia and in those patients with frontal lobe lesions. This suggests that the cognitive deficits in schizophrenic patients are caused by dysfunction in the frontal lobe feedback mechanisms.

In a review of studies of attentional deficits Levin et al (1989) found that the results were consistent with the model proposing frontal system dysfunctions in schizophrenia. In addition, it was found that there were differences in test performance between patients displaying positive and negative symptoms. Chronic patients with negative symptoms produced errors of omission and large numbers of incorrect responses on tasks such as dichotic listening and digit span. Patients with positive symptoms displayed errors of commission and performed better than normals.
Schizophrenic patients have also been found to display significant impairments on tests of executive function. The executive function consists of cognitive processes that allow the subject to respond and adapt to his or her environment (Walsh 1985). These functions include the preparation and execution of action, the initiation and modulation of activity levels and the integration of behaviours into purposeful activity. As noted earlier, these processes are mediated by the frontal lobes although it must be acknowledged that whilst executive functions are mediated by the frontal lobes they may involve other cortical and subcortical sites connected to the frontal lobes.

Tests of executive function are generally those used as a measure of frontal lobe function (see section 1.1). Impairments in frontal lobe functioning has been documented in several studies. Kolb and Whishaw (1983) assessed schizophrenic and healthy controls on a battery of tests previously validated with neurological patients. They found that the performance of schizophrenic patients was impaired on tests measuring right and left frontal and temporal lobe functions.

Gruzilier et al. (1988) produced a more comprehensive study using both schizophrenic patients and affective disorder patients as experimental groups. They found that performance on the tasks was related to clinical symptomology: schizophrenic patients with positive symptoms demonstrated intact performance on tests of left frontal function (verbal fluency) but had deficits on tests of right hemisphere function. The reverse pattern was found in subjects displaying negative symptoms.
In a study by Liddle (1987b) the author divided the subjects according to their symptoms into three syndromes (see earlier). Psychomotor poverty syndrome was associated with impairments on tests of conceptual thinking, object naming and long-term memory. These deficits are associated with damage in the mediobasal areas of the frontal cortex. Disorganisation syndrome was related to impairments on tests of word learning and concentration also associated with the frontal lobes. Reality distortion symptoms were associated with poor figure-ground perception which may reflect temporal lobe impairments.

Weinberger et al (1986) used cerebral blood flow as a measure of functioning for a sample of schizophrenic patients performing the Wisconsin Card Sort Test. They found that patients differed from normal control subjects in the relative changes in prefrontal blood flow during the task. The authors showed an inverse relationship between blood flow and perseveration errors on the test suggesting that impairments of the dorsolateral prefrontal cortex may be linked to task failure.

A summary of the research would suggest that schizophrenics have significantly impaired executive functions. The results are consistent with dysfunction in the network of processes mediating arousal and attention. Whilst these results appear to be very consistent it must be noted that many different designs have been used in the research and it is not yet clear whether prefrontal impairment is specific to schizophrenia or exists in other mental health patients such as those with bipolar or affective disorders.
Studies of memory in schizophrenics have found that patients demonstrate an impaired performance on tests of verbal memory (Koh 1978). In addition, investigators have found that poor recall is related to severity of symptoms and that recall improves with structured tasks (Calev, 1984). The patterns of impairment found suggest that memory deficits are related to poor organisation in the encoding of the memory. It is proposed that deficits in memory of schizophrenic patients is secondary to dysfunction in the executive function (organisation of information) and poor attention.

Disturbances in affect regulation are common in schizophrenic patients, specifically inappropriate or incongruent affect and flattening of affect. Neurological studies have shown that patients with negative symptoms are particularly impaired in the production of vocal affect and the recognition of facial emotion (Levin et al; 1985). In terms of the brain areas implicated in this research it is thought that these deficits are associated with problems in visual decoding which are subserved by a number of brain areas.

In summary, research into the neuropsychology of schizophrenia has implicated general cognitive impairments, frontal lobe and temporal lobe deficits. Confusion over the specific site of damage linked to the disease may be resolved if the time span of the illness and symptom profiles are taken into consideration. It is not, however, clear whether the particular deficits noted are specific to schizophrenia or if they may be found in other disorders.

1.4 Violence - The problem of classification
In any investigation of violent behaviour it would seem to be essential to describe the exact nature of the behaviour under scrutiny. Unfortunately, the literature associated with violence has consistently failed to distinguish different types of violence and has thus been unable to provide conclusive evidence for the factors influencing the aetiology of violence.

There are a number of possible ways to categorise violent offences. Firstly, the object of the attack, that is person vs. property. Within the category of personal violence there is the issue of victim preference and the intimacy of their relationship with the offender. The legal classification of offences takes into account my second category, that is whether the violence was intentional (mens rea) or a consequence of poor judgement and ignorance of the consequences of actions. The third possible category is that of the degree of violence involved, for example a simple assault as compared with murder or manslaughter, sexual assault or rape. A further category is that of persistency, that is the number of violent episodes the offender has committed. A final consideration, particularly when considering mentally disordered offenders is whether the violence was performed in response to a delusional belief.

Very little research has considered possible ways to classify violent behaviour. Volavka (1992) reviews this question, citing studies which have used index offence or impulsivity of the act as a way of characterising behaviour. Robertson et al. (1987) designed a scale taking into account a violence rating for the index offence and a rating of overall violence. By their criteria murder/manslaughter is considered the most extreme form of violent behaviour on both scales.
In terms of looking specifically at the type of violence associated with schizophrenia, studies seem to identify two distinct types of behaviour: the consistently assaultative patient and those who produce single violent episodes (which may be extremely severe) when they are acutely psychotic. Volavka and Krakowski (1989) state that much of schizophrenic violence occurs in recently admitted acutely psychotic patients. It is common that this behaviour will disappear following the treatment of the psychosis with neuroleptic drugs. A small percentage of these cases do not respond to medication and, when transferred to special care wards, continue to be assaultative. The authors add that the persistently assaultative patients have a greater degree of neurological impairment although they do not define the exact nature of the pathology.

In relation to the present study, the criteria for selection for violent patients was their conviction for murder/manslaughter. This type of patient reaches the maximum severity of violence on the Robertson et al. scale and corresponds to the more 'treatable' schizophrenic outlined by Volavka. Persistently assaultative patients were not chosen due to potential difficulties in the testing procedures.
1.5 Mental illness and violence

There have been a number of studies examining the links between mental illness and violent behaviour. The primary focus of this work has been to examine the role of mental illness plays as a contingent risk factor for criminal violence. Recent research into violent behaviour has focused on the concept of risk assessment or "dangerousness". In essence, this research has looked at the likelihood of a past offender damaging property or endangering life if he is released into the community. A number of factors need to be considered including social, environmental and, in the case of disordered offenders, the severity of symptomology. Cirincione (1992) states that the single most effective predictor is a past history of violence: future violence is impossible to predict in the absence of a violent past. In essence this criteria is difficult to utilise effectively due to its property of being relevant only after the event. It is evident that more research needs to be undertaken to provide objective measures of dangerousness which may be used as a screening mechanism to prevent offending.

Whilst it is widely accepted by the general public that mental illness predisposes its sufferers to dangerous and violent behaviour (Monahan, 1988) the experimental evidence to support this view is equivocal. Estimating the contribution of mental illness to violent behaviour has been a major dilemma for researchers who have run into numerous methodological and ethical difficulties. Specifically, attempting to establish the differences between "true" rates of mental disorder and crime and their "treated " rates, that is, those who are confined within a secure psychiatric hospital or a prison (Monahan and Steadman, 1983). As such, it has been extremely difficult to discover whether the base rate for violence is higher for the mentally ill than for the general
population. Statistics may vary as a function of the diagnostic criteria for schizophrenia used, socio-economic status, demographics and unknown factors such as whether schizophrenics are more likely to be caught than non-mentally ill offenders or that they are less likely to be convicted.

Monahan and Steadman (1983) reviewed 200 studies on the association between crime and mental disorder. Their conclusions may be summarised as:

"the relationship between... crime and mental disorder can be accounted for largely by demographic and historical characteristics that the two groups share. When appropriate statistical controls are applied for factors such as age, gender, social class and previous institutionalisation, whatever relations between crime and mental disorder are reported tend to disappear." (p.152)

Since their original paper, Monahan and Steadman have conducted further reviews of the literature and comment that methodological rigour has led to superior research over the past 10 years. In a revised paper Monahan (1993) concludes that there does appear to be a relationship between violent behaviour and mental disorder. However, he emphasises that this relationship is only apparent for those patients experiencing psychotic symptoms (Link et al. 1992). In addition, approximately ninety percent of those patients who are currently suffering from a mental illness are not violent. In terms of the perception of mental disorder as a risk factor for criminal violence its contribution must be seen as relatively small when compared to factors such as gender, age, socio-economic status and substance abuse.

Thus, whilst it is probable that the base rate of violent behaviour is greater for mentally disordered patients than for the general population, by no means all the mentally ill are
violent and their "dangerousness" must be assessed with respect to numerous other known contributory factors.

Research focusing specifically on the contribution of schizophrenic illness to criminal behaviour is rare. The few studies that have addressed this issue have tended to consider the issue in terms of epidemiological rather than aetiological data.

The figures quoted for the proportion of schizophrenics who are violent range between 8 and 45% (Shader et al. 1977) with estimates being affected by both diagnostic and demographic factors. Taylor and Gunn (1984a, 1984b) produced a comprehensive study of 1264 men who represented all those remanded on violence charges in Brixton prison. A more detailed investigation was performed on 203 of the men, 90 of whom were regarded as having schizophrenia.

It is interesting that the criminal activities of the psychotic men differ quite significantly from those of the non-psychotic in terms of the degree of violence, the antecedents to the offence and the characteristics of the victims. The major factor that the study highlights is that among those schizophrenic men who were violent, 79% had first acted "in a way that could have been construed as aggressive" after the onset of their illness. In addition, when considering actual injury inflicted (rather than random violence) in 88% of cases the assault followed the onset of the schizophrenia. In the 40 cases sampled, Taylor & Gunn (1984b) found that the peak of violent behaviour usually occurred between 5 and 10 years after the onset of the illness.
Thus, it is possible to conclude that there is some aspect of schizophrenic symptomology which increases the likelihood of violence in patients who had no previous history of violence prior to the onset of the disorder. The nature of this link is one that needs to be explored. Specifically, there is a need to tease out the organic and social sequelae that may be predictive of violence.

The Taylor and Gunn (1984a) study also provides information about the rates of homicide in schizophrenic men. It was found that there was a significantly higher diagnosis of schizophrenia in men charged with murder/manslaughter (11%) than would be expected in the general population (0.1-0.4%).

The contribution of delusions and hallucinations to criminal violence by schizophrenics has also been explored. In Taylor's study 112 out of 121 men with a psychotic illness had positive symptoms at the time of the offence. Taylor (1985) found that 40% of the psychotic men had been acting on delusional beliefs when they committed the offence. However, Virkkunen (1974) found that only about one third of violent offences are committed during a psychotic episode. Krakowski et al. (1986) proposed that the type of violence may vary as a functions of the schizophrenic symptomology. For example, paranoid delusions may lead to well planned, specifically directed and dangerous violence. In contrast, more disorganised psychotic states may produce less focused and consequently less dangerous behaviour.

In general, attempts to associate different types of violence with subtypes of schizophrenia have been inconclusive. In a review of 8 studies it was found that five
showed paranoid schizophrenics were more violent than non-paranoid patients. Two studies reported the opposite result and one found no difference. It is suggested that discrepancies may be accounted for by examining the time span of the illness in terms of increases in violence during the acute phases of the illness which disappear as patients become chronic and hospitalised. In addition, the distinction between paranoid and non-paranoid schizophrenia is rather crude and is being replaced by more sophisticated models of schizophrenic subtypes (see earlier section.)

The role of command hallucinations in offences is considered minor (Taylor 1985; Hellerstein et al. 1987), as is the role of delusional misidentification.

Essentially, it is not clear as to the exact contribution of positive symptoms to violent behaviour. Whilst many acts of extreme violence may be committed under the influence of a paranoid delusional state, by no means all violent offences are mediated in this way.

1.6 Violence and its cognitive correlates.

The literature on the neuropsychology of violence has produced a number of contradictory and inconclusive results. This confusion may well be attributed to the unsatisfactory definitions of violence used by researchers (discussed earlier in this paper). Whilst it is clear that there are a wide range of factors, such as social and environmental considerations, that may contribute to the aetiology of violent behaviour, there is clear evidence that cerebral pathology plays an important role in many instances. The nature of the impairments, in terms of their location and behavioural
sequelae, are yet to be fully explored. However, a large number of studies have been undertaken and their results reviewed below.

Robertson, Taylor and Gunn (1987) studied the relationship between cognitive function and violence in 76 remanded prisoners without psychiatric illness. Subjects completed a WAIS, verbal fluency, visual retention and visual recognition tasks. Their findings showed that whilst violent offenders tended to be of lower general intelligence than the non-violent group there was no relationship between specific patterns of cognitive impairment and violence.

In contrast to these results, there have been a number of studies suggesting that violent behaviour is the expression of a variety of insults to the central nervous system. (Mark and Ervin 1970; Monroe 1970). Violent prisoners showed significantly more brain dysfunction than non-violent offenders on a neuropsychological test battery (Spellacy 1978). Martell (1992) conducted a study of 50 male patients at a maximum security forensic hospital. He collected data relating to 5 indicators of cerebral pathology including cognitive impairments and abnormal neurological findings. The results revealed that at least one indicator was present for 64% of the sample. In addition, subjects with such a history were significantly more likely to have committed a violent criminal act. In terms of the other behaviours typically characterising the brain injured behaviours, Martell identified poor judgement, impulsivity and explosive rage as indicators of the sorts of brain injury leading to violence.
With regard to the localisation of neurological impairment studies have produced results supporting both temporal and frontal lobe abnormalities.

The temporal lobes have frequently been linked to violent behaviour due to the findings of EEG recordings linking electrical discharges with violent outbursts. In addition there is a strong link between Temporal Lobe Epilepsy and violent behaviour. Volavka et al. (1992) conclude, however, that these factors probably contribute very little to an understanding of violent crime.

Neuropsychological studies looking at frontal lobe function in violent offenders have also produced evidence for a link. The role of the frontal lobes in human behaviour have been discussed in some detail above. Their role in the pathogenesis of violent behaviour is linked to their function as an executive control in the regulation of behaviour. In particular, in controlling, planning and understanding the consequences of actions. Tancredi and Volkow (1988) used Positron Emission Tomography (PET) to look at brain activity in 4 violent inpatients. Half of the sample had dysfunction in the frontal cortex.

Yeudall (1977) studied aggressive psychopaths in comparison to depressed criminal patients. His results indicated neuropsychological impairments in the frontal cortex for both samples. Violent behaviour has also been linked to frontal lobe lesions in Heinrichs (1989) study of neuropsychiatric patients which used CT scans to localise the damaged areas.
Neuropsychological studies of delinquent behaviour have also shown strong links between deficits in executive function and antisocial behaviour. Whilst delinquent behaviour is not defined in the literature (thus leading to confusion over the levels of violence exhibited) it is assumed that delinquency necessarily involves a number of dangerous and/or anti-social acts. Miller (1988) reviews the studies concluding that subjects show deficits in verbal skills, concept integration, impulse control, anticipation of the consequences of action and the utilisation of feedback to modify behaviour. All these processes are thought to be mediated by the frontal lobes.

Berman and Siegal (1976) found significant deficits in the performance of delinquents on tasks such as Trails B (a test of mental flexibility) and the category test (a concept formation task requiring integration of past information). Spellacy (1977) specifically looked at violent adolescents in contrast to a non violent sample. The violent group were more impaired on tests of visual memory and organisation and language related tests.

There have been few studies which specifically address the neuropsychology of violent behaviour in schizophrenic patients. Krakowski et al (1989) produced a well executed study dividing schizophrenic patients into high, low and non-violent groups. The criteria were based on frequency of assault behaviour as opposed to the severity/extreme nature of the violence itself. The study found significantly more impairments in the high violence group specifically on visuo-spatial functions such as block design, digit symbol (from the WAIS) and the Benton visual retention test. Impairments were
specific and indicated discrete areas of functional deficit; specifically the integration of sensory and motor information thought to occur in the frontal lobes.

Rasmussen and Levender (in press) conducted a study of violent and non-violent schizophrenic patients and healthy controls using symptom rating scales, personality inventories and neuropsychological tests. No differences were found in symptom ratings. Neuropsychological tests suggested that violent schizophrenic patients performed worse on tests of frontal lobe function than the other groups. Whilst this study is relevant to the present investigation it is important to note that the authors did not define their criteria for violence and all subjects were taken from a medium secure unit. In addition, the tests used were a new computerised battery which has not yet been sufficiently standardised to allow effective comparisons to be made.

1.7 The present study

The investigation described in this paper is an attempt to clarify some of the issues discussed in the literature and to answer some outstanding questions. What is clear from the studies reviewed in this paper is that there is very little research looking specifically at schizophrenia and violence from a neuropsychological perspective.

It is evident that the presence of a schizophrenic illness increases the likelihood of violent behaviour in its sufferers. Whilst this increased risk is present, it is by no means predictive in itself and the vast majority of schizophrenic patients never commit a violent act. The role of symptomology in the incidence of violent behaviour is unclear but there are a significant number of patients who do not commit violence in response to
positive symptoms. The Taylor study (1985) finds significant evidence that violent behaviour generally occurs following the onset of the disease thus implicating the disease process itself in the aetiology of violent behaviour. The studies also suggest that the incidence of schizophrenia in men who commit extreme violence, murder/manslaughter, is higher than the figures for the general population would suggest. The question that arises from this research is: What features of the illness place certain sufferers at increased risk of violence?

Looking at the question from a neuropsychological perspective, it seems clear that there is some evidence to support a hypothesis implicating frontal lobe impairments in both schizophrenia and violent behaviour. Research has shown that schizophrenic patients and violent offenders tend to perform badly on tests which measure frontal lobe functions. In particular, subjects show deficits on tasks of abstraction and concept integration, response suppression, and long term planning.

Studies may be criticised on two counts;

1. The type of behaviour considered violent is not adequately defined leading to research into heterogeneous 'violent' populations

2. Researchers have used schizophrenic patients without taking into account differences in symptomology which may be reflected in the pattern of cognitive impairments found.
The present investigation uses the criteria of an index offence of murder/manslaughter to provide a precise definition of the level of violence under investigation. This criteria is consistent with the most extreme form of violence on Robertson et al. (1987)'s scale. In addition, there may be a specific link between murder/manslaughter and schizophrenia (indicated in Taylor 1985).

In order to counter the second criticism, the investigation is using the SANS (Andreason 1982) and the Manchester Scale (Krakowski et al. 1977) to provide a profile of the symptoms of the subjects used in terms of their presence/absence and their severity.

1.8 Hypotheses

The aims of the present study are to test the following hypotheses:

1/ Schizophrenic patients who have committed murder will show impairment on tests pertaining to frontal function.

2/ The pattern of impairments on the tests will differ between the two groups. This is purely an exploratory hypothesis and specific differences relating to behaviour are not predicted at this stage.
SECTION 2 - METHODOLOGY

2.1 Design

The study presented is a comparison of two groups of subjects on measures of neuropsychological functioning, specifically performance on tests of frontal lobe function. The experimental group consists of schizophrenic male patients from a maximum security psychiatric hospital, namely, Broadmoor Special Hospital, whose index offence (i.e. the offence which led to their admission to the hospital) is murder or manslaughter on the grounds of diminished responsibility. This selection criteria is based on Robertson et als'(1987) scale for assessing levels of violent behaviour. In addition, the incidence of murder/manslaughter is increased in schizophrenic patients than for the general population.

The control group is a sample of schizophrenic outpatients from Queen Mary's Hospital, Roehampton who have never been convicted of a violent offence nor been resident in a maximum security hospital.

2.2 Subjects

A total of 28 subjects took part in the study. Each of the two groups had 14 participants.

Research and Ethics Committee approval was obtained from both the Broadmoor and Queen Mary's Hospital Ethics Committees.
2.2.1 Experimental Group

Subjects for the experimental group were taken from a maximum security psychiatric hospital according to the following selection criteria:

1. A diagnosis of schizophrenia according to DSM-III-R (American Psychiatric Association 1987) as recorded in their medical records.

2. Index offence of murder or manslaughter on the grounds of diminished responsibility.

3. No increase in psychotropic medication in the preceding month.

4. No diagnosis of seizure disorder. Forms of epilepsy are known to be associated with violent behaviour and their inclusion in the study may have compounded the results.

5. Aged less than 55 years. Elderly patients were excluded to avoid possible confounding effects of age-related deterioration in cognitive ability.

6. Admission to the hospital within the past 5 years. This criteria was included in order to minimise the effects of institutionalisation.

7. A NART IQ of over 80. This criteria was added in order to exclude a diagnosis of learning disability in the sample. In addition none of the control patients had an IQ below 80 and it would have thus presented problems in matching the samples.
The medical records office of the hospital provided a list of patients who were diagnosed as mentally ill with their date of birth and dates of admission. Forty-four patients were below age 55 and had been admitted in the past 5 years. Eight of the sample were not diagnosed as schizophrenic and were thus excluded. Three more patients who were consistently assaultative and held in the intensive care wards were also excluded due to difficulties in gaining consent for testing.

The medical notes, including results of previous neuropsychological tests were then investigated for the remaining 33 patients. Eight of the patients had an IQ lower than 80 (as tested by the NART) and were excluded from the study.

In order to gain permission to test the subjects, written consent was required by the Responsible Medical Officer for each patient. The RMO was also required to state whether the patient was capable of giving informed consent to the testing. (See Appendix 2.) Consent was not given for two of the patients who were considered by their RMO to have been 'over-tested' in recent months. In addition, four of the patients were on the admissions ward and their RMO did not feel that they were in a stable enough condition to be tested accurately as they had only been at the hospital for 3 months.

The nineteen remaining patients were contacted and times arranged for testing to take place. Written consent was required from each of the patients prior to testing. Three
patients refused to do the tests. One further patient was excluded during testing due to the fact that he could not read nor write.

Only one female patient met the selection criteria and was not included in the study due to potential difficulties in analysing the results for one subject.

A total of fourteen patients were tested at the hospital. The average age of the patients was 36 years (range 25-54). The mean NART IQ was 97 (range 81 to 116).

2.2.2 Control Group

Control subjects were chosen from a sample of schizophrenic outpatients at Queen Mary's Hospital, Roehampton. Patients' names were taken from a list of subjects used for a study undertaken in 1993 by Dr. E. Joyce. The selection criteria for the previous study were identical to those used in selection for the present study excluding numbers 2 and 6. For the present study an additional criteria was included: No history of arrest for violent behaviour prior to or since being diagnosed schizophrenic.

Control subjects were matched on the basis of age, sex and premorbid IQ (as tested by the NART). The mean age of the control subjects was 39 years (range 27 - 54) and their mean IQ was 104 (range 92 - 124).
2.3 Procedure

Testing of subjects took place between February and May 1994. For the Special Hospital sample, permission to see the patients was required from the Responsible Medical Officer (RMO) prior to testing (see appendix 2). In addition the RMO was asked to confirm that the patients themselves were capable of giving informed consent.

Whilst permission was being sought, previous test results performed by the hospital's neuropsychologist were recorded on a database and a checklist of missing results compiled for each patient. Due to the varying test-retest timespans it was considered acceptable to use these existing results. All the tests used had standard administration procedures thus minimising complications of different result interpretations. None of the previous testing had been performed more than 2 years prior to the present investigation. Results were taken from the psychology case notes and confidentiality was assured by using a password-secure database.

Additional information recorded on the database included: Name, age, date of birth, year of first diagnosis, date of admission to Broadmoor, ward, date of testing and handedness (see Appendix 6).

Meetings were arranged with the patients via ward staff. The purpose of the investigation was presented to each patient (see Appendix 3&4) and they were made
welcome to ask any questions about the study that they wished. Written consent was obtained from all the patients tested.

Testing took place on the ward where the patient was resident in a room away from any disturbances. The procedure took between 15 and 35 minutes depending on the number of tests which needed to be completed. All the patients had to do at least three new tests during the procedure. None of the patients had to do the NART test as these had all been completed previously. In addition, only one subject had to be tested on the Weschler Memory Scale subtests.

Prior to testing subjects were asked which hand they used for writing. In addition, they were asked if they had needed a change in medication any time in the preceding month.

Tests were administered in a random order with the exception of the Weschler Memory Scale subtests. For these tests immediate recall was taken at the start of the testing session and delayed recall at the end (or after half an hour, whichever was the longer period).

Following the completion of the neuropsychological tests, Dr. T. Exworthy (Senior Registrar) arranged to meet the patients to administer the Scale for Assessment of Negative Symptoms (SANS; Andreason, 1982) and the Manchester Scale (Krawiecka et al., 1977). These tests were also undertaken on the ward with the help of nursing staff.
The control subjects were chosen from a list provided by Dr. E. Joyce at Queen Mary's Hospital, Roehampton. All were outpatients from the hospital and were first contacted by letter to inform them about the study. Many of the sample were tested when they were visiting the hospital for DEPO medication whilst others were attending activities at the hospital day centre.

The control subjects had already been tested on the NART the previous year for the purposes of a research project. In addition, results for the Verbal Fluency test were available in the hospital records as well as symptom ratings using the SANS and the Manchester Scale (administered by Dr. P. Crighton). These results were transferred to the database along with other demographic information and the date of testing.

Control subjects were also required to give written consent to the study and were invited to ask questions if they were unclear about the procedure.

All testing of controls took place in an office at Queen Mary's Hospital.

2.4 Neuropsychological Tests Used

*National Adult Reading Test (NART)* (Nelson and O'Connell, 1975)
The NART was devised to test the residual level of vocabulary in patients with dementing conditions. Vocabulary correlates highly with general ability and tends to resist the dementing process.

The NART comprises a list of 50 words which are phonetically irregular (e.g. Debt). Patients are asked to read the list and their errors of pronunciation are noted. Correct pronunciation of a word is used as evidence for a premorbid familiarity with that word. The error score may be converted into a predicted IQ score using tables provided with the test. Unlike the WAIS the ceiling score is 125 rather than 150.

*Verbal Fluency Test*

The procedure used in the present investigation was based on the Thurstone Word Fluency test. Subjects were first asked to think of as many words as they could in one minute. Proper nouns were allowed. Subjects were asked to close their eyes during this part of the test to prevent cueing from the environment.

The second part of the test involved asking the subject to state as many items from a particular category in one minute. The categories were furniture and animals and were alternated between patients to get a random spread of scores.

The final part of the test asked subjects to state as many words as they could beginning with the letter 'S' in one minute.
The Verbal Fluency test is considered to be a measure of the changes in speed and ease of verbal production which may follow brain injury. Patients with left frontal lesions tend to have a greatly diminished verbal output and perform poorly on this test.

*The Stroop Test (Trenerry et al. 1989)*

The material for this test consists of two white sheets of paper with colour names - BLUE, GREEN, RED and TAN - printed in random order in four lists (28 words down) across the page. The words themselves are randomly printed in one of these four colours. The test used is based on the original Stroop (1935).

Initially, the subject is asked to read down the columns as fast as possible. The time taken to read all 112 words is recorded. In the second part of the test (the contrast condition) the subject is given the second sheet and asked to read "the colour the word is written in" rather than the colour name. The subject is timed for two minutes and the number of words read noted. In addition, the number of errors and corrected errors are recorded.

The Stroop test is a measure of the ease with which the patient can shift his perceptual set to conform to conceptual demands. It is noted that frontal patients have difficulty in making mental or behavioural shifts necessary to complete this task accurately. Patients have difficulty in inhibiting the automatic response (that is, the colour name written) and take longer to complete the task. Frontal patients also make more errors on the test than healthy subjects.
The Rey-Osterrieth Complex Figure Test. (Rey, 1941; Osterrieth, 1944)

This test was devised by Rey to investigate perceptual organisation in brain damaged subjects. The material consists of a copy of Rey's figure and a set of coloured felt-tip pens. The subject is asked to copy the figure as accurately as possible. The figure is placed in the horizontal plane by the experimenter. The experimenter watches the subject's performance and, at the completion of a 'section' the patient is handed a different coloured pen, noting the order of the colours used. This procedure is undertaken so that the experimenter can look back at the order in which the drawing was completed. For the purposes of this investigation, the drawings were rated using Osterrieth scoring. This method ranks the drawings from I-VII dependent on the order of copying and the final result.

Performance on the Rey figure copying test is thought to be impaired in patients with frontal lesions who may have difficulty in effectively planning and implementing a copying strategy. Thus, such patients may copy the figure in a piecemeal fashion producing a poor overall result.

Trail Making Test (Reitan, 1958)

The trail making test is administered in two parts, A and B. Part A requires the subject to draw lines to connect consecutively numbered circles on a work sheet. The beginning and end are indicated and the sequence goes from 1 to 25. The subject is
asked to complete the task as quickly but as accurately as possible without taking their pen from the paper. A trial run is provided on the reverse of the paper. The time taken to complete the task and the number of errors are recorded.

In part B, the subject is given a second work sheet which has consecutively numbered letters and numbers. Starting with number 1 the patient is asked to join up the circles, alternating between the numbers and letters. (e.g. 1-A-2-B), again as accurately and as quickly as possible. The time taken to completion and the number of errors are noted.

The Trail Making Test is considered to be highly sensitive to the effects of brain injury. Patients with frontal lesions are known to be impaired on this test as a result of difficulties in making behavioural shifts which the task demands.

*Weschler Intelligence Scale for Children - Mazes subtest. (Weschler 1949)*

The Maze subtest of the WISC consists of nine mazes, three simple trials and six test mazes which increase in complexity through the test. The first trial maze is completed by the experimenter who indicates that they are starting from the centre and completing the maze without crossing any barrier lines nor going back on themselves. The subject is instructed to complete the other two trial mazes and that timing will start when they move onto the test mazes. The subject is asked to complete the mazes as quickly and as accurately as possible. The time taken for the subject to complete the test is noted as well as the number of complete, correct mazes and the number of errors made. Errors include breaking through a barrier line and going backwards.
Maze tests are often used as a measure of frontal lobe function due to the large planning component involved in effectively completing the maze. In addition, frontal patients are known to 'break rules' and tend to have a higher proportion of errors than healthy subjects.

*Classical Weigl (Weigl 1941)*

The material used in this test involves a set of coloured, cardboard shapes (made by the experimenter). The required shapes are: four triangles, four circles and four squares each coloured in red, blue, green and yellow.

The experimenter lays out the shapes in a random array in front of the subject who is required to "place the pieces into piles in a way that is sensible". If this is achieved satisfactorily (i.e. using shape or colour as a sorting rule) then the experimenter again lays out the pieces in a random way and asks the subject to think of a different way of sorting the pieces into piles. In order to pass the test the subject must sort the pieces by shape and colour separately in the two parts of the test. If the subject uses shape initially and then colour *but retains the shape sequence in the pile* then they are awarded a secondary pass (P2).

There are a number of prompts which the experimenter may need to provide: If the subject arranges the shapes into lines then the experimenter asks them to put the shapes into piles. Often subjects use the same sorting category in both sections of the test but
will use a design rather than piles in order to arrange them in a "different way". The experimenter may prompt the subject once by pointing out that they have used the same sorting category (i.e. shape or colour) and ask them to do it differently. If the subject does not pick up the second category after the initial prompt then the experimenter may tell them the second way of sorting. The subject is given a score of 4 or 5 which indicates a failure on the test.

The Weigl task is the original task from which the WCST was developed. The Classical Weigl was used for the present study as it is the test which is generally used for admissions assessments to Broadmoor. It was found that 30% of admissions patients failed the Weigl and it was thus suggested that this was sufficiently discriminatory for the sample being tested. The WCST is a very long battery which is used for patients with high premorbid IQ. Broadmoor patients tend to be of low IQ and the WCST would not be discriminatory for them.

*Cognitive Estimates Test (Shallice and Evans 1978)*

This test is considered to be a measure of failure in 'judgement' and in dealing with novel situations frequently shown in patients with frontal lobe impairments. The test involves asking subjects to give the 'best guess you can' in answer to a series of questions. Subjects are informed that they are 'almost certain not to know the exact answer'. The test consists of ten questions. Patients are given as long as they need to answer and the question may be repeated. Scoring is based on the reasonableness of the
guess given. Shallice states that the test requires a subject to select an appropriate
cognitive plan and to utilise items of common knowledge in a novel way.

_Hayling Sentence Completion Task (in Burgess, P.W., and Shallice T., 1993)_

This test is presented in two parts. In part A, the subject is presented with a series of
sentences which have the last word omitted and asked to provide the word which
completes the sentence. In each case the last word is cued by the sentence frame, for
example, Bloom and Fischler (1980) found a probability of .99 that the sentence "He
mailed the letter without a ..." would be completed with the word "stamp".

In part B, the subject is asked to produce a word which makes no sense in the sentence
context. If the subject is unable to do this or is merely producing opposites, the
experimenter may prompt the subject by saying, "very good, but can you think of a
word that is more nonsensical". Scoring is based on the level of abstraction the subject
achieves; an actual completion response receives an error score of 3, a word
semantically related to a word in the sentence (and opposites) are given a score of 1 and
an unrelated word receives a score of 0.

The test is considered to measure both a subject's ability to initiate words and also the
ability to inhibit the automatic response and initiate a novel reply. Frontal lobe patients
tend to perform poorly on this test.
Weschler Memory Scale - Revised. Subtests (Weschler 1988)

Logical Memory

In this test the subject is read two stories and asked to recall as much information as they can remember immediately after each presentation. Half an hour later (at least) the subject is asked to recall the information again. The recalled information is written down verbatim and scored as per the WMS-R scoring criteria.

The Test is thought to be a test of memory. In addition, frontal patients may perform poorly on the test due to impairments in the ability to organise material effectively for memorisation.

Design Recall

Subjects are shown a series of 4 designs which become gradually more complex. Each design is seen for 20 seconds and then the subject is asked to draw what they can remember. As with the logical memory task, a delayed recall test is performed after half an hour.

Symptom Rating Scales

SANS

The SANS is a scale developed specifically to act as a standard instrument to assess negative symptoms of schizophrenia. The symptoms are defined by objective behavioural indices and have excellent inter-rater reliability. There are 5 symptom
complexes assessed by the scale: Affective flattening, alogia, avolition, anhedonia and attentional impairment. Each of the symptoms within the complexes is given a score from 1 to 5 by the rater. A summary score and composite score are also calculated to give an overall rating of severity.

*The Manchester Scale*

This scale provides a standardised psychiatric assessment for rating chronic psychotic patients. The scale has three sections: ratings made in replies to questions, ratings made by observation and medication side effects. The scale is short, easy to administer and has high inter-rater reliability.

Copies of the scales are provided on the data sheets in Appendix 6.
SECTION 3 - RESULTS

The results analysed in this paper are the raw scores for the neuropsychological tests and the symptom severity scores. All the data is normally distributed and the statistical tests used automatically check for equality of variance (using Levine's Test) prior to calculating the significance of the results.

There are 2 sets of missing data: One subject did not complete the Stroop test because he was colour blind; one other subject did not complete the Verbal Fluency tests.

All statistics were calculated using SPSS for Windows.

3.1 Matching

The two groups were matched for age, NART IQ and year of diagnosis. The means and standard deviations are shown in Table 3.1. A series of t-tests were performed on these scores to confirm that the subjects had been matched effectively. The results showed that there were no significant differences between the two groups on these three measures.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Means for violent group</th>
<th>Standard Deviations for violent group</th>
<th>Means for non violent group</th>
<th>Standard Deviations for non violent group</th>
</tr>
</thead>
<tbody>
<tr>
<td>NART IQ Score</td>
<td>103.64</td>
<td>10.16</td>
<td>103.93</td>
<td>9.83</td>
</tr>
<tr>
<td>Age -Years</td>
<td>35.71</td>
<td>7.65</td>
<td>38.71</td>
<td>8.30</td>
</tr>
<tr>
<td>Year of First Diagnosis</td>
<td>1984</td>
<td>6 years</td>
<td>1981</td>
<td>8 years</td>
</tr>
</tbody>
</table>

Table 3.1 - Means and Standard Deviations for Matching Variables.
3.2 Neuropsychological Test Scores

3.2.1 Means and Standard Deviations

The results show that both the violent and non violent schizophrenic patients show similar patterns of impairment on the neuropsychological tests. It is, however, interesting to note the high standard deviations found in the out patient sample on a number of the tests. It would appear that there is a large amount of variation in the scores within each group suggesting that individual performances vary greatly across the two groups. The general trend of the results shows that the Broadmoor sample perform slightly better than the Queen Mary's subjects. The exceptions to this are the WISC Mazes Time score and the immediate recall on the WMS-R designs subtest.
<table>
<thead>
<tr>
<th>Variable</th>
<th>Means for violent Group</th>
<th>Standard Deviations for violent group</th>
<th>Means for non-violent group</th>
<th>Standard Deviations for non-violent group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verbal Fluency</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nouns</td>
<td>20.86</td>
<td>9.03</td>
<td>25.08</td>
<td>13.67</td>
</tr>
<tr>
<td>Animals</td>
<td>15.71</td>
<td>5.88</td>
<td>13.46</td>
<td>5.55</td>
</tr>
<tr>
<td>'S'</td>
<td>11.86</td>
<td>4.82</td>
<td>12.54</td>
<td>5.95</td>
</tr>
<tr>
<td>Stroop</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. in 120 sec.</td>
<td>81.62</td>
<td>22.13</td>
<td>67.79</td>
<td>27.36</td>
</tr>
<tr>
<td>Errors</td>
<td>4.31</td>
<td>3.64</td>
<td>17.29</td>
<td>31.30</td>
</tr>
<tr>
<td>Weigl</td>
<td>2.07</td>
<td>1.49</td>
<td>3.86</td>
<td>1.61</td>
</tr>
<tr>
<td>Trail Making</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A - Time Sec.</td>
<td>47.57</td>
<td>15.88</td>
<td>55.29</td>
<td>19.99</td>
</tr>
<tr>
<td>A-Errors</td>
<td>.21</td>
<td>.58</td>
<td>.50</td>
<td>.94</td>
</tr>
<tr>
<td>B - Time Sec.</td>
<td>116.57</td>
<td>51.26</td>
<td>126.86</td>
<td>65.93</td>
</tr>
<tr>
<td>B - Errors</td>
<td>1.29</td>
<td>1.86</td>
<td>3.85</td>
<td>5.16</td>
</tr>
<tr>
<td>WISC Mazes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. Correct</td>
<td>4.93</td>
<td>1.07</td>
<td>4.79</td>
<td>1.53</td>
</tr>
<tr>
<td>Errors</td>
<td>1.71</td>
<td>1.86</td>
<td>5.79</td>
<td>4.10</td>
</tr>
<tr>
<td>Time</td>
<td>235.57</td>
<td>101.71</td>
<td>177.64</td>
<td>54.03</td>
</tr>
<tr>
<td>Rey Figure</td>
<td>3.5</td>
<td>1.344</td>
<td>3.64</td>
<td>1.60</td>
</tr>
<tr>
<td>Cog. Estimate</td>
<td>8.29</td>
<td>4.10</td>
<td>8.57</td>
<td>4.69</td>
</tr>
<tr>
<td>Errors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weschler Memory scale</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prose - immed. recall</td>
<td>15.68</td>
<td>7.45</td>
<td>15.86</td>
<td>7.60</td>
</tr>
<tr>
<td>Prose - del. recall</td>
<td>11.71</td>
<td>7.45</td>
<td>10.43</td>
<td>7.27</td>
</tr>
<tr>
<td>Design - Immed. recall</td>
<td>24.71</td>
<td>11.15</td>
<td>30.71</td>
<td>7.25</td>
</tr>
<tr>
<td>Design - del. recall</td>
<td>17.36</td>
<td>10.66</td>
<td>22.64</td>
<td>11.70</td>
</tr>
</tbody>
</table>

Table 3.2 Means and standard deviations for each dependent variable
An assortment of normative data may be found in Appendix 5. Whilst the results cannot be compared directly it would appear that both samples are performing below the mean score expected on most of the tests. A summary of these comparisons is provided below:

1/ Both samples perform below the adult average on all three of the Verbal Fluency tests. As no standard deviations are available it is not known how significant this poor performance is in relation to healthy populations.

2/ The mean scores for the Stroop for the Broadmoor sample fall into the bottom 5th percentile. The mean for the non-violent subjects is in the lowest 2% of the population.

3/ The only norms available for the Weigl are from Broadmoor data which show that 54% of the admissions receive a score of 1. The means of the two groups in the present study both fall below this.

4/ Norms for the Trail Making tests would suggest that all the subjects in the present study perform below the 25th percentile which is mildly suggestive of brain damage.

5/ The WISC maze scores for the two groups show that both produce more errors than the admissions sample but that the times and number of mazes correct are similar.
6/ The subjects in the present study perform poorly on both the Cognitive Estimates task and the Hayling Sentence completion as compared with research samples by Burgess and Shallice. The standard deviations for the Cognitive Estimates task show that both subject groups in the present study fall below 2 standard deviations of the control mean.

7/ All the subjects performed relatively poorly on the WMS-R, with both group means falling below the 50th percentile on all the subtests.

3.2.2 t-Tests

A series of t-tests were performed in order to compare the mean scores of the groups for each of the tests administered. Only two of the tests produced significant results:

1/ The mean scores on the Classical Weigl for the two groups were significantly different (t value = -3.04; df=26; P= .005 two-tailed). The scores showed that the control group scored significantly worse on the task than the Broadmoor sample.

2/ The Error score on the WISC Mazes was significantly different between the two groups. (t value = -3.39; df=26; 2 tail sig. = .002). As with the Weigl the control group performed more poorly on the task producing more errors than the Broadmoor sample.

These results are not consistent with Hypothesis 1, that the Violent group would show greater impairments on the frontal tasks than the non violent group. However, on the
two tests quoted the performance of the two groups differed significantly thus supporting hypothesis 2.

### 3.2.3 Discriminant Function Analysis

Unlike the t-tests, the discriminant function analysis uses correlations between the scores of the two groups rather than the mean values. The test provides a measure of the ability to predict which group an individual may fall into on the basis of their neuropsychological test data.

<table>
<thead>
<tr>
<th>Actual Group</th>
<th>No. of Cases</th>
<th>Predicted Group</th>
<th>Membership</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Group 1</td>
<td>14</td>
<td>11</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>78.6%</td>
<td>21.4%</td>
</tr>
<tr>
<td>Group 2</td>
<td>14</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td></td>
<td>21.4%</td>
<td>78.6%</td>
</tr>
</tbody>
</table>

Percent of group cases correctly classified: 78.57% N=28

**Table 3.3 - Discriminant Function Analysis**

The above results show that it is possible to predict the group membership with 78.57% accuracy on the basis of an individuals' neuropsychological test performance.

The test identified only one function. The results which related significantly to the function were high scores on the Weigl (that is, poor performance), high numbers of errors on the Trail Making Test B, and low scores on the Rey figure. The Broadmoor
sample were found to score low on function one (Group Centroid -.87486) whilst the control group scored highly (.94776).

These scores do not support Hypothesis 1 as they suggest that the experimental group perform better than the control sample on many of the tasks.

Whilst the results appear to support hypothesis 2, in that they suggest that the two groups show different patterns of impairments, they must be regarded in the context of the t-test results which would not suggest that there are significant variations between the scores of the two groups on the neuropsychological tests. The discriminant function analysis is based on correlations and the results may have been affected by correlations between the tests used.

3.2.4 Correlations

It would be expected from the nature of the tests used that there would be some correlations between the test scores. Due to constraints on space in this document Table 3.4 provides a summary of those correlations which are significant.
<table>
<thead>
<tr>
<th>Variable</th>
<th>Correlated with..</th>
<th>Correlation Coefficient</th>
<th>2-tailed significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verbal Fluency- Nouns</td>
<td>Verbal Fluency -Animals</td>
<td>.49</td>
<td>.028 *</td>
</tr>
<tr>
<td></td>
<td>Verbal Fluency-'S'</td>
<td>.59</td>
<td>.006 **</td>
</tr>
<tr>
<td>Verbal Fluency - Animals</td>
<td>Stroop - no. of word read in 120 sec</td>
<td>.18</td>
<td>.022 *</td>
</tr>
<tr>
<td></td>
<td>Weigl</td>
<td>-.41</td>
<td>.032 *</td>
</tr>
<tr>
<td></td>
<td>WMS - Prose Delayed Recall</td>
<td>.58</td>
<td>.002 ***</td>
</tr>
<tr>
<td></td>
<td>WMS - Prose recall immed.</td>
<td>.43</td>
<td>.023 *</td>
</tr>
<tr>
<td>Verbal Fluency - 'S'</td>
<td>Cognitive Estimates</td>
<td>-.50</td>
<td>.008 **</td>
</tr>
<tr>
<td></td>
<td>WMS- Prose, Del.</td>
<td>.51</td>
<td>.006 **</td>
</tr>
<tr>
<td>Stroop - No. read in 120 secs.</td>
<td>Trail Making Test A - errors</td>
<td>-.59</td>
<td>.001 ***</td>
</tr>
<tr>
<td>Stroop - errors</td>
<td>Weigl</td>
<td>.38</td>
<td>.05 *</td>
</tr>
<tr>
<td>Weigl</td>
<td>TMTB - errors</td>
<td>.65</td>
<td>.000 ***</td>
</tr>
<tr>
<td></td>
<td>WISC errors</td>
<td>.50</td>
<td>.007 **</td>
</tr>
<tr>
<td></td>
<td>WMS - prose. Del.</td>
<td>-.40</td>
<td>.036*</td>
</tr>
<tr>
<td>Trails Test A - sec.</td>
<td>TMTB-sec</td>
<td>.58</td>
<td>.001 ***</td>
</tr>
<tr>
<td></td>
<td>Sentence Completion</td>
<td>.40</td>
<td>.036 *</td>
</tr>
<tr>
<td></td>
<td>WMS- Prose Del</td>
<td>-.49</td>
<td>.008 **</td>
</tr>
<tr>
<td>Trails Test B-sec</td>
<td>Sentence Completion</td>
<td>.49</td>
<td>.008 **</td>
</tr>
<tr>
<td></td>
<td>WMS - prose Imm</td>
<td>-.50</td>
<td>.006 **</td>
</tr>
<tr>
<td></td>
<td>WMS-prose Del.</td>
<td>-.53</td>
<td>.003 **</td>
</tr>
<tr>
<td>Trails Test B - err</td>
<td>Sentence Completion</td>
<td>.43</td>
<td>.025 *</td>
</tr>
<tr>
<td>WISC - Time</td>
<td>WMS design. Imm</td>
<td>-.48</td>
<td>.010 **</td>
</tr>
<tr>
<td></td>
<td>WMS design Del</td>
<td>-.38</td>
<td>.043 *</td>
</tr>
<tr>
<td>Rey Figure</td>
<td>WMS Design. Imm</td>
<td>-.38</td>
<td>.047 *</td>
</tr>
<tr>
<td></td>
<td>WMS Design. Del</td>
<td>-.50</td>
<td>.007 **</td>
</tr>
<tr>
<td>Sentence Completion</td>
<td>WMS Prose Delayed Recall</td>
<td>-.39</td>
<td>.038 *</td>
</tr>
<tr>
<td>WMS - Prose Immed. Recall</td>
<td>WMS- Design Immed Recall</td>
<td>.41</td>
<td>.032 *</td>
</tr>
<tr>
<td>WMS Des. Immed Recall</td>
<td>WMS -Design Delay Recall</td>
<td>.81</td>
<td>.000 ***</td>
</tr>
</tbody>
</table>

* denotes significance at the .05 level  
** denotes significance at the .01 level  
*** denotes significance at the .001 level

Table 3.4 - Significant Correlations between the Neuropsychological Tests
The correlations shown in table 3.4 show some interesting findings:

1/ There are certain correlations which may be predicted as they are between different sections of the same tests. For example, the three tests of verbal fluency correlate with each other as do the two Design recall measures (Immediate and delayed) and the Trail Making Test speeds.

2/ It is interesting to note that there are a number of tests which correlate with the Weigl task, a test which showed significant differences between the groups. In general all the tasks which correlated with the Weigl are considered tests of response inhibition and cognitive flexibility and it would be expected that they would correlate with each other.

3/ There were correlations between tests of verbal abilities such as the verbal fluency test and the prose recall section of the WMS-R. On the other side, the Design tasks such as the Rey figure and the WMS-R Design recall also correlated with each other but not with the verbal tasks.

4/ The Sentence Completion task, designed to be a measure of response inhibition did not correlate with other tasks of this functions such as the Stroop and the Weigl. It must be noted, however, that it did correlate with the Trail Making Test B which is thought to be a measure of the same function. The Sentence Completion task surprisingly correlated with the Trails Test part A which is a relatively pure test of motor and cognitive speed not linked to frontal function.
The WMS-R subtests used correlated with each other and with many of the Frontal tests. This would suggest that there is a frontal component to these tasks which require not only memory but also organisation in encoding (Note: Correlation with the Rey Figure) and initiation for retrieval (Note: Correlation with Verbal Fluency tests).

Overall, the results from the correlations confirm that the frontal tests do measure the functions which they claim to be testing and the correlations are generally as would be expected when using the present range of tests.

3.3 Symptom Rating Scales

3.3.1 Means and Standard Deviations

Table 3.5 shows the means and standard deviations of the scores on the Manchester Scale and the SANS for the two groups. The information is represented graphically in Graph 1. In contrast to the neuropsychology test results the two groups do not show similar profiles on the rating scales.
<table>
<thead>
<tr>
<th>Symptom Scale</th>
<th>Means for violent Group</th>
<th>Std. Dev. for violent group</th>
<th>Means for non-violent group</th>
<th>Std. Dev. for non-violent group</th>
<th>t-value (df=27)</th>
<th>2-tail significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Manchester Scale</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>0.50</td>
<td>1.17</td>
<td>1.00</td>
<td>1.04</td>
<td>-1.16</td>
<td>.26</td>
</tr>
<tr>
<td>Anxiety</td>
<td>0.92</td>
<td>1.24</td>
<td>1.93</td>
<td>1.33</td>
<td>-2.00</td>
<td>.057</td>
</tr>
<tr>
<td>Delusions</td>
<td>0.58</td>
<td>1.38</td>
<td>1.71</td>
<td>1.38</td>
<td>-2.08</td>
<td>.048*</td>
</tr>
<tr>
<td>Hallucinations</td>
<td>0.25</td>
<td>0.87</td>
<td>1.29</td>
<td>1.73</td>
<td>-1.88</td>
<td>.072</td>
</tr>
<tr>
<td>Incoherence</td>
<td>0.42</td>
<td>0.67</td>
<td>0.64</td>
<td>0.92</td>
<td>-.70</td>
<td>.490</td>
</tr>
<tr>
<td>Poverty of Speech</td>
<td>0.67</td>
<td>0.89</td>
<td>1.71</td>
<td>1.14</td>
<td>-2.58</td>
<td>.016*</td>
</tr>
<tr>
<td>Flat affect</td>
<td>0.83</td>
<td>0.72</td>
<td>1.71</td>
<td>1.14</td>
<td>-2.31</td>
<td>.030*</td>
</tr>
<tr>
<td>Psychomotor Retardation</td>
<td>0.36</td>
<td>0.51</td>
<td>1.71</td>
<td>1.27</td>
<td>-3.32</td>
<td>.003**</td>
</tr>
<tr>
<td><strong>Side effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tremor</td>
<td>0.33</td>
<td>0.65</td>
<td>0.14</td>
<td>0.36</td>
<td>.94</td>
<td>.357</td>
</tr>
<tr>
<td>Rigidity</td>
<td>0.17</td>
<td>0.39</td>
<td>0.64</td>
<td>0.75</td>
<td>-1.99</td>
<td>.058</td>
</tr>
<tr>
<td>Dystonia</td>
<td>0.00</td>
<td>0.00</td>
<td>0.29</td>
<td>0.73</td>
<td>-1.36</td>
<td>.187</td>
</tr>
<tr>
<td>Akathisia</td>
<td>0.83</td>
<td>0.77</td>
<td>0.36</td>
<td>0.75</td>
<td>1.65</td>
<td>.112</td>
</tr>
<tr>
<td>Vision Probs.</td>
<td>0.36</td>
<td>0.65</td>
<td>0.64</td>
<td>0.75</td>
<td>-.97</td>
<td>.343</td>
</tr>
<tr>
<td>Other</td>
<td>0.75</td>
<td>0.87</td>
<td>0.38</td>
<td>0.65</td>
<td>1.20</td>
<td>.243</td>
</tr>
<tr>
<td><strong>SANS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Affective Flattening - Subscale 1</td>
<td>6.58</td>
<td>4.03</td>
<td>16.50</td>
<td>8.69</td>
<td>-3.63</td>
<td>.001**</td>
</tr>
<tr>
<td>Alogia-Subscale 2</td>
<td>2.67</td>
<td>3.53</td>
<td>12.36</td>
<td>4.47</td>
<td>-6.07</td>
<td>.000***</td>
</tr>
<tr>
<td>Avolition - Subscale 3</td>
<td>3.42</td>
<td>3.15</td>
<td>13.36</td>
<td>4.4</td>
<td>-6.47</td>
<td>.000***</td>
</tr>
<tr>
<td>Ahedonia - Subscale 4</td>
<td>8.00</td>
<td>3.86</td>
<td>17.64</td>
<td>6.87</td>
<td>-4.31</td>
<td>.000***</td>
</tr>
<tr>
<td>Attentional Impairment-Subscale 5</td>
<td>3.67</td>
<td>3.03</td>
<td>8.0</td>
<td>4.17</td>
<td>-2.99</td>
<td>.006**</td>
</tr>
<tr>
<td>Total Score</td>
<td>29.50</td>
<td>13.46</td>
<td>61.29</td>
<td>23.69</td>
<td>-4.11</td>
<td>.000***</td>
</tr>
</tbody>
</table>

* - denotes significance at the .05 level  
** - denotes significance at below .01 level  
*** - denotes significance at below .001 level

**Table 3.5 - Means and standard deviations for Symptom Rating Scales with t-values and significance**
3.3.2 t-tests

The final two columns of table 3.5 summarise the results of a series of t-tests comparing the group means for the symptom scale scores. There is a significant difference between the groups on all the subscale scores on the SANS. These results reveal that the Broadmoor patients show significantly less severe negative symptoms than the non-violent subjects. A more detailed investigation into the individual scores making up the subscale totals showed that virtually all of the ratings found the non-violent sample to have more severe symptomology than the violent patients. The exceptions to this general trend were: Decreases in Spontaneous movement, Inappropriate affect and impersistence at work. For these three characteristics no significant difference was found between the two groups.

The Manchester Scale results showed less diversity between the two groups. On the symptom observations significant differences were found between the groups for Poverty of Speech, Flattened affect and Psychomotor retardation. These results are consistent with the SANS with the non violent group being more severely effected than the violent group.

For the observations of positive symptoms (delusions and hallucinations) there was a significant difference between the level of delusions expressed by the subjects and borderline significance (0.063) for experiences of hallucinations. In both cases the trend showed more severe symptoms in the non-violent sample.
There were no significant differences between the side effects experienced by the two groups. This would suggest that there were no great differences in the levels of medication being given to the two groups.

3.4 Correlations

A number of correlations were undertaken in order to examine the relationship between the symptom scores and the neuropsychological test results. Again, in order to save space, only the significant correlations are listed in table 3.6.
<table>
<thead>
<tr>
<th>Symptom Rating Scale</th>
<th>Correlated with...</th>
<th>Correlation Coefficient</th>
<th>2-tailed significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Manchester Scale</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hallucinations</td>
<td>WISC Mazes-Error</td>
<td>.54</td>
<td>.004 **</td>
</tr>
<tr>
<td>Delusions</td>
<td>WISC Mazes-Error</td>
<td>.52</td>
<td>.007 **</td>
</tr>
<tr>
<td>Incoherence of Speech</td>
<td>Sentence Completion</td>
<td>.44</td>
<td>.024 *</td>
</tr>
<tr>
<td>Poverty of Speech</td>
<td>Trails Test B- Time</td>
<td>.40</td>
<td>.04 *</td>
</tr>
<tr>
<td></td>
<td>WMS Prose-Immed.</td>
<td>.53</td>
<td>.016 *</td>
</tr>
<tr>
<td></td>
<td>WMS Des- Del.</td>
<td>.43</td>
<td>.027 *</td>
</tr>
<tr>
<td>Psychomotor Retardation</td>
<td>WMS-Des. Imm</td>
<td>.44</td>
<td>.027 *</td>
</tr>
<tr>
<td></td>
<td>WMS-Des. Del</td>
<td>.58</td>
<td>.002 **</td>
</tr>
<tr>
<td>Rigidity</td>
<td>Rey Figure</td>
<td>-.41</td>
<td>.038 *</td>
</tr>
<tr>
<td>Dystonia</td>
<td>Stroop-corrected errors</td>
<td>.56</td>
<td>.003 **</td>
</tr>
<tr>
<td></td>
<td>WMS-Des. Delay</td>
<td>.40</td>
<td>.043 *</td>
</tr>
<tr>
<td></td>
<td>TMTA - Time</td>
<td>.44</td>
<td>.025 *</td>
</tr>
<tr>
<td></td>
<td>TMTA- errors</td>
<td>.43</td>
<td>.03 *</td>
</tr>
<tr>
<td>Akathisia</td>
<td>WISC Maze errors</td>
<td>-.41</td>
<td>.04 *</td>
</tr>
<tr>
<td></td>
<td>WMS Des. Immed.</td>
<td>-.42</td>
<td>.034 *</td>
</tr>
<tr>
<td></td>
<td>Stroop-words</td>
<td>-.41</td>
<td>.039 *</td>
</tr>
<tr>
<td>Other Side Effects</td>
<td>WMS- Prose. Immed.</td>
<td>-.43</td>
<td>.03 *</td>
</tr>
<tr>
<td></td>
<td>Stroop-Corrected errors</td>
<td>.41</td>
<td>.04 *</td>
</tr>
<tr>
<td><strong>SANS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alogia - Scl. 2</td>
<td>WISC Mazes-Time</td>
<td>-.49</td>
<td>.03 *</td>
</tr>
<tr>
<td></td>
<td>WMS-Des. Immed.</td>
<td>.40</td>
<td>.039 *</td>
</tr>
<tr>
<td>Avolition - Scl.3</td>
<td>Stroop - No. of Words in 120s</td>
<td>-.5346</td>
<td>.015*</td>
</tr>
<tr>
<td>Attentional Impairment-Scl 5</td>
<td>WMS- Design delayed recall</td>
<td>.44</td>
<td>.49 *</td>
</tr>
<tr>
<td></td>
<td>TMTA - errors</td>
<td>.46</td>
<td>.43 *</td>
</tr>
<tr>
<td><strong>SANS Totals</strong></td>
<td>TMTA - errors</td>
<td>.66</td>
<td>.002 **</td>
</tr>
<tr>
<td></td>
<td>TMTA - Time</td>
<td>.46</td>
<td>.042 *</td>
</tr>
</tbody>
</table>

Table 3.6 - Correlations between Symptom Scales and Neuropsychological Tests.
There are a number of interesting findings that arise from these results:

1/ Both Delusions and Hallucinations were significantly correlated with the number of errors on the WISC Mazes. The correlations are positive suggesting that the positive symptom severity is linked to a higher error rate on the test. This link is important to explore as there was a significant difference between the two groups on the WISC maze error score and between the severity of positive symptoms between the two groups. The number of errors on the mazes was not linked to the severity of negative symptoms.

2/ The scores on the WMS are correlated with negative symptom ratings on both the SANS and the Manchester Scale. Poverty of Speech and Psychomotor retardation on the Manchester scale were both correlated with the memory scores for delayed recall of design. This test was also correlated with subscale 5 on the SANS (attentional impairment) and subscale 2 (alogia). This result is interesting considering that there were significant differences between the two groups on the SANS scales but not on the WMS-R design test.

3/ The Hayling Sentence Completion task is positively correlated with Incoherence of speech suggesting that a greater number of errors were produced by those subjects with more severe incoherence and irrelevance in their speech due to the schizophrenic symptomology. There was no significant difference between the group means for 'incoherence of speech' on the Manchester Scale. This result may have affected the
neuropsychological test finding that there was no difference between the groups on the sentence completion task.

4/ There were a number of significant correlations between the neuropsychological test scores and the side effect ratings on the Manchester Scale. This result is interesting in the context of the T-test results which show no significant differences between the groups on the side effect ratings. In addition, none of the neuropsychological test scores which are correlated with the side effect ratings showed significant group differences on the t-tests. There is a possibility that if side effect ratings had shown significant differences then there may have been differences in the neuropsychological test scores for those tests which correlate with the side effect ratings.

5/ Overall, there were very few correlations between symptoms and neuropsychological test scores. This is surprising as it has often been noted by researchers that different symptom patterns are related to different types of neuropsychological profiles. This result would suggest that the symptom severity and the neuropsychological test results are functioning relatively independently of each other. Thus, the failure to find significant differences between the neuropsychological test scores is not related to the symptom severity differences between the two groups.
Graph 1: Mean Score for Manchester Scale Symptom Rating

- Depression
- Anxiety
- Delusions
- Hallucinations
- Incoherence
- Pov. Speech
- Flat. Affect
- Psycho. Ret.
- Tremor
- Rigid.
- Dystonia
- Akathisia
- Vis. Probs.
- Other

Legend:
- ■ Violent
- × Non-violent
Graph 2: Mean Scores on the SANS

Mean Scores


Violent Non-violent

2 4 6 8 10 12 14 16 18
SECTION 4 - DISCUSSION

The results of the present investigation present a complicated and inconclusive picture of the relationship between neuropsychological test performance and violent offending. The following discussion of the study will look at the neuropsychological test results and the symptom ratings in the context of the behavioural differences between the groups. In addition, methodological considerations will be discussed as well as the implications of the study for future research into violent offending.

4.1 Discussion of Neuropsychological Results

The neuropsychological profiles for the two groups were very similar. The non-violent sample showed significantly more impairment on two of the tests than the violent offenders. The general trend of the results shown in graph 1 reveals that this is a consistent pattern. These results were not consistent with the hypotheses for the study which predicted greater impairments in the violent sample and general differences between the group profiles. The standard deviations for the two groups on many of the tests show that whilst the Broadmoor patients present quite a stable pattern of results, the non violent sample showed considerable variation in the range of scores produced.

It is important to note that, by comparison with the normative data available, both groups performed significantly worse on almost all the tests than would be expected in a healthy population. This finding is consistent with the proposals in the current literature which implicate the frontal lobes in the pathogenesis of schizophrenia. It would be
interesting to have used a matched control group in order to statistically confirm these findings. In addition, the use of a control group with frontal lobe damage may have given an indication of the severity of frontal impairments in the schizophrenic samples.

In terms of the correlations between the neuropsychological tests, the relationships found were those expected from analysis of the tasks involved. It is interesting to note, however, that the Sentence Completion test did not seem to correlate with those tests of response inhibition which it would have been expected to do. Having administered the test myself to all the patients in the two samples it was evident that all the subjects found it very difficult. Only two of the subjects tested effectively managed to invent completely insensible answers to part B. In addition, many of the subjects were not able to complete part A accurately and could not think of a word to finish off the sentences. An example of this inability is the response of one patient to the test sentence "It's hard to admit when one is...". He finished the sentence with the word "basketball" which was completely inappropriate for part A of the test. Essentially it would appear that the test is very difficult to administer to a schizophrenic population due to the nature of their symptomology. A failure to successfully complete part A would also seem to invalidate error scoring on part B (inhibition).

The t-test results show that two tasks, the Weigl and the WISC Mazes error score, showed significant differences between the violent and non-violent samples. Both these findings were robust and it is interesting to look at the particular functions which these tests are thought to measure.
The Classical Weigl was a forerunner to the Wisconsin Card Sort test which has been widely used as a measure of a patient's ability to abstract a concept in order to devise a rule. In addition it is used as a test of mental flexibility, that is, the ability to change strategy following negative feedback from the experimenter. In the present study, the test was found to correlate with Verbal Fluency - Animals, the number of errors on the Stroop, the number of errors on the WISC mazes, errors on the Trail Making Test part B and the WMS-R delayed Prose Recall.

It is possible to show that these test are measuring quite similar functions. In each case the subject is required to switch from an existing pattern of responding to a new one. In the case of the VF-Animals test the subject has to stop thinking of Nouns and try to specifically generate animal/furniture names without cueing from the environment. Those subjects who performed poorly on the Weigl task produced less words in this condition of the Verbal Fluency test. For the Stroop test the subject again has to switch from reading out colour names to reading the colour of the print. Many subjects produced errors on this test due to a failure to effectively inhibit the more instinctive response. Errors on the WISC Mazes test were frequently committed due to the subject continuing to follow the existing path and having to turn back when realising it was a dead end. The Trails Test part B is a test of the subjects' ability to shift response set from letters to numbers alternately. Like the Weigl this test requires cognitive flexibility and the ability to inhibit a previous response set.

It is possible to view all of these tests as measuring perseverative behaviour and the inability to shift response set in response to environmental cues. These findings would
be compatible with Shallice's model of 'Supervisory Attentional System' impairments in frontal patients. The Weigl is a relatively pure test of this ability whilst the other correlated tests also include other components. This may account for the fact that only the Weigl showed a significant difference between the two groups. Thus, it is suggested that the non-violent subjects showed more perseverative behaviours than the violent group. In terms of the 'executive function' it would appear that the non-violent subjects are more severely impaired in this frontal function than the Broadmoor subjects.

The WISC Mazes error score was also significantly different between the two groups. This test correlated only with the Classical Weigl score and their similarities have been considered in the above section.

4.2 Relationships between Neuropsychological Test Results and Violent Behaviour

The relationship of the executive function to violent behaviour is unclear especially when it is considered that the non-violent subjects displayed greater impairments. In the literature concerning delinquent behaviour (Moffitt and Henry; 1989) it is proposed that delinquent subjects display greater executive function deficits than normal adolescents. They propose that these deficits limit the ability to which the subject can hold in the mind "abstract ideas of ethical values and future contingencies while they attend to immediate rewards, and may be unable to inhibit inappropriate behaviour or to adapt their behaviour readily to social feedback" (p. 105). The authors cite a number of studies (Pontius, 1972) that demonstrate significant impairments of delinquents on tests such as the WCST, Porteus Mazes and Verbal Fluency.
Whilst these results are very interesting it is difficult to apply the theory to the present study. It has been widely acknowledged that ALL schizophrenic patients suffer from executive function deficits yet only a very small percentage of them commit violent offences. Thus, it is impossible to implicate executive deficits as the sole cause of antisocial behaviour. In addition, many schizophrenic patients may show 'delinquent' behaviours in the sense that they may be disinhibited and socially inappropriate. However, there is no definition of delinquency to suggest that it necessarily involves violent behaviours.

In the light of these contradictions it is suggested that violent behaviour is not necessarily linked to executive function deficits in the way Moffitt and Henry describe. It is possible that a minimum amount of cognitive flexibility is necessary in order to carry out a violent act and that if executive function is extremely impaired the subject is incapable of producing such behaviour.

4.3 Discussion of Symptom Rating Scale Results in Relation to Neuropsychological Test Scores

It is interesting to look at the neuropsychological test results in the context of the symptom severity scores. There were significant differences between the symptom ratings of the two groups on all the scales of the SANS and on a number of the Manchester scale ratings. The most significant differences were between the levels of negative symptoms and delusional ideation between the groups. There are a number of methodological factors to consider when looking at these results.
1/ The symptom rating scales were administered by different psychiatrists which may account for the differences found. This possibility seems unlikely when it is considered that the scales used are standardised and have been proven to have high inter-rater reliability. Both the raters were Senior Registrars from the Institute of Psychiatry who had received very similar training throughout their careers. In addition, it was evident from subjective observation by the experimenter (who tested both groups on the neuropsychological tests) that the Queen Mary's Hospital sample were more clinically impaired than the Broadmoor patients particularly in terms of their negative symptoms.

2/ It may be possible that the non violent sample were a particularly severe group of patients and that this fact affected the results. Again it would seem that this possibility is unlikely due to the fact that the patients were randomly selected from an outpatient sample who had been living in the community for the duration of their illness. As previously mentioned, the patients had been used for a study in 1993 by Dr. E. Joyce who considered them to be representative of the general population of schizophrenic patients.

The rationale behind using Symptom Rating scales in conjunction with the neuropsychological test scores was to examine whether:

1/ The severity of symptomology was related to impairments on the neuropsychological test battery, and
To ensure that any differences in neuropsychological functioning were related to differences in violence levels rather than a reflection of different symptomology between the two groups.

The findings were rather surprising as there were few differences in neuropsychological functioning but there were significant differences in the symptomology of the two groups. These results are considered even more remarkable when it is considered that the two groups were matched for age and year of diagnosis. Thus, the results would suggest that the two groups show different patterns in the course of their illnesses.

It is interesting to look at the correlations between the symptom scales and the scores from the neuropsychological tests. The Weigl test does not correlate with any of the symptom rating scores suggesting that the differences found between the groups are a result of the differences in the level of violent behaviour and not related to symptomology. The WISC maze error score is, however, correlated with the positive symptom ratings on the Manchester Scale which are significantly more severe in the non-violent subjects. It would appear that the number of errors is related to the severity of the delusions and hallucinations experienced by the patient and not necessarily a difference relating to violent behaviour. The relationship between the positive symptoms and the WISC maze error score is consistent with the literature relating disorganised behaviour to frontal lobe pathology (Liddle 1987b).

The positive symptom ratings did not suggest greater potential for delusions and hallucinations in the violent group. This result is interesting as it contradicts the
proposals of Taylor (1985) and Link et al. (1992), which state that schizophrenic patients only commit violent acts whilst in a psychotic state. This factor may be interesting to consider further by looking specifically at the mental states of the offenders at the time of their crime.

There were very few correlations between the SANS scores and the neuropsychological tests which is, again, a surprising finding in the context of existing literature which relates negative symptomology and frontal lobe damage. Whilst all the subjects are impaired, by comparison with healthy controls, on the frontal tests, those with significantly worse negative symptoms are not necessarily more affected than patients with less severe symptoms. It is important to note, however, that the previous studies have divided symptom clusters into syndromes which have then been compared against each other. The present study did not analyse the ratings in such detail but it was evident that the non violent sample were more severely affected generally and there were no obvious differences in the patterns of symptomology between the two groups.

There were significant correlations between a number of the symptom ratings and the memory scales. Poverty of speech and Psychomotor retardation from the Manchester Scale and the overall ratings for alogia and attentional impairment on the SANS were all correlated with recall on the design section of the WMS-R. Poorer recall was related to greater severity of these symptoms. Poor performance on the memory for designs subtest is be consistent with damage to the hippocampal, frontal or temporal regions of the right hemisphere. This finding is interesting when related to the literature on the brain imaging studies of schizophrenic patients. It has been noted that patients
displaying more chronic, negative symptomology are more likely to have enlarged ventricles with thinning in the hippocampal area. Thus, these results are consistent with the literature in that they link negative symptoms with memory impairments.

It is important to note that there were no significant differences between the side effect rating scores for the two groups. This factor is particularly relevant when it is considered that many of the neuropsychological tests correlate with severity of side effects. Thus, it is probable that the results of the neuropsychological tests were not affected by differences in the severity of medication side effects experienced by the groups. Whilst no record was kept of the levels of medication taken by the patients, it would seem that there are no significant differences in the levels as there is no evidence from the side effect rating scales.

4.4 Violence - Reconsidered

Overall it is a surprising finding that the non violent sample had more severe symptoms and neurological impairments than the Broadmoor subjects. It is generally assumed by the general public (and many mental health professionals) that patients incarcerated in Broadmoor suffer from the most severe mental disorders which have led to their violent behaviour. The results from the present investigation would suggest that this is a misleading generalisation. The violent sample showed no significant neuropsychological impairments by comparison with a non-violent sample and had significantly milder symptom profiles.
The neuropsychological results do not support Hypothesis 1 of the study and contradict the findings of neuropsychological studies of violent schizophrenic patients (Rasmussen et al, in press). It is possible that these contradictions may be accounted for by looking at the nature of the violent behaviour examined in the studies. In the present investigation the criteria for violent behaviour was defined as an offence of Murder/Manslaughter as this was considered the most extreme form of aggression which may be perpetrated. The Rasmussen et al. study does not provide a definition of violent behaviour and it is possible that the experimenters selected a sample of consistently assaultative patients rather than those who had committed a specific offence. In terms of the research into this area it has been proposed that assaultative violence is associated with neurological impairment and treatment resistance which may have led to their findings. In order to examine this possibility it would be necessary to conduct further research comparing assaultative, treatment resistant schizophrenic patients with those who had committed an act of extreme violence but had stabilised their psychosis with medication. Unfortunately there may be difficulties in testing assaultative patients due to the risk involved for the experimenter.

The above discussion highlights the issues which were mentioned in the introduction to this paper concerning the problems in defining violent behaviour. Whilst it is known that all the violent patients in the present sample committed an index offence of murder further investigation into the circumstances of the crime were not accounted for. Thus, some of the patients may have committed a number of offences prior to the murder whilst others committed only the one offence. Also relevant may be the 'randomness' of the crime, that is, whether it was directed against a significant other or a stranger, and
the presence of delusional ideation at the time of the offence. It may be possible that some of the patients murdered in response to command hallucinations whilst others committed the offence when they were relatively free of psychotic symptoms.

All these factors need to be considered and it may be more helpful in future investigations of this type to take a 'case study' approach in order to look in more detail at the nature of the offence committed. In terms of the present study, the standard deviations for the violent group show a wide range of scores within each of the group means. This individual variation may be accounted for by looking more closely at the characteristics of the violent offending for each of the subjects.

Whilst it is possible to criticise the present investigation for the specificity of the criteria for offending behaviour there are certain factors which must be present in order to be convicted of murder by an English court. The first of these is that the violence must have been of a severity to have resulted in the death of the victim. In addition, the perpetrator must have had 'Mens Rea', that is, the intention to kill the victim. It is usually assumed that there must be an element of pre-planning of the crime in order for 'Mens Rea' to exist. All the subjects in the violent sample had committed murder or Manslaughter on the grounds of diminished responsibility. Thus all were perceived by the courts to have had Mens Rea even if the offence was committed whilst they were mentally ill.

In terms of the neuropsychology of murder, it is evident that there are prerequisite mental and behavioural functions which need to be relatively intact in order to commit
the offence. Perpetrators must be able to plan their actions, although this does not include looking at the long term consequences of the act, and have the co-ordination, motivation/volition to complete the killing successfully. With regard to schizophrenia and frontal lobe disorder it would seem that the offender would need to be relatively unimpaired in order to commit the offence. The only frontal feature which may be impaired would be that of inhibition, that is, the offender would possibly need to be disinhibited to undertake the act. Thus, I would propose that there may be a prerequisite amount of intact functioning necessary for a murder to be committed by an individual.

Whilst both the violent and non violent groups are impaired on frontal functioning compared to healthy populations, the violent offenders are slightly less affected and do not suffer the same extremes of negative symptomatology that the non violent sample display. Thus, with regard to symptomatology the non violent group display greater alogia, apathy, anhedonia and affective flattening (paucity of gesture, decreased spontaneous movement) than the violent group which may effectively render them incapable of planning and executing a murder.

A further issue arising from the findings is that of diagnosis. It would appear that the Broadmoor sample showed significantly less schizophrenic symptomatology than the outpatient sample to the point where it may be debatable, in certain cases, as to whether the diagnosis of schizophrenia is appropriate. In order to examine this proposal it would be necessary to compare a larger sample of out-patient or inpatient schizophrenic patients with those incarcerated in a Special Hospital. It is possible that there is a general over diagnosis of schizophrenia amongst offender populations. Whilst this is unlikely, it
must be acknowledged that Forensic Psychiatrists have the important task of influencing the disposal of offenders and may therefore be over-inclusive in their diagnosis due to fears that a mentally ill offender may be left untreated in a prison setting.

In a more general sense, it is impossible to consider violence from only one perspective. The present study has shown that the violent offenders do have neurological impairments by comparison with healthy populations but not when compared with other schizophrenic patients. Thus, it is possible that the frontal lobe impairments found do contribute to the violent behaviour exhibited by the Broadmoor sample, but this is by no means the whole story. The resultant violent behaviour may be a function of a complex interaction of social and familial factors in combination with neurological impairments. It is evident that the control group could not possibly have been matched on the basis of upbringing, level of exposure to violence nor premorbid personality, all of which may be contributing factors to the murders committed by the violent group.

4.6 Methodological Considerations

There are a number of methodological difficulties in the present study which need to be considered when interpreting the findings presented.

1/ The small sample sizes used limit the extent to which the findings may be generalised to the schizophrenic and offender populations. In addition, the small sizes limited the number of statistical tests which could be performed on the data.
However, it is important to note that the study is one of the few reported in the literature which uses a completely non-violent control sample.

2/ The present study did not control for medication dose and it is not known whether there were differences between the groups on this criteria. In general, the vast majority of schizophrenic patients are being treated with neuroleptic medication and in any study of this population it is difficult to completely exclude the effects of medication on the cognitive test scores.

It is, however, unlikely that medication differences influenced the symptom profiles of the two groups as it is generally accepted that medication has relatively little effect on negative symptomology.

3/ The violent patients' length of incarceration was taken from the time of admission to Broadmoor and did not take into account any other periods of institutionalisation either in a prison or another hospital. This factor needs to be taken into account when looking at the effectiveness of the matching process. Obviously, if the violent offenders have been in an institution setting for a long period of time it is difficult to say that the control subjects differed from controls solely on the violence criteria. The two groups have had very different lifestyles and this may have affected the results.

In general, there is always a difficulty in matching control subjects effectively with an experimental group. In this case, difficulties arose due to an inability to control for the effects of institutionalisation. Obviously one must be cautious in forming generalised conclusions from the results found in one paper.
It is also probable that the Broadmoor sample were being treated within the hospital either with medication or psychotherapy. The treatments received were likely to be consistent with very little scope for non compliance with medication. In contrast, the subjects in the community were not being treated with psychotherapy and were expected to come to the DEPO clinic for their medication. It is very possible that a number of the subjects used in the study had missed their medication from time to time.

A further aspect of institutionalisation effects that needs to be considered is that of stress in the environment. It could be argued that an institutionalised setting with a fixed routine may be less stressful than fending for oneself in the community. Brown and Birley (1970) produced a study looking at the effects of crises and stress on schizophrenic symptomology. They found that 60% of the cases they studied experienced a stressful life event in the 3 weeks prior to being admitted for a psychotic breakdown. Other neurological diseases have also been found to deteriorate at times of stress, for example Multiple Sclerosis and Parkinsonism. Thus, it is possible that exposure to life stresses in the community may have affected the severity of the symptom profiles of the outpatient sample.

4/ Many of the patients at Broadmoor Hospital and in the community have abused drugs and alcohol at some point in their lives. It is known that this abuse may lead to cerebral damage and neuropsychological impairments. Unfortunately, it was impossible to exclude these patients from the study as there were very few, from either Broadmoor of
Queen Mary's who had never indulged in substance abuse. Obviously this factor may have influenced the results.

5/ The Broadmoor sample were subjected to a number of rigorous selection criteria in order to counter affects of institutionalisation and dementia. In addition, a number of patients were excluded on the basis of having a NART IQ below 80. Thus, the sample used in the present study may have been unrepresentative of schizophrenic patients at the hospital by virtue of their generally higher intelligence. It would be interesting to investigate further the possibility that violent offenders have a lower IQ than non violent schizophrenic patients. If this were the case, it would support the literature on Delinquency (Moffitt et al. 1989) which proposes that delinquent adolescents have a lower verbal IQ than their non delinquent counterparts.

4.7 Implications of the study

What may be said about the violent group in this study is that they appeared to have less severe symptomology than a non-violent group and that the only differences in neuropsychological functioning were on the Weigl test which is thought to be a measure of cognitive flexibility. These differences may have contributed to the violent offending of the Broadmoor sample but it is important to take into account the whole picture of the individual's upbringing and the details of his offence in order to draw any further conclusions.

It is possible that the study could have implications for the prediction of violence in schizophrenic offenders. Further research is necessary in order to confirm whether there
is a prerequisite level of executive functioning which renders an individual capable of violent behaviour. It would certainly seem that there is a maximum level of negative symptomology necessary to commit a violent act, past which the subject is incapable of having the volition to undertake the offence.

The differences between the two groups on symptomology also have implications for the study of different sub-types of schizophrenia. It would appear that despite being of similar ages and having the same lengths of illness the two groups show very different symptom profiles. Future research may be able to investigate whether there are differences between the diagnoses of Forensic as opposed to general Psychiatrists.

In general, the present investigation highlights the need for more specific definitions of violent offences and further studies looking at the different characteristics of different styles of violent behaviour.
REFERENCES


Miller, E. (1984) Verbal Fluency as a Function of a Measure of Verbal Intelligence and in Relation to Different Types of Cerebral Pathology. *British Journal of Clinical Psychology, 23*, 359-369


Osterrieth, P.A. (1944) Le Test de Copie D'une Figure Complexe. *Archives de Psychologie, 30*, 206-356.


*Australian and New Zealand Journal of Psychiatry, 24*, 113-132


Weschler, D. (1945) A standardised Memory Scale for Clinical use. Journal of Psychology, 19, 87-95


APPENDIX 1

Letter to the Responsible Medical Officer providing Information about the Research.
Dear

Research into the neuropsychological correlates of violence in a schizophrenic population

I am a psychologist in Clinical Training. As part of my MSc. studies I am currently undertaking research into the neuropsychological correlates of violence in schizophrenic patients. I am writing to ask for your permission to test patients under your care in the study.

The study will use 2 groups of patients:
1. 20 (approx.) schizophrenic men whose index offence is murder/manslaughter. These patients will be selected from Broadmoor Hospital.
2. 20 (approx.) schizophrenic men who have never received a conviction for violent behaviour. This group will be selected from an outpatient population at Queen Mary’s Hospital, Roehampton and inpatients at the Horton hospital.

The research will involve using information from the patients’ medical records (age of onset of illness, previous hospitalisations, present medication) and the use of Dr. Mary Hill’s (Neuropsychologist) data from previous testing. In addition, I will be doing a small number of further neuropsychological tests specifically related to frontal lobe functioning. Dr. Tim Exworthy (Senior Registrar) has agreed to complete the Scale for Assessment of Negative Symptoms (SANS) and The Manchester Scale in order to establish the characteristic symptoms for each patient. The research proposal has been passed by the Ethics and Research committees at Broadmoor Hospital and Queen Mary’s Hospital.

I enclose a list of the patients who meet the selection criteria for the study some of whom are under your care. I would be grateful if you could complete the attached consent forms and return it to me as soon as possible. The patients will also be asked for consent prior to testing.

Thank you in advance for your co-operation in this matter. Please do not hesitate to contact me if you have any queries or require further information.

Yours sincerely

Fiona Barber
Psychologist in Clinical Training.
APPENDIX 2

Consent Form for Responsible Medical Officers
SECTION A

I ___________________________ Responsible Medical Officer to

________________________________________ hereby give my approval to the involvement of the

above named patient in the research project conducted by Fiona Barber.

I have received an explanation of the study.

Signed __________________________ Date __________________________

SECTION B

I ___________________________ Responsible Medical Officer to

________________________________________ am satisfied that the patient is capable of
giving consent to his involvement in the proposed research project.

Signed __________________________ Date __________________________
APPENDIX 3

Debrief Letter and Consent Form for Subjects at Broadmoor
INFORMATION SHEET

I am currently carrying out a study as part of my professional training qualification. The research aims to investigate aspects of mental function, such as intelligence and memory, in patients who have mental health problems. The study intends to compare two groups of patients:

1/ Outpatients at Queen Mary’s Hospital, Roehampton.

2/ Inpatients at a special hospital.

If you agree to participate, you will be given a series of tests that require you to answer questions and do simple tasks, such as copying a drawing. The tests are quite easy and not like school tests in that they do not require any specialist knowledge. Testing should last for 20 minutes (max.) Dr. Tim Exworthy (Senior Registrar) will then come and interview you for a further 20 minutes. He will be asking questions about the type of symptoms that you have experienced. The information that you give is entirely confidential and will not be used outside of this study.

Participation is voluntary and you may withdraw consent at any time. If you refuse to consent it will in no way affect the care and attention that you receive from your doctors.

Your name will not be used on any of the answer sheets and all information will be kept confidential.

If you have any questions about the study, please ask me.

If you agree to take part in the study, please sign below.

Consent Form

I understand the study as presented and have had the opportunity to ask questions about it.

I understand that participation in the study is voluntary and that I may withdraw consent at any time and, that if I do, it will not affect the future care that I receive from my doctors.

NAME: __________________

SIGNED: __________________ DATE: __________

WITNESSED: __________________
APPENDIX 4

Debrief Letter for Subjects at Roehampton
INFORMATION SHEET

I am currently carrying out a study as part of my professional training qualification. The research aims to investigate aspects of mental function such as intelligence and memory in patients who have mental health problems. The study intends to compare 2 groups of patients:

1/ Out patients at Queen Mary’s Hospital
2/ Inpatients at a Special Hospital.

If you agree to participate, you will be given a series of tests that require you to answer questions and do simple tasks, such as copying a drawing. The tests are quite easy and are not like school tests in that they do not require any specialist knowledge. Testing should last for 20 minutes (max). The information that you give is entirely confidential and will not be used outside of this study.

Participation is voluntary and you may withdraw consent at any time. If you do refuse to consent it will in no way affect the care and attention you recieve from your doctors.

Your name will not be used on any of the answer sheets and all information will be kept confidential.

If you have any questions about the study please ask me.

If you agree to take part in the study, please sign below.

______________________________
NAME:

______________________________  DATE:
SIGNED:

______________________________
WITNESSED:
APPENDIX 5

Normative Data Available
NORMATIVE DATA

Trail Making Test. (in Seconds)

<table>
<thead>
<tr>
<th>Age</th>
<th>20-39</th>
<th>40-49</th>
<th>50-59</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>B</td>
<td>A</td>
</tr>
<tr>
<td>Mildly Suggestive of Brain Damage (25%)</td>
<td>42 A</td>
<td>94 B</td>
<td>45 A</td>
</tr>
<tr>
<td>Moderately Suggestive of Brain Damage (10%)</td>
<td>50 A</td>
<td>129 B</td>
<td>59 A</td>
</tr>
</tbody>
</table>


Stroop (No. of Words in 120 sec)

<table>
<thead>
<tr>
<th>Raw Score</th>
<th>Percentile</th>
</tr>
</thead>
<tbody>
<tr>
<td>64</td>
<td>84</td>
</tr>
<tr>
<td>65</td>
<td>85</td>
</tr>
<tr>
<td>66</td>
<td>86</td>
</tr>
<tr>
<td>67</td>
<td>87</td>
</tr>
<tr>
<td>68</td>
<td>88</td>
</tr>
<tr>
<td>69</td>
<td>89</td>
</tr>
<tr>
<td>70</td>
<td>90</td>
</tr>
<tr>
<td>71</td>
<td>91</td>
</tr>
<tr>
<td>72</td>
<td>92</td>
</tr>
<tr>
<td>73</td>
<td>93</td>
</tr>
<tr>
<td>74</td>
<td>94</td>
</tr>
<tr>
<td>75</td>
<td>95</td>
</tr>
<tr>
<td>76</td>
<td>96</td>
</tr>
<tr>
<td>77</td>
<td>97</td>
</tr>
<tr>
<td>78</td>
<td>98</td>
</tr>
<tr>
<td>79</td>
<td>99</td>
</tr>
<tr>
<td>80</td>
<td>100</td>
</tr>
<tr>
<td>81</td>
<td>101</td>
</tr>
<tr>
<td>82</td>
<td>102</td>
</tr>
<tr>
<td>83</td>
<td>103</td>
</tr>
</tbody>
</table>
### WMS-R

<table>
<thead>
<tr>
<th>Test</th>
<th>Age</th>
<th>Percentile and Raw Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prose Recall - Immediate</td>
<td>20-24</td>
<td>50% score 26 or below</td>
</tr>
<tr>
<td></td>
<td>25-34</td>
<td>51% score 26 or below</td>
</tr>
<tr>
<td></td>
<td>35-44</td>
<td>49% score 29 or below</td>
</tr>
<tr>
<td></td>
<td>45-54</td>
<td>47% score 23 or below</td>
</tr>
<tr>
<td>Prose Recall - Delayed</td>
<td>20-24</td>
<td>49% score 22 or below</td>
</tr>
<tr>
<td></td>
<td>25-34</td>
<td>50% score 22 or below</td>
</tr>
<tr>
<td></td>
<td>35-44</td>
<td>51% score 22 or below</td>
</tr>
<tr>
<td></td>
<td>45-54</td>
<td>53% score 20 or below</td>
</tr>
<tr>
<td>Design Recall - Immediate</td>
<td>20-24</td>
<td>54% score 34 or below</td>
</tr>
<tr>
<td></td>
<td>25-34</td>
<td>45% score 33 or below</td>
</tr>
<tr>
<td></td>
<td>35-44</td>
<td>48% score 33 or below</td>
</tr>
<tr>
<td></td>
<td>45-54</td>
<td>50% score 31 or below</td>
</tr>
<tr>
<td>Design Recall - Delayed</td>
<td>20-24</td>
<td>50% score 31 or below</td>
</tr>
<tr>
<td></td>
<td>25-34</td>
<td>44% score 30 or below</td>
</tr>
<tr>
<td></td>
<td>35-44</td>
<td>48% score 30 or below</td>
</tr>
<tr>
<td></td>
<td>45-54</td>
<td>53% score 29 or below</td>
</tr>
</tbody>
</table>

Taken from WMS-R Manual (Russell, T.W. 1975a)

### Complex Figure Test

In Osterrieth' 1944 sample 83% of the adult control subjects followed procedure types I and II. 15% Used type IV. In the traumatically brain injured group 63% also followed procedures I and II. One aphasic and one presenile dementia patient followed a type V procedure.

Taken from Lezak, M.D. *Neuropsychological Assessment* 2nd Edition 1983)

### Cognitive Estimates Task

<table>
<thead>
<tr>
<th>Research Scores</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
</tr>
<tr>
<td>Standard Deviation</td>
</tr>
</tbody>
</table>

### Hayling Sentence Completion Research results (Burgess, 1993)

<table>
<thead>
<tr>
<th>Part B errors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
</tr>
<tr>
<td>Standard Deviation</td>
</tr>
</tbody>
</table>
Normative Data for Broadmoor Admissions Sample:

NART IQ Mean = 93.5

WISC Mazes

<table>
<thead>
<tr>
<th>Time</th>
<th>211.6 Secs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Errors</td>
<td>2.7</td>
</tr>
<tr>
<td>Number Correct</td>
<td>4.4</td>
</tr>
</tbody>
</table>

Wiegl

1 54%
2 14%
3 3%
4 3%
5 12%
6 12%

Verbal Fluency - Averages

Nouns 28
Animals 18
'S' 18
APPENDIX 6

Sample Data Record Sheets
### DEMOGRAPHICS

<table>
<thead>
<tr>
<th>Name</th>
<th>Date of Birth</th>
<th>Year of lst Diag</th>
<th>Location</th>
<th>Date of 1st Test</th>
<th>Age</th>
<th>Dominant Hand</th>
<th>Date of Re-Test</th>
<th>Date of Admission</th>
<th>Research No</th>
</tr>
</thead>
</table>

### FRONTAL TESTS

- **Verbal Fluency (60 Secs) - Nouns**
  - Category: 
  - S-Score: 
  - Errors: 
  - Corrected: 

- **Stroop (120 Secs) - Completed**

- **NART - Predicted IQ**

- **Weigl Score**

- **Trail Making Test (Part A) - Time**
  - Errors: 

- **Trail Making Test (Part B) - Time**
  - Errors: 

- **WISC Mazes - Number Correct**
  - Number Errors: 
  - Time Taken: 

- **Rey Figure: Six Element Task: Sentence Completion A: B:**

### MEMORY TESTS:

- **WMS-R**
  - Prose Recall - Immediate: 
  - Delayed: 
  - Design Recall -Immediate: 
  - Delayed: 

### MANCHESTER SCALE

#### SYMPTOM

<table>
<thead>
<tr>
<th>RATING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ratings made by replies to Questions... (0 = Absent... 4 = Severe)</td>
</tr>
<tr>
<td>Depressed</td>
</tr>
<tr>
<td>Anxious</td>
</tr>
<tr>
<td>Coherently Expressed Delusions</td>
</tr>
<tr>
<td>Hallucinations</td>
</tr>
</tbody>
</table>

#### SYMPTOM

<table>
<thead>
<tr>
<th>RATING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ratings by Observation... (0=absent...4 = Severe)</td>
</tr>
<tr>
<td>Incoherence/Relevance</td>
</tr>
<tr>
<td>Poverty of Speech</td>
</tr>
<tr>
<td>Flattened/Incog Affect</td>
</tr>
<tr>
<td>Psychomotor Retardation</td>
</tr>
</tbody>
</table>

#### SYMPTOM

<table>
<thead>
<tr>
<th>RATING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Side Effects... (0=Absent, 1=Mild, 2=Marked)</td>
</tr>
<tr>
<td>Tremor</td>
</tr>
<tr>
<td>Rigidity</td>
</tr>
<tr>
<td>Dystonia</td>
</tr>
<tr>
<td>Akathisia</td>
</tr>
<tr>
<td>Vision Problems</td>
</tr>
<tr>
<td>Other (specify)</td>
</tr>
</tbody>
</table>
**Broadmoor Violence Study**

**Name:**

**Research No:**

**SANS - SCALE**

**AFFECTION FLATTENING / BLUNTING**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Code 0 = Absent</th>
<th>Code 5 = Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unchanging Facial Expression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decreased Spontaneous Movement</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paucity of Gesture</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poor Eye Contact</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Affective non-responsivity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inappropriate Affect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lack of Vocal Inflection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subjective Rating</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global Rating</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subscale Score</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**ALOGIA**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Code 0 = Absent</th>
<th>Code 5 = Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poverty of Speech</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poverty of Content of Speech</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blocking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased Latency of Response</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subjective Rating</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global Rating of Alogia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subscale Score</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**AVOLUTION - APATHY**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Code 0 = Absent</th>
<th>Code 5 = Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grooming and Hygiene</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impersistance at Work/School</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical Anergia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subjective Rating</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global Rating of A/A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subscale Score</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**AHEDONIA - ASOCIALITY**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Code 0 = Absent</th>
<th>Code 5 = Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recreational Interests/Hobbies</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sexual Interest/Activity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ability to feel Intimacy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relationships - Friends/Peers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Awareness of Anhed /Asociality</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global rating of Anhed/Asociality</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subscale Score</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**ATTENTIONAL IMPAIRMENT**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Code 0 = Absent</th>
<th>Code 5 = Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Work Inattentiveness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inattentiveness in Testing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complaints of Inattentiveness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global Rating of inattentiveness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subscale score</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**SUMMARY SCORE (GLOBAL RATINGS)**

**COMPOSITE SCORE**
RESEARCH DOSSIER

AN INVESTIGATION OF THE PERFORMANCE OF PATIENTS WITH SCHIZOPHRENIA ON A REPEATED PRESENTATION STROOP PARADIGM

FIONA BARBER

SUBMITTED IN PART FULFILMENT OF THE REQUIREMENTS FOR THE PSYCHD. CONVERSION COURSE

UNIVERSITY OF SURREY

1998
ABSTRACT

Neuropsychological and information processing investigations of patients with schizophrenia have consistently reported deficits in selective attention (Frith, 1979; Gray, 1991). As such, patients fail to attend to relevant aspects of the environment and are unable to inhibit the intrusion of irrelevant information into conscious awareness. One of the most widely used tests of selective attention is the Stroop task (Stroop, 1935) in which subjects are required to inhibit the more familiar skill of word reading in favour of the less automatic one of colour naming. Studies of attention in would suggest that patients with schizophrenia would have difficulty with two aspects of the paradigm: First, in inhibiting automatic word reading and second, that in a repeated presentation design they would fail to benefit from previous exposure to stimuli and as such fail to show practice effects which are observed in normals (Jensen, 1965; Harbeson et al, 1982). A computerised Stroop paradigm was administered with 12 trials, six where the words and colours were congruent and six incongruent, alternating in an ABAB design. The results showed no differences in the Stroop effect between the two groups. However, there was an interaction between trial and group such that while normal subjects became faster in both conditions across trials, the patients with schizophrenia showed no differences in their reaction times across the 6 presentations in both conditions. Analysis of individual subject data showed considerable variations in performance between subjects with some patients being able to improve with practice on the incongruent condition to the same extent as normals while for others, there was a decrease in speed across trials. There was a trend for these individual differences to be related to symptoms and medication type.
AN INVESTIGATION OF THE PERFORMANCE OF PATIENTS WITH SCHIZOPHRENIA ON A REPEATED PRESENTATION STROOP PARADIGM

CONTENTS

SECTION 1 - INTRODUCTION
1.1 The Fronto-Striatal Hypothesis of Schizophrenia 2
1.2 Attention in Schizophrenia 8
1.3 Attention and Cognitive Theories of Schizophrenia 14
1.4 The Stroop Task 22
1.5 Practice Effects and the Stroop 28
1.6 Stroop Studies in Schizophrenia 29
1.7 The Functional Anatomy of Visual Attention 34
1.8 The Anterior Cingulate and the Stroop Paradigm 40
1.9 Schizophrenia and the Anterior Cingulate 45
1.10 The Functional Anatomy of Practice 48
1.11 The Present Study 50

SECTION 2 - METHODOLOGY
2.1 Design 54
2.2 Subjects 54
2.2.1 Experimental Group: Patients with schizophrenia 55
2.2.2 Healthy Control Subjects 56
2.3 Procedure 57

SECTION 3 - RESULTS
3.1 Matching 59
3.2 Error Data 59
3.3 Reaction Time Data 59
3.3.1 Repeated Measures ANOVA 61
3.4 Interaction Effects 62
3.5 Symptom Rating Data 62
3.6 Analysis of Individual Subject Data 64
3.7 Congruent versus Incongruent Learning 68

SECTION 4 - DISCUSSION
4.1 Discussion of Results 69
4.1.1 Data Relating to the First Hypothesis 70
4.1.2 Data Relating to the Second Hypothesis 74
4.2 Methodological Considerations 76
4.2.1 Experimental Design 76
4.2.2 Subject Numbers 77
4.2.3 Test Administration 78
4.3 Medication and Hospitalisation 78
4.4 Implications for Further Research 79
4.5 Conclusions 81

SECTION 5 - REFERENCES 82
List of Graphs and Tables

Table 3.1. Mean age and NART IQ score for the two groups 59
Table 3.2. Mean number of correct responses made by the two groups 60
Graph 1. Reaction Time for Congruent and Incongruent Conditions across 6 Trials 46
Table 3.3. Mean reaction times on congruent and incongruent trials for the two groups 61
Table 3.4. Mean reaction times for first 3 trials vs second 3 trials for the two groups 63
Table 3.5 Individual subjects' difference scores and medication for patients with schizophrenia 65
Table 3.6 Mean difference scores for learners and non-learners for patients with schziophrenia and normals 66
Table 3.7 Spearman's rank correlations for difference scores and other variables for patients with schizophrenia 67
SECTION 1 - INTRODUCTION
Research into the neuropsychology of schizophrenia has identified a number of cognitive deficits associated with the disease. One such impairment that has been consistently identified is in the area of attention and research has shown that patients with schizophrenia have difficulty in sustaining attention and in selectively attending to relevant material (Frith, 1979; Gray, 1991).

The dysfunction of selective attention in patients with schizophrenia has been observed on a number of neuropsychological tests including the classical Stroop paradigm. This task involves the naming of the ink colour in which colour words are written. In the incongruent condition, the word ‘RED’ may be written in blue ink and subjects consistently take longer to name the ink colour in this condition than if the word and ink colour are the same (Stroop, 1935). Patients with schizophrenia have been found to perform poorly on this test, displaying significantly longer reaction times in the incongruent condition than normals (Joyce et al, 1996; Buchanan et al 1994b). In cognitive terms, it has been hypothesised that their poor performance is associated with a difficulty in selectively attending to the relevant aspects of the stimuli and actively inhibiting the automatic process of word reading.

There have been many recent studies of Stroop performance in normal populations using functional imaging (Pardo et al, 1990; Bench et al, 1993). A significant difficulty associated with functional imaging research is that in order to obtain enough meaningful data subjects are often required to complete multiple repetitions of the same test. In those studies using the Stroop paradigm, it has, therefore, been necessary to assume that performance on the test is not affected by practice and that the subject is approaching the task in exactly the same way each time it is presented. Intuitively, it would seem unlikely that this is the case. Given the recent growth of research in this area it is critical to clarify the factors which influence performance under these repeated presentation conditions.
There have been few studies on the effects of practice on Stroop performance in normals and none concerning practice effects in the case of patients with schizophrenia. Those studies which have looked at practice effects suggest it is possible for reaction times to improve in normal subjects following repeated presentation of Stroop stimuli. In addition, there have been some studies which have suggested that the interference effects may be reduced as subjects improve more in the incongruent than the congruent condition (Jensen, 1965). In the case of patients with schizophrenia, one recent theoretical model attempting to formulate a neuropsychology of schizophrenia proposed that "schizophrenia arises from a weakening of the effect of previous experience on the selection of environmental stimuli to which to attend and respond" (Gray, 1991 p. 19). Studies investigating mechanisms of learning such as latent inhibition and negative priming have indicated that some patients with schizophrenia, particularly those with positive symptoms, fail to demonstrate normal learning patterns.

The present study aims to investigate the performance of patients with schizophrenia on an identical version of the Stroop paradigm used for an activation study in healthy controls. The effects of practice on performance of the Stroop paradigm will be investigated in both normals and patients with schizophrenia. Furthermore, some inferences will be made about the brain areas associated with Stroop performance based on the activation data obtained for the normals. Literature relating to the hypothesised neural substrates of schizophrenia and studies of attention in schizophrenia provide a context for the present study. In addition, a discussion of practice effects on Stroop performance and learning is provided.

1.1 The Fronto-Striatal Hypothesis of Schizophrenia

Attempts to localise the site of dysfunction in schizophrenia have dominated research since the first characterisations of the disease by Kraepelin (1919) and Bleuler (1913). Researchers from a number of different scientific schools have examined brain structure, neurotransmitter functioning and cognitive function in patients with the
disease in order to address this fundamental issue. The primary source of difficulty in finding a 'site' of dysfunction is that schizophrenia consists of a number of diverse symptoms and it is unlikely that one area of damage will account for the variety of behaviours seen. However, in recent years attempts to identify brain regions associated with schizophrenia have been advanced by two major research findings: the characterisation of anatomically segregated, functionally distinct cortico-striatal loops; and factor analytic studies of the heterogeneity of schizophrenic symptomatology.

It is interesting that even in the earliest descriptions of the disease, comparisons between patients with schizophrenia and patients with frontal lobe damage highlighted similarities between the behaviours associated with these conditions. This notion of dysfunction in the frontal lobe in patients with schizophrenia has been a major focus of research interest for decades and despite criticism has remained a prominent theory. Robbins (1990) in his review of the literature in this area comments not only on the large number of studies in which the frontal lobes are implicated but also the fact that evidence comes from diverse sources including neurochemical, neuroanatomical and neuropsychological research.

One of the primary neuropsychological deficits reported in patients with schizophrenia is that of impairment in 'executive functioning'. This term has been widely used in cognitive neuropsychology to describe the co-ordination of the cognitive processes involved in the execution of complex tasks. As such, intact executive functioning is necessary for the planning of complex tasks, generation of strategies for action and the monitoring of behaviour in response to environmental feedback. In addition, the executive plays a role in the maintenance of goal directed attention such that irrelevant information is ignored. Patients with frontal lobe pathology have been found to show specific deficits on tasks of executive functioning and their neuropsychological profile is characterised by impairments in planning, generation of behaviour, maintenance of goal directed behaviour and behavioural inflexibility.
Patients with schizophrenia have been found to perform poorly on many tasks of executive functioning. Pantelis et al (in submission) used a computerised version of the Tower of London task to assess planning ability in patients with chronic schizophrenia and those with frontal lobe damage. The results showed that although the patients with schizophrenia and those with frontal damage were able to complete as many problems as the normal controls, they needed more moves to reach a correct solution. This result would suggest a deficit in both these groups’ ability to plan effectively and to effectively utilise feedback. In addition, both groups had significantly longer thinking times while doing the task as opposed to initial planning time prior to commencing. This result would also indicate a failure to create a plan of action. In support of these findings, Goldberg et al (1990), used the original, Tower of Hanoi task and found impaired planning on both the three and four disc problems.

Another commonly used measure of executive functioning is verbal fluency which requires the generation of lists of words from a particular category (e.g. animals, furniture) or beginning with a specific letter (F, A, S). Joyce et al (1996) found reduced verbal fluency in patients with schizophrenia for both category and letter fluency. The study also showed that patients with schizophrenia performed worse than would have been predicted form their premorbid IQ scores. Impairments in verbal fluency have also been found in numerous other less recent studies (Kolb and Wishaw, 1983; Gruzelier et al 1988; Crawford et al, 1993).

A further task which has been used widely to assess executive function in schizophrenia is the Wisconsin card sort test (WCST). It has frequently been reported that patients with schizophrenia achieve fewer sorting categories and make more perseverative errors than normal controls (Weinberger et al; 1986, 1988; Kolb and Wishaw, 1983; Taylor and Abrams,1984). In addition, functional imaging studies have shown hypometabolism in the dorsolateral prefrontal cortex while completing the task. A more sophisticated, computerised version of the task has also supported the finding that patients with schizophrenia have difficulty in shifting attentional set.
between different stimulus dimensions and tend to perseverate with previously correct solutions despite receiving corrective feedback (Elliott and Sahakian, 1995).

In a case study approach to neuropsychological assessment, Shallice et al (1991) assessed five patients with schizophrenia using a range of tests which included a battery for 'frontal functions' incorporating the Stroop, verbal fluency, WCST and the Trail Making Test. The most prominent finding was that of heterogeneity in the 5 patients tested for both the severity and pattern of impairment. All five patients were found to have impaired performance on tests sensitive to frontal lobe functioning while three of the five also had more widespread cognitive impairment including one patient with a visual agnosia.

In addition to findings implicating the prefrontal cortex in schizophrenia, there have also been a large number of findings that suggest dysfunction in the subcortical structures. Studies have found anatomical differences in the thalamus and basal ganglia, for example, Vogt and Vogt (1952) found dwarf cell changes in the mediodorsal nucleus of the thalamus. Neurochemical pathology has shown changes in the temporal lobe, frontal cortex and striatum which are specific to schizophrenia (for review see Kleinman et al. 1988). Neuropsychological studies have found parallels between the performance of patients with schizophrenia and those with the so-called subcortical dementias (Parkinson's disease, supranuclear palsy, Huntingdon's Chorea) on a number of tests and there are behavioural similarities between these disorders and schizophrenia (Pantelis et al, 1992), for example, cognitive and motor slowing.

The neuropsychological studies have found that patients with subcortical dementias also perform poorly on tests of executive functioning, however, there are significant differences in the profiles of these patients and those with schizophrenia. An example is that of performance on the computerised Tower of London task reported earlier (Pantelis et al, in submission). While patients with schizophrenia have slowed thinking times during completion of the task, patients with Parkinson's disease have slowed initial thinking times, that is, prior to commencing the task. One explanation
for this result would be that the Parkinsonian patients take time to plan their strategy and have slowed thinking while the schizophrenia sample do not plan and therefore have to generate strategies as they go along. Obviously, in experiments which measure 'thinking times' it is difficult to hypothesise the processes occurring inside the patients' minds without collecting more qualitative data regarding their performance. It is possible that other factors such as initiation difficulties and attention may also be relevant.

A further example of differences in executive abilities comes from the computerised attentional set shifting task based on the WCST. In this paradigm, subjects are required to complete nine test stages, at stage eight, an extra-dimensional set shift is necessary in order to pass on to the next level. An extra-dimensional shift occurs when subjects are able to switch attention from one stimulus dimension to another, for example, in the WCST it is a shift from sorting by shape to colour. Patients with frontal lobe lesions are impaired at performing this final shift (Owen et al 1991) as are patients with schizophrenia and those with Parkinson's disease (Downes et al 1989; Elliott et al, 1995). A more sophisticated version of the computerised set shifting test allows for distinguishing between failure at the extra dimensional shift stage due to perseveration (found in patients with frontal lesions; Owen et al 1993) and learned irrelevance, that is, having learned that a dimension is irrelevant being unable to attribute significance to it. Patients with Parkinson's disease fail due to 'learned irrelevance', (see Owen et al 1993 for full description of task) while those with schizophrenia fail as a result of perseveration (Elliott et al, 1995).

Thus, while there is evidence to implicate the subcortical structures in the pathogenesis of schizophrenia, it is unlikely that this is the primary site of dysfunction, as it is in the subcortical dementias. Robbins (1990) hypothesises that the dysfunction in schizophrenia is a result of a combination of cortical and subcortical changes. The characterisation of cortical striatal loops by Alexander et al (1986) has provided some insight into the possible mechanisms by which this combination of changes may occur. There are five loops which project from different regions of the
cortex with related functions, for example, control of movement, which converge to regions of the striatum, feedback to the substantia nigra/globus pallidus, then the thalamus and back to one of the regions of the cortex where they originated. The five cortical regions are the supplementary motor cortex, the dorsolateral prefrontal cortex, the lateral orbitofrontal cortex and the anterior cingulate cortex.

In the context of schizophrenia, these loops may provide vital clues as regards the heterogeneity of the disease and a way of explaining how schizophrenia can share features of both frontal lobe and subcortical pathology. Functional imaging and neuroanatomical research has implicated the dorsolateral prefrontal cortex, the orbitofrontal cortex and the anterior cingulate cortex in the pathology of the disease and thus it would seem that the loops may be of significant interest to schizophrenia research (Robbins, 1990). The most interesting aspect of this hypothesis is that it allows for the diversity of symptoms that are associated with the disease. Liddle (1987a&b) in his cluster analysis of schizophrenic symptomatology isolated three major clusters labelled reality distortion, disorganisation and psychomotor poverty syndromes. These were associated with damage to different areas of the cortex, with the latter syndrome being linked to dorsolateral prefrontal cortex pathology, disorganisation being associated with the orbitofrontal cortex and reality distortion being associated with temporal lobe damage. While these areas are currently hypothetical, the theory does fit in with Robbins’ proposal that different combinations of cortical-subcortical loops may be damaged in different subtypes of the disease.

In summary, there is evidence to suggest that damage to the frontal cortex is important in understanding the nature of schizophrenic symptomatology. However, the symptoms cannot be accounted for by frontal or subcortical damage alone and it is proposed that damage to cortical-subcortical connections may account for the behaviours associated with the disease. The heterogeneity of schizophrenic symptomatology may be attributed to ‘an altered balance in the flow of information between different cortico-striatal loops each of which is controlled by input from the frontal lobes’ (p399). Of particular interest to schizophrenia are those loops which
receive input from the dorso-lateral prefrontal cortex (DLPFC), the lateral orbitofrontal cortex and the anterior cingulate cortex.

1.2 Attention in Schizophrenia

Deficits in attention have been noted behaviourally in patients with schizophrenia since the earliest descriptions of the disease (Bleuler, 1913; Kraepelin, 1919). Patients have been described as ‘highly distractible’ and ‘having great difficulty in maintaining concentration’ and the symptoms themselves have also been linked to dysfunction of attentional mechanisms. For example, Gray et al (1991) has proposed that positive features of the disease arise due to a failure in regulation of attention. In addition, Frith (1979) has proposed that symptoms such as auditory hallucinations arise as a failure in regulation of automatic responses such that they enter consciousness and are misperceived as being external to the individual. In both these cases, positive symptoms are attributed to a failure of attentional mechanisms to select which items are to be entered into conscious awareness. In each of these theories it is important to note the assumptions that: 1/ the human brain has a limited capacity to process information (Mesulam, 1981) and 2/ that there is active inhibition of stimuli to prevent them from gaining access to central processing mechanisms (Broadbent, 1971). As such, research on attention in schizophrenia has focused on the mechanisms and consequences of the failure of ‘selection’ systems.

The study of attention in schizophrenia can be divided into three broad areas: Neuropsychological studies which use standardised cognitive tests in order to look at associations in performance of patients with schizophrenia and those with distinct areas of brain damage; the information processing approach which uses experimental tests in order to establish the specific routes by which information is attended and processed; and combining that of functional imaging which examines brain behaviour relationships. This latter area is discussed in a later section (1.8).
The concept of attention in the neuropsychology of schizophrenia has remained largely unspecified despite frequent reports of 'attentional' deficits in this population. The majority of studies have based these statements on poor performance on tests which have an attentional component but which are not specifically designed to measure this cognitive process alone. As such, the deficits found have been subsumed within the global category of 'executive dysfunction' and there has been little attempt to provide brain-behaviour models which link the impairments to the pathophysiology of schizophrenia.

Support for the notion of an attention deficit in schizophrenia has come from neuropsychological studies which have found that patients perform more poorly than normals on tasks which demand complex information processing e.g. the WCST, maintenance of attention or rapid psychomotor speed (Maruff and Curie, 1996). As reported earlier, patients with schizophrenia display consistent deficits on tests of executive function such as the WCST and rCBF studies have shown that this is related to hypometabolism in the DLPFC (Weinberger, 1986). The WCST has been widely used in schizophrenia research and patients consistently produce fewer sorting categories and make more perseverative errors than matched controls. Poor performance has been attributed to a failure to inhibit previously successful modes of responding in the face of conflicting external feedback (Nelson, 1976). However, recent studies have questioned this explanation and failure on the task may be a result of a more general deficit in comprehending task demands and generalising learning (Pantelis et al, in preparation; Goldberg and Weinberger, 1995). Thus, it is difficult to isolate the role of attention in task performance other than that it is a long, complex procedure which requires sustained concentration. The extent to which these aspects of attention are responsible for overall task failure is unclear.

Slowness in performance on timed tests has frequently been cited as indicative of attentional impairment in patients with schizophrenia. Deficits on tests such as the digit symbol subtest (taken from the WAIS-R) have been reported frequently (Eysenck, 1968; Shallice et al, 1991; Heaton et al, 1994) along with slowing on the
Trail Making Test parts A and B (Watson et al, 1968; Heaton et al 1994), the AMIPB cancellation task and reaction time (RT) tests (Goldberg et al, 1990). More detailed investigation of these deficits has revealed that they are present even in the earliest stages of the disease (Saykin et al, 1994) even prior to disease onset (Cornblatt et al, 1989) and are highly correlated with severity of negative symptomatology (Nelson et al, 1990). Brain areas implicated in these studies include the frontal lobes, as these are associated with negative symptoms (Nelson et al, 1990) and the basal ganglia which are commonly associated with bradyphrenia (Pantelis et al, 1992).

Beyond these comparisons with brain damaged populations, neuropsychological studies provide little information regarding the nature of the attentional deficit in schizophrenia. Tests such as the trail making test and digit symbol task have different cognitive requirements beyond attention alone and any inferences made about brain areas should be treated with caution as subjects may be performing poorly on the tasks for a variety of different reasons. Thus, the concept of attention is poorly specified and it is not clear which aspects of attention are hypothesised as being impaired. For example, it is possible that subjects are unable to concentrate on the task for the requisite period of time, implying a deficit in sustained attention, or that they have difficulty inhibiting irrelevant material resulting in a selective attention deficit. At face value the studies can only propose the argument that patients with schizophrenia have an attentional deficit because they perform poorly on tasks which have an attentional component.

The only neuropsychological study to have investigated attentional processes in schizophrenia using a strong theoretical model is Shallice (1991). His model of executive functioning has been explained in detail elsewhere (Maruff and Curie, 1996; Barber, 1994; Shallice, 1989). The important aspects of the theory are that a limited number of cognitive and motor programs for action exist and that they are activated by situational cues. The most highly activated program for routine situations is selected by a ‘contention scheduling’ system. In novel situations selection of action is qualitatively different and is modulated by a supervisory attentional system which
may override the contention scheduling system by activating or inhibiting particular programs. In his case study approach, Shallice found that all patients showed deficits in executive functioning. According to his model he proposed that the deficits in schizophrenia may occur as a failure of the Supervisory Attentional System to inhibit responses to irrelevant stimuli. As such, the slow and inaccurate performance on the trail making test would be due to a failure to attend to the relevant stimuli and to inhibit competing responses during the B part of the task. Thus, the important aspect of the study is the idea of 'inhibition' and that failure to selectively attend to stimuli occurs as a consequence of not only an impaired mechanism for selection but also a deficit in inhibition of competing responses.

Thus, neuropsychological studies of patients with schizophrenia have revealed that patients display deficits on tasks which require complex information processing and timed tasks which require both cognitive and motor speed. While Shallice attributes these deficits to a failure of executive control which prevents inhibition of inappropriate responding, the other neuropsychological studies are concerned with inferring potential sites of cerebral dysfunction from comparisons with brain injured populations and do not provide a clear picture of the specific attentional processes that are dysfunctional in schizophrenia. Studies have failed to use tasks which are highly specified in terms of their attentional demands and the 'attentional deficits' found in patients with schizophrenia are poorly defined in terms of the underlying deficit.

Information processing approaches to the study of attention in schizophrenia address some of the difficulties inherent in neuropsychological research. Studies in this area are based on experimental paradigms that have been used widely with normal populations and are designed to assess specific aspects of attention. However, these approaches are not based on brain-behaviour models and as such provide little insight into the neural networks which are responsible for the deficits found (Maruff and Curie, 1996).
Attentional information processing deficits in schizophrenia have been widely reported since the earliest findings using simple reaction time (RT) experiments (Shakow, 1962; Kornetsky and Mirsky, 1966). However, there has been continuing debate in the literature as to the exact nature of these processing difficulties and the confusion has been exacerbated by poor specification of the mechanisms for normal information processing (Laplante et al, 1992). One aspect of processing that has been universally agreed is that in order for a limited capacity information processor to selectively attend to relevant stimuli, distracting stimuli must undergo a process of active inhibition. The issue which has been a continuous source of debate is where in the chain of processing this selection and inhibition occurs. Opinion is divided as to whether the selection occurs at the early (Broadbent, 1971; Kahneman and Treisman, 1984) or later stages of processing (Allport, 1980; Posner and Snyder, 1975) and as such, it has been difficult to establish the nature of the deficits in processing by patients with schizophrenia.

Much of the research on attention in schizophrenia has been influenced by the ‘defective filter’ theory of Broadbent (1971). This theory attributes attention deficits to a failure in the ability to exclude irrelevant information from entering consciousness (early selective deficit). Due to the limited capacity of the central processor, relevant information is given too little space making processing error prone and slower than in normals. While this theory still governs attention research, it is subject to the same debate that is faced by normal information processing research, that is, at which specific stage in processing the defective filtering occurs. Thus, while it is possible that irrelevant stimuli enter consciousness at the first stages of processing it is also hypothesised that the failure of selection could occur at the output stages of processing.

Current research into information processing in schizophrenia has employed a number of different experimental paradigms. One which has been widely used is the Continuous Performance Test (CPT) which involves the presentation of a sequence of stimuli at the rate of approx. 1 per second. Subjects are required to respond to only
certain specified targets, for example, 'X', usually by pressing a key. Patients with schizophrenia have been found to have lower accuracy rates and a greater number of false positives than healthy controls (Orzack and Kornesky, 1966). When the requirements of the task are manipulated such that the processing demands are increased, for example, increased speed of presentation (Nuechterlein and Dawson, 1984) and using degraded stimuli (Nuechterlein, 1983), performance deficits in patients with schizophrenia become more apparent. The deficits on the task have been attributed to a failure to sustain focused attention over time and the results of studies using degraded stimuli would imply that patients with schizophrenia have difficulty in encoding the relevant information.

There have been a large number of studies which examine the nature of the inhibitory deficit in patients with schizophrenia. The earliest of these used the cross over reaction time (CORT) paradigm which requires subjects to respond to a warning stimulus or cue after a time interval. In normal subjects, knowledge of the length of this interval improves performance, however, in patients with schizophrenia, performance only benefits if the inter-stimulus interval is less than 7 secs. It is thought that the patients are unable to prevent interference from previous trials over a time interval and fail to filter out or inhibit this irrelevant material (Nuechterlein, 1977).

In essence, information processing studies of attention in schizophrenia have consistently reported deficits in patients' ability to perform attentional tasks. Unlike the neuropsychological paradigms, the tasks are designed to specifically measure attentional processes and are based on a theoretical model. The results of the studies indicate two findings; that patients with schizophrenia have a deficit in sustaining attention over long periods of time and that they have an impairment in selectively attending to relevant material such that they respond inappropriately. The theory governing the research in this area is that selective attention requires inhibition of
unattended material and that this ‘inhibition’ is impaired in patients with schizophrenia.

Despite the differences in paradigms and approaches of neuropsychology and information processing research, the inhibition theory above is similar to that of Shallice (1989; described above) who proposes that patients have an executive deficit, or in other words, an inability to inhibit inappropriate responses. While Shallice appears to attribute poor performance on executive tasks to competition in response selection, the filter theory focuses more on the failure to screen out irrelevant material at the input stages of processing.

In summary, both neuropsychological and information processing approaches to schizophrenia have highlighted attentional deficits in this population. Both have found that patients have difficulty in sustaining attention, lowered speed of processing and an inability to screen out inappropriate information. According to existing theories of attention, the primary cause of these deficits is a failure to select relevant material in the face of competing stimuli. Thus, an executive deficit is suggested which may be localised in the frontal lobes. However, in themselves, the research findings lack specificity; poor concentration is found in a large number of disorders, including depression, and the findings tell us little about how the attentional deficits are specifically related to schizophrenic behaviour. In order to address this cognitive theories have been developed which attempt to explain some of the symptoms of the disorder in terms of an underlying attentional deficit. These are discussed below.

1.3 Attention and Cognitive Theories of Schizophrenia

Despite many years of research into schizophrenia the disease remains a ‘mystery’ in the sense that the aetiology and neural substrates are as yet unspecified and there are many conflicting theories about the disorder. The contribution of psychology to the debate in recent years has been to provide a description of the symptomatology in terms of an underlying cognitive deficit. At present there are two models which have
been proposed which are concerned with processes of attention, both are primarily aimed at explaining positive symptoms rather than negative features of the disease. The first is that of Frith (1987) who hypothesised that there is a disruption of 'willed intention' in schizophrenia such that actions and goal states are not effectively monitored. By this model, auditory hallucinations for example are explained as a failure to monitor inner speech which is therefore misinterpreted an external voice. The second hypothesis proposed by Hemsley (1987a) is that positive symptoms arise from a 'weakening of the influences of stored memories of previous input on current perception'. It is this latter hypothesis that will be discussed here as it has some relevance to the issue of practice and the role of attention in schizophrenia.

Hemsley's original hypothesis was based on work by Broadbent and Neuechterlein (described in the previous section) who describe attentional difficulties in schizophrenia in terms of a failure to select relevant material such that more processing capacity is given to stimuli that are irrelevant to the goal state. This phenomena may be described as 'overattention' in the sense that all aspects of the environment are attended to indiscriminately. Of relevance to Hemsley's theory is the notion that in normal cognition information processing gradually changes from a controlled to an automatic process as irrelevant information is inhibited from conscious awareness (Schneider and Schriffin; 1977). That is, through experience, the irrelevance of certain stimuli is learned and they become inaccessible to conscious attention. In essence, Hemsley's theory proposes that, in patients with schizophrenia the occurrence of 'overattention' is a result of patients failure to use stored memories of past experiences as a means of guiding selective attention.

In terms of its ability to account for symptoms of schizophrenia, the model proposes that delusions are the result of misperceptions of causality between stimuli and environmental events. Due to 'overattention' the patient will notice aspects of the environment that are incidental and will attempt to formulate causal relationships between them in order to explain the co-occurrence of events. If previous memories of experiences are not accounted for in these formulations then such attempts to make
sense of the world may be confused and result in misperceptions of the relatedness of stimuli.

Less convincingly the model has also been applied as an explanation for hallucinations. Hemsley accounts for these as 'intrusions' from memory which enter conscious awareness. While normal cognition would be capable of preventing these memories from entering conscious awareness, faulty attentional mechanisms in patients with schizophrenia make these phenomena more likely to occur.

Experimental evidence for Hemsley's hypothesis and the background to the theory are described in Gray et al (1991). In their paper the authors expand on Hemsley's original theories and provide an anatomical basis for the model. In order to substantiate the theory that the symptoms of schizophrenia represent a reduced influence of stored memories on current perception Gray and colleagues cite three experimental findings: The failure of some patients with schizophrenia to show latent inhibition, the negative priming effect and Kamin blocking. In each of these paradigms previous experience affects current performance.

Latent inhibition is a phenomena that has frequently been reported in animal experiments. In essence, experiments involve preexposing subjects to an irrelevant stimuli which later becomes relevant (the conditioned stimulus, CS). In normal subjects subsequent learning of associations with the CS is retarded due to the preexposure. However, experiments with rats have shown that animals given amphetamines which increase dopaminergic function, fail to show latent inhibition. Patients with schizophrenia are known to have increased dopaminergic activity primarily because the symptoms of the disease respond to drugs which decrease dopamine re-uptake.

Baruch et al (1988) conducted a latent inhibition experiment with a group of 53 patients with schizophrenia. Diagnostic criteria classified the sample as 26 acute and 27 chronic patients. Subjects within these groups were randomly assigned to pre-
exposed and non-preexposed groups. All groups were initially presented with a tape recorded list of nonsense syllables. In the preexposure groups 30 bursts of white noise were superimposed over the tape at random intervals. All subjects were instructed to choose one syllable and count how many times they heard it repeated. In the next phase of the experiment all subjects were presented with the recording involving nonsense syllables and white noise. At the same time they were instructed to look at a scoreboard in front of them being told that the scores would rise dependent on the noises they were hearing on the tape. When they became aware of the rule which guided the raising of the points they were to raise their hands and were then to raise their hands when they next expected the scores to rise. In all cases the rule was dependent on the hearing of the white noise.

The results showed that normal subjects in the pre-exposed group showed latent inhibition, that is, learning of the association between the white noise and the scoreboard was retarded by comparison with the non-preexposed group. For the patients with schizophrenia, chronic patients in the pre-exposed group showed latent inhibition while those patients classified as acute failed to show latent inhibition in the pre-exposed condition. In fact, this group showed facilitated learning in that they learnt the association faster than the non-preexposed group (although this effect was not significant).

A further source of experimental evidence to support Hemsley's theory is cited in Gray et al and concerns negative priming. This phenomena occurs when a previously suppressed response becomes relevant. In the Gray experiment (cited as a personal communication) normal subjects were given oral amphetamines in an attempt to determine whether the effects shown in rats could be replicated in humans. In this experiment subjects were asked to name one of two line drawings depending on the colour. Stimuli were arranged such that the target stimulus was the response that had been suppressed in the previous trial. In these instances naming latency was slower. Negative priming was shown not to occur after administration of the amphetamine.
Negative priming has also been shown in patients with schizophrenia. LaPlante et al (1992) used a Stroop experiment to examine the effect in patients with schizophrenia and found that there was less negative priming in schizophrenics, that is, they were less affected by the previous exposure which they had been required to inhibit. However, in contrast to the results with latent inhibition they found that this effect was greater for chronic than acute patients. The details of this experiment are discussed in a later section (section 1.6).

The final source of experimental evidence cited in the Gray paper is that of Kamin blocking. The experimental paradigm described by Gray et al is based on that of Jones et al (1990). As with latent inhibition experiments subjects are divided into two groups. In the first stage of the experiment subjects are presented with a series of shapes on a computer monitor. For the blocking condition, the appearance of yellow squares is always preceded by a blue square while for control subjects a random sequence of triangles (of any colour) are presented. In the second phase, both groups are presented with a sequence in which yellow squares are preceded by a blue square and also by a second stimulus, in this case two white squares presented at the edge of the monitor. In the final stage of the experiment, the appearance of the yellow square is predicted by the second stimulus (i.e. the two white squares) while the blue square is presented randomly. Subjects are required to predict the appearance of the yellow square and learning is measured by the number of trials needed to reach reliable prediction. Normal subjects in the blocking condition show retarded learning of the association (Jones 1989; Jones et al 1990). When this experiment was conducted with patients with schizophrenia it was found that patients with acute symptoms of the disease did not show the Kamin blocking effect, that is, they were able to learn the association of the white squares and the yellow square as quickly as subjects who had been in the control condition. (Jones 1989).

The most widely accepted explanation for the Kamin blocking effect, latent inhibition and negative priming is that in normal subjects, mechanisms of selective attention prevent the entrance of previously irrelevant information into conscious awareness.
thus retarding future learning when previously irrelevant material subsequently becomes salient. In essence, it is thought that these paradigms demonstrate the process of active inhibition of irrelevant material which is fundamental to selective attention. The evidence from the above studies would suggest that patients with acute symptoms of schizophrenia demonstrate a failure of selective attention resulting in the non-occurrence of these effects. Gray et al describe this as attending to all stimuli regardless of their importance.

There have been numerous criticisms of Hemsley's model and the experimental evidence that he cites. One of the most fundamental of these is his interpretation of the results of latent inhibition and Kamin blocking as a 'weakening of the influence of past regularities on current perception'. Dawson and Hazlett (1991) cite evidence from habituation and orienting studies which also require the use of 'past memories'. Habituation and orienting are described by Pavlov (1927, as cited in Dawson and Hazlett) as an 'investigation' or 'what is it?' response and may be measured by skin conductance responses to novel stimuli. By the Gray model patients with acute symptoms could show either greater than normal habituation due to overattention or impaired habituation due to a weakening of the influence of past memories. Studies have shown that some patients fail to show an orienting response (Dawson and Neuechterlein, 1984; Ohman, 1981) and for those who do respond some show faster habituation than controls while others habituate more slowly. The authors emphasise that this heterogeneity of responding is not incorporated into the Gray model.

Frith (1991) takes these criticisms a step further stating that the failure of patients with schizophrenia to show latent inhibition infers that they continue to attend to stimuli when this is no longer appropriate. An extension of this explanation is that failure to show latent inhibition is primarily the absence of habituation. While failure to habituate may be described in terms of a weakening of the influence of past memories on current behaviour, other processes such as stimulus identification and stimulus significance are also involved. It is likely therefore that latent inhibition also
comprises many more complex processes that the Gray model does not take into account.

Further, Dawson and Hazlett point out that what is common to latent inhibition and Kamin blocking is that they require not only the use of past memories but also reversal learning, that is, they require the subject to detect a change in the relevance of a stimulus. The authors and Frith comment that if patients had deficient use of past memories to guide current perceptions then they would have considerable difficulty in learning associations at all, let alone reversal learning. In this sense, latent inhibition could be seen as a failure to shift attentional set. However, given that set shifting deficits are reported in patients with schizophrenia (Elliot et al. 1995; Weinberger, 1986) it would be expected that they would display greater latent inhibition rather than none at all.

Frith comments that Gray et al provide little explanation of the concept of relevance and suggests that relevance is determined largely by context. Thus, failure to show latent inhibition may be related to deficits in inferring relevance when there are no explicit contextual changes. Thus, the Gray et al model fails to look at the effects of context which may have considerable impact on the process of latent inhibition.

Some of these criticisms are addressed in part by Jones et al (1991). In this study, the authors use a design which involves naming a target letter which is flanked by two others, for example, XAX and YBY. In ten percent of the presentations the flankers are reversed, that is, YAY and XBX (termed 'invalid' trials) and it has been reported that normal subjects perform more slowly on these trials (Eriksen and Eriksen, 1974). The authors propose that this experimental design allows for discrimination between failure due to inefficient prior learning, that is, that subjects fail to learn the previous regularity and that of 'overattention', that is, they attend to all aspects of the environment and do not inhibit selective attention to the invalid trials. In the first instance, patients would not show the normal slowing on invalid trials because they had failed to learn the first regularity. If the second explanation were true, patients
would be abnormally sensitive to context effects and therefore show greater slowing than normals. The results showed that while normals and chronic patients showed slowing on the invalid trials, patients with acute symptoms showed no slowing of reaction times. However, they did show a significantly higher number of errors on the invalid trials. The authors were therefore unable to draw any firm conclusions.

While the Jones et al (1991) experiment attempts to delineate further some of the component processes which may be responsible for patients’ failure to show latent inhibition and Kamin blocking effects, it fails to reach any firm conclusions. In addition, the distinction they make between the prior learning hypothesis and that of a broadening of selective attention appears confusing given that Hemsley’s original model would imply that prior learning is disrupted because of a broadening of selective attention. Thus, if prior learning is inefficient then it is likely that selective attention mechanisms are also disrupted and the two processes would be indistinguishable. Further, the assumption that a broadening of selective attention would result in patients being abnormally sensitive to context is poorly explained and is not supported by experimental findings.

The previous criticisms imply that to some extent Gray et al have been selective in terms of the experimental evidence chosen in support of the model. As mentioned previously, habituation studies appear to be highly relevant to the proposed hypotheses and yet they are not mentioned by Gray et al. In addition, results form the Laplante (1992) negative priming study showed that chronic rather than acute patients were more likely to show retarded negative priming which is contrary to the findings from the latent inhibition experiment. Further, some more recent studies have not supported the theory. One example is O’ Carroll et al (1993) who used a proactive interference study as an explicit test of the Gray et al model. Proactive interference occurs when new learning is retarded due to previous learning. However, acute schizophrenic patients showed no difference in proactive interference to depressed and healthy controls.
In essence, the model has been criticised for its basis on somewhat flimsy and selective evidence and not taking into account numerous other potential explanations for their findings. Very little evidence is presented and, certainly in the Baruch et al experiment quite low subject numbers are used (less than 15 in each experimental group). Further criticism may also arise from its focus on positive symptomatology without providing sufficient explanation for the negative features of the disease.

In terms of the present investigation the Gray et al model is relevant in that the selective attention deficit discussed in the previous section may have significant effects on the performance of schizophrenic patients on a repeated presentation Stroop paradigm. Evidence would indicate that some patients may fail to show any benefits in terms of practice effects from repeated presentation of Stroop stimuli. The experimental evidence would suggest that this may be due to a failure to selectively attend to relevant aspects of the environment such that the processes whereby behaviours become automatic are disrupted.

1.4 The Stroop Task

In 1935 John Ridley Stroop published a paper describing a task which is now widely known by psychologists around the world as the Stroop colour-word naming test. Since the publication of his landmark study there have been hundreds of further investigations of the task manipulating the experimental variables in order to determine the exact mechanisms by which the ‘Stroop effect’ occurs. In this section, some of these studies are described and an the cognitive theories which have been proposed to account for the findings are discussed.

The original studies presented by Stroop were preceded by a body of literature investigating the finding that naming objects (and colours) takes a longer time than to read aloud the corresponding written word. For example naming a patch of colour as ‘red’ is slower than reading the word ‘red’ aloud (Cattell, 1886). The explanation
proposed for this finding was that word reading is a more automatic process due to the extent to which it is practiced in every day life while colour naming requires voluntary effort as it is a less familiar task.

Nearly fifty years after the publication of Cattell’s work, the idea that words and colours could be combined was first presented by J.R. Stroop. His original study consisted of three experiments: the first required subjects to read aloud 100 words presented on a 10x10 stimulus card. Four cards were presented: in the 2 experimental conditions, the card was printed with five colour words (red, blue, green, brown and purple) each written equally often in one of the other 4 colours. The 2 control conditions required the subjects to read aloud the same colour names but all were written in black ink. As would be expected, there was no significant difference between the time taken to read the control stimuli as to read the words written in coloured inks.

In his second experiment, Stroop used the same experimental stimulus cards but the task required subjects to name the colour the word was written in rather than read the word itself. The control conditions required subjects to name blocks of colour which were substituted for words on the stimulus cards. His findings showed what he termed a ‘marked interference effect’ whereby subjects took an average of 47 seconds longer to name the colours of the incongruent words than to name the blocks of colour.

In a final experiment, Stroop required his subjects to practice reading the incompatible words over a period of 8 days. His results showed that average naming times reduced by 16.8 secs for a 50 word card. Interestingly, subjects were then required to return to the original task of word reading and Stroop found that the 8 days practice on the naming task interfered with the reading such that their times slowed by 24.6 secs. This phenomena was termed the ‘reverse Stroop effect’ and disappeared quickly after a second test.
Since the original studies by Stroop a further phenomena has been identified known as ‘facilitation’ (for review see MacCleod, 1991). When the colour words and the ink colour are congruent the rate of responding increases. While the facilitation effect is small and may not reach significance the two effects are separable and studies have shown that they are dissociable in normal and brain injured populations.

The Stroop effect has been found to be a robust phenomena and replications of the original experiments have produced very similar results (MacCleod, 1991). One of the reasons for the popularity of Stroop’s paradigm is that it is not only replicable but also open to a wide range of experimental variations whereby the demands of the task are manipulated in order to dissect the cognitive processes which underlie the effect. Examples of these manipulations include practice effects, variations in the order of presentation, negative priming of stimuli and use of different control tasks. Despite the variety and inventiveness of the Stroop experiments which have been designed in the past 50 years there is still some debate concerning the causes of the Stroop interference effect and a number of explanations have been proposed.

Information processing accounts of the Stroop effect have focused on two theories: the speed-of processing account and the Automaticity theory. The former explanation proposes that interference occurs because word reading is faster than colour naming. The two processes are hypothesised to occur in parallel and they compete to be the response which is actually produced. Thus, the general interpretation is that competition occurs at the output stage and the result of this competition is ‘interference’. In their description of the speed of processing explanation Posner and Snyder (1975) specify that interference occurs in the direction of the relative speeds of processing, that is, the slower process receives greater interference from the faster than the reverse. This theory is supported by much of the experimental research which has emerged (for review see MacCleod; 1991), however, there is one direct test which has shed some doubt on the veracity of the explanation. Stimulus onset asynchrony (SOA) was first attempted by Dyer (1971). The method is essentially a means of
manipulating the speed of processing demands of the Stroop by presenting a block of colour prior to the presentation of the colour-word. It would be expected that this delay would enable the slower colour naming process to be given a 'head start' in the race to be the response output. The results showed that interference and facilitation still occurred if the word was presented within a 100ms window of the ink colour. Further studies have shown similar results. In addition, SOA had no impact on word reading and no experiment found evidence of a reverse Stroop effect such that colour naming interfered with word reading. In the light of this finding, the speed-of-processing hypothesis has been discredited and other explanations have received greater attention.

The automaticity account of the Stroop interference effect is based on the premise that different processes are on a gradient of automaticity that is developed by learning. Thus, word reading which is a highly practiced skill is very automatic while colour naming is less automatic due to its comparatively low usage. In a task where there are competing responses, the more automatic processing interferes with the less automatic processing. This explanation has been widely favoured and accounts for experimental findings including semantic priming. The priming task will automatically spread activation and thus will interfere with the ensuing response.

There are, however, a number of results which are not accounted for by the automaticity hypothesis. One difficulty is that there is no role for attention in the automaticity hypothesis and as such strategies that interfere with the allocation of attention should not have an effect on the degree of interference that occurs. This is not the case and experiments that have manipulated the number of congruent and incongruent trials have been shown to alter interference. In addition, studies which have introduced distractor stimuli for example, Kahneman and Chajczyk (1983) who placed irrelevant words on the display, have shown that interference increases if attention is allocated away from the task, even when the distractor is a more 'automatic' process such as word reading.
The main criticism of the automaticity hypothesis is that the notion of 'automaticity' is not subject to direct test in the way that speed of processing was able to be assessed by SOA paradigms. The degree to which a process is perceived as 'automatic' is currently based on test performance and predictions of interference are made after the fact.

From a neuropsychological perspective, the Stroop paradigm is seen as a direct measure of selective attention and the ability to inhibit an automatic process, that is, word reading. Rafal and Henik (1994) perceive that practice not only increases the automaticity of word reading but also allows increased control over the skill such that it may be inhibited when necessary. Thus, reduced interference on the Stroop task is indicative of a highly developed reading skill that is able to be controlled.

This aspect of the Stroop task is evident in studies which have used bilingual subjects to examine the effects of language competence on Stroop performance. Tzelgov, Henik and Leiser (1990) used two groups of subjects; one fluent in Arabic with Hebrew as a second language and a second group of Hebrew speakers with a knowledge of Arabic. The first experimental condition had 80% of Stroop stimuli in one language and 20% in the other while in the second condition the proportions in each language were reversed. When the expected language (i.e. the language of 80% of the trials) was their preferred language, subjects were able to inhibit the Stroop effect and took less time to name the ink colour in the incongruent condition than when their second language was expected. Thus for Arabic speakers, the effect for Arabic stimuli, was smaller (76 msecs) when Arabic was expected than when Hebrew was the expected language (121 msec). For Hebrew stimuli the effect was smaller when Hebrew was expected 109 sec than when Arabic was expected (122 msec). For Hebrew speakers the opposite pattern was found; for Hebrew stimuli the effect was smaller when Hebrew was expected (100 msec) than when unexpected (136 msec, i.e. when Arabic was expected). There was no difference for Hebrew speakers when Arabic stimuli were expected (61 msec) or unexpected (56 msec).
Thus, in each condition, there was a Stroop effect, that is, it took longer to name the colour in the incongruent condition than when colours and words were congruent. The study also showed that control of the magnitude of this effect is possible in certain circumstances and that language competence is a factor affecting this control. The experiment also raised the issue of how expectations could affect performance on the Stroop. In a later study, Tzelgov, Henik and Berger (1992) used a Stroop paradigm involving neutral words as well as colour words. They found that the interference effect increased when there was a larger proportion of neutral words suggesting that different expectations of the stimulus affected the ability of the subject to control the interference effect. Interestingly, increasing the number of neutral words did not alter the facilitation effects.

Further studies which have investigated the effects of trial expectation have also shown interesting effects on performance. Logan and Zbrodoff (1979) used a spatial analogue of the Stroop paradigm and found that as the frequency of incongruent trials increased, response times for these trials grew faster while response times for congruent trials became slower. The authors proposed that this result was due to the subjects’ dividing attention between the two dimensions (spatial location and word name) as the incongruent trials increased. In a series of studies, Tzelgov et al (1990, 1992) altered the proportions of congruent, incongruent and neutral trials orthogonally. They found that altering the congruent vs. incongruent expectations affected the facilitatory component while changing the proportion of neutral words affected interference magnitude. Rafal and Henik propose that when the proportion of congruent trials is high, the subject attends more closely to the word than the colour as it is informative in itself and may aid processing.

These studies provide an insight into the ‘automaticity’ element of the Stroop. While word reading is ‘automatic’ and interferes with colour naming, it is evident that a degree of control may be exerted on the process. Thus the ‘automaticity’ is mediated by subjects’ expectations of the stimuli and their language proficiency. These
Experiments highlight that practice/learning not only leads to proficiency in the skill and automaticity, but also the ability to control performance when necessary.

### 1.5 Practice Effects and The Stroop

Since Stroop's original experiment looking at the effects of practice on interference effects (described earlier) there have been relatively few studies in this area. In addition, many of these studies have sought to compare the effects of practice using alternative versions of the Stroop paradigm, for example, sorting tests (Flowers and Stoup, 1977) or semantic tasks (Menard-Buteau and Cavanagh 1984) which are not relevant to the present investigation.

The principle focus of studies has been to establish whether the magnitude of the Stroop effect decreases with practice. Based on the hypothesis that the Stroop effect occurs because reading is a more 'practised' skill than colour naming, if colour naming is also practised then Stroop interference may decrease. This was not the case in a study by Alperson (1967) who found that practice of the incongruent condition did not lead to a decrease in the Stroop interference effect. However, it is notable that he conducted only 3-50 practice trials which is a very small number compared to that of Stroop's original experiment.

Harbeson et al (1982) conducted a study using 19 subjects aged between 19 and 24 years. They presented the Stroop paradigm on a series of 10x10 slides requiring a push button response with the measure being the number of responses produced in 30 seconds. Alternative forms of the test were presented fifteen times on consecutive days. Stimuli were always presented in the same order: Black and white colour words, coloured blocks and colour incongruent words. Means of all scores showed learning curves which stabilised after 4-6 days. It was noted that on all testing occasions incongruent colour word naming was slower than the other two conditions.
The issue of whether incongruent naming improves more than other naming is still under debate. Hollingworth (1915) tested 19 subjects up to 100 times over 10-40 days. He found a 30% increase in speed of responding in the incongruent condition however, it always remained 37% slower than naming in the congruent condition. It is not clear however whether congruent naming was practised as in the Harbeson experiment. Jensen (1965) used 10 administrations of the test and found that there was a greater improvement in the incongruent condition as compared to congruent naming. By contrast, Warner Brown (1915, as cited in Jensen and Rohwer's review, 1966) stated that both conditions improved to the same extent with practice.

What is interesting about all these studies is that performance became faster with practice. Thus, even though there was no reduction in the Stroop effect in any of the experiments except for Stroop's original study in which the 'reverse Stroop effect' was only transitory, subjects became more efficient at the task after repeated presentations. While it would be interesting to establish whether these improvements are more significant in the incongruent condition or whether the same reaction time improvements could be found for congruent stimuli it is still important to note that general changes occur in efficiency over time.

As yet, no studies have looked at the effects of practice on the task in patient groups. For the purposes of the present investigation however, it is relevant that normal subjects do show improvements and it is important to establish whether patients with schizophrenia are able to show the same improvements in efficiency. In terms of functional imaging experiments which have frequently used the Stroop paradigm changes in performance related to practice effects must be taken into account when analysing brain-behaviour relationships.

1.6 Stroop Studies in Schizophrenia

The Stroop paradigm has been used widely in schizophrenia research as a measure of selective attention in order to assess the patients' ability to inhibit automatic
responding and select the appropriate response category. In addition, the task has been used by neuropsychologists as a measure of frontal lobe functioning and as such, inferences as to the localisation of brain areas associated with schizophrenia have been made from studies using the Stroop paradigm. While there have been a number of studies which have used the Stroop as part of a battery of tests aimed to assess executive functioning, the most informative studies have provided detailed analysis of specific aspects of performance on the task.

It is generally acknowledged that patients with schizophrenia perform poorly on the Stroop task such that many studies have used it in studies of medication effects in order to assess changes in cognition post-medication (Verdoux et al 1995; Buchanan et al, 1994a). In both these studies, the patients with schizophrenia performed more poorly on the task than normal controls, even after improvements had occurred due to medication. A further study by Buchanan and colleagues (1994b) comparing deficit and non-deficit forms of schizophrenia also found that both groups performed more poorly than normal controls on the Stroop test. A recent study by Joyce et al (1996) also used the Stroop task in an experiment examining verbal fluency. They found again, that the patients with schizophrenia performed more poorly on the task than healthy controls. It should be noted that in all of these studies the decrement in performance of patients with schizophrenia was based on their slowed performance relative to controls. In Buchanan et al’s study (1994b) a score was calculated based on congruent reading times to predict the incongruent colour naming time. The discrepancy of these scores from the actual obtained time was calculated as a measure of performance. This method is interesting but the notion of generating a predicted score for the Stroop effect is without precedent. In addition, no indication of the number of errors made was given in the literature and it is unclear whether patients with schizophrenia are able to do the task, albeit slowly, or whether they are unable to overcome the demands of the task and make perseverative or other types of error.

One of the questions which has been highlighted in Stroop research in schizophrenia has been the issue of whether attentional deficits are a result of a limited processing
capacity or if the deficits seen are a result of cognitive fatigue due to the high attentional demands of the task. Harvey, (1984) observed that in normal subjects, Stroop performance was less effortful if the instructions were repeated prior to each trial and when the cognitive operation to be performed varied from one trial to the next. He hypothesised that delayed responding in the normal Stroop paradigm occurs because some of the cognitive resources are taken up with remembering the instructions, and/or that fatigue increases with repetition of the same cognitive process over time. Everett and colleagues (1989) compared schizophrenic patients, depressed patients and healthy controls on two versions of the Stroop task; the standard 100 item version, and a shortened, 10 item task. They found that both groups of psychiatric patients were slower on all three parts of the Stroop test (word reading, colour naming and word-colour incongruency) than the controls. However, the patients with schizophrenia showed the greatest increase of all three groups from performance of the incongruent condition in the short version to performance in the standard task. The results suggest that patients with schizophrenia do have difficulty in selectively attending to the relevant dimension of the stimulus but that this deficit is not specific to schizophrenia and is found in other psychiatric patients. However, the patients with schizophrenia do show an increased difficulty in maintaining selective attention over time which is not present in the nonschizophrenic group.

A further issue which has been a subject of debate in attentional research is the nature of the inhibitory deficit in schizophrenia. Despite the behavioural and experimental evidence suggesting that patients with schizophrenia display diminished inhibitory ability, there is also evidence to suggest that they also show an excess of inhibition. Mialet (1981) interpreted the results from the CORT (cross over reaction time) experiments (described in previous section) differently from previous authors, suggesting that there are two processes involved in performance; first an activation of the appropriate response network and second, inhibition of this activation until the imperative stimulus occurs. Thus, a long time delay would lead to an excess of inhibition in the patients with schizophrenia and they would be unable to respond when necessary. Shagass et al (1978) proposed that excessive inhibition was
responsible for a pattern of evoked potentials found after consecutive presentation of two identical stimuli. In healthy controls, the evoked potential to the second stimuli is less than to the first due to an inhibitory mechanism applied after the first response. If the time interval between presentations is increased the evoked potentials become similar in size. However, in patients with schizophrenia this recovery of the second event related potential (EP) does not occur and the authors attributed this to an excessive inhibitory mechanism in these patients.

In order to address the question of persistent vs. insufficient inhibition, Laplante and colleagues (1992) investigated ‘negative priming’ using a paradigm in which Stroop like stimuli are arranged in order to create a ‘distractor-suppression effect’. As with the standard Stroop experiment, the subject is required to suppress word reading in favour of colour naming, however, the items are arranged so that the response that was inhibited on the previous trial is activated on the next. For example, the word BLUE written in green (correct response) is followed by a trial where the correct response is blue. Studies have shown that response time on the second item is increased in normal subjects and that the effect occurs because of the active cognitive inhibition that has occurred on the previous trial.

Laplante and colleagues investigated the negative priming effect in eight positive, 10 negative (chronic) patients with schizophrenia, 21 depressed patients and 35 healthy controls. The results showed that both groups of patients with schizophrenia did not show a distractor-suppresser effect, that is, response times did not increase when the previously irrelevant stimuli became the correct response. The authors proposed that this was due to inadequacy of inhibitory responses. In addition, the experiment had a further condition in which the response-stimulus latency was increased. Patients with schizophrenia were able to display a suppresser effect if given 1300ms for the inhibitory effect to build up. This effect was quicker to build up in patients with acute symptoms than for the chronic population. The authors proposed that there are two levels of inhibition necessary for the task; the first is the level which allows correct responding to the colour-word stimulus, the second is a higher level that is required
for a distractor-suppresser effect to occur. Overall, the results supported the notion of insufficient inhibition in patients with schizophrenia and that this insufficiency was more marked for patients with chronic negative features of the disease.

A further Stroop experiment which has supported the notion of insufficient inhibition in patients with schizophrenia is that by Carter and colleagues (1992). In contrast to many other studies of Stroop performance in these patients the authors did not find evidence for increased Stroop interference. However, they did find that patients with schizophrenia displayed greater facilitation than healthy controls such that naming of colour-congruent words was faster than in normals. The authors attributed this to a selective disruption of inhibitory processes that were separate to those involved in the interference effect. They compared the effect to that of semantic priming which is also increased in patients with schizophrenia and has been attributed to a lack of inhibition in the semantic network (Kwapil et al, 1990).

Carter and colleagues went on to produce a second experiment (1993) which examined the facilitation effect across different illness subtypes. They found that undifferentiated patients (those with prominent delusions, hallucinations or grossly disorganised behaviour) were responsible for the increased facilitation effect, while patients meeting criteria for the paranoid subtype (one systematised delusion/hallucination, flat/inappropriate affect) displayed an interference effect with normal facilitation.

Thus, the studies of patients with schizophrenia on the Stroop task show that this population has difficulty in completing the interference condition of the task. Performance on the incongruent naming task is slower than in healthy controls and is more error prone. Studies which have manipulated the experimental variables in order to assess specific aspects of performance have shown that poor performance may be attributed to a failure to inhibit competing responses. This failure to inhibit effectively may also account for the increased facilitation effect in patients with schizophrenia. Thus, the results from the Stroop studies are consistent with other studies of attention...
in schizophrenia and indicate that these patients have a deficit in the executive control of action. In addition, they are consistent with the Hemsley model described earlier which uses the concept of ‘overattention’ to describe patients’ inability to stop irrelevant material entering conscious processing. In summary, the above studies highlight two important issues in schizophrenia research: that there is a great deal of variability between the cognitive deficits associated with different symptom subtypes and, that although there are deficits present, these are not necessarily specific to schizophrenia and may be found in other psychiatric populations.

1.7 The Functional Anatomy of Visual Attention

The notion that cognitive processes are localised in areas of the human brain has been the fundamental assumption of the majority of neuropsychological study. In the main, localisation of function has been achieved by comparing subjects’ performance on neuropsychological tests against that of a patients with a known brain lesion. However, there are two major difficulties with this approach; 1) that the tasks have to be highly specified in terms of the cognitive processes which they are assessing otherwise it is possible that poor performance may be attributable to different cognitive deficits and therefore different brain areas and; 2) that it is rare to find patients with small isolated brain lesions and the numerous connections in the brain make it difficult to establish exactly what areas may be affected by a particular lesion.

These difficulties have been addressed to some extent by the work of cognitive neuropsychologists who have focused on specifying the cognitive processes involved in complex operations such as speech and word reading (Coltheart, 1978). In addition, the advent of functional imaging techniques has led to the ability to assess brain activity during the performance of tasks and thus localise the areas associated with a particular cognitive operation.

An area which has been studied in some detail is that of visual attention and the selection of attention. Studies in this area have focused on patients with ‘neglect’ a
phenomena that is characterised by an inability to attend to objects in one side of space. Mesulam (1981) proposed that there are three cortical regions involved in the directed attention; sensory representation in the posterior parietal cortex, a schema for movement in the frontal cortex and motivational aspects in the cingulate cortex.

More recently, Posner and colleagues (1982, 1987, 1984; Posner, 1988) have proposed a model of the brain areas responsible for directed attention comprising a spatial attention system and an executive attention system in the anterior cingulate which is concerned with inhibiting competing responses. In addition, a vigilance system located in the right anterior hemisphere is responsible for sustaining attention and alerting to the presence of new stimuli. This model is presented in the following section and is based on research using monkeys, brain lesioned subjects and PET. While the model represents only one proposed system of visual attention it is backed by substantial experimental evidence and has provided a useful basis for studies investigating disturbed attentional processing.

The most striking example of an attentional deficit is found in subjects with ‘neglect’ and studies using single cell recording from alert monkeys have shown that neglect can occur after damage to three distinct brain areas: the posterior parietal lobe, a portion of the thalamus (part of the pulvinar) and areas of the midbrain that control eye movements.

Studies of visual orienting of attention have used a paradigm which involves the cueing of attention using arrows pointing to a stimulus future location or brief flashes which indicate where a target stimulus will next occur. The experimental variables are manipulated by providing false cues or no cue at all. When the target stimulus appears subjects are required to attend by moving their eyes towards it as quickly as possible and pressing a response key. Attention is assessed by the efficiency with which they are able to process targets at cued and uncued locations. In normal subjects, reaction times are lower if the target occurs in the cued location.
Posner and colleagues found that there were variations in visual attention that occurred as a result of the three different lesion sites. Patients with lesions in the parietal lobe take a longer time to orient to stimuli on the opposite side to the lesion only when their attention has previously been cued to a stimulus on the lesion side. However, when the cue and target are both on the contralesional side there is no increase in reaction time. These results led the authors to propose that the posterior parietal lobe plays a role in the disengagement of attention to stimuli and that the results suggest that the patients were unable to disengage from a cue that was on the lesion side if the target appeared on the contralesional side.

Patients with midbrain damage have been found to experience a transient loss of voluntary eye movements that, after recovery, appear as a delay in movement. Patients tested on the cued paradigm displayed a general increase in reaction time. In addition, faster responding to cued locations than uncued locations did not occur. The authors proposed that the midbrain plays a role in attentional movements and thus control of eye movements to the cues and targets was lowered in the lesioned patients.

Patients with thalamic damage were found to have slower reaction times to targets that appeared on the side opposite the lesion. This occurred whether the target was cued or uncued. Posner et al hypothesised that this result reflected a failure to engage attention and use it to enhance processing speed.

Thus, the visual spatial attention system involves three distinct operations: disengagement from a previously attended stimuli, eye movement towards a to-be-attended target, and engagement / processing of the new stimuli. Thus, the system is responsible for shifting attention towards a target and sending the visual information forward in the brain.

Once the orienting process has occurred, the second part of Posner’s model, the executive attention network, is called into action. The network has the role of ‘detection’, that is, ‘bringing an object into conscious awareness’ (Posner and Raichle,
The type of processes involved in the executive network have been termed 'attention for action', that is, the type of attention that is required so that meaningful stimuli are assigned more resources than irrelevant stimuli and are used for the direction of action (Mesulam, 1981). As such, this form of attention limits the large number of conflicting actions that may occur in response to a stimuli and assists in the generation of the appropriate operation.

The brain areas associated with this network are considered to be in the frontal lobes (Posner and Raichle, 1994). Patients with frontal lobe lesions have been found to lack the ability to follow instructions and act in a seemingly random way without concern for achieving a particular goal state (Posner and Raichle, 1994). One area that has been considered to be fundamental to the executive attention network is the anterior cingulate gyrus. Studies which have examined patients with bilateral lesions to the anterior cingulate have displayed a disorder termed 'akinetic mutism' in which patients have extreme difficulty in initiating voluntary activity including speech. While orientation to visual stimuli is intact other voluntary actions are retarded (Posner and Raichle, 1991, Janer and Pardo, 1991).

The question that has concerned researchers in this area is the role of the visual spatial orienting system and the executive attention network during word reading. Studies have found that some patients with right parietal lesions may neglect the first few letters of a non-word. However, when a real word is presented with the same number of letters in the same spatial location, the patient is able to read it accurately suggesting that words are not scanned by the visual spatial attention system described above (Sieroff, 1988).

Posner and colleagues report a series of experiments using PET to measure cerebral blood flow in different brain areas while performing word reading tasks (Posner et al 1989). The first task required subjects to passively look at nouns that were foveally presented at the rate of one per second. The control experiment involved passive fixation on a point on the screen. The results showed that there were five areas of
activation in the occipital lobes. When words were presented orally no activation was
found in this area and the authors concluded that the activations were specific to
visual word forms and that this process is performed entirely in the occipital lobes.

More complex word form processing was found to activate different areas of the
brain. The authors presented the subjects with 40 nouns and asked them to generate a
use of the word (for example, ‘hammer’ the response would be hit). As a control
condition, subjects were required to repeat nouns, i.e., there was no generational
component or semantic processing. A second task required subjects to passively read
a list of 40 presented nouns and note which were dangerous animals. No verbal
output was required and subjects were asked to estimate the number at the end of the
test. In both of these experimental conditions the same two areas of the cortex were
activated: the anterior left frontal lobe and a more anterior, inferior region
corresponding to the anterior cingulate gyrus. In the condition requiring verbal output
there was also activation in the medial frontal lobe corresponding to the
supplementary motor area.

Previous research has shown that damage to the first of these areas, the left lateral
frontal region, is associated with poor verbal fluency and is activated only during
tasks which require semantic processing. Thus, it is hypothesised that the area is
responsible for forming word associations. The latter area, the anterior cingulate
gyrus appears to be responsible for the selection of responses.

In order to test the role of the two areas, Posner et al. employed a paradigm whereby
in the first condition, subjects were required to decide whether words belonged to the
category of dangerous animals or not. In the second condition, subjects were required
to note the presence of dangerous animals in a word list without producing a verbal
response. According to cognitive theory, the second condition has a greater
attentional requirement. The authors found that in the second condition, the larger the
number of target words (that is, dangerous animals) in the list the greater the
activation in the cingulate cortex. The left frontal area displayed little difference in
blood flow between the two conditions.

Thus, the results from these experiments would suggest that there are a number of
processes involved in word reading. The forming of visual word forms occurs in the
occipital lobe and this process does not involve the parietal/midbrain system of visual
spatial attention. When words are processed semantically, blood flow increases in the
left anterior frontal lobe and it is thought that this area is responsible for forming word
associations. A further important component to word reading and processing is the
anterior cingulate cortex which is hypothesised to be responsible for higher level
attentional tasks which require ‘attention for action’. This area appears to be
responsible for selecting and generating appropriate action by focusing resources on
the relevant stimuli.

Posner and colleagues therefore hypothesise that the executive attention network
consists of a number of frontal brain areas including the anterior cingulate cortex and
the lateral frontal lobe. In addition, Goldman-Rakic (1987), has discovered an area of
the lateral, upper prefrontal cortex in monkeys that is responsible for maintaining the
spatial position of objects in memory when they are removed from view.

The third network proposed by Posner is the vigilance network which is responsible
for sustaining attention and alertness. An example of a task requiring this network is
one where subjects are reading a list of words and are asked to pick out those
corresponding to particular category. If the target words appear infrequently it is
darker to remain alert and the body has a physiological response which slows the heart
rate and electrical activity recordings from the scalp indicate lowered levels.
However, according to PET studies, the right side of frontal and parietal lobes
becomes activated at these times. In addition, activation in the anterior cingulate
decreases as right frontal blood flow increases.
Fiona Barber PsychD.

Pardo et al (1991) measured brain blood flow while subjects were required to perform tasks requiring sustained attention to sensory input, a somatosensory task and a visual task. In the former, subjects were required to focus attention on their right or left big toe and monitor pauses in suprathreshold touches with a von Frey hair. In the visual task, subjects were required to detect luminance changes in a central fixation mark. During both tasks there was activation in the prefrontal and superior parietal cortex, primarily in the right hemisphere, regardless of the modality of the sensory input. The anterior cingulate was not activated during either experimental condition. The authors concluded that there is an anatomically distinct neural system which mediates sustained attention to sensory stimuli.

1.8 The Anterior Cingulate and the Stroop Paradigm

The notion of the anterior cingulate as an important component in attentional systems has been assessed using both functional imaging techniques and studies of patients with cingulotomies. Due to its important role in the processing of words, many of these studies have used the Stroop paradigm which combines high level attentional requirements with a word reading component.

One of the first of these studies was performed by Pardo et al (1990) who used the classical Stroop paradigm to investigate the brain areas associated with cognitive operations in attentional conflict paradigms. Eight healthy volunteers were asked to name the colour of words presented to them on a video monitor for 1300msec with a 350ms interval. In the congruent condition all the colour names were the same as the colour presented (that is, red written in red). In the second condition the names and colours were incongruent. The difference in brain activity between the two scans was measured and it was hypothesised that this process would reveal the brain areas involved in the interference effect. Their results revealed that the largest responses occurred in the right anterior cingulate cortex. Additional foci of activity occurred in the bilateral peristriate region, supplementary motor area, inferior anterior cingulate and right temporal areas.
While the Pardo et al study provided the first insights into the brain areas implicated in attentional processes there are some problems with the design of the experiment. The primary difficulty is that the study did not have a control condition and thus the differences in activation between the congruent and incongruent conditions represents the areas associated with facilitation and interference not interference alone. As it has been hypothesised that there are specific neural networks associated with facilitation that are separable from those involved in interference effects (see earlier section, Carter et al, 1992) it is important that these processes are assessed separately. A further criticism of the study is that detail of the experimental design is poorly specified in the write-up of the study. It is unclear how many scans were performed overall and the number of words presented per scan.

An attempt to replicate the Pardo et al study was performed by Bench et al (1993). An additional aim of the study was to obtain baseline data for a study of patients with depression who have been found to show resting state cingulate abnormalities. The study used 2 experimental paradigms: the first comprised 3 tasks, A) naming the colour of coloured crosses (of varying lengths), B) naming the colour of neutral words (top, down, front, back) and C) naming the incongruent colour of a colour word (the standard Stroop task). Six scans were taken for each subject in a ABCCBA design. In the second experiment condition A) was again, naming coloured crosses, B) naming colour congruent words and C) the standard Stroop task. Again the conditions were presented in an ABCCBA design.

The results of the experiments were analysed as a series of comparisons, that is, task C vs Task A, to assess interference, Task C vs Task B, to assess interference vs. word reading and Task B vs Task A to contrast colour naming and word reading. In both experiments, reaction times in the interference condition were greater than for the neutral/congruent word and coloured cross conditions. In terms of rCBF, the results of the first experiment were somewhat surprising in that increased rCBF occurred in the right orbitofrontal region and the cingulate cortex during the interference condition as
compared to the coloured crosses. The comparison of neutral words as opposed to colour crosses produced increased blood flow in the cingulate cortex and left middle frontal gyrus.

In the second experiment, there was significantly greater activation in the right anterior cingulate cortex in the interference condition as compared to naming coloured crosses. These areas were not activated in the comparison of the interference vs. congruent task.

The authors commented that while the results of the second experiment replicate the findings of Pardo et al, the first experiment results do not show a similar pattern of activation, not even in the identical condition of coloured crosses vs. the standard Stroop task. The authors attribute these findings to differences in the experimental paradigms. Most notably the Bench et al study trained the subjects on the task prior to scanning in order to collect reaction time scores. Thus, the patterns of activation may have been disrupted by practice effects and habituation to the task. A further experimental variable that was altered in the Bench et al study was the order of presentation which allowed for between scan habituation. While the Pardo et al study presented the conditions in pairs in a strict congruent-incongruent order, the Bench et al study had three conditions that were ordered in order to control for time effects. The failure to find significant right anterior cingulate activation in the first study was attributed to a relative increase in blood flow to this area during the second congruent task (scan 5). This increase may have been due to the subjects' expectations of the content as it occurred after rather than before the interference condition.

The authors concluded that the results provided evidence for the involvement of the anterior right hemisphere and medial frontal structures in attention. The finding of parietal activation during the scans also supported the Posner model of a dual action attention system with both posterior and anterior involvement. However, the study also highlighted the importance of experimental design in PET paradigms and the complexity of measuring attentional systems.
A later study by George and colleagues (1994) measured regional cerebral blood flow using a Stroop paradigm which was similar to that of Bench et al. In this design, three different tasks were used: the standard Stroop, a Stroop using sad words (e.g. grief, misery) and a control task using coloured bars. The inclusion of the sad Stroop had a dual purpose in that it acted as a neutral condition because the words were not directly competing with the colour naming task; and that it acted as a baseline measure for future PET studies using depressed patients. A further modification to the study was that word presentation was self-paced by the subject who was asked to respond as quickly as possible. The results showed that there was increased activation in the left midcingulate region during the standard Stroop task as compared with the colour bar control task. The reaction time for performing the standard Stroop was correlated with the activity in the left anterior cingulate and midcingulate regions.

Again, the study found discrepant results to the original Pardo et al study. In their discussion, the authors attributed this to the experimental design, particularly the self paced nature of responding which led to presentation being faster than in the other studies. George et al, cite a PET study by Taylor et al (1993) who also used a self paced presentation in a study which required inhibition of inappropriate responding which also found midcingulate activation. The authors attribute the findings to the emphasis on response selection which self pacing produces. They comment that the midcingulate has connections to the primary motor areas and language areas (Van Hoeson et al, 1993) and is thus implicated in the selection of appropriate motor and verbal responses. In contrast, they cite evidence that the anterior cingulate is implicated in tasks involving emotional responses as well as selective attention.

In summary, the three studies which have investigated the functional anatomy of selective attention using the Stroop paradigm provide some support for the notion that the anterior cingulate is implicated in higher order ‘attention for action’. Two of the studies found that the right anterior cingulate received comparatively greater blood flow during the interference condition than a neutral naming task (Bench et al) or
congruent naming task (Pardo et al). However, the studies highlight the difficulty in designing experiments for PET scanning and the necessity to control a large number of experimental parameters in order to focus on the selective attention component of the task. Bench et al. improved on the Pardo design by using a control condition, however, by ordering the conditions with incongruent preceding congruent a different pattern of activation was found such that right anterior cingulate activity increased in the congruent condition as well. The George study also stressed experimental design as a factor in the activation patterns found, particularly allowing subjects to self pace. While they believed that response selection is emphasised in self paced tasks, it may also be argued that self pacing adds additional variation to responses and thus makes it more difficult to establish the role of selective attention. For example, if required to self pace, subjects may be more concerned with speed of responding rather than accuracy, alternatively, some subjects may habituate to the task at a faster rate than others. This latter criticism may be directed at all the studies as individual variations in task performance cannot be accounted for when measuring activation and as such, subjects may be using different strategies to complete the task. Thus, while the three studies provide some information regarding the brain areas associated with selective attention, the clarity of the results is affected by complications of experimental design and poor specification of the cognitive components of the task.

While the above PET studies have provided important information regarding the brain areas associated with attention and word reading it is important to note that while functional imaging techniques have improved considerably since their early usage, many believe that their accuracy is questionable and that the finding should be considered with some reservations (Coltheart, Functional Imaging Conference, 1995). In terms of the scanning techniques used, the accuracy of localisation is relatively poor due to the low spatial resolution of the images produced. In addition, blurring techniques used to map the images lead to an even greater decrease in spatial resolution. Images from different subjects are averaged and then mapped on to an anatomical grid. This method does not take into account the considerable individual
differences in individual brain anatomy. This difficulty is being addressed by MRI 
co-registration, that is, mapping PET activation on to previously taken MRI scans of 
that same individual’s brain, however, this is a complicated and more expensive 
procedure.

A further criticism of the studies is the difficulty in controlling for all the experimental 
variables which may affect brain activation. In even the simplest tasks it is difficult to 
ascertain that all the subjects are performing the task in exactly the same way and 
there is no way to control for the different thoughts or strategies that may be occurring 
during scanning.

One further difficulty that must be noted is that all subjects are volunteers who have 
been given warnings about the dangers of injecting radioactive material. As such, the 
individuals who participate are generally from a skewed sample who perceive 
themselves to be at a low risk. For example, women are not advised to participate if 
they intend to have children in the near future. In addition, the scanning procedure is 
generally extremely uncomfortable for the subjects who must lie still for hours at a 
time with their heads in a restricting mask.

1.9 Schizophrenia and the Anterior Cingulate

In an earlier section, the neural substrates of schizophrenia were discussed in terms of 
dysfunction in fronto-subcortical connections. Different subtypes of the disease were 
attributed to disruption of different pathways within this system. One of the fronto-
subcortical loops isolated by Alexander et al (1986) involved connections to the 
anterior cingulate and the relevance of this area in schizophrenia is discussed in this 
section.

The deficits in attention observed in schizophrenia, found in information processing 
and neuropsychological research, have been reported earlier. However, in addition to 
the deficits in sustained attention it has been noted that never medicated, first-episode
patients with schizophrenia are slower to shift attention to the right visual field, even when cued. According to the Posner model of attention, the patients behaved like those with damage to the left parietal lobe displaying a failure to disengage from stimuli on the lesion side. Interestingly, this deficit is only manifest in patients with hallucinations and delusions (positive symptoms) while chronic patients do not show specific deficit in the right visual field (Posner et al, 1988).

However, as reported earlier, the overwhelming body of research in schizophrenia implicates frontal and subcortical areas of the brain not the parietal lobes. Early, (1987) conducted resting PET scans of unmedicated patients and found abnormality in the left globus pallidus, an area of the basal ganglia which connects to the frontal lobe and it would seem most likely that the visual orienting deficit found is due to dysfunction in the attentional network that connects parietal areas to the executive attention system via the basal ganglia.

Posner and Raichle attempted to mimic the right visual field deficit in normals by requiring them to shift attention to visual cues while engaging in a secondary task of repeating back a story as they heard it. The secondary task involves the executive attention network while the former uses the visual spatial orienting network. The subjects did show deficits in orienting to targets in the right visual field in a way that was similar to that found in schizophrenia patients. Thus, the authors concluded that the executive attention system has some influence over the visual orienting network and that the deficit in schizophrenia could be due to executive dysfunction. According to the Posner model, this would implicate the anterior cingulate which is the fundamental part of the executive attention system.

The previously reported findings of right anterior cingulate involvement in Stroop performance also implicates this area as relevant to schizophrenia. Patients with schizophrenia have great difficulty in performing the interference condition of the test and as the anterior cingulate is most active in this condition, its dysregulation could account for the impairments in schizophrenic patients. This assumption is, however,
Graph 1. Reaction Time for Congruent and Incongruent Conditions across 6 Trials
difficult to substantiate as no study to date has investigated performance of patients with schizophrenia using an identical paradigm to that used in normals under PET activation.

While there is an increasing interest in using PET to study patients with schizophrenia, the majority of studies have looked at attentional processing in healthy subjects and used these results in order to hypothesise about the possible areas of dysfunction in schizophrenia. However, Andreason and colleagues (1992) used a Tower of Hanoi puzzle to examine executive deficits in patients with schizophrenia under PET. In healthy subjects, the task activates the anterior cingulate, however, in unmedicated patients with schizophrenia, who have great difficulty with the task, the anterior cingulate failed to activate.

Other studies implicating the anterior cingulate in schizophrenia have focused on the role of this area in the semantic analysis of words. Patients with schizophrenia show deficits in tasks such as verbal fluency which require intact connections between the anterior cingulate and left lateral frontal areas. Dysfunction in these networks may also be responsible for symptoms such as word salad and neologisms.

Dolan et al. (1995) conducted an interesting PET study investigating the role of dopamine on cortical functioning in normal and unmedicated patients with schizophrenia. The subjects performed a paced verbal fluency task and a paced verbal repetition task before and after the injection of dopaminergic agonist, apomorphine, or a placebo. In healthy subjects, the verbal fluency task activates the left dorso lateral prefrontal cortex, the thalamus and the anterior cingulate cortex. In patients with schizophrenia, the anterior cingulate cortex failed to activate. Following apomorphine injection, the patients displayed a significant increase in anterior cingulate activity that was not present in the normal controls. The effect was specific to the fluency condition and no increase in anterior cingulate activity was found during the repetition task. The authors propose that the regional specificity of the effect adds weight to the argument that the anterior cingulate cortex is implicated in the pathology of
schizophrenia. They suggest that the area may participate in cortico-cortical integration, that is, in maintaining the integrity of the connections between cortical areas and that schizophrenic symptoms may occur as a result of a failure in this integration.

1.10 The Functional Anatomy of Practice.

To date there have been few studies which have attempted to look at practice effects under activation conditions. However, the notion that practice leads to improved performance on a novel task is widely acknowledged and it is important to investigate the brain areas which may be responsible for this phenomena. Peterson and colleagues (1988) conducted a research experiment investigating the different brain areas associated with generating verbs from observed nouns and a control task of speaking a seen noun. The authors found that the former task was associated with activation in the left prefrontal cortex, the anterior cingulate and the right cerebellar hemisphere while the latter activated the sylvian-insular cortex bilaterally. However, after 15 minutes of practice on the experimental task, the brain areas activated during both the tasks were indistinguishable. Behavioural data showed that there was a considerable reduction in response times and improved task accuracy suggesting a practice effect.

These results led the authors to conduct a further study investigating differences between naive and practised performance on a verbal response selection task. Raichle et al (1996) performed the same experiment as described above. On the first scan the subjects fixated on a screen point while words were presented. On the second they were instructed to repeat the nouns as they appeared on the screen and in the third scan the subjects were required to generate an action word that described what the presented noun might do. The list of 40 words was then presented repeatedly for 8 blocks of trials lasting 10-15 minutes. Following the practice, the subjects were scanned again on the experimental task and then performed the two control scans
again. Finally, a novel list of words was presented, initially for repetition and then verb generation.

The activation during the task supported the hypothesis of two separate pathways exist for the execution of the task. The first involves the anterior cingulate, left prefrontal and temporal cortices and the right cerebellum. These areas were activated during the first generation condition (that is, before practice) and in the novel generation condition at the end of the session. The second pathway consisted of bilateral sylvian insular cortex activation and activity in the left medial striate cortex. These areas were activated during the repeat task and in the generation task after practice had occurred. The authors account for the differences in activation in terms of the degree to which a task is learned or automatic.

In terms of the areas implicated in each pathway, activity in the left lateral prefrontal cortex has been reported in a number of studies (e.g. Frith et al, 1991, Friston et al 1991) suggesting that this area is concerned with internalised knowledge which is necessary to guide behaviour in the absence of informative external cues. In Shallice's model it therefore corresponds to some extent with the role of the supervisory attentional system. The possible role of the anterior cingulate cortex has been discussed earlier. The authors speculate that in their experiment its role in spontaneous speech is of some importance and also as a component of the anterior control of attention during complex tasks which require inhibition of inappropriate responding. In both instances, when the task becomes more practised there is no longer any requirement of these functions.

Additionally, there was strong right cerebellar activity. This result has been found previously (Fiez et al 1992; see earlier) who found impaired non-motor learning and low error detection following cerebellar damage. The results suggest that the cerebellum plays an important role in learning and that its activation is not restricted to purely motor tasks.
In terms of the present investigation, the results of the Raichle et al activation study has distinctly different learning demands to the learning involved in the practice of the Stroop paradigm. In their study, subjects practised the same response repeatedly and thus it may be perceived as a learning test of declarative knowledge. Studies of practice of the Stroop effect merely show an increase in efficiency of responding over time rather than the learning of a particular skill as evidenced by the continued appearance of the Stroop interference effect. As such, improvements may be likened more to the association learning shown in latent inhibition and Kamin blocking experiments discussed earlier. In these paradigms the learning is of associations between stimuli and, as Frith proposes, the use of context effects that may be impaired in patients with schizophrenia. Thus, any effect of practice in the Stroop paradigm may be attributed to becoming more aware of the demands of the paradigm and predicting the sequence of presentations, that is, knowing that the next set of words will be incongruent if the previous set was congruent.

Gray et al present a detailed theoretical model of the neuroanatomy which may underpin association learning involving the septo-hippocampal system, the amygdala and subcortical pathways. However, the model has been criticised for being too detailed given the knowledge base that exists. In addition, it is considered that this information is irrelevant to the present paper as the similarities between the association learning experiments cited by Gray et al and the Stroop experiment presented here are limited. For the purposes of the present investigation it is the fact that patients with schizophrenia show some deficit in using previously stored information to guide selective attention that is the most salient issue.

1.11 The Present Study

The present investigation aims to investigate the performance of patients with schizophrenia on a repeated presentation Stroop paradigm. The previous review of the literature in this area has identified a number of issues. First, patients with schizophrenia have been observed to have deficits in selective attention (Frith, 1979;
Gray et al (1991) and second, this has been attributed to a failure to inhibit irrelevant stimuli that are competing for processing resources (Broadbent, 1971). Third, detailed investigations of the Stroop paradigm have shown that it is a robust design that assesses subjects' ability to inhibit automatic word reading in favour of the less well practised skill of colour naming (MacCleod, 1991). Patients with schizophrenia are known to have difficulty with the task (Joyce et al, 1996; Buchanan et al 1994b).

Recent cognitive theories of the deficits underlying schizophrenic symptomatology have proposed that the fundamental difficulty is in integrating stored memories of past regularities of experience with current motor programmes in the control of selective attention (Gray et al 1991). Thus, it is hypothesised that patients with schizophrenia will not only have difficulty coping with the demands of the incongruent condition but also, that they may be unable to show the same improvements as normals when presented with repeated presentations of the same experimental paradigm.

Detailed study of the functional anatomy of attention has proposed that there is a high level 'attention for action' system that is used in situations which require selective attention and inhibition of competing responses. Lesion studies and functional imaging have supported the suggestion that the anterior cingulate cortex is a fundamental part of this executive attention system (Posner and Raichle, 1991; Janer and Pardo, 1991). PET studies of Stroop performance in healthy subjects have shown that in a classical Stroop paradigm the right anterior cingulate cortex is activated in the interference condition (Bench et al 1991; Pardo et al 1990). It is therefore proposed that the Stroop task requires an intact executive attention system for successful performance.

Importantly, while these studies have used repeated presentations of the Stroop paradigm in order to gain sufficient data, none to date have looked at changes at a behavioural level which occur with practice. Experiments with normal subjects have repeatedly found improvements in the speed of responding after repeated presentations of Stroop stimuli and it is vital that these effects are investigated as they
may have significant impact on brain functioning. In addition, before functional imaging is conducted with patients with schizophrenia it is important to establish whether they are able to show similar practice effects. The Gray et al model would suggest that this may not be the case in which case, interpretation of imaging data must be able to take these differences into account.

Furthermore, while authors have inferred that poor performance of patients with schizophrenia on the Stroop task is indicative of dysfunction in the anterior cingulate, this claim cannot be substantiated unless patients are tested using the same Stroop paradigm as that used for healthy subjects. As the literature on PET studies of the Stroop shows, experimental variables have a great deal of influence on the areas of activation and it is vital that these parameters are controlled before inferences about patients with schizophrenia can be made.

The present study employs a Stroop paradigm that is very similar to that of Pardo et al. In order to investigate the effects of practice on performance a repeated presentation paradigm is employed.

In essence, the present investigation aims to investigate the effects of practice on the performance of the Stroop paradigm in both normals and patients with schizophrenia. In addition, the study aims to obtain behavioural data of the performance of patients which can be compared to that of normal controls who have been tested under activation conditions. This comparison is necessary in order to ascertain whether patients with schizophrenia perform differently on the task to normal controls and, if so, in what ways. It is important to look at this information prior to undertaking an expensive scanning procedure so that it is clear what aspects of performance the scanner is measuring.
There were two experimental Hypothesis:

1/ That some patients with schizophrenia will show less practice effects than normal controls after repeated presentation of the Stroop paradigm as shown by no decrease in reaction times over trials.

2/ That patients with schizophrenia will display greater Stroop interference than the normal controls as indicated by slower reaction times and greater numbers of errors.
SECTION 2 - METHODOLOGY

2.1 Design

The study is a comparison of two groups of subjects on a Stroop colour-word naming paradigm. The experimental group consists of patients with schizophrenia selected from a resident population at Royal Park Psychiatric Hospital, Parkville, Victoria, Australia.

The control group is a sample of 16 healthy volunteers, 10 of whom agreed to be tested in a PET scanner while performing the task. The scans were conducted as part of a separate experiment and the results are not reported in the present study. PET scanning was undertaken at the Austen Hospital, Heidelberg, Victoria, Australia.

The presentation of words consists of two conditions:

A) Colour words that are written in a congruent colour (e.g. ‘RED’ in red print)

B) Colour words that are printed in an incongruent colour (e.g. ‘RED’ written in green)

There were a total of 12 presentations, six of the congruent condition and 6 of the incongruent condition. Order of presentation was an ABABABABABABABAB design. Each presentation consisted of 36 words.

2.2 Subjects

A total of 33 subjects took part in the study, 16 in the control group and 17 in the patient group. Due to initial difficulties with the voice key method of reaction time measurement, the data from one subject was excluded. In the patient group the ratio male to female was 10:6 while in the normal controls it was 11:5.
Research and Ethics committee approval was obtained from Royal Park Hospital and the Austen Hospital where the scans were conducted.

2.2.1 Experimental Group: Patients with schizophrenia

Patients were selected from a group of who were involved in a rehabilitation programme based at Royal Park Hospital. The patients were resident at the hospital and were, at that time, considered to be too low functioning to live in sheltered accommodation in the community. The average length of stay on the rehabilitation ward is 20.36 months. The average age of onset of illness for the patients was 19 years (range, 15-24 years). Patients were all taking neuroleptic medications: seven were on Clozapine, one on Haloperidol, one on Pimozide, two on Stelazine, one on Seroquel, one on Risperdone, one on Remoxipride and two on Flupenthixol.

Patients were selected according to the following criteria:

1. DSM III-R diagnosis of schizophrenia, as recorded in medical records (American Psychiatric Association, 1987)

2. No increase in psychotropic medication in the past month

3. Aged less than 55 years

Patients were recruited by the experimenter from the wards at the hospital. All the patients were also involved in another neuropsychological research study and were asked to participate in the present investigation as an adjunct to this experiment. As such, no additional consent form was considered necessary as all patients had previously signed their consent for neuropsychological testing and confidentiality was assured. Dates and times for testing were arranged for the convenience of the patients.
2.2.2 Healthy Control Subjects

Ten control subjects were recruited from the Melbourne area using a poster campaign in the Hospitals, research institute and general notice boards for a PET activation study. Subjects were offered a picture of their brain as a means of encouragement. These subjects were presented with detailed information regarding the risks involved in PET studies. As reported earlier, the activation data was taken for a separate study and is not recorded in this volume.

Six further control subjects were recruited from the nursing staff on the rehabilitation wards at Royal Park Hospital. These subjects were tested under the same conditions as the patients with schizophrenia. These subjects were all naive to the Stroop paradigm. Due to the different testing conditions between the PET scanner and the hospital it was necessary to compare the results of these six controls and the other ten who were tested under scanning conditions. There were no significant differences in the performance of these two groups and they were therefore combined into a group of 16 controls.

Exclusion criteria for the normal controls were:

1/ No history of schizophrenia or bipolar psychiatric disorder
2/ No previous history of neurological disorder (including epilepsy)
3/ No history of excessive alcohol consumption as defined by no admissions to hospital for alcohol related disorders.
4/ Aged under 55 years
5/ For the ten subjects who were scanned, females were excluded if pregnant

2.3 Procedure
The testing occurred between February and November of 1995. Ten of the normal subjects were completed by April 1995 due to time constraints in using the PET scanner.

The ten subjects who were scanned were tested at the Austin Hospital. They were required to lie on a bench wearing a facial mask to prevent movement which could lead to artefact. The monitor was suspended above their heads so that they could read the presented stimuli from a horizontal position. There was a 10 minute delay between each presentation in order to allow for the radioactive isotope to decay.

The remaining subjects were all tested on the rehabilitation ward at Royal Park Hospital. The ward consisted of a series of bungalows and one of these was designed for leisure activities rather than living and had a room in which the computer could be installed. The room was quiet and free from distractions. Subjects were not tested if other activities were going on in the bungalow.

The experimental paradigm involved the presentation of individual coloured words on an IBM video monitor. The subject was seated in a comfortable chair in front of the screen and asked to look at a white cross fixation point presented in the centre of the screen. The measurement of reaction time was accomplished using a voice key which responded to threshold noises. The voice key was calibrated at the beginning of the experiment in order to be sensitive to the volume of the subjects’ voice. When the subject responded to the stimuli, the reaction time was recorded by the computer in milliseconds and the next stimulus was presented. Due to some initial problems with the voice key method (described in the discussion) data for one of the patients with schizophrenia was excluded from the study.

In both experimental conditions the subject was given the instruction to ‘Read the colour that the word on the screen is written in’. At the beginning of each trial this instruction was repeated. Each trial consisted of the presentation of 36 colour words, either written in the same colour as the word (red in red letters) or not (red in green
letters) depending on the experimental condition. The fixation point was present at all
times beneath the word. The experimenter had a written list of the correct responses
in front of them and marked the errors on the sheet as they occurred.

The order of the stimuli for all trials was randomised such that for each trial the order
of presentation of the words was never the same.

There was a two minute (approx.) break between each trial during which the subject
was allowed to relax and walk around if necessary. The testing procedure took
approximately 45 minutes for each subject.

The NART 2nd Edition (Nelson, 1991) was not administered to the patients with
schizophrenia as all had been tested within the past 3 months for another research
project. This project involved the administration of a large neuropsychological battery
of tests and compared chronic, prodromal and first episode cases. The healthy controls
were tested on the NART prior to the administration of the Stroop paradigm.

Patients with schizophrenia were assessed on the Manchester Scale for severity of
symptomatology. This assessment was performed by a consultant psychiatrist at
Royal Park Hospital (Dr. D. Velakoulis). For the purposes of this investigation only
six measures were taken relating to the three syndromes of schizophrenia identified by
Liddle (1987a & 1987b). Namely, Reality distortion, (delusions and hallucinations)
Disorganisation (incoherence of speech and incongruity of affect) and Psychomotor
poverty (blunting of affect and poverty of speech). Each symptom is rated 0-4 with 4
being the most severe. A score of 2 means that the symptom is clinically significant
but mild, 1 indicates it is not clinically relevant but present and 3 indicates moderate
severity.
SECTION 3 - RESULTS

Average scores for reaction time on each trial, measured in milliseconds were calculated by the computer. Thus, the score for each trial is a mean reaction time for the 36 word presentations. The computer automatically excluded outlying data, that is, reaction time scores over 1000ms and it was replaced with the mean result for that trial. The number of errors made on each trial was also recorded.

There was no missing data. All statistics are calculated using SPSS for windows, version 5.0.

3.1 Matching
The subjects in the two groups were matched for age and NART IQ score. The means and standard deviations for the two groups are shown in table 1. A one-way analysis of variance showed that there was no difference in age or IQ between the two groups (F(1,30)=.0943, ns; F(1,30)=.405, ns, respectively).

<table>
<thead>
<tr>
<th></th>
<th>Schizophrenia</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE</td>
<td>30.5 (s.d. 7.67)</td>
<td>29.75 (s.d. 6.04)</td>
</tr>
<tr>
<td>NART</td>
<td>109.87 (s.d. 6.87)</td>
<td>111.56 (s.d. 8.07)</td>
</tr>
</tbody>
</table>

Table 3.1. Mean age and NART IQ score for the two groups

3.2 Error Data

The number of errors made on each trial was recorded by the experimenter on a score sheet. Examination of the scores shows that no errors were made on the first 5 congruent trials by any of the subjects.
Errors were made on the incongruent trials. The number of errors made ranged from 1 to 6 on each of the 6 trials. The mean number of correct scores on each trial is shown in table 3.2.

Due to the fact that the data for errors was not normally distributed non-parametric Mann-Whitney U tests were performed. The results showed that the healthy controls made significantly more errors than the patients with schizophrenia on trial 6 of the congruent series, $Z=-2.66$, $p=.0079$.

On the incongruent trials, only trial one showed a significant difference between the groups, $Z=-3.05$, $p=.0023$, with the patients with schizophrenia producing more errors than the healthy controls.

<table>
<thead>
<tr>
<th>TRIAL</th>
<th>Schizophrenia</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean No. of</td>
<td>Std Dev</td>
</tr>
<tr>
<td></td>
<td>Correct</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Responses</td>
<td></td>
</tr>
<tr>
<td>Congruent 1</td>
<td>36</td>
<td>0</td>
</tr>
<tr>
<td>Congruent 2</td>
<td>36</td>
<td>0</td>
</tr>
<tr>
<td>Congruent 3</td>
<td>36</td>
<td>0</td>
</tr>
<tr>
<td>Congruent 4</td>
<td>36</td>
<td>0</td>
</tr>
<tr>
<td>Congruent 5</td>
<td>36</td>
<td>0</td>
</tr>
<tr>
<td>Congruent 6</td>
<td>36</td>
<td>0</td>
</tr>
<tr>
<td>Incongruent 1</td>
<td>33.81</td>
<td>2.0</td>
</tr>
<tr>
<td>Incongruent 2</td>
<td>35.56</td>
<td>.89</td>
</tr>
<tr>
<td>Incongruent 3</td>
<td>35.56</td>
<td>.89</td>
</tr>
<tr>
<td>Incongruent 4</td>
<td>35.75</td>
<td>.77</td>
</tr>
<tr>
<td>Incongruent 5</td>
<td>35.87</td>
<td>.34</td>
</tr>
<tr>
<td>Incongruent 6</td>
<td>35.81</td>
<td>.54</td>
</tr>
</tbody>
</table>

*Table 3.2. Mean number of correct responses made by the two groups*
### 3.3 Reaction Time Data

The data for reaction time is normally distributed and automatically checked for equality of variance (Levene’s Test).

Reaction time data was automatically recorded by the computer as the Stroop task was presented. For each trial there were 36 responses and these were averaged by the computer so that there was only one reaction time score per subject on each of the 12 trials.

Mean scores for reaction time on each trial are shown in table 3.3. They are represented graphically in Graph 1.

<table>
<thead>
<tr>
<th>TRIAL</th>
<th>Patients with Schizophrenia</th>
<th>Healthy Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (msec)</td>
<td>Std. Deviation</td>
</tr>
<tr>
<td>Congruent 1</td>
<td>609.92</td>
<td>117.3</td>
</tr>
<tr>
<td>Congruent 2</td>
<td>621.99</td>
<td>102.65</td>
</tr>
<tr>
<td>Congruent 3</td>
<td>624.79</td>
<td>116.27</td>
</tr>
<tr>
<td>Congruent 4</td>
<td>587.07</td>
<td>98.43</td>
</tr>
<tr>
<td>Congruent 5</td>
<td>605.92</td>
<td>115.01</td>
</tr>
<tr>
<td>Congruent 6</td>
<td>613.3</td>
<td>102.61</td>
</tr>
<tr>
<td>Incongruent 1</td>
<td>757.35</td>
<td>178.4</td>
</tr>
<tr>
<td>Incongruent 2</td>
<td>777.05</td>
<td>183.56</td>
</tr>
<tr>
<td>Incongruent 3</td>
<td>780.89</td>
<td>98.83</td>
</tr>
<tr>
<td>Incongruent 4</td>
<td>784.88</td>
<td>103.99</td>
</tr>
<tr>
<td>Incongruent 5</td>
<td>804.56</td>
<td>100.89</td>
</tr>
<tr>
<td>Incongruent 6</td>
<td>772.89</td>
<td>111.31</td>
</tr>
</tbody>
</table>

Table 3.3. Mean reaction times on congruent and incongruent trials for the two groups
3.3.1 Repeated Measures ANOVA

Main Effects

A repeated measures ANOVA was performed with three levels: one between subjects variable and two within subjects variables. The former was group which had two levels, healthy and schizophrenic. The latter were trial, which had six levels and condition, which had two levels, incongruent and congruent.

The ANOVA showed that there was a main effect of Group, $F(1,30)=6.38$, $p=.017$, showing that there was a significant difference between the reaction time scores of the healthy controls and the patients with schizophrenia. The mean scores show that the healthy subjects were significantly faster overall than the patients with schizophrenia.

The ANOVA revealed a significant effect of trial, $F(5,150)=4.39$, $p=.001$, such that the speed of reaction time increased over the six trials.

There was also a main effect of condition, $F(1,30)=61.85$, $p<.0001$, such that reaction time for the congruent condition was faster than for the incongruent condition.

3.4 Interaction Effects

There was one significant interaction effect: The ANOVA showed a significant interaction of group by trial, $F(5,150)=6.31$, $p<.0001$. In order to investigate the nature of this effect further, a mean score for the first three trials and the last three trials was calculated for each subject in each condition. The mean scores are shown in table 3.4. These scores were then compared using t-tests.
A paired samples t-test showed that, for the control subjects on the congruent trials the first three trials were significantly slower than the last three, \( t(15)=3.07, p=.008 \). However, for the patients with schizophrenia there were no differences between the scores for the first three trials and the latter three, \( t(15)=1.21, \) ns. The same was true for the incongruent trials, the healthy controls speeded up their performance over the 6 trials, with a significant decrease in reaction time between the first three and the last three trials, \( t(15)=7.45, p<.0001 \). The patients with schizophrenia showed no difference in reaction time scores between the two sets of trials, \( t(15)=-.74, \) ns.

Further analysis of between groups differences across trials using independent samples t-tests showed that there was no difference in speed of responding between the two groups on the first three congruent trials \( t(30)=-1.44, \) NS. However, on the second three congruent trials, there was a significant difference between the two groups, \( t(30)=-2.44, p=.021 \), with the patients with schizophrenia being slower than the normal controls.

This discrepancy was also evident on the incongruent trials. There was again no difference between the two groups in the first three trials, \( t(30)=-.62, \) ns; but on the last three incongruent trials, significant differences emerged between the two groups, \( t(30)=-3.06, p=.005 \).
3.5 Symptom Rating Data

Data from the Manchester Scale was compiled such that each patient was given three scores relating to reality distortion (comprising scores for delusions and hallucinations), disorganisation (scores for incoherence of speech and incongruity of affect) and a score for Psychomotor poverty (scores for blunting of affect and poverty of speech). These syndrome scores were derived according to Liddle (1987a). A correlation was then performed between reaction time scores on each of the trials and symptom rating scores. There were no significant correlations between any of the symptom ratings and reaction time scores on either congruent or incongruent trials.

3.6 Analysis of Individual Subject Data

Due to the small number of subjects participating in the study, an analysis of individual results was considered important in order to establish whether only a selected number of the patients with schizophrenia failed to become more efficient at the task over repeated presentations and, if so, whether there were any differences in terms of medication type or symptomatology between these patients and those that behaved in a similar way to the normals.

For each of the subjects a ‘difference’ score was calculated by subtracting the mean reaction time for the last three trials from the mean reaction time for the first three trials for both congruent and incongruent conditions. Thus, in those subjects who had become more efficient at the task this number would be greater than zero while for those subjects who showed no practice effect, the number would be zero or less. The results are shown in table 3.5.
Due to the small sample sizes, non-parametric Mann-Witney U tests were performed on the data. Surprisingly, in the congruent condition more of the healthy subjects fail to show learning than do the patients with schizophrenia (7 normals vs. 3 patients with schizophrenia). The mean speed of responding for the normals who learned and those who failed to do so was not significantly different (Z=-.1588, NS). However, it is noteworthy that the extent of the improvement in those subjects who did speed up over trials exceeded that of the patients with schizophrenia, that is, the difference scores were significantly greater (Z=-2.44, p=.015). In addition, for the three patients

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Congruent - Difference Score</th>
<th>Incongruent - Difference Score</th>
<th>Medication Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sx</td>
<td>Normal</td>
<td>Sx</td>
<td>Normal</td>
</tr>
<tr>
<td>1</td>
<td>-94.36</td>
<td>-46.88</td>
<td>71.67</td>
</tr>
<tr>
<td>2</td>
<td>-93.64</td>
<td>-27.26</td>
<td>71.67</td>
</tr>
<tr>
<td>3</td>
<td>-43.04</td>
<td>35.39</td>
<td>73.4</td>
</tr>
<tr>
<td>4</td>
<td>1.7</td>
<td>23.25</td>
<td>73.4</td>
</tr>
<tr>
<td>5</td>
<td>8.76</td>
<td>26.26</td>
<td>29.33</td>
</tr>
<tr>
<td>6</td>
<td>8.76</td>
<td>26.26</td>
<td>29.33</td>
</tr>
<tr>
<td>7</td>
<td>14.33</td>
<td>-57.41</td>
<td>29.33</td>
</tr>
<tr>
<td>8</td>
<td>16</td>
<td>76.69</td>
<td>6.03</td>
</tr>
<tr>
<td>9</td>
<td>19.32</td>
<td>61.34</td>
<td>-8.17</td>
</tr>
<tr>
<td>10</td>
<td>30.82</td>
<td>-10.16</td>
<td>59.17</td>
</tr>
<tr>
<td>11</td>
<td>32.89</td>
<td>26.3</td>
<td>40.17</td>
</tr>
<tr>
<td>12</td>
<td>59.98</td>
<td>-119.02</td>
<td>40.17</td>
</tr>
<tr>
<td>13</td>
<td>59.98</td>
<td>-119.02</td>
<td>60.87</td>
</tr>
<tr>
<td>14</td>
<td>63.10</td>
<td>-241.21</td>
<td>79.87</td>
</tr>
<tr>
<td>15</td>
<td>92.13</td>
<td>47.24</td>
<td>53.8</td>
</tr>
<tr>
<td>16</td>
<td>92.13</td>
<td>47.24</td>
<td>53.8</td>
</tr>
</tbody>
</table>

Table 3.5 - Individual subjects Difference scores and medications for patients with schizophrenia
with schizophrenia who failed to display an improvement over time, the decrement in their performance was greater than that of the normals ($Z=-2.77, p=.005$). The mean difference scores for learners, that is, those who improved with practice and non-learners, those subjects’ whose performance showed no improvement or worsened after practice, are shown in table 3.6. Number of subjects in each group is shown in brackets.

<table>
<thead>
<tr>
<th></th>
<th>Congruent</th>
<th>Incongruent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Learners</td>
<td>Non-learners</td>
</tr>
<tr>
<td>Normals</td>
<td>86.72 (9)</td>
<td>-11.16 (7)</td>
</tr>
<tr>
<td>Schizophrenics</td>
<td>38.45 (13)</td>
<td>-77.01 (3)</td>
</tr>
</tbody>
</table>

Table 3.6 - Mean difference scores for learners (those who improved with practice) and non-learners (those who failed to improve) for normal and schizophrenic subjects

In the congruent condition only one healthy control failed to improve the speed of their performance with practice. By contrast, seven patients with schizophrenia showed either no increase in speed with practice or an actual worsening of performance over trials. While two of these patients also failed to improve on the congruent trials, the other five were capable of increasing the speed of their performance with practice on the congruent trials but not on the incongruent trials.

In contrast to the congruent condition, there was no significant difference between the patients with schizophrenia and healthy controls in terms of the extent of the improvement in those subjects who were classified as ‘learners’ ($Z=-1.40, p=.16$, NS).

The next stage of the investigation, therefore was to examine possible explanations for the finding that seven of the patients were unable to improve with practice while the other nine were able to learn to the same extent as the normals on the incongruent trials. A number of Spearman’s rank correlations were performed looking at
difference scores for incongruent trials and age, NART IQ, age of onset of the illness, and symptomatology.

<table>
<thead>
<tr>
<th></th>
<th>Age of onset</th>
<th>Age</th>
<th>Reality Distortion</th>
<th>Poverty</th>
<th>Disorganisation</th>
<th>NART IQ</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Congruent Trials</strong></td>
<td>p = -.05</td>
<td>p = .27</td>
<td>p = -.04</td>
<td>p = -.07</td>
<td>p = -.04</td>
<td>p = -.01</td>
</tr>
<tr>
<td>Difference Scores</td>
<td>p = .98</td>
<td>p = .13</td>
<td>p = .87</td>
<td>p = .12</td>
<td>p = .99</td>
<td>p = .95</td>
</tr>
<tr>
<td><strong>Incongruent Trials</strong></td>
<td>p = -.005</td>
<td>p = .15</td>
<td>p = -.44</td>
<td>p = -.46</td>
<td>p = -.19</td>
<td>p = -.13</td>
</tr>
</tbody>
</table>

Table 3.7 - Spearman's rank correlations between difference scores and other variables for patients with schizophrenia.

The results shown in Table 3.7 indicate that there were no significant correlations between difference scores and any of the variables selected. There was no correlation between difference scores for congruent and incongruent trials. There was, however, a trend for symptoms of reality distortion, that is, hallucinations and delusions, and the symptoms of psychomotor poverty syndrome (that is, blunting of affect and poverty of speech) to be negatively correlated with difference scores on the incongruent trials. That is, the higher the symptom rating the lower the difference score. Due to the small number of subjects involved in the analysis it is difficult to ascertain the significance of this result.

In terms of medication, a 2x2 Chi-square was performed between learners and non-learners and patients on atypical versus typical antipsychotic medications. Atypical medications were classified as: Clozapine, Seroquel, Risperidone and Remoxipride. Dr. C. Pantelis a consultant psychiatrist at Royal Park Hospital provided this
information. The results showed a trend for patients on atypical medications to be non-learners (likelihood ratio Chi square = 3.06, p=.08). No statistics relating to quantity of medication were possible as chlorpromazine equivalents were not provided for the present investigation. However, due to the small subject numbers it is difficult to draw any clear conclusions about the possible effects of medication on performance.

3.7 Congruent versus Incongruent Learning

Previous studies have shown some disagreement as to whether there is greater improvement in normal subjects on incongruent as opposed to congruent trials. In order to address this question a linear regression was conducted for each of the control subjects to determine the slope of their performance across the 6 congruent and the 6 incongruent trials. The slope scores were then compared using a paired samples t-test. The results showed that the improvement was significantly greater for incongruent than congruent trials t(15)=2.68, p=.017. This result is consistent with the results shown above that reveal that several of the normal subjects failed to learn at all on the congruent trials.
SECTION 4 - DISCUSSION

The results of the study support the first alternative hypothesis that patients with schizophrenia do not show the same practice effects as normal controls on the repeated presentation Stroop test. However, more surprisingly given the existing literature, the second hypothesis was not supported; there were no differences in the degree of Stroop interference shown in the incongruent condition between normal and schizophrenic subjects. The results are discussed in the following section in the context of the literature presented in the introduction.

4.1 Discussion of Results

The ANOVA showed that there were three main effects present: that of condition, group and trial. The first of these showed that there was a Stroop effect present, that is, the subjects took longer to respond when the words and colours were incongruent than when they were the same. This result was expected in view of the large body of literature, cited earlier, that attests to the robust nature of the Stroop effect since its discovery in 1935. The graph of the results shows that the Stroop effect was present in both groups of subjects cross all six trials.

The second main effect was that of trial. The speed of responding of the subjects altered significantly across the six trials.

A further main effect found was that of group. There were significant differences between the speed of responding of the normal subjects and the patients with schizophrenia. The graph shows that this was true for only the last five trials, while on the first trial there was no difference in the speed of responding between the two groups on either the congruent or incongruent condition.
4.1.1 Data relating to the First Hypothesis

The nature of the differences found in the main effects may be elucidated by looking at the interaction effects shown by the ANOVA. The interaction effect that was found in the study supports the first alternative hypothesis. A group by trial interaction was found indicating that while healthy controls decreased their reaction time in both congruent and incongruent conditions over the 6 trials, the patients with schizophrenia showed no evidence of increased speed of responding in either condition over time. This result would indicate that the patients with schizophrenia were unable to benefit from practice and use their prior learning experience to increase task competency. Previously, only Carter et al. (1992) have administered a repeated Stroop task in the past to patients with schizophrenia and they did not report any evidence of learning in either normal controls or patients with schizophrenia.

While the effect in the present experiment is fairly robust, analysis of individual performance shows that there was considerable variation in scores. In the congruent condition, more normal subjects failed to show a learning effect than did patients with schizophrenia. However, the magnitude of this effect was slight, that is, they showed only minor slowing over time with the maximum non-learner difference score found being only -23.67 ms. One explanation for this finding is that these subjects showed a ceiling effect in that they reached a maximum speed of responding early in the test. However, this is contra-indicated by the fact that those who did learn were not slower overall in their performance than the non-learners. Alternatively, the minor slowing over time could be due to fatigue although it would be expected that this would be evident across both congruent and incongruent conditions.

By contrast, only two patients with schizophrenia failed to improve their performance over time in the congruent condition. However, their difference scores were significantly greater than those of the normal controls who failed to learn (maximum difference score being -94.36 as compared to -23.67 for the normals). Further, those
subjects who did show practice effects on the congruent trials had significantly lower difference scores than the non-schizophrenic controls. Thus, even though the majority of the patients showed no decrement in performance over time on the congruent condition, there was a general trend of impaired learning as compared to controls.

The data for the incongruent trials shows that only one of the normal controls failed to improve with practice. This was a relatively small effect (difference score, -8.17). However, the data for schizophrenic subjects shows that the performance of seven patients worsened over trials, that is they got slower over consecutive presentations of Stroop stimuli. Interestingly, the learning shown by the other nine patients was equivalent in magnitude to that of normal controls.

In terms of the literature relating to practice effects on the Stroop test in normals, there has been some controversy regarding whether improvements are greater on the incongruent than the congruent trials. Analysis of the slope of learning for each of the control subjects showed that they displayed significantly greater learning over the six trials of the incongruent rather than the congruent condition. This would be consistent with the findings of Jensen (1967).

The findings relating to performance of some patients with schizophrenia are consistent with the Gray et al model presented in the introduction. Patients showed retarded learning in the congruent condition and nearly half of the subjects showed a decrement in performance over time in the incongruent condition. There are a number of potential explanations for this finding. / Subjects who failed to improve with practice were unable to use their previous exposure to the test to aid performance. Aspects such as the test situation and predictability of the order of presentation may not have been integrated into task performance such that each trial was, in effect, entirely novel. The concept of overattention is relevant in that the patients may have been selectively attending to irrelevant aspects of the environment which prevented improvements in performance. However, the fact that speed of performance actually deteriorated does not entirely fit with these explanations. One way of incorporating
this fact is to propose that by not attending to relevant aspects of the task patients pay more and more attention to irrelevant information which slows their performance.

2/ Some patients show a failure to habituate, that is, they show no habituation to the stimuli and as above, treat each presentation as if it is novel. However, as Frith (1991) comments, failure to habituate may be related to a number of different factors which are difficult to disentangle.

3/ The attentional demands of the test are particularly tiring for some patients and as such, their performance becomes slower over time. Contrary to this, however; is that they do not make more errors over trials. It is possible that, due to the high number of errors made on the first trial some patients use a speed accuracy trade-off approach in order to maintain error free performance.

4/ Some patients show slower learning than others and as such require more than six trials to improve their performance. This is unlikely, however, as performance was deteriorating rather than slowly improving across trials.

The symptom rating and medication data potentially explain the differences in performance within the schizophrenic group. Those subjects who failed to show practice effects showed a trend towards having higher levels of positive symptoms, that is, hallucinations and delusions and higher levels of poverty symptoms. This would be inconsistent with the results of Baruch et al who found that only patients classified as acute failed to show latent inhibition whereas in the present investigation both positive and negative symptomatology was associated with poor learning. This discrepancy may be related to difficulties with definition between the studies. In the Baruch et al study patients were classified as acute or chronic according to diagnostic criteria whereas in this study ratings of individual symptoms were taken. The only types of symptoms that were not related to performance were those of disorganisation, that is, incoherence of speech and incongruity of affect. It is evident from looking at the symptom rating data that poor performance was not related to symptom severity.
per se as the three subjects who scored highly on disorganisation measures were able to learn effectively. It is important to note, however, that these results show merely a trend and that the effect would need to be replicated with much larger subject numbers.

In terms of medication, subjects on atypical neuroleptics showed a trend to be non-learners. It is likely that this is related to symptom ratings as patients with persistent severe symptoms are more likely to be on atypical medications. Again larger subject numbers are required in order to establish whether this is a significant finding.

In summary, the results are quite complex when examined at an individual subject level. Some normal subjects fail to improve on the congruent condition while patients with schizophrenia show some retarded learning and two with a significant decrement in performance. The failure of normal subjects to improve is unexpected and difficult to explain.

On the incongruent trials, all but one normal subject improved their performance over trials. However, seven of the patients with schizophrenia show a decrement in performance over trials. This may be related to the Hemsley hypothesis which suggests that there is a reduced influence of previous experience on current perception in patients with schizophrenia. If this were the case then patients fail to selectively attend to relevant aspects of the environment and continue to attend to irrelevant material over time rather than inhibit this from conscious awareness.

What is interesting is that those patients who are able to improve with practice are able to do so at an equivalent rate to normal controls. The differences in performance between patients may be explicable in terms of symptom differences although only statistical trends were found.
4.1.2 Data relating to the Second Hypothesis

Interestingly there was no group by condition interaction which supports the second null hypothesis in the present investigation, that is, that the difference in reaction time between congruent and incongruent conditions will be greater for the patients with schizophrenia than for normal controls. Studies in the literature have suggested that patients with schizophrenia perform more poorly than normal subjects on the Stroop task due to a failure to inhibit inappropriate responding. This is not evident in the data and is illustrated in graph 1 which clearly shows that the magnitude of the Stroop effect is identical for both normals and patients.

However, some support was found for the notion that the patients with schizophrenia had more difficulty in undertaking the incongruent task was derived from the error data. The results from the analysis of error scores shows that the patients with schizophrenia made more errors on the first of the incongruent presentations than the normal controls. This would suggest that they had greater difficulty in grasping the demands of the task and in the initial process of inhibition.

The failure to find an increased reaction time for the incongruent condition in patients with schizophrenia was also found in the study by Carter et al (1993). In their study the authors found that the magnitude of the Stroop effect was related to symptom profiles with patients who have predominantly negative symptomatology showing increased Stroop interference while those with positive symptomatology displayed increased facilitation with no increase in the Stroop effect.

It is difficult to compare symptom rating scores between the present study and the Carter et al studies as the subjects in this investigation were not simply classified as being acute or chronic. However, there was no correlation between any of the symptom rating scores and patient performance on either congruent or incongruent trials.
A further issue that needs to be addressed in relation to these findings is that of IQ. Due to the high IQ of the healthy controls it was necessary to find patients with schizophrenia whose IQ scores were relatively preserved. As has been reported in the literature (Barber et al, 1996) patients with schizophrenia, particularly those with chronic symptoms of the disease, generally have a current IQ score 10 to 15 points below their predicted premorbid levels. In most studies matching is based on NART IQ scores which provide a predicted premorbid score rather than a measure of current functioning. While the NART score of the patients with schizophrenia in the present investigation was in the average range and similar to scores found in previous experiments (for example, Joyce et al had an average NART IQ of 107 for the schizophrenics), there is no indication of the extent of deterioration and the present sample may have been particularly well preserved in terms of their present functioning.

One factor that may also account for the discrepant results between this and other studies is the style of Stroop paradigm employed. Since the creation of the first Stroop paradigm in 1935 there have been a large number of variations on the task, for example, in the Joyce et al study (1996) the Stroop was administered as the number of colours that could be read, then named in 1 minute. By contrast, other authors have used a conventional Stroop with control conditions of coloured crosses and colour words written in black and white. Many experiments have not been specific about the type of paradigm used and these differences may account for the decrement findings of the present study. However, all the paradigms used are fundamentally similar to the present experiment in that they involve congruent followed by incongruent conditions, sometimes interspersed with control tasks, and it is unlikely that the minor differences could account for the failure to find an increased interference effect in this study.
4.2 Methodological Considerations

4.2.1 Experimental Design

The present study was designed in order to replicate the findings of Pardo et al. (1991) and therefore used an ABAB... series of presentations. However, the use of this design precludes getting an accurate measure of facilitation and interference as there is no neutral condition. The interference effect is measured as the difference between congruent and incongruent conditions rather than the difference between neutral and incongruent. Thus, facilitation and interference are subsumed into one effect.

The previous studies by Carter et al (1992, 1993) would indicate that patients with schizophrenia show a marked facilitation effect on the congruent condition as compared to normal controls. It would therefore have been interesting to have examined this effect in the present study. However, the latter Carter et al study found an increased facilitation effect only in patients with undifferentiated symptomatology (that is, more positive symptoms such as hallucinations and disorganisation) while those with paranoid symptomatology displayed greater interference effects than the controls.

In the present study it is unlikely that the patients are displaying a significant facilitation effect. If this was the case, the measured difference in reaction time between congruent and incongruent conditions would be partly due to facilitation for the congruent words and partly due to the interference effect. As such, it would be expected to be greater for the patients with schizophrenia than for the normal controls which is not the case. There is no significant difference in the congruent-incongruent reaction time difference between the two groups. In addition, the patients with schizophrenia do not show faster reaction times on the congruent condition than the normal controls.
While it would be very interesting to examine facilitation effects in patients with schizophrenia the confines of the present study precluded their measurement. As the present study was designed to examine the behaviour of patients with schizophrenia on a Stroop test prior to their being used in an activation study it was imperative to use the same experimental design that had been used for the normal controls. This design had been found to produce activation patterns consistent with current understanding of brain function while a study using a counterbalanced control condition (Bench et al, 1993) had found rather surprising and inconsistent results.

4.2.2 Subject Numbers

A further methodological issue is the small number of subjects who were tested in the present experiment. Due to the failure to find increased interference effects in the present sample of patients with schizophrenia a power analysis was conducted using the results of the patients with paranoid symptomatology from Carter et al 1993. The results showed that a sample size of 32 in each groups would be required a difference in means of -147.3 (the difference between a group 1 (normal) mean of 226.9 and a group 2 (schizophrenic) mean of 374.2) assuming that the common standard deviation is 206.38 using a two-group t-test with a 0.05 significance level. Thus, the present investigation only has 50% power using these statistics. However, the estimate given in the power analysis is likely to be an overestimate as the standard deviation of the differences had to be based on using the standard deviations for raw without subtracting the covariance which may have significantly increased the standard deviation scores used and thus the desired number of subjects. In addition, the statistics used from the Carter paper are for paranoid schizophrenics which is not the diagnosis used in the present study. Further, the Carter et al study used only 25 subjects overall, meaning that there were significantly less subjects in each group than in the present investigation. Overall, it would appear that no firm conclusions can be drawn from the present data relating to the absence of an increased Stroop effect in patients with schizophrenia as the power analysis indicates that the small number of subjects would give only a 50% chance of finding the effect.
4.2.3 Test Administration

One issue that was evident during the first administration of the Stroop paradigm was the difficulty in using the voice key to measure reaction time. In the present study, the presence of the stimuli was cued by the voice key being activated. However, the monitor reacted regardless of what was said so if a subject produced the wrong response and then said 'whoops' or a further comment, the next stimuli would appear and disappear in response to the verbalisation rather than because the subject had responded appropriately. Thus, both the reaction time and error scores would be affected. Data for the first subject was discarded because of this difficulty. In order to avoid this occurring again, subjects were requested not to produce any comments other than those which were a direct response to the stimuli. This strategy was successful and no further incidents occurred.

4.3 Medication and Hospitalisation

In all studies attempting to look at neuropsychological performance in patients with schizophrenia there is the issue of medication and length of hospitalisation. These issues have been discussed in some detail previously, (King, 1990) but the general consensus of research would suggest that medication tends to have a beneficial effect on neuropsychological test performance, particularly attentional tests. King and Green (1996) reviewed the results from a large number of studies and drew the following conclusions: First that neuroleptic medication does not account for the cognitive deficits found in schizophrenia. Second, tests which depend on attention, have been found in general to improve with neuroleptic treatment independent of clinical response, that is, improvement in symptomatology. However, the explanations for this remain varied and include the possibility that they improve motivation and lessen the distraction from hallucinations. Alternatively, medication may have a direct effect on information processing and improve filtering or set shifting by reducing dopamine release in the limbic system.
Due to the fact that many patients in the present study were on Clozapine it is important to address whether this, atypical neuroleptic has different effects to more traditional forms of treatment. King and Green review four studies which have looked at cognitive performance following Clozapine treatment and found contradictory results with two studies reporting improvements in cognition and two studies showing only minor gains. However, the authors conclude that those studies which found improvements following Clozapine treatment were more methodologically sound and would therefore indicate some gains in general cognitive functioning. There was no evidence to suggest specific gains in attention based tasks.

In terms of the effects of institutionalisation, the results are confounded by the fact that only the most severe and chronic patients are placed in permanent hospital care and will therefore most likely perform more poorly on neuropsychological tests for this reason. It is very difficult to separate out these effects. In the present sample, it is unlikely that institutionalisation was a factor affecting the results as the patients with schizophrenia had not been in hospital for long periods of time. In addition, the rehabilitation at the hospital aimed to maximise community involvement and to foster independent living to as great an extent as possible.

4.4 Implications for Further Research

The present study highlights two major areas for further research; first, the failure of patients with schizophrenia to improve the efficiency of performance with practise and second, the variables which influence the Stroop effect in patients with schizophrenia.

In terms of the effects of practise, the present study provides some evidence to support Hemsley’s hypothesis that patients with schizophrenia have a reduced influence of previous regularities on current perception. However, practise effects are quite different from other experimental evidence which Hemsley uses such as latent
inhibition and Kamin blocking. It is of interest to further examine the performance of patients on other repeated presentation experiments to investigate whether the effect can be replicated, for example in other reaction time experiments where normal subjects' can improve their performance. It may be possible to use this data to address some of the criticisms of the model, particularly those which suggest that it does not provide enough delineation of all the component cognitive processes occurring when patients fail to show learning effects.

Other explanations for the failure to show practice effects are presented earlier in the discussion. These include failure to habituate to novel situations, cognitive fatigue and slowed learning. These hypotheses need to be examined in the future. The last of these could be examined by using more trials in order to investigate whether patients' performance eventually reaches the same level as normals.

The issues regarding individual differences in performance are also important. The work by Gray et al would indicate that only patients with acute schizophrenia show deficits in selective attention that lead to learning difficulties. However, in the present investigation subjects with both positive and negative symptoms were more likely to be non-learners. This effect needs to be replicated in a larger sample.

Failure to show improvements in efficiency following practice have implications for rehabilitation of patients with schizophrenia. If this result represents a general failure to generalise learning or even to acquire associations in the first instance then some patients with schizophrenia may require more specialised rehabilitation programmes which take into account these deficits.

A further implication of the present research is for functional imaging studies. Many of these studies require repeated presentations of stimuli in order to acquire enough data for statistical purposes. More research is being undertaken using patient populations and thus these practice effects need to be taken into account when interpreting activation data.
The issue of the failure to show Stroop interference effects also needs further investigation. The present study showed that this is unlikely to be linked to symptom factors although other studies (Carter et al) have found this to be the case. Again, larger subject numbers are required to assess this in more detail. In addition, the possibility of the effect being linked to IQ or experimental design needs to be assessed.

4.6 Conclusions

In summary, the aims of the present study were to investigate whether patients with schizophrenia showed deficits of selective attention on a repeated presentation Stroop paradigm. It was hypothesised that deficits would result in 1/ Reduced effects of practice and 2/ increased Stroop interference. The first hypothesis was supported while the second was surprisingly not.

Given the body of evidence inferring frontal lobe dysfunction in these patients and a large number of previous studies which have found impaired inhibition and attention in patients with schizophrenia, the results are incongruous. Proposed explanations of the findings suggest that IQ, symptoms and experimental design may potentially explain this anomaly.

The finding that some patients did not improve with practice and in fact showed a decrement in performance over trials may be related to Hemsley's hypothesis which suggests that patients with schizophrenia have reduced influence of previous experience on current perception. As such, each presentation of the stimuli is effectively novel. Other explanations include cognitive fatigue, reduced speed of learning and failure of habituation.

The significant differences within the schizophrenic population which were found may be related to differences in symptomatology and symptom severity. In addition, medication type may be a factor influencing performance.
SECTION 5 - REFERENCES


Bleuler, E. (1913) *Dementia Praecox or the Group of Schizophrenias.* New York: International Universities Press


Cattell, J.M. (1886) The time it takes to see and name objects. *Mind, 11,* 63-65


Everett, J., Laplante, L. & Thomas, J. (1989) The selective attention deficit in schizophrenia: Limited resources or cognitive fatigue? *Journal of Nervous and Mental Disease*, 177, 735-738


Kolb, B., & Whishaw, I.Q. (1983) Performance of schizophrenic patients on tests sensitive to left or right frontal, temporal or parietal functioning in neurological patients. *Journal of Nervous and Mental Disease*, **171**, 435-443


Logan, G.D. & Zbrodoff, N.J. (1979) When it helps to be misled: Facilitative effects of increasing the frequency of conflicting stimuli in a Stroop-like task. *Memory and Cognition, 7*, 166-174


emission tomographic studies of the cortical anatomy of single word processing. *Nature*, **331**, 585-589.


