Memory in depression

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MEMORY IN DEPRESSION.

ABSTRACT.

The memory of clinically depressed psychiatric patients was compared with that of anxious patients and control subjects. The depressed patients had impaired ability to learn new material and to remember past public events; they retained information in memory as well as controls and did not have a more conservative response bias. These impairments were not attributable to the psychotropic medication the patients were receiving or to the after-effects of ECT. The retarded depressed patients were most severely ill and most impaired; the neurotic patients were only impaired on the more difficult tests. The anxious patients' scores were not significantly different from those of either the depressed or control subjects. The relative effects of depression and anxiety on performance were assessed using regression analysis; depression was related to performance on the easier tests, whilst something common to both depression and anxiety was related to performance on the more difficult tests. The retarded depressed subjects reported more cognitive failures than the other subjects whilst both the depressed and anxious subjects complained of significant deterioration in memory. There were statistically significant, although modest, correlations between these self-assessments of memory and performance on the memory tests. Anxiety was related to self-assessments of memory but depression was not. The memory of depressed general practice patients for information given to them by their general practitioners was investigated directly; they did not in fact have impaired memories in this everyday situation.

These results suggest that the degree of memory impairment shown in depression depends both on the severity of depression and the difficulty of the task. They are discussed in the light of the suggestion by Johnson and Magaro (1987) that memory impairments may not be specific to depression but instead be related to the overall level of psychopathology. The working memory capacity model of memory in anxiety (Eysenck, 1982) is also discussed and extended to depression, as is a model developed by Williams and Teasdale (1982) which argues that effort expenditure is largely determined by perceived task difficulty. Finally, it is concluded that the best understanding of memory in depression will come from the concurrent use of experimental studies, metamemory questionnaires and studies of memory performance in everyday life.

Memory in Depression

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None of the material offered in this thesis has been previously submitted for a degree in this or any other University.

MEMORY IN DEPRESSION: A REVIEW

1.1 INTRODUCTION

Memory problems are a classic symptom of clinical depression. McAllister (1981) notes that clinicians have been taught for years that severely depressed patients will complain of, and often have, impairments in memory. Alterations in memory and concentration are included as diagnostic symptoms for Major Depression in both DSM-III (American Psychiatric Association, 1980) and the Research Diagnostic Criteria (RDC; Spitzer, Endicott and Robins, 1978). Despite the acceptance of memory problems as a symptom of depression, there are many unanswered questions: what groups of depressed people will have such problems, what types of memory are affected and what causes the impairment. Memory changes in clinical depression are therefore the focus of this thesis.

Three aspects of memory in depression are investigated: the performance of clinically depressed people on laboratory memory tests; their reports of memory problems in everyday life and the relationship of these reports to performance on the laboratory tests; and finally, their memory performance in an important everyday situation - the general practice consultation.

This chapter has two components. The first explores what is meant by 'clinical depression' and reviews classifications of depression. The second reviews the literature on memory in depression. The literature review, like the thesis, is concerned solely with memory in depression, and not with other aspects of cognition such as intellectual performance and decision making which may also be impaired in depression (see Miller (1975); Willner (1984) for reviews of cognitive functioning in depression).

1.2 CLINICAL DEPRESSION

This thesis is concerned with clinical depression, not with the mild lowering of mood and sadness which most people experience in their daily lives, usually in response to loss or disappointment.

The term 'clinical depression' is frequently used, but it is not immediately clear what it means. It can refer to the type and severity of depression usually seen by psychiatrists, without implying any theoretical view of the relationship between this and normal mood disturbances. It is clear from epidemiological studies of depression that the term is frequently used in this sense: reliable ways are sought of identifying groups of depressed people who have levels of symptoms similar to those in depressed psychiatric patients and therefore qualify as 'cases' of clinical depression (Wing et al, 1978; Brown and Harris, 1978).

Clinical depression can, however, be taken to mean an illness which is qualitatively different from low mood or sadness as normally experienced. Snaith (1987) takes this view and therefore stresses the importance of finding ways of reliably differentiating between clinical depression, which is presumed to have biological causes and to respond to medical treatment, and normal despondency in response to adverse events. He believes that many of the 'cases' of clinical depression identifed in community samples do not qualify for the term and instead are merely despondent or unhappy.

There are also examples of the term being restricted to those being treated by psychiatrists. For instance Weissman, Prusoff and Pincus (1975) found that 'unhappy' unemployed women had levels of depression only slightly lower than those in women being treated by the psychiatrist, and indeed had more severe depressed mood; but because they were not having psychiatric help they were not considered by the authors to be clinically depressed.

In this thesis clinical depression refers to symptoms of depression which are as severe as those experienced by depressed patients under the care of psychiatrists. This does not necessarily imply that the subject is being treated by a psychiatrist: in Chapter Eight, for instance, the subjects were general practice patients with levels of depression comparable to those seen in psychiatric patients. Whether clinical depression is different in kind from depressed mood and sadness will be discussed below.

1.2.1 The Symptoms of Clinical Depression

Depressed or dysphoric mood is usually taken to be the central feature of clinical depression, although it has been recognised that some people do not complain of this, but instead have a pervasive lack of interest or pleasure (Malt, 1983; American Psychiatric Association, 1980). Other symptoms included in the criteria for depression listed in DSM-III (American Psychiatric Association, 1980) are feelings of worthlessness, self reproach or excessive or inappropriate guilt; poor concentration; poor appetite or weight loss; insomnia or hypersomnia; loss of libido; psychomotor retardation or agitation; loss of energy or fatigue; and recurrent thoughts of death, suicidal ideation or suicide attempts. Some depressed people have hypochondriacal preoccupations and depressive delusions (Hamilton, 1982). According to Andreasen (1982) there have been repeated descriptions of a syndrome with these symptoms since ancient times; the first comprehensive description of it in English was published in 1586 (Bright, 1586).

1.2.2 The Relationship of Clinical Depression to Normal Depressed Mood

It is not clear where the boundary between clinical depression and normal depressed mood lies, or indeed whether there is one. On the one side of the supposed boundary is depressed mood which, as Bebbington (1987) has noted, can be accounted for with varying degrees of plausibility in terms of social experience and which most people would regard as normal and explicable. On the other is clinical depression, which many see as qualitatively different and best conceptualised as an illness (Snaith, 1987; Kraepelin, 1921). Bebbington (1987) concluded that 'while this distinction may represent the application of considerable clinical intuition, it cannot be said to have been validated'.

Interest in how to distinguish between 'depression as an illness' and normal depressed mood is not new. For instance Griesinger (1861) stated that melancholia could be distinguished from normal emotional reaction by reason of its severity, duration, independent development and presence of other specific symptoms, and because there are no psychosocial precipitants at all. There is evidence to support some of his assertions.

There is evidence that levels of depression in patients diagnosed as having clinical depression are higher than those in depressed people who are not considered to have it. For instance, as already noted, Weissman, Prusoff and Pincus (1975) found that women treated by a psychiatrist had higher overall levels of depression than 'unhappy' women seeking career guidance, while Sturt (1981) found that psychiatric patients tended to have higher scores on the Present State Examination (PSE: Wing, Cooper and Sartorius, 1974) than depressed people identified during a community survey. There is also evidence that clinical depression does have a longer duration than most depression in non-clinical populations. Hammen (1980) interviewed students with high scores on the Beck Depression Inventory (BDI; Beck et al, 1961) two to three weeks after they had completed the questionnaires in order to see what diagnostic group they would fall into. By this time 79% of the sample were no longer moderately depressed, and 53% scored in the normal range on the BDI. Such subjects would probably not reach the criteria for clinical depression contained in DSM-III which states that the symptoms need to be present for at least two weeks. It is not surprising that patients clinically diagnosed as being depressed differed from non-diagnosed depressed people in the community in terms of severity and duration as these are two factors which determine whether people seek psychiatric help and are therefore diagnosed as clinically depressed (Dew et al, 1988).

Griesinger's notion that melancholic patients would have specific symptoms not seen in normal emotional reactions is partly substantiated. Weissman, Prusoff and Pincus (1975) found that the psychiatric patients had higher levels of somatic complaints and somatic anxiety than the 'unhappy' women. Sturt (1981) found that psychiatric patients had a higher rate of certain symptoms than depressed people identified during a community sample: these included suicidal plans or acts, guilty ideas of reference, pathological guilt and morning depression. She also found that the presence of these symptoms was associated with the presence of a high number of other symptoms; that is that they were related to the severity of the depression. In both these examples the psychiatric patients had higher levels of certain symptoms but the same symptoms were also found in the groups which were not diagnosed as having clinical depression. In addition the occurrence of these symptoms was related to the severity of the overall depression. They do not, therefore,

indicate that clinical depression is different in kind to other types of depression: the differences may solely be due to differences in severity.

It has been suggested that depressed mood in clinical depression is not the same as normal depressed mood but has a 'distinct quality', as described by Kraepelin (1921; Akiskal, 1983). According to Ramos-Brieva et al (1987) this distinct quality or pathological sadness has always been central to the diagnosis of depression in Continental Europe, but has tended to be overlooked in recent criteria for clinical depression because it is difficult to describe or measure reliably. They therefore used a semantic-differential task to identify how depressed patients with this distinct quality to their depression described their sadness and compared the results to those of parents who were sad because their children had been admitted to hospital. They then constructed a pathological sadness index which they argue can be used to differentiate between normal and pathological sadness. If they are correct in believing that depressed mood in clinical depression has a different quality to normal depressed mood then this would substantiate Griesinger's notion that there are symptoms unique to clinical depression, or melancholia.

However, the authors did not use an unselected group of patients with a diagnosis of clinical depression, but instead selected only those who were judged to have this 'distinct' quality to their depression. There do not seem to be grounds for deciding that only clinically depressed patients with this quality to their sadness are 'really' depressed and it cannot therefore be argued that this symptom differentiates between clinical and normal depression. Eventually this quality may be found to be associated with biological changes, showing that such patients are qualitatively different from other depressed people, but at present such evidence is lacking. The claim that depressed mood in clinical depression is qualitiatively different to normal depression is therefore not yet substantiated.

Snaith (1987) recognised that normal and clinical depression can appear very similar and, like the above authors, asserted that they can be differentiated on the basis of one clinical feature. He suggests that anhedonia, the loss of the ability to experience pleasure, is the central and most reliable symptom of what he labels 'hypermelancholia', by which

he seems to mean mild states of clinical depression, as opposed to unhappiness, despondency etc. This assertion is not as yet supported by evidence.

The suggestion that clinical and normal depressed mood can be distinguished not phenomenologically but because the latter is a response to circumstances while the former has no precipitant is not supported by the results of research. Matussek, Søldner and Nagel (1981) found that it was possible to identify a precipitant of depression in 75% of endogenous depressed patients, while Jablensky (1987) concluded that the results of epidemiological research focusing on psychosocial contributions to the aetiology of depression (Brown and Harris, 1978; Brown and Prudo, 1981) pointed to an almost ubiquitous role of life events in the precipitation of depression. Bebbington (1987) has also pointed out that factors other than adversity can affect normal mood, and that sometimes there is no apparent cause for changes in mood. Clinical depression and normal depressed mood cannot, therefore, be distinguished on aetiological grounds, despite Snaith's (1987) assertion that clinical depression is caused by biological factors.

There are differences between the severity and duration of symptoms found in people diagnosed as being clinically depressed and those found in depressed people without such a diagnosis. There are also some differences in symptoms which seem to be related to the severity of depression (Sturt, 1981). Such differences do not show that clinical depression is a separate entity, but rather suggest that it is situated on the same dimension as normal depression and differs in quantity, rather than quality.

Evidence that there are categorical differences between clinical and normal depression could come from the distribution of scores on self-rating depression scales if they showed a discontinuity at some point. Two studies have shown a continuous distribution of scores which suggest the appropriateness of a dimensional view of the relationship between clinical and normal depressed mood (Radloff, 1977; Dent and Salkovskis, 1986). However, they do not provide strong evidence because neither included groups of clinically diagnosed depressed people. If they had, a discontinuity may have been observed.

In summary, clinical and normal depression differ in severity and probably duration, but the available evidence from self-rating depression scales shows a unimodal distribution, with no point of rarity between mild and severe depression to support the assertion that they are qualitatively different. There do seem to be some symptoms which occur with greater frequency in diagnosed groups of depressed patients, but these differences seem to be related to severity and therefore do not necessarily show that clinical and normal depression are separate conditions. There is currently no evidence for different aetiologies: Bebbington (1987) argues that attention must be directed to this if the relationship between clinical and normal depression is to be clarified.

It may be necessary to take sub-divisions of depression into consideration when investigating the relationship between normal and clinical depression. For instance Kiloh et al (1972) held that 'psychotic' or 'endogenous' depression is a categorical entity, with a restricted range of clinical manifestations consistent with an imputed genetic or biochemical basis, and that it is qualitatively different from both other sub-types of depression and from normal depressed mood. In contrast he suggested that 'so-called neurotic depression' can be conceptualised as a continuum from mild to severe, and that it differs from normal mood disturbances only in terms of severity. It may therefore be that there are both qualitative and quantitative differences between normal and clinical depression, depending on which sub-type of clinical depression is under consideration. Sub-classifications of depression are discussed in the next section.

1.3 SUB-CLASSIFICATIONS OF DEPRESSION

Many attempts have been made to sub-classify clinical depression in order to identify disorders with characteristic prognosis, course and aetiology. However, there is no consensus as to how it should be sub-classified: as Andreasen (1982) has put it 'although affective disorders have been recognised for thousands of years, clinicans and researchers have still not reached agreement on the basic concepts of these disorders and the best methods of classifying them'.

1.3.1 Bipolar versus Unipolar Depression

Kraepelin (1921) subsumed depression and manic disorders into one class: manic-depressive illness. However, Leonhard (1979) proposed that patients who had both depressed and manic episodes (bipolar) should be separated from those who only had episodes of depression (unipolar). This division has been widely accepted and has been included in DSM-III (American Psychiatric Association, 1980). The evidence supporting this division is reviewed in detail elsewhere (Depue and Monroe, 1979; Andreasen, 1982; Malt, 1983) and will be summarised here.

Although there is considerable overlap in symptoms, bipolar patients exhibit more retardation and are more likely to have hypersomnia while unipolar patients have more agitation and tend to suffer from insomnia. It has been suggested that the two groups can be reliably differentiated on the basis of their psychomotor and/or sleep patterns in as many as 85% of cases (Depue and Monroe, 1979). Bipolar patients tend to be first hospitalised at a younger age than unipolar patients (Angst et al, 1973) and the sex ratio is equal, while more females than males suffer from unipolar illness. Bipolars tend to have more episodes and to be more likely to commit suicide (Depue and Monroe, 1979). Twin studies suggest that genetic factors may be more important in the occurrence of bipolar illness than unipolar (reviewed in Malt, 1983) and studies of response to treatment also support the distinction between the two groups, with lithium having a better prophylactic effect in bipolar than unipolar illness (eg Dunner, Stallone and Fieve, 1976). There is some preliminary evidence that there are physiological differences between the groups, for instance decreased urinary methoxyhydoxyphenylglyol (MHPG) and low platelet MAO in bipolars (see Andreasen, 1982). However, Jablensky (1987) has recently concluded that no such biochemical variable has yet been shown to distinguish reliably between the two groups.

There are several problems with the distinction between bipolar and unipolar depression. For instance some studies have shown that the relatives of unipolars have less risk for depression or mania than the relatives of bipolars, who are at risk for both unipolar and bipolar forms of the illness (reviewed in Akiskal, 1983). This may be because it is often difficult in practice to distinguish between the two groups; the

diagnosis must be based on observations over a number of years as manic episodes can develop after many episodes of depression (Malt, 1983). It may, however, be because the two groups are more closely related than once thought.

1.3.2 Sub-classifications of Unipolar Depression

There is less agreement on how unipolar depression should be sub-classified. One proposal is that primary depression (an episode of depression in people without a prior history of any other psychiatric disorder) should be distinguished from secondary depression (which occurs in those who have had an antecedent psychiatric disorder). This is intended to identify a relatively homogenous population of people with depression uncontaminated by other disorders (Andreasen and Winokur, 1979). However, despite the value of this distinction for researchers who are seeking to identify homogenous groups it has not been well studied and validated. Andreasen (1982) reviewed the available evidence and concluded that there is little evidence to suggest that this distinction is useful in predicting course, response to treatment, or familial prevalence and that it could not yet be considered to be either valid or invalid.

The most widely used classifications of unipolar depression have proabably been the dichotomous classification systems, for instance endogenous versus reactive and psychotic versus neurotic depression. There is some confusion as what these terms signify as they have been used to mean different things by different researchers. For instance Spitzer, Endicott and Robins (1978) noted that the term 'psychotic' might be used to indicate that there is evidence of delusions, hallucinations or stupor, or it might be used synonymously with 'endogenous', or it might refer to an incapacitating major depressive disorder. Similarly 'endogenous' was originally used to indicate that the depression 'arose from within', but as there is now evidence that many severe depressions considered to be endogenous have some psychosocial cause (Matussek, Søldner and Nagel, 1981), it has tended to lose these aetiological connotations and instead refer simply to a syndrome of severe depression. According to Zimmerman et al (1986) the terms 'endogenous', 'autonomous', 'incapacitating', 'melancholic', 'psychotic', 'vital', and 'severe' have all

been used more or less synonymously to refer to severe depression, while milder depressions have been labelled 'mild', 'neurotic', non-melancholic', 'non-psychotic', and 'reactive'. Because of this confusion DSM-III reverted to the historical term 'melancholia' to refer to severe depression. However, despite the disagreements over what it should be called there is evidence for a fairly well defined syndrome of severe depression. Some of this evidence is reviewed next.

In order to establish the existence of this syndrome and to investigate its characteristics Nelson and Charney (1981) reviewed the results of twenty studies which used factor analysis to describe diagnostic groups, nine studies which used cluster analysis to find new diagnostic classifications and to confirm pre-existing ones, and four studies which used discriminant analysis to validate diagnostic groupings. They also reviewed studies of symptom profiles and response to treatment. They concluded that the evidence from these studies for a syndrome of severe depression was substantial. This has been supported by a recent study using a new technique especially intended to study disease: Grade of Membership analysis (Davidson et al, 1988). This found that one of the five disease types identified in 190 depressed patients corresponded to the syndrome of severe depression.

Nelson and Charney (1981) found that psychomotor changes, both retardation and agitation, were the symptoms most consistently related to the severe depression syndrome, which they labelled 'autonomous' depression. Several studies had also shown an association between it and the severity of depressed mood, a lack of reactivity, depressive delusions, self-reproach and loss of interest. There was less evidence to associate it with a distinct quality of mood, morning worsening and difficulty concentrating. The authors concluded that disturbances in appetite and sleep were common to both autonomous and non-autonomous depression and therefore were not useful in distinguishing between the two. This has been disputed by Sinaikin (1985), who maintained that these symptoms should not be excluded as other studies investigating ways of discriminating between autonomous and non-autonomous depression have weighted them heavily. For instance Feinberg and Carroll (1982) used a discriminant function based on clinical features to distinguish between

endogenous (autonomous) and non-endogenous depression and found that decreased appetite was a particularly important discriminator.

Evidence from treatment response studies supports the distinction between endogenous and non-endogenous depression. For instance Carney and Sheffield (1972) showed that depressed patients scoring in the endogenous range on the Newcastle Scale (Carney, 1986) did significantly better in terms of social and clinical recovery both immediately and three months after treatment than other depressed patients treated with ECT. Endogenous depression has also been shown to predict a good response to antidepressant drug therapy; for instance Carney, Reynolds and Sheffield (1986) found that endogenous depressed patients, classified according to their scores on the Newcastle Scale, did significantly better in anti-depressant drug trials than non-endogenous depressed patients.

There is some evidence that depressed individuals with this syndrome can be identifed on the basis of neurochemical and neuroendocrine tests. For instance Coppen et al (1983) found that 88% of those classified as having endogenous depression (again according to scores on the Newcastle Scale) were unable to suppress the production of cortisol in the Dexamethosone Suppression Test (DST), compared to only 49% of non-endogenous depressed patients. The validity of the DST test has been questioned, largely because results have been found to be influenced by measures such as the stress of hospitalisation, weight changes and current medication (Watkins et al, 1988). This led Sinaikin (1985) to conclude that 'the more extensively DST is tested, the more controversial its use becomes'. Interest in this test continues however, and a recent study (Watkins et al, 1988) has shown that more than half of a group of patients diagnosed as being DSM-III 'melancholics' were non-suppressors, in contrast to 29% of patients diagnosed as belonging to the more inclusive group of International Classification of Diseases 'manic-depressive illness - depressed' (ICD-9: World Health Organisation, 1978). Other laboratory tests being investigated to see if they can distinguish between groups of depressed patients include CSF 5-hydroxyindole-acetic acid; MHPG; rapid eye movement latency; and thyrotropin releasing hormone (TRH) stimulation (Sinaikin, 1985). Eventually these may provide both more evidence to substantiate the existence of an 'endogenous' subgroup and a reliable way of identifying patients with the syndrome.

There is, therefore, substantial evidence to support the existence of a syndrome of severe depression characterised by altered psychomotor activity, depressive delusions, self-reproach, severe depressed mood and possibly anorexia and insomnia, which is associated with a good response to ECT and anti-depressant medication. There is not, however, complete agreement as to the criteria which should be used to diagnose this syndrome. For instance, as already stated, Carney, Reynolds and Sheffield (1986) found that scores in the endogenous range on the Newcastle Scale predicted a good response in an anti-depressant drug trial. However this result was not found when the same subjects were divided into melancholic and non-melancholic groups according to DSM-III criteria. This indicates that the Newcastle definition of an endogenous depressed subject is not completely comparable with the DSM-III melancholia criteria, despite the fact that both are intended to identify the same group of patients. More work is therefore needed to identify more clearly the exact characteristics of patients with this syndrome of severe depression. Nevertheless despite this controversy the evidence for such a syndrome is substantial.

There is less agreement about how non-melancholic depressions should be classified. These have traditionally been labelled 'neurotic depression' but several different meanings of this term have been identified. For instance, Akiskal et al (1978) studied a group of 100 depressed patients and found that clinicans used the term 'neurotic depression' with the following meanings: mild illness; non-psychotic; non-endogenous; neurotic symptoms such as phobias present; reactive; and characterologic by which they meant a tendency to overreact to stress. Over the next three to four years 40% of this sample developed full-blown 'endogenous', 'manic' or 'psychotic' episodes. The authors concluded that the term 'neurotic depression' was applied to a heterogenous group of depressions and that a substantial proportion were precursors of major affective illnesses.

There is evidence that some patients labelled as having 'neurotic depression' might well be diagnosed at another time as suffering from an anxiety neurosis. Tyrer et al (1987) followed up 78 psychiatric patients with diagnoses of depressive, anxious or phobic neurosis for a period of two years. They found that while phobic symptoms were relatively constant symptoms of anxiety and depression varied greatly over time and that

these symptoms were poorly related to diagnosis: patients diagnosed as having depression did not consistently have more symptoms of depression than symptoms of anxiety. In only three of the 78 patients did the same symptom group, anxiety, predominate at all four testing sessions. The authors conclude that the classification of neurotic disorders on the basis of the predominant symptom at the time of presentation is unsatisfactory and that most patients could be classifed as members of a single mixed disorder. This is supported by the poor agreement between different systems of classification, including RDC and PSE-ID-CATEGO, in assigning cases from a community sample to categories of depression or anxiety (Dean, Surtees and Sashidharan, 1983) which, according to Goldberg et al (1987) reflects the fact that neurotic patients, at least those identified in community samples, do not form themselves into natural groupings. This is supported by Goldberg et al's (1987) exploration of the relationship between psychiatric symptoms encountered in primary-care settings. It may therefore be that the classification of neurotic depression should not be considered separately from the classification of anxiety neuroses as many patients might be most appropriately classified as having a mixed anxiety-depression disorder.

As reviewed above, the results of multi-variate studies have consistently identified a class or syndrome of severe depression (endogenous depression). There is, however, less consistency in how the remaining cases of depression are grouped or classified. The number of clusters additional to that of the severe depression cluster identified in cluster analysis studies has varied from one (Pilowsky, Levine and Boulton, 1969) to three (Paykel, 1971). Blashfield and Morey (1979) reviewed eleven cluster analysis studies and suggested that three groups could be synthesised in addition to endogenous depression: these were 'hostile depression', 'anxious depression' and 'other forms'. The results of Paykel's study (1971) fits in with this as his categories included anxious depressives, hostile depressives and young depressives with a personality disorder, while those of Raskin and Crook (1976) included agitated depressives, neurotic depressives and young depressives with personality disorders and were therefore quite similar. However, the results of other studies are not consistent with this. For instance Davidson et al (1988), who used Grade of Membership analysis, identified four types in addition to endogenous depression: one severe and one less

severe group containing depressed patients who were also agrophobic; a mildly symptomatic hypochondriacal group and a highly neurotic, obsessive, anxious non-phobic group. It is clear that there is still no consensus on how non-endogenous depressed patients should be sub-classified.

Because there is such confusion about the meaning of the term 'neurotic depression' it was dropped completely from DSM-III. Instead, depressed patients who do not fulfil the criteria for melancholic depression are allocated to one of the following classes according to their symptoms: major depression without melancholia; dysthymic disorders; atypical depression, or adjustment disorders with depressed mood. Malt (1983) concludes that although there is at present no conclusive evidence on the most valid way of sub-classifying non-melancholic depression, the classifications provided by DSM-III should help to clarify the situation by at least providing explicit diagnostic criteria for the sub-types.

1.3.3 The Relationship Between Categories of Depression

There has been much debate about whether different categories of depression are qualitatively distinct or whether they form a continuum or dimension, with severe or psychotic forms at one end and milder forms at the other, and therefore differ only in severity. Lewis (1938) was one of the earlier authors to support the latter view, which has more recently been strongly supported by Kendell (1976). The view that categories of depression are distinct illnesses was originally proposed by Kraepelin (1921) and was later developed by Roth and his colleagues (Carney, Roth and Garside, 1965; Gurney et al, 1972). Some of the evidence on both sides of the argument will now be reviewed.

An early study collected clinical ratings on depressed in-patients which were then subjected to multiple regression analysis: the distribution of scores on the resulting function was shown to be bimodal rather than unimodal (Carney, Roth and Garside, 1965). Carney and Sheffield (1972) replicated this finding in a group of both in- and out-patients. This was taken as evidence of two discrete categories, one corresponding to endogenous and one to neurotic depression. Other

attempts to replicate this finding have, however, failed (Kendell, 1969; Kendell and Post, 1973; Post, 1972). Garside and Roth (1978) argued that a unimodal distribution could result from a bimodal distribution obscured by other factors, and therefore the failure of Kendell and Post to replicate the finding of bimodality did not mean that the distribution was not really bimodal. In an attempt to prove this Garside (1973) pooled the data of Kendell (1969) and Post (1972) data and found that the resulting frequency distribution departed significantly from normal and had a dip in the middle. The results of these studies investigating the distribution of depression scores in order to support categorical (bimodal) or dimensional (unimodal) views of depression do not, therefore, seem to have produced much undisputed evidence for either view.

Some recent evidence has clearly not supported either view. For instance Zimmerman, Coryell and Pfohl (1985) calculated RDC endogenous scores for over 200 depressed in-patients and found a unimodal distribution of scores, which would appear to support the dimensional view of the relationship between endogenous and non-endogenous depression. However, if this was so the authors expected to find a linear relationship between the number of endogenous symptoms and independent variables believed to be related to endogenous depression (such as treatment response, family history of depression and DST results); for instance, the more endogenous symptoms the patients had the more likely they were expected to be to respond to antidepressant treatment. This was not the case. The results of this study do not, therefore, support either view of depression.

There is some evidence that the differences between the sub-groups of depression are not differences in severity alone. For instance, in their study using a discriminant function to distinguish between endogenous and non-endogenous depression, Feinberg and Carroll (1982) adjusted the raw scores for the overall level of severity, as measured by the Hamilton Depression Rating Scale (HDRS) and argued that the results showed that differences in severity did not account for differences between the two groups. Carney, Reynolds and Sheffield (1986) found that endogenous and non-endogenous patients, as assessed by the Newcastle Scale, who participated in a drug-trial did not differ in overall severity of depression but, as already noted, did differ significantly in their

response to the anti-depressants. They argue that evidence for the binary view of depression is provided by this and other treatment studies which show a discontinuity of outcome between endogenous and non-endogenous depression. Preliminary evidence for this view of depression is also provided by studies such as those reviewed above which find a higher level of DST suppression in endogenous depressed patients than in other depressed patients.

The original belief that endogenous depression was caused from within while non-endogenous depression was caused by events outside of the patient has not been substantiated (see above). Andreasen (1982) argues that identifying a specific aetiology is the strongest validator of any category or class, but as yet no such aetiology specific to a particular category of depression has been identified, despite the assertion of Snaith (1987) and others that the syndrome of endogenous or melancholic depression has a biological cause. It seems likely that the debate as to the relationship between different categories of depression, and the related debate on the relationship between clinical depression and normal depressed mood, will not be resolved until some specific aetiological agents are identified. It is quite possible that, as Kiloh et al (1972) suggested, endogenous or melancholic depression will be shown to have a genetic or biochemical cause and therefore to be a separate discrete entity, while the relationship between other types of clinical and normal depression will be shown to be one of severity.

The lack of agreement as to how depression should be classified and the related fact that 'there are still a plethora of diagnostic 'systems' and *ad hoc* provisions for classifying affective disorders, which employ concepts and terms that are far from being equivalent or synonymous' (Jablensky, 1987) has implications for research into memory in depression. It makes it difficult to know what types of depressed subjects were used in what studies and it is therefore hard to be certain that experiments supposedly investigating the same aspect of memory in depression in similar groups of subjects are in fact comparable. It is therefore not surprising that conflicting results have been obtained in some areas.

The remainder of this chapter is taken up with a review of experimental investigations of memory in depression.

1.4 COMPARISONS BETWEEN THE MEMORY OF DEPRESSED AND NON-DEPRESSED SUBJECTS

A number of studies have found significant impairments in depressed subjects. An early study by Rapaport (1945) found that psychotic depressives were significantly impaired on the WAIS Performance tests, a Digit span test and on the Babcock Story Recall tests.

Cronholm and Ottosson (1961) looked at the performance of 'endogenous depressives' on the 30 word-pair test, 20 figure test and 30 personal data test. They got significantly lower scores for immediate and delayed reproduction on all three tests than normal controls matched for sex, age and educational level. The groups did not differ in the amount of information forgotten between immediate and delayed recall.

Sternberg and Jarvik (1976) used similar tests with patients with endogenous depression and replicated the findings of Cronholm and Ottosson. In addition they computed mean registration scores which they found also differed significantly between the depressed subjects and normal controls. Steif et al (1986) used the word-pair test orginally used by Cronholm and Ottosson to compare depressed in-patients with controls comparable for sex, age, educational level, social-economic status and estimated pre-morbid IQ. They again found that the depressed subjects were impaired on tests of immediate and delayed recognition, but there was no difference between the groups in the amount of information forgotten.

Breslow, Kocsis and Belkin (1980) compared Wechsler Memory Scale (WMS) scores of depressed in-patients with those of controls matched for age and educational level. There was substantial evidence for memory deficit in the depressed subjects on both the verbal learning and visual reproduction subscales. However, five of the depressed subjects were tested during the initial stages of treatment with tricyclics. As there is some evidence that these drugs can cause confusional states in the early

stages of treatment (Davies et al, 1971) the observed differences may have been due to the effects of treatment, rather than to depression.

Frith et al (1983) compared the memory of a group of severely depressed endogeneous patients with that of attenders at a psychiatric out-patient clinics who were not depressed but had mild anxiety, transient situational disturbances or circumscribed phobias. The depressed subjects were significantly impaired on tests of remote semantic and episodic memory, word list recall and recognition, learning labels for faces and concentration/vigilance.

Calev and Erwin (1985) found that a group of unipolar depressed in-patients were significantly impaired on verbal recognition and recall tests. Wolfe et al (1987) compared the performance on the Rey Auditory Verbal Learning Test of depressed patients and non-depressed subjects of a similar age and educational level. Their depressed subjects were also significantly impaired on both recognition and recall tasks.

Siegfried, Jansen and Pahnke (1984) looked at the performance of depressed geriatric in-patients on tests measuring arousal, attention, perceptual group, short- and long- term memory and complex reaction time. The data was factor-analysed which produced three factors: cognition and learning, complex reaction abilities and short-term memory. Stepwise discriminant analysis was then used and showed that the depressed subjects could be reliably differentiated from geriatric in-patients who were not depressed on the basis of their cognitive performance: as expected the depressed subjects has significantly lower scores.

Robertson and Taylor (1985) compared the memory of manic depressed, unipolar psychotic, reactive depressed and non-depressed prisoners on tests which included the WAIS, and tests of verbal fluency, visual retention and visual recognition. The depressed subjects were significantly impaired on all tests except the WAIS vocabulary and similarities subscales.

Calev et al (1986) found that depressed in-patients were impaired on tests of verbal and non-verbal memory. Similarly Cutting (1979) found that depressed patients achieved lower scores than controls on the WMS

Paired Associate task and a pattern recognition memory task. Similar subjects were found by Kopelman (1986) to have significantly lower scores than non-depressed controls on the immediate and delayed recall trials of the WMS Logical Memory test, and on a test of Paired Associate learning.

Depressed patients were shown by Watts, Morris and MacLeod (1987) to be significantly impaired compared to controls comparable for age, educational level and IQ on tests of verbal recognition memory. Hart et al (1987a) found a similar impairment in a group of elderly depressed patients who showed normal forgetting on a nonverbal recognition task, but needed longer exposure times to acquire the same information as controls matched for age and educational level. In contrast Watts and Sharrock (1987) found that depressed patients did not differ from controls on a recognition memory test, although they were significantly impaired on both a free recall and a cued recall test. However, once scores on all three tests were standardised there was no evidence that recognition memory was less impaired than free or cued recall and therefore it is not clear whether or not recognition memory was impaired in these patients.

The study by Ellis et al (1985) differs from those reviewed above in that the subjects were students in whom depressed mood was induced using the Velten mood induction procedure. The subjects studied a list of sentences before mood was induced, rating them for complexity, and then were given an unanticipated cued recall test of target adjectives. Subjects who had received the depressed mood induction recalled fewer adjectives than other subjects, showing an effect of depressed mood on retrieval from memory.

Abrams and Taylor (1987) gave neuropsychological tests with known cut-off points for severe cognitive impairment to depressed inpatients and to controls who were not depressed. The tests included tests of orientation, registration. attention and recall. Half of the depressed subjects showed moderate to severe impairment on the battery, compared with none of the controls. Zung, Rogers and Krugman (1968) also looked at the performance of depressed subjects on established tests frequently used by neurologists. They found that the depressed patients performed at levels which were normally presumed to indicate the presence of some

cerebral pathology or 'organicity'. Newman and Sweet (1986) found that depressed in-patients scored significantly lower than controls on two summary scales derived from the Luria-Nebraska Neuropsychological battery. In addition, 40% of the depressed patients were classified as brain damaged as opposed to 5% of the controls when scores above the recommended cut-off point on two of the fourteen scales were taken as the criteria for brain damage. Fisher, Sweet and Pfaetzer-Smith (1986) found that depressed patients achieved significantly lower scores than controls on ten of the fourteen tests in a neuropsychological test battery which included the WMS.

These papers all indicate that depressed patients have significantly impaired memory compared to non-depressed controls. There is, however, some conflicting evidence. Some studies suggest that the degree of impairment shown by depressed subjects is minimal, while others have found no evidence of impairment.

Friedman (1964) compared the performance of psychotic depressives on a battery of cognitive, perceptual, vigilance and psychomotor tests, with that of controls matched on a variety of variables (age, educational level, sex, religion, marital status and scores on a vocabulary test). The depressed subjects were significantly impaired on only nine of the 82 test scores, a number which, as indicated by Johnson and Magaro (1987), could have arisen by chance. The author concluded that depressed subjects showed relatively minor deficits of cognitive, perceptual and psychomotor function.

Coughlan and Hollows (1984) also concluded that depression had relatively little effect on performance on a battery of cognitive tests. The scores of the depressed patients were more than two standard deviations below that of controls of similar ages on only three out of eleven tests. The critiera for impairment set by these authors were stringent and the two groups might have been significantly different on more tests if mean scores had been directly compared. The possibility that these subjects were significantly impaired cannot, therefore, be ruled out.

Other studies have found no evidence of memory impairments due to depression. For instance Cole and Zarit (1984) used medical in-patients found to meet the RDC criteria for a severe depressive episode. Their performance on the WAIS Digit Symbol Substitution test, a recognition memory test and the Krauss Card Classification task was found to be significantly poorer than that of a group of healthy community volunteers. However, they did not differ from a group of medical in-patients who were not depressed but had some anxiety about medical procedures etc. The authors concluded that the observed memory disruption was due to hospitalisation rather than to depression.

Davis and Unruh (1980) did not find significant differences between the verbal recognition and recall performance of non-psychotic depressed out-patients and a control group recruited from a private counselling agency. The choice of control group in this study, and that of Cole and Zarit (1984), may have made it unlikely that an effect of depression would be found. The controls used by Davis and Unruh were having counselling and therefore were likely to have some psychiatric morbidity themselves which could have been affecting their performance. The performance of the hospitalised controls in the Cole and Zarit study may also have been affected by their current levels of anxiety. If this was the case it would have reduced the likelihood of detecting impairments due to depression.

Hasher et al (1985) found that mildly depressed college students, selected on the basis of their scores on the BDI, performed as well as nondepressed controls on tests of prose passage recall. The authors concluded that the mild levels of depression seen in this study were not substantial enough to disrupt performance on this task. Ellis (1985) suggested that an impairment might have been seen on a more difficult task which lacked the meaning and structure of the material used here.

Rush et al (1983) looked at the performance of depressed in- and out-patients on a standard neurological test battery which measured the accuracy of visual and auditory information processing and memory, visual filtering and visual-motor performance. The battery had been validated in other groups, including elderly and psychiatric patients, and normative data had been obtained from more than 10,000 subjects. There were no

significant differences between the accepted norms on these tests and the performance of the endogenous and non-endogenous groups of subjects. As there were only nine subjects in the former group and eleven in the latter it is possible that the numbers were too small to allow the detection of real differences between the performance of these subjects and the norms on these tests.

Gass and Russell (1986) looked at the effects of organicity and depression on short- and long- term memory in a group of 135 patients assessed by neurologists to have an organic diagnosis, and a similar number who were considered to have functional impairment. Half the subjects in each group were found to have Minnesto Multiphasic Personality Inventory (MMPI) depression scale scores of above 70, and were defined as depressed. There were no significant differences between the performance of the depressed and non-depressed subjects in either group. The depressed subjects had not been diagnosed as being clinically depressed but were defined as depressed solely on the basis of scores on the MMPI-D. They may, therefore, differ in severity or type of depression from subjects who have come under psychiatric care for their distressing mood disorders.

Depressed subjects with a mean age of 75 were found by Miller and Lewis (1977) to have strict decision criteria but normal 'pure' memory function on a visual recognition test, compared to controls matched for age and sex. It is not clear how depression was assessed, whether or not the subjects had a primary diagnosis of depression or how severe the depression was. These subjects may not have been comparable with the depressed in-patients with major depressive disorders used as subjects in most of the studies reviewed above which found a significant effect of depression.

Both Popkin et al (1982) and O'Hara et al (1986) looked at the performance of elderly depressed subjects on verbal learning tests. The performance of these subjects did not differ significantly from that of non-depressed controls. O'Hara et al suggest that the free recall task used in their study may have been difficult for all elderly subjects, and that differences between the groups may be found on easier or more relevant tasks. This may also have been true of the similar materials

used by Popkin et al (1982). However, an alternative explanation is that the subjects in the latter study were out-patients while those in the former were identified during a large epidemiological study of the elderly. As with the Miller and Lewis study (1977), these subjects may not be comparable with the psychiatric in-patients used in the majority of studies showing memory deficits, even though in these cases they fulfilled RDC criteria for primary depression (Section 1.2.2).

Kahn et al (1975) also failed to find any significant differences between the performance of elderly depressed and non-depressed subjects on a battery of memory tests. In this case the subjects were geriatric out-patients and their families who were allocated to the depressed or non-depressed group according to whether or not their score on the Hamilton Depression Rating Scale (HDRS) was above the median for the whole group. Thus they had not been diagnosed as being clinically depressed and were therefore not comparable with the subjects used in studies showing memory deficits.

In summary, a number of studies have demonstrated significant differences between the memory of depressed subjects and non-depressed controls. In all cases the depressed patients have had impaired memories. Some studies have suggested that the impairment may be minimal (Friedman, 1964; Coughlan and Hollows, 1984) while a minority have failed to find a memory deficit. In some cases this may have been due to using control subjects with memories impaired by anxiety (Davis and Unruh, 1980; Cole and Zarit, 1984). In the remainder the subjects were out-patients, recruited from an epidemiological survey or defined as depressed on psychometric grounds (Kahn et al, 1975; Gass and Russell, 1986; Hasher et al, 1985). As the subjects in studies showing significant impairment were predominately psychiatric in-patients, these results suggest that a memory impairment may only be found in subjects with symptoms severe enough to warrant psychiatric in-patient care. If this is the case, however, it is difficult to see why students subjected to a depressed mood induction were impaired (Ellis et al, 1985) while out-patients and depressed medical patients were not. This may have been a function of the different types of memory tests used in different studies or to the tighter experimental control in the induced mood study. It does,

however, indicate that as yet it is not clear which depressed people will demonstrate memory impairment.

1.5 THE RELATIONSHIP BETWEEN SEVERITY OF DEPRESSION AND MEMORY IMPAIRMENT

This relationship has been explored in several studies. Cohen et al (1982) found a highly significant negative relationship between depressed mood, as measured by the POMS depression scale, and the number of trigrams recalled after varying recall intervals in a group of subjects consisting of two patients with bipolar depression, nine patients with unipolar depression and five normal controls. A similar relationship was found between the HDRS and Beck Depression Inventory (BDI) scores of the eleven depressed patients and recall.

Siegfried, Jansen and Pahnke (1984) factor-analysed HDRS scores obtained from 100 geriatric in-patients, half of whom had a primary depressive disorder. This resulted in five factors, three of which were found to be highly negatively correlated with factors derived from a battery of cognitive tests (Section 1.4). These factors were a general factor of depression, anxiety and agitation, and vitalised depressive symptoms. However, no evidence is presented that the same relationship would be found if the analysis was restricted to the depressed subjects: these results may therefore just reflect differences between the depressed and nondepressed groups.

Fisher, Sweet and Pfaetzer-Smith (1986) looked at the relationship between the BDI scores of their entire sample of subjects, comprising fifteen depressed in-patients and fifteen controls, and scores on ten neuropsychological measures which had been shown to differ significantly between the two groups (Section 1.4). Amongst other significant correlations, they found a significant negative correlation between BDI scores and two memory measures derived from the WMS: Immediate and Delayed Figural Memory. However, as the correlations are not presented for the depressed group alone the same criticism applies to this study as that by Siegfried, Jansen and Pahnke (1984).

Wolfe et al (1987) looked at the relationship between scores on the BDI and scores on the Rey Auditory Verbal learning test in twenty unipolar and twelve bipolar depressed patients. They found significant negative relationships between depression scores and delayed recall and recognition scores in the unipolar group only. There were no significant correlations in either group with immediate recall and recognition scores.

Watts and Sharrock (1987) found that depression scores derived from the Levine-Pilowsky depression severity scale correlated significantly with the free recall and cued recall scores of twenty-one depressed in-patients: as expected higher depression scores were associated with poorer performance.

As depressed patients had been found to differ significantly from non-depressed controls in all of these studies it may seem unsurprising that performance was related to the severity of depression in each case. However, six other studies have shown significant differences between depressed and control groups, but failed to find a significant relationship between the severity of depression and memory.

Friedman (1964) rated the severity of depression in the depressed subjects in his study, and correlated these with one key score from seventeen of the tests in his extensive test battery, including four in which the depressed group had shown a deficit. No significant relationships were found with any of the tests. Negative results were also obtained by Coughlan and Hollows (1984), who correlated the scores of depressed subjects on the depression subscale of the McNair and Lorr self-rating mood scale with their performance on a battery of verbal and visual memory tests. Newman and Sweet (1986) found no evidence of a significant relationship between scores on two summary scales derived from the Luria-Nebraska Neuropsychological battery and scores on two depression scales derived from the Schedule for Affective Disorders and Schizophrenia (SADS; Spitzer and Endicott, 1977).

Silberman et al (1985) gave two verbal learning tasks to depressed patients and to non-depressed controls: there was a significant difference between the groups on only one of the tasks. They then divided the depressed group into DST suppressors and non-suppressors. There were

no significant correlations between scores on either learning task and the scores of either depressed group on the Zung Depression Scale, Zung Anxiety Scale or Beck Hopelessness scale. Kopelman (1986) failed to find any significant correlations between the performance of sixteen depressed patients on a variety of memory tests and their scores on the HDRS. Steif et al (1986) found that scores on this scale obtained for subjects after a course of ECT were not significantly related to performance, even though the subjects were impaired compared to controls. This study differed from others in that the effects of social-economic status, age, education and IQ were controlled for.

Two studies which did not find significant differences between the performance of depressed patients and controls on memory tests have also looked at the relationship between the severity of depression and performance. Rush et al (1983) did not find any significant correlations between scores on a standardised neuropsychological test battery and scores on the BDI or on the HDRS. Similarly Gass and Russell (1986) did not find a significant relationship between the scores of 270 medical in-patients on the MMPI-D subscale and their performance on tests of short- and long-term memory.

Several studies have not included a control group, and instead have just looked at the relationship between the severity of depression and memory. Stromgren (1977) compared performance on three scales derived from the WMS (Mental Control; Verbal Learning; Visual Reproduction) with three components of depression (Depressive Appearance; Content of Ideas; Agitation) in endogenous depressed patients. There were significant negative correlations between both Depressive Appearance and Agitation and all three memory scales, and between Content of Ideas and Visual Reproduction. Henry, Weingartner and Murphy (1973), in a slightly different type of study, found that a group of bipolar and unipolar depressed patients showed a significant decrease in learning on days when they were severely depressed compared to their own performance on days when less depressed, as assessed by the Bunney-Hamburg ward rating scale.

The remaining studies which have looked at the correlation between memory and the severity of depression have either found negligible relationships, or no relationship at all.

Donnelly et al (1982) correlated scores on the WAIS with the MMPI-D scores of 65 depressed patients: the correlations with full-scale IQ, verbal IQ and performance IQ were all non-significant. Cavanaugh and Wettstein (1983) found a very small relationship between scores on the Mini Mental State Examination (MMSE) and the BDI in randomly selected medical in-patients. However, this relationship disappeared in patients under the age of 65 when the effects of sex, race and social-economic status were controlled for. La Rue, Spar and Dessonville Hill (1986) used the MMSE with depressed in-patients in a psychogeriatric in-patient unit and correlated scores with nurses' ratings of anxiety, depression and global assessment scores. Lower MMSE scores were linked with anxiety and global ratings, but not with depression. Other studies have shown depressed patients to be impaired on the MMSE (McHugh and Folstein, 1979) and therefore the lack of relationship in these studies is not likely to be due to depressed patients performing normally on this measure.

In summary, out of eighteen studies which have reported the relationship between the severity of depression and performance, only seven found a significant relationship between the two: in each case memory declined as the severity of depression increased. In six of the remaining studies there was no significant relationship between memory and depression even though the scores of the depressed patients on the memory tests had been shown to differ significantly from those of controls who were not depressed. There are several reasons why these studies may have failed to find a significant correlation.

Small numbers of subjects may be the cause in some cases. For instance Silberman et al (1985) had seventeen subjects in one depressed group and ten in the other. Kopelman (1986) used sixteen subjects while Steif et al (1986) used nineteen. However some of the studies finding a significant relationship had similar subject numbers. For instance, Watts and Sharrock (1987) had twenty-one subjects, while Cohen et al (1982) found a highly significant correlation between memory and depression with

only eleven depressed subjects. In addition some of the studies which failed to find a relationship had very large numbers: 170 in one study (Gass and Russell (1986) and 289 in another (Cavanaugh and Wettstein, 1983).

The measures of depression used in some of these studies may have been inadequate. The Zung Depression Scale, used by Silberman et al (1985) has been extensively criticised and two reviews have concluded that it should not be used to measure depression (Kearns et al, 1982; Boyle, 1985). La Rue, Spar and Dessonville Hill (1986) used nurses' ratings of depression which may be of doubtful validity and reliability, while it is not clear how Newman and Sweet (1986) derived their depression scales from the SADS or how Friedman (1964) rated depression in his patients. Two studies (Gass and Russell, 1986; Donnelly et al, 1982) used the MMPI-D, which has been criticised for being a narrow measure of depression (Boyle, 1985) and therefore may not have been sufficiently sensitive for this task.

However, this cannot be the reason for the lack of correlation between performance and depression in all cases as some of the studies did use appropriate measures of depression. For instance some used the Hamilton Depression Rating Scale (HDRS) which is considered to be valid and reliable, and sensitive to changes in depression (Kearns et al, 1982). It is an interviewer rating scale which is considered to be preferable to self-rating scales in severe depression (Prusoff, Klerman and Paykel, 1972). This raises the question of why studies using this scale failed to get a relationship between depression and memory scores. Kopelman (1986) had a narrow range of scores on the HDRS, with a mean of 28.1 and a standard deviation of 4.1, which would have reduced the likelihood of finding a significant correlation. Steif et al (1986) do not give the range of HDRS scores in their study, however they do state that many of the subjects were substantially improved following ECT and this may have resulted in a narrow range of depression scores. Rush et al (1983) used the HDRS, but as there was no significant difference between depressed patients and controls on their test battery it is not surprising that no relationship was found between depression scores and test battery scores.

In summary, some studies may not have found a significant relationship between levels of depression and memory scores because of inappropriate measures of depression, a narrow range of depression scores and/or a small number of subjects. However some of the studies which found a significant effect can be criticised for combining depressed and nondepressed subjects which means that the correlation may be due to between group differences. The question of whether the degree of memory impairment in depression is related to the severity of depression therefore remains unanswered.

1.6 REMISSION OF DEPRESSION AND CHANGES IN MEMORY PERFORMANCE

Several studies have looked at whether the memory deficits observed in depression disappear on the remission of the depression. Whitehead (1973), using a battery of verbal learning and memory tasks, compared the performance of 26 depressed patients with their performance on the remission of the depressive illness. Their scores increased significantly on remission on three of the eight tasks. It is not clear how it was decided that the patient was in remission and it is possible that some of the patients were still depressed at follow-up: this may account for the failure to find improvement on the other tests.

Fromm-Auch (1983) gave depressed patients a neuropsychological assessment which measured learning, abstraction, motor speed and sensory-perceptual function. Thirty-three were retested once they showed significant signs of improvement following antidepressant treatment. Significantly fewer patients had abnormal profiles on the test battery after treatment, but some remained impaired. Again, as no information is given on the extent of clinical improvement, it is speculated that the residual deficit was due to unremitted depression.

Fisher, Sweet and Pfaetzer-Smith (1986) tested depressed in-patients on a neuropsychological test battery on admission and shortly before discharge. They showed significant improvement on three of the ten tests they were originally impaired on. Control subjects were retested after a similar interval and showed no significant improvement, thus ruling out a

practice effect. As before, the remaining deficit in the depressed patients may have been due to residual depression.

Sternberg and Jarvik (1976) looked at depressed patients who were judged by a clinician, and on the basis of Zung depression scores, to have shown at least some improvement in depression after 26 days' treatment with imipramine hydrochloride or amitriptyline hydrochloride. They showed significant improvements in immediate reproduction, delayed reproduction and computed registration compared with their own performance before treatment. The greatest improvement in memory was shown by those patients who showed the greatest improvement in depression. Control subjects who were retested after 26 days did not show significant changes in performance, again ruling out a practice effect.

Glass et al (1981) also evaluated the effects on memory of clinical improvement resulting from treatment with imipramine hydrochloride. Out-patients with non-psychotic and mainly non-endogenous primary depression were tested on two psychomotor tasks and on Sternberg's Item Recognition Procedure before treatment, after treatment with a placebo, and after receiving the anti-depressant. Treatment with imipramine led to a signi-ficant reduction in the number of errors on the memory task compared to the subjects' own performance when taking a placebo. This improvement occurred without any apparent improvement in depression, as measured by the HDRS, thus suggesting that anti-depressants can produce improvement in cognitive function as a forerunner of clinical improvement.

Siegfied, Jansen and Pahnke (1984) compared the effects of the anti-depressant nomifensine and a placebo in depressed and non-depressed elderly geriatric patients. They used stepwise discriminant analysis firstly to identify changes in the symptoms of depression, as measured by the HDRS, after treatment with nomifensine as compared to placebo, and secondly to identify changes in scores on a cognitive test battery after treatment. They then analysed the discriminant functions of changes in the depressive and cognitive symptoms and found that they were very highly correlated: improvements in depression were very closely related to improvements in cognitive performance.

These studies show that remission following anti-depressant treatment is associated with improvements in memory. Other studies have looked at the relationship between remission following treatment with ECT and improvements in memory.

Cronholm and Ottosson (1961) examined changes in memory in endogenous depressed subjects who responded to ECT. There was a positive relationship between improvement in depression and improvement in immediate and delayed reproduction, but none of the coefficients reached statistical significance. Despite this the authors claim that the greater the improvement in the depressive state, the greater the improvement in learning. Stromgren (1977) also looked at the relationship between changes in depression and changes in memory after ECT. He found that the larger the change in depression scores, the larger the change in scores on the WMS. Recovery from depression was associated with the elimination of the impairment in memory evident before treatment (Section 1.5).

Frith et al (1983) compared the performance of depressed patients on a battery of tests with that of control subjects who were not depressed, both before and after treatment with ECT. As reviewed above (Section 1.4), the depressed subjects were significantly impaired on all aspects of memory and concentration which were investigated before treatment. Six months after treatment significant differences between the groups remained on only two tasks: word list recall and recognition. The authors argue that these continued deficits were probably due to the moderate levels of depression still found in some patients, rather than to ECT, as patients who had received sham ECT also had deficits on these tasks. McAllister et al (1987) found a significant improvement in scores on both a prose passage recall test and a visual memory test after treatment compared with the patients' own performance before treatment. This was associated with a significant reduction in scores on the HRDS, BDI and Brief Psychiatric Rating Scale.

In summary, the results of studies investigating changes in memory following successful treatment with ECT or anti-depressant drugs are consistent: remission in depression is associated with improvement in memory. It might have been expected to find less evidence of a relationship in studies using ECT because ECT itself can affect memory

(Frith et al, 1983), and the detrimental effects of this might obscure the ameliorating effect of the improvement in depression. This was not the case. It can be concluded from these studies that memory deficits observed in depression are temporary.

1.7 THE NATURE OF THE MEMORY DEFICIT IN DEPRESSION

A number of studies have attempted to delineate the exact nature of the memory impairment in depression. First, studies based on some version of a 'stages' model of memory (Atkinson and Shiffrin, 1968) will be reviewed.

1.7.1 'Stages' models of memory

Most studies of memory in depression have implicitly or explicitly been based on these models. They assume that information processing occurs in a number of discrete stages, involving the initial registration of information in a sensory register; its transfer to a short-term store of limited capacity and then to long-term memory; its retention and, finally, its retrieval. An impairment in any one of these stages of information processing would result in a failure to recall information adequately.

Henry, Weingartner and Murphy (1973) looked at the performance of hospitalised bipolar manic-depressed and unipolar psychotic depressed patients on a serial learning task on days when they were less depressed and more depressed. The first trial of the memory task was considered to measure immediate recall, while the remaining five trials measured the shift of information from short-term to long-term memory. The subjects showed impaired learning on trials two to six, but not on the first trial. The authors concluded that the deficit in depression was best understood as an inability to shift information from short-term to long-term memory.

Sternberg and Jarvik (1976) considered memory performance to be a function of registration, retention and retrieval and compared these stages in depressed in-patients and controls who were not depressed. Mean computed registration scores and immediate recall were significantly lower in the depressed patients but there was no change in forgetting.

They concluded that endogenous depression was associated with impairments in short-term memory, but not in retention. The same conclusions were reached in two other studies which used very similar tasks (Cronholm and Ottosson, 1961; Steif et al, 1986).

These results conflict with those of Henry, Weingartner and Murphy (1973). As Johnson and Magaro (1987) have recently observed, this may be due to differences in the type of subject used. Henry, Weingartner and Murphy (1973) used both unipolar and bipolar depressed patients. As reviewed above (Section 1.3.1) the two conditions appear to differ significantly in a number of ways, including symptomatology, response to treatment and age of onset. Studies using both bipolar and unipolar depressed subjects (Henry, Weingartner and Murphy, 1973) may not, therefore be comparable with studies restricted to unipolar subjects.

1.7.2 Short-term memory scanning

Other studies interested in identifying the precise nature of the deficit in depression have looked at detailed aspects of short-term memory. They have all used additive factor methodology (Sternberg, 1975) which permits several elementary aspects of information processing in reaction time tasks to be isolated and measured. It differentiates between four sequential stages of short-term memory processing: initial encoding; memory scanning; response selection and response execution.

Hilbert, Niederehe and Kahn (1976) used this procedure in their investigation of depression and altered brain function in people over 50 years of age. People defined as depressed on the basis of their scores on the HDRS (ten or more) were compared with controls who were not depressed and had no signs of altered brain function. The slope of reaction time function (a measure of the speed of memory scanning) did not differ significantly between the depressed subjects and the controls. However, the intercept was significantly higher in the depressed subjects, indicating slowing of the non-scanning stages of processing (encoding, decision processes, motor response). The same results were obtained by Hart and Kwentus (1987) who compared elderly depressed psychiatric patients with normal controls matched for age.

Glass et al (1981) did not find a significant difference between the slope of reaction time function of a group of depressed subjects (unipolar, non-psychotic and mainly non-endogenous) and a group of matched controls. Again, the depressed subjects were significantly slower overall. After treatment with imipramine hydrochlorine the depressed subjects made significantly fewer errors compared with their performance before treatment. This suggests that previously they were maintaining accuracy comparable with controls at the expense of speed.

Koh and Wolpert (1983) used schizophrenics, rather than subjects without significant psychopathology, as the control group. They argue that this is appropriate because previous studies had shown that schizophrenics' short-term memory processing is comparable to that of normals. The performance of unipolar and bipolar depressed patients was therefore compared with that of a group of schizophrenics. All patients were free from psychotic disturbance at the time of testing. The short-term memory scanning of both the unipolar and bipolar subjects was as good as that of the schizophrenics, and therefore presumably as good as that of normal subjects. Previous studies had shown that the overall reaction time of schizophrenics was significantly slower than that of normals: there was no significant difference between the overall reaction times of the schizophrenic and depressed subjects in this study, which suggests that the depressed subjects would also be slower than normals. The chain of inference is rather long, however.

In contrast to the above studies Brand and Jolles (1987) did find that unipolar depressed subjects had significantly higher slopes of reaction time function than controls who were not depressed. They compared the performance of groups of unipolar and bipolar depressed patients with that of anxious patients and controls who were neither anxious or depressed on two tests similar to those used above, which involve the tachistoscopic presentation of stimuli, and two pen and paper tests which were also based on the additive factor methodology. As in the previous studies the unipolar depressed subjects were slower than controls on all tasks. They were also significantly slower than the anxious subjects. They also had significantly higher slopes on both of the pen and paper tasks, and on one of the tachistoscopic tasks. They therefore showed slower

memory scanning than the controls. Bipolar subjects did not show the same evidence of impairment.

These results are in conflict with those from earlier studies using the same methodology. Brand and Jolles (1987) suggest that this may be because they made the tasks more acceptable to depressed subjects by reducing the number of trials in the tachistoscopically presented tasks and by using pen and paper tests which were probably less demanding than the usual tasks. This explanation implies that subjects will show impairment on acceptable tasks but perform as well as controls on more difficult ones: this seems unlikely. The issue of whether memory scanning is impaired in depression has not, therefore, been resolved.

1.7.3 'Levels of Processing' Models of Memory

The 'levels of processing' model (Craik and Lockhart, 1972; Craik and Tulving, 1975) underlies some studies of memory performance in depression. This model differs from the 'stages' framework in that it considers memory to be a single continuum, rather than consisting of separate, discrete stages. The type of processing carried out at the time of acquisition determines whether or not the event will be learnt. In this view memory fails because encoding operations are weak or inefficient, not because of an impairment in a discrete stage of memory.

Weingartner et al (1981) examined the stategies depressed patients used to process and organise information. In Experiment One subjects were asked to respond either to the meaning (semantic processing) or the sounds (acoustic processing) of words which were then recalled. The patients were impaired on the semantic processing condition compared to matched controls, but not on the acoustic processing condition; this suggests that they were unable to make use of the more elaborative encoding stategy. In Experiment Two subjects were asked to group words and then to recall them: the words were either highly related or random. Both the kinds of organisation imposed on information and the recall of the more difficult (random) words were impaired in the depressed subjects. In Experiment Three subjects processed sets of words which differed in degree of organisation. As in Experiment Two, the depressed patients were able to organise material efficiently when the relationships

were obvious, but were deficient when they were not. The authors concluded that the depressed patients 'failed to use encoding operations that would be useful in organising input and that would then facilitate later recall'. These results suggest that the memory deficits observed in depressed subjects may result from processing deficits due to inefficient subject-imposed organisation.

Silberman et al (1985) compared the performance of a group of depressed in-patients with that of non-depressed controls on two tests which examined the processing stategies used by subjects. In the first subjects rated a list of words for emotionality and were then given an immediate recall test and a delayed recognition test. The second test looked at subjects' memories as a function of the level of organisation of stimulus material. The depressed subjects got significantly lower scores than the controls on the first test. This indicated that they had processed the information shallowly, and had not made use of the emotionality of the words, which would increase recall in normal subjects. The depressed and control subjects did not differ significantly on the second test, despite the fact that other depressed subjects had been impaired on similar tests (Weingartner et al, 1981). The authors suggest that this may be because the subjects in this study were being treated in a private hospital, and therefore may have differed in severity or type from those used in the earlier study.

The final study to look at processing stategies in depression differed from the studies reviewed so far in that the subjects were students in whom depressed mood was induced using the Velten mood induction technique (Ellis, Thomas and Rodriguez, 1984). In the first experiment subjects read sentences that varied in elaborative detail and then had to recall a word in the sentence. Subjects who had received the depressed mood induction did not benefit from the elaboration, which should have resulted in deeper processing and therefore better recall. The second experiment investigated the effects of semantic orientating instructions on the free recall of a word list. The depressed students recalled fewer words overall than the neutral mood group, but both groups performed better in the semantic condition. The third experiment was based on the finding of Tyler et al (1979) that difficult words in sentence completion tasks were recalled better than easy words, presumably because more

resources are allocated to process them. The results again showed that the depressed group were impaired overall, and that unlike the controls they did not recall more difficult words than easy ones. This study is important in that it found impairments in learning in subjects with induced depressed mood, rather than clinical depression. It also suggests that such subjects may process information less efficiently than subjects in a neutral mood.

There is, therefore, some evidence that depressed subjects process information less efficiently than non-depressed individuals, and that this may account for the memory deficts observed in depression.

The final group of studies to look at the specific nature of the impairment in depression have looked at the role of the hedonic tone of the material to be learnt.

1.7.4 The Role of Hedonic Tone

Breslow, Kocsis and Belkin (1981) asked depressed patients and matched controls to read a short story which consisted of ten positive, ten negative and six neutral themes and then to recall it afterwards. The recall of the passage correlated highly (r=.75) with recall of the WMS prose passage which was administered to the same subjects and shown to be recalled significantly less well by the depressed subjects (Breslow, Kocsis and Belkin, 1980). In this case there was again an overall deficit, but there were no significant differences between the groups in the recall of the negative or neutral themes. In contrast there was a highly significant difference in the number of positive themes recalled, and the overall difference between the groups disappeared when this was controlled for.

The authors suggest that these results show that the affective tone of material is an important organising principle for memory in depressed patients. Alternatively they suggest that the depressed subjects may be able to recall the positive themes but do not report them because from their perspective they seem so unimportant as not to be worth reporting. The same subjects were significantly impaired on the WMS (Breslow, Kocsis and Belkin, 1980) and it is difficult to see how this could be accounted

for by a memory deficit restricted to items with a positive affective tone. However, this study does indicate that not all types of material will be equally affected by depression.

Dunbar and Lishman (1984) compared the performance of hospitalised depressed patients and matched controls on a recognition memory task in which a third of the words had positive hedonic tones, a third neutral, and a third negative. The data was analysed using a signal detection procedure (McNicol, 1972) which allowed 'pure' memory processes (d') to be separated from the subjects' response criteria (ß). There was no overall difference between the groups in the sensitivity of memory but there were differences in the type of material they recognised with ease: the depressed subjects had higher d' scores for negative words than the controls while the reverse was true for positive words. In addition the depressed subjects found the negative words easier to recall than the positive words; again the reverse was true for the controls. There was also evidence that the depressed patients had more conservative response criteria than the controls for positive and neutral words. The results suggest that the strength of a memory trace varies according to the hedonic tone of the material and the affective status of the subject. Again, this study indicates that the amount of memory impairment found in depressed subjects may depend on the nature of the material to be remembered.

There has been considerable interest in the effect of hedonic tone on memory in depression, especially since Beck (1967, 1976) developed his Cognitive Theory of Depression. This states that depression is primarily a thought disorder, and that changes in the way depressed people think about, interpret and remember events underlie all the other symptoms of depression. Kovacs and Beck (1978) suggested that these distortions were caused by depressive self-schemata which develop early in life in response to loss and are later activated by events similar to those which caused their original development. Once activated they organise incoming information to be consistent with negative thoughts and feelings, and direct attention to information consistent with a negative view of oneself. There is some evidence for such schemata (eg Davis, 1979a, 1979b) although there is less evidence to support the assertion that

these schemata lie dormant between episodes of depression (see Johnson and Magaro (1987) for a review).

This theory has generated a considerable body of research into the effects of depression on the recall of material of different hedonic tones. (This is summarised here and is reviewed in more detail in Blaney (1986) and Johnson and Magaro (1987)). A number of studies have shown that depressed people, both those diagnosed as having clinical depression and those in a depressed mood, tend to selectively recall negative stimuli and events. For instance Nelson and Craighead (1977) found that depressed students (as identified by the BDI) recalled less positive feedback and more negative feedback than non-depressed subjects, while Gotlib (1981) found that clinically depressed patients incorrectly recalled having self-administered more punishers and fewer reinforcers than normal control subjects. Derry and Kuiper (1981) asked unipolar depressed patients, non-depressed psychiatric patients and normal controls to rate depressed and non-depressed content adjectives on a number of dimensions, and then to recall them. Depressed subjects showed superior recall for depressed content words while both control groups recalled more non-depressed content words than the depressed group.

A number of other studies have also demonstrated that congruence between mood state and the emotional tone of material to be remembered increases the probability of recall. For instance Teasdale and Russell (1983) found that induced mood at the time of recall differentially affected memory for previously presented negative and positive trait words: more positive words were recalled in a good mood than in a negative mood, while more negative words were recalled in the negative mood than in the positive mood. Similar results have been obtained from other studies (Teasdale and Fogarty, 1979; Fogarty and Hemsley, 1983; Clark and Teasdale, 1982).

Blaney (1986) summarised the available evidence and concluded that two basic phenomena underlie these effects of depression and depressed mood on memory. These are state dependent memory and mood congruence. The former asserts that what one remembers in a given mood is determined in part by what was learnt when previously in that mood: the emotional tone of the material to be learnt is not important. In contrast, mood

congruence states that material congruent with the present mood is most likely to be learned or recalled: the mood at the time the material was learned is not important. The Network theory (Bower, 1981) underlies both phenomena. This suggests that each emotion has a node in memory which is linked to other aspects of the emotion. The activation of the node spreads activation throughout the memory structures to which it is connected, spreading subthreshold activation. This means that when the node for depression is activated negative memories will also be activated and will therefore be more accessible and more likely to be recalled.

Research on the effects of depression on the recall of emotionally-toned material has, on the whole, been carried out by different researchers to those interested in the more general deficit in depression. There has, therefore, been little attempt to explain the general deficit in terms of theories put forward to account for the effects of the emotional tone of material on recall, and vice versa. One possibility is that negative material, but not positive and neutral material, is protected from the general memory deficit because of mood congruence and the operation of depressive schemata, which select material consistent with a negative perspective. However, the recall of neutral material is not always impaired in the studies which find impaired recall of positive themes (Teasdale and Russell, 1983; Breslow, Kocsis and Belkin, 1981) and the recall of negative material is similarly not always enhanced when the recall of positive material is diminished (Breslow, Kocsis and Belkin, 1981). The hypothesis that the recall of positive and neutral themes is affected by the same factors as those responsible for the general memory deficit while negative themes are protected cannot, therefore, explain all the relevant data. Equally, it is difficult to see how state-dependent learning and mood congruence can explain all the results from studies of the general deficit in depression. These studies usually involve the acquistion and recall of material in the same session and therefore presumably in the same mood state: if state-dependent learning was operating it should therefore enhance the recall of all material learned in that session and would therefore make memory deficit less likely, rather than explain why one is found. Mood congruence helps to explain why some material is more accessible in depression, but does little to explain why most material is learnt less well than usual and is less accessible.

There is therefore some difficulty in combining the results of research on general memory deficits in depression with those from research on the effects of the emotional tone of material on memory in depression. Nevertheless it can be concluded that there is considerable evidence that the amount of memory impairment shown by depressed subjects will depend on the emotional characteristics of the material to be remembered.

1.7.5 Summary

Several different models of memory have been used to direct research into the nature of memory deficits in depression. On the whole, studies based on the 'stages' models of information-processing have shown that registration and short-term memory are impaired in depression, but that once information has been learned depressed subjects do not forget it any faster than other subjects. Studies investigating detailed aspects of short-term memory have, in all but one case, shown that memory scanning was intact but that the overall reaction times were significantly longer, presumably due to deficits in encoding, decision- making or motor response. The results of studies investigating the strategies used to process information in depression suggest that depressed subjects fail to use encoding stategies that would facilitate later recall. Finally, there is evidence that the extent of impairment found in depression will vary according to the emotional tone of the material to be remembered.

1.8 CAUSES OF MEMORY IMPAIRMENT IN DEPRESSION

In his review of psychological deficits in depression Miller (1975) noted that three basis hypotheses had been proposed to explain the observed deficits. The first of these was 'Reduced Motivation'. This states that depressed subjects are simply not motivated to do well on tasks, or, alternatively, that they are motivated but are unable to sustain any prolonged motivation. The second hypothesis was 'Cognitive Interference', which postulated that deficits are the consequence of distracting thoughts, worries and poor self-esteem which compete for the depressed person's attention and therefore disrupt his/her performance on a variety of different tasks. The final hypothesis combined both

cognitive and motivational factors and was described as 'Learned Helplessness'. According to this hypothesis 'the perception of reinforcement as response independent, the expectation that responding is useless, and reduced motivation, are thought to produce the depressive deficit' (p257). Miller went on to state that although there was some evidence for each of these hypotheses, the issue was far from resolved.

Most explanations of the deficit in depression are still based around these three hypotheses, although 'Learned Helplessness' has been modified and is now more accurately labelled 'Response Bias'. The possibility that memory is intact in depression and that deficits are due to response difficulties caused by psychomotor retardation has also been considered, as has the possibility that the deficits result from changes in hemispheric function in the brain. Each of these hypotheses will now be reviewed. First, the evidence for 'Reduced motivation' is reviewed, followed by two other hypotheses which have connections with this: 'Response Bias' and 'Psychomotor retardation'.

1.8.1 Reduced Motivation

The possibility that memory deficits in depression are a consequence of reduced motivation or arousal and associated problems in sustaining attention has been considered by many researchers.

Friedman (1964) observed that many of the depressed patients in his sample protested before testing that they would be unable to do anything, yet after 'gentle persuasion' they usually became motivated and showed little evidence of deficit. Stromgren (1977) argued that the attitude of the tester to the patient was one of the most important determinants of whether a deficit would be seen because a supportive and encouraging approach could help the patients to overcome their lack of motivation and encourage them to expend more effort on the task.

Breslow, Kocsis and Belkin (1980) found most impairment in depressed patients on the 'Mental Control' subfunction of the WMS. They argued that this indicated that depressed patients had difficulty in sustaining attention. Siegfried, Jansen and Pahnke (1984) concluded that the memory

deficit in depression is quite generalised and is best conceptualised as a result of decreased arousal or activation.

Hart et al (1987a) concluded that depressed patients needed longer than controls to reach the same level of learning because of attentional and motivational deficiencies. They drew similar conclusions from their study looking at the performance of depressed subjects on a selective reminding procedure (Hart et al, 1987c). This showed that tasks requiring more effort for completion were impaired in depression. They also attributed their finding that elderly depressed patients showed psychomotor slowing on the Sternberg short-term memory scanning procedure to motivational deficiencies (Hart and Kwentus, 1987).

Glass et al (1981) found that depressed patients and controls did not differ in speed of memory scanning, a tapping speed test, and a simple reaction time test. However, the depressed group had significantly longer overall reaction times on the short-term memory test. Since they did not differ on the psychomotor tasks, the authors argued that this could not be due to simple motor slowing or reduced attention, but instead was due to the memory task being more complex: impairments would be found on any sufficiently complex task, presumably because such tasks demand more motivation and effort to complete than simpler tasks.

Several studies have looked at whether depressed subjects are more impaired on tasks presumed to require more motivation and effort. Calev and Erwin (1985) compared the performance of hospitalised depressed patients and controls who were not depressed on a recognition test and a recall test comparable in level of difficulty. Depressed patients were impaired overall, and performed better on the recognition items than the recall, in contrast to the controls who performed alike on both tasks. The authors suggest that this may be because the depressed patients found the recognition task easier because the words were provided for them and therefore it required less effort to complete.

As reviewed above, Weingarner et al (1981) found that depressed patients failed to use encoding operations that would be useful in reorganising input and would facilitate later recall, presumably because to do so required more effort than they could exert, but that they were

able to make use of organisation when material was clearly presented in a structured way. Ellis, Thomas and Rodriguez (1984) also found some evidence that tasks requiring more effort for completion were particularly impaired, in this case in students subjected to a depressed mood induction procedure. However, Watts and Sharrock (1987) found that free and cued recall were equally affected by depression despite the fact that cued recall was presumed to demand less effort than free recall. They concluded that there might be other types of effort involved in these tasks which made them less different on this dimension than anticipated.

Cohen et al (1982) tested the hypothesis that impairment in memory performance in depression is related to an inability to sustain effort. They correlated performance on a simple motor task (which measured the ability to sustain effort) with performance on a simple memory test. Depressed patients were significantly impaired on sustained motor performance and on the memory test. There was a very high correlation between the two measures and both were highly correlated with the intensity of depressed mood. The authors suggest that a general deficit in motivation in depression would account for these findings, and that deficits will be found in any cognitive task providing it is sufficiently complex and demands enough effort.

Interest in the concept of 'effort' has in part developed from the work of Kahneman (1973) who proposed a capacity model of attention and suggested that the amount of capacity or effort supplied to a task will depend on the difficulty of the task. As Eysenck (1982) has noted, Kahneman's key concept of effort is not precisely defined: sometimes it seems to be synonymous with concentration whilst at other times it is used in a broader sense, as when Kahneman refers to 'a nonspecific input, which may variously be labelled 'effort', 'capacity' or 'attention'. Tyler et al (1979) defined 'effort' as the amount of the limited-capacity central processor which is engaged in performing a task, and therefore used the term to encompass attention. However, it may be more appropriate to distinguish 'effort' and 'attention' so that effort denotes a deliberately initiated activation of information processing power, which results in a certain amount of attention or processing capacity being focused on a particular task (Dornic, 1977; Eysenck, 1982). There is, therefore, some confusion as to what the term means.

Kahneman (1973) also suggested that attentional capacity varies both within and among individuals. This led Hasher and Zacks (1979) to devise a model of cognitive operations which integrated research on memory in young children, the elderly and individuals under stress with that on normal college students. This model draws distinctions between cognitive processes that require effort or cognitive capacity (terms used synonymously) for completion, and processes that can be accomplished automatically without sustained intent or focused attention. Effortful processes include imagery, rehearsal, organisation and mnemonic techniques while automatic processes encode fundamental aspects of the flow of information such as spatial, temporal and frequency-of-occurrence information. The two types of processing are considered to be qualitatively different; for instance, performing one effort-demanding process limits the efficiency of other such operations that may be processed at the same time, while this is not true of tasks requiring automatic processing. In addition effortful processes are intentional and benefit from practice, while automatic processes occur without intention and do not benefit from practice.

Hasher and Zacks (1979) proposed that depression, like age and high arousal, reduces the amount of attentional capacity available and therefore would be accompanied by a deficit on effortful tasks, while leaving performance on automatic tasks intact. Evidence for this pattern of deficit is given below; however it does not necessarily confirm Hasher and Zacks' (1979) assertion that processing capacity is reduced in depression. If, as Dornic (1977) suggested, the concepts of attention and effort are separated it can be hypothesised that reduced motivation would cause this pattern of results by reducing the amount of effort put into the task and therefore the amount of attentional capacity used: attentional capacity may be intact but not utilised due to a lack of effort. Hasher and Zack's assertion that processing capacity is reduced in depression therefore needs confirmation, but there is some evidence that they were correct to suggest that effortful tasks are impaired in depression while automatic tasks are protected.

This was investigated by Roy-Byrne et al (1986). The performance of hospitalised unipolar and bipolar depressed patients and non-depressed controls was compared on two tasks, both of which included tests of

intentional and unintentional learning: the former was included as a measure of effortful processing, and the latter as a measure of automatic processing. The depressed subjects differed significantly from the controls on both effortful tasks, but not on the automatic tasks. The authors concluded that these results supported the view that depressed individuals are impaired on tasks that require effort.

Hart et al (1987c) also investigated effortful and automatic processing in depressed patients. Like Roy-Byrne et al (1986) they found that a task which required effort was impaired in depressed patients compared to non-depressed controls, but there was no difference on an incidental learning test. Hasher and Zacks (1979) found no difference between depressed students chosen on the basis of their BDI scores and nondepressed controls on the judgment of how often various pictures were presented over eight trials: this was presumed to reflect automatic processing. In an associated study using similar subjects, they found that the depressed students were impaired on a task presumed to demand effortful processing (Hasher and Zacks, 1979).

Some researchers suggest that depressed patients can overcome their lack of motivation if they are given sufficient encouragement (e.g Friedman, 1964; Stromgren, 1977) and that it is not that they are unable to make the effort necessary to complete difficult tasks, but that they are unwilling to do so. In contrast, other researchers postulate that there are cognitive, neurochemical or other physiological changes in depression which mean that depressed people have reduced levels of motivation and arousal, or reduced attentional capacity, and are unable to sustain effort even if they try. As already noted, Hasher and Zacks (1979) suggested that attentional capacity (which they believed to be synonymous with effort) was reduced in depression and therefore depressed subjects could not exert more effort if they wanted to. This hypothesis has yet to be confirmed. Others have suggested that the inability to sustain effort is due to changes in motivation and arousal which are mediated by the catecholaminergic systems (Weingartner et al, 1981; Cohen et al, 1982; Hart et al, 1987b; Roy-Byrne et al, 1986). Further research is needed to clarify this issue. It has also been suggested that changes in balance between the cerebral hemispheres are responsible for the link between effortful processing and memory failure (Weingartner and

Silberman, 1982). The evidence for laterality changes will be reviewed later in the chapter: the link between these and the decreased effort shown by depressed subjects remains highly speculative. What mechanism underlies the relationship between motivation, effort and memory in depression is not known; what is clear is that depressed people are particularly impaired on tasks requiring effort for successful completion.

1.8.2 Response style

One of the hypotheses put forward by Miller (1975) was labelled 'learned helplessness'. This suggested that depressed subjects view reinforcement as response independent, expect responding to be useless, and lack the motivation to do so; they therefore may not produce an answer even when they know it. From this perspective the memory of the subjects may be intact; what is affected by depression is the likelihood of them responding. The memory deficit seen in depression is therefore regarded as a consequence of response style. This explanation is related to the 'reduced motivation' hypothesis (Section 1.8.1) in that it is also a motivational explanation; however in this case it only explains the effects of motivation on retrieval, not on learning.

This hypothesis is supported by the study of Miller and Lewis (1977) who used signal detection analysis (McNicol, 1972) to distinguish between basic memory capacity (d') and decision stategy (β) on recognition memory tests in depressed psychogeriatric patients who were demented or depressed, and in community controls. The depressed patients had significantly higher d' scores than the demented patients; however they did not differ significantly from the controls. β scores were significantly higher in the depressed group than in either of the other groups, indicating that the depressed patients had adopted a more conservative response criterion. Thus the depressed patients did not differ from the community controls in their ability to recognise geometric figures, but they needed to be very certain that they had correctly recognised a figure before they responded positively.

This finding is supported by the study by Dobson and Dobson (1981) of problem-solving in depressed students. Students scoring above 10 on the BDI were less efficient problem solvers than students scoring below

10: this was due to a conservative response style whereby they needed a lot of information to confirm previous information before they would respond.

Not all studies have found evidence of a conservative response style in depression. Hilbert, Niederehe and Kahn (1976) undertook a signal detection analysis of accuracy data derived from a Sternberg short-term memory procedure given to elderly depressed and non-depressed subjects. They found that both the highest memory efficiency and the strictest criterion level were found in the control subjects who were not depressed, rather than in the depressed group. Wolfe et al (1987) found that unipolar depressed patients made more false positive responses and fewer false negative responses than non-depressed controls on the recognition section of the Rey Auditory Verbal Learning Test: the reverse would be expected if the depressed subjects had a more conservative response style. Calev and Erwin (1985) checked the number of false positive answers given on a recognition memory task by depressed in-patients and matched controls in order to rule out the possibility of response bias: there was no significant difference between the two groups.

Watts, Morris and MacLeod (1987) gave verbal recognition memory tests to a group of predominantly endogenous depressed patients and controls matched for age, educational level and verbal intelligence. The depressed subjects had significantly lower d' scores, showing a strong effect of depression on recognition memory. There was no difference in β scores between the two groups, indicating that the depressed patients did not have a more conservative response style. Half the subjects had been asked to vocalise words as they were presented in order to ensure some encoding: the results showed that depressed patients gave more false positive responses than controls in this condition, but fewer than the controls in the non-vocalisation condition. The authors speculate that this may have been because the former condition required additional processing and it suggests that procedural variables determine whether depressed subjects will make more or less false positive errors than controls; the authors note that this makes it extremely difficult to demonstrate a clear effect of depression on β.

Dunbar and Lishman (1984) have demonstrated that both 'pure memory' and the response criteria of the depressed subject will vary according to the hedonic tone of the material. There was no overall difference in recognition rates on a verbal recognition task, but depressed and non-depressed subjects did differ in the type of material they could recognise with ease. Depressed in-patients had lower d' scores for positive words and higher d' scores for negative words than non-depressed controls. There was no difference between the groups for neutral words, suggesting that 'pure memory' was not affected by depression. Depressed subjects had higher β scores for positive and neutral words, indicating a conservative response criteria. The authors concluded that there was evidence for high β scores in depression, but it was not a universal effect and varied with, amongst other things, the emotional tone of the material being handled.

In summary, there is conflicting evidence regarding response criteria in depression. Miller and Lewis (1977) reported evidence that elderly depressed patients had conservative response criteria. Although this study was criticised by Watts et al (1987) for using geometric material which may be less sensitive to the memory impairment in depression than verbal stimuli, its conclusions have been supported by the findings of Dobson and Dobson (1981) from an investigation of problem solving. Other studies, however, have not supported these conclusions. They have either not found an effect of depression on the likelihood of subjects making false positive errors or having high β levels, or have found that the response criteria and pure memory processes both vary according to the type of material being processed. The issue of whether memory impairments in depression are artifacts resulting from conservative response criteria has not therefore been resolved.

1.8.3 Psychomotor Retardation

It is generally accepted that many depressed individuals experience some degree of psychomotor retardation (reviewed by Miller, 1975), and that this is particularly true of those with endogenous depression (Nelson and Charney, 1981). It is possible that the impairments shown by depressed people on memory tests are a consequence of their slower

thought processes or motor response, rather than an impairment in memory itself.

According to Weckowicz et al (1972) psychomotor retardation may result from physiological mechanisms such as a 'central inhibitory state', or from complex cognitive and motivational mechanisms such as lack of interest, inattention, anxiety or intrusive thoughts. Psychomotor retardation should perhaps be seen as a mechanism by which reduced motivation and/or intrusive thoughts produce impairment, rather than a cause of impairment in its own right.

Several studies have found that depressed patients showed psychomotor slowing on a variety of cognitive tests. Weckowicz et al (1972) gave tests of intellectual functioning and speed of performance to depressed in-patients, and to normal controls matched for social-economic background. The depressed subjects performed significantly slower than controls of comparable ages on the majority of the tests. They concluded that as the retardation was probably due to cognitive and motivational mechanisms depressed people might be able to perform as well as controls if given sufficient encouragement.

Caine (1981) gave depressed in-patients a detailed neuropsychological screening test, and found that, amongst other things, they were impaired on tests of motor processing speed. Rush et al (1983) also used standardised neuropsychological tests: endogenous depressed patients were significantly impaired on a test of psychomotor retardation but non-endogenous depressed patients were not. Rosen and Fox (1986) found that depressed patients had significantly slower response times than normal controls on a serial sevens test, even when demographic variables such as age, sex and educational levels were controlled for. Depressed patients have also been found to perform significantly slower than normal controls on the Digit Symbols test from the WAIS (Hart et al, 1987b). Several studies have shown that they have a slower overall response rate on a Sternberg's short-term memory scanning procedure, without a concomitant slowing in speed of memory scanning: this reflects slowing in initial encoding, decision making or time to respond (Glass et al, 1981; Hart and Kwentus, 1987; Hilbert, Niederehe and Kahn, 1976; Koh and Wolpert, 1985).

Abrams and Taylor (1987) administered a variety of neuro-psychological tasks to depressed in-patients and normal controls. They found that performance speed, as assessed by reaction times, accounted for a significant proportion of the poor performance of depressed patients on these tasks. However, the depressed patients were still significantly impaired compared to controls when the effects of retardation were controlled for. This indicated that although psychomotor retardation contributed to impairment in these subjects, it was not the only cause.

In summary, depressed patients have been shown to respond more slowly than control subjects who were not depressed on a variety of cognitive tests. Psychomotor retardation was clearly related to poor performance on a battery of neuropsychological tests (Abrams and Taylor, 1987) but did not completely explain the difference found between depressed and control subjects; other factors were also affecting performance. Retardation may be due to reduced levels of motivation, or to other symptoms of depression such as intrusive thoughts, which themselves have been shown to affect memory performance (Sections 1.8.1 and 1.8.4). It is perhaps most appropriate, therefore, to view retardation as a means by which other factors such as reduced motivation exert their influence on memory performance.

1.8.4 Cognitive Interference

Early research on memory in depression (Cronholm and Ottosson, 1961) suggested that impairment was a result of constant interference from depressive thoughts and a reduced ability to concentrate. Sternberg and Jarvik (1976) also hypothesised that depressed patients were subject to constant interference from ruminative depressive thoughts and that this, together with reduced motivation, accounted for the impairments they had detected.

There has been virtually no research on the role of cognitive interference in causing memory impairment in depression. It has been speculated that such interference lies behind the supposed reduction in attentional capacity in depression (Hasher and Zacks, 1979; Ellis et al, 1985) but this possibility has not been investigated and, as indicated

above (Section 1.8.1) it is not clear that attentional capacity is reduced in depression.

Watts and his colleagues have looked directly at the link between intrusive thoughts, concentration and memory (Watts and Sharrock, 1985; Watts, MacLeod and Morris, 1988). In their first study, depressed in-patients were interviewed about their experience of concentration problems and then asked to read a short passage and to indicate each time they lost their concentration. This was usually due to mind-wandering. Concentration lapses on this task were found to be signif-icantly correlated with both free and cued recall (r=-0.37 and -0.34 respectively).

In their second study they explored the distinction between different kinds of lapses of concentration in depressed patients and found that patients distinguished between 'mind-wandering' and 'blanking' (where the mind goes blank). As in the earlier study mind-wandering was the most common lapse reported by depressed patients, and the proportion of such lapses on a reading task was found to correlate significantly with the reports of the frequency of mind-wandering in everyday life.

The two types of concentration problems had different task per-formance correlates. Reports of mind-wandering, but not of blanking, were found to be significantly correlated with poor prose passage recall, while blanking, but not mind-wandering, was related to slow planning times on the 'Tower of London' task. The authors suggest that this indicates that not all performance deficits in depression can be attributed to interference from competing thoughts.

The authors applied Shallice's model of the regulation of attention to this data (Shallice, 1978). According to Watts, MacLeod and Morris (1988) this model assumes that many cognitive processes can be performed with-out conscious attention but are done more efficiently with it. At any one time there are a number of processing structures associated with particular tasks ('action systems') competing for dominance; the one that is dominant receives conscious attention. Watts, MacLeod and Morris (1988) suggest that mind-wandering can be seen as a loss of dominance of the action system concerned with the task in hand in favour of another

action system. This may be because no one action system is dominant, perhaps because depressed patients lack motivation and do not give adequate priority to tasks of low personal importance; the associated action systems therefore do not retain their dominance. This suggests that intrusive thoughts are able to obtain dominance in the attentional system because of a lack of motivation and therefore links the 'intrusive thoughts' and 'motivational' explanations of impairment in depression.

Shallice also postulated the existence of the Supervisory Attentional System (Norman and Shallice, 1986) which is hypothesised to be a super-ordinary planning system which gives assistance to action systems which need help to maintain dominance. It does this partly by inhibiting competing action systems. Watts, MacLeod and Morris (1988) suggest that this inhibitory action may in some cases become generalised and that this would give rise to the phenomenon of 'blanking'. They speculate that this may be particularly likely to happen on effortful tasks and that if this is the case then findings that depressed patients tend to adopt processing strategies which demand little effort (Weingartner and Silberman, 1982) may in part be because this is an adaptive stategy which avoids the generalised inhibition that might result from cognitive effort.

This research by Watts, MacLeod and Morris (1988) is concerned primarily with concentration rather than with intrusive thoughts; it has already shown that not all lapses in concentration are caused by interference from such thoughts. There seems to have been no other research on concentration and cognitive interference in depressed subjects; it therefore remains a possibility that cognitive interference is a cause of memory deficits in depression, even if, as Watts, MacLeod and Morris (1988) suggest, it cannot explain all types of deficit.

1.8.5 Lateralised Hemispheric Dysfunction

Weingartner and Silberman (1982) suggested that depressed people are frequently impaired on tasks requiring effortful processing because there are changes in the usual balance between the cerebral hemispheres. They argue that there is evidence to support characterization of left hemisphere cognitive style as detailed, serial or intentional, and that of

the right as holistic, parallel and incidental: they speculate that the deficits in effortful processing found in depressed subjects may represent a decrement in left hemisphere function.

This speculation is based on two bodies of data, both of which are reviewed elsewhere (Wexler, 1980; Fromm-Auch, 1983) and will not be reviewed in detail here. The first of these links the left hemisphere with the mediation of positive affective states, and the right with negative affective states. This is based upon diverse experimental sources, such as studies of lateral eye movements, electroencephalographic studies, animal studies and studies of patients with unilateral brain lesions. The hypothesis that depression may be a manifestation of right hemisphere activity is not firmly established and the evidence as yet is preliminary (Fromm-Auch, 1983). The second body of data concerns shifts in hemispheric function as a result of depression. Wexler (1980) concluded that the initial results from studies of laterality produced a picture that was very confused, presumably due to the large number of different experimental measures and designs: these included dichotic listening and galvanic skin response in addition to those listed above. Some studies had found evidence for right hemispheric dysfunction, some for left hemispheric dysfunction, and others for a shift away from left hemisphere towards right hemisphere function.

The results of more recent studies which have used neuropsychological tests designed to locate lesions to right, left or both hemispheres, or other psychological tests, are equally confused. Some have concluded that there is evidence of right hemisphere dysfunction (Fromm-Auch, 1983; Abrams and Taylor, 1987; Taylor and Abrams, 1987), whilst others have found no evidence to support this (Taylor, Greenspan and Abrams, 1979; Calev et al, 1986; Sapin et al, 1987). It has also been suggested that processing normally carried out in the left hemisphere is shifted to the right (Silberman et al, 1983) and that there is evidence for bilateral dysfunction (Taylor and Abrams, 1983). Weingartner and Silberman (1982) concluded that 'a great deal more data need to be collected before the presence of laterality changes in depression are firmly established' and this remains the case.

In summary, the evidence for lateralised hemisphere dysfunction in depression is limited. It does, however, raise the possibility that changes in cerebral function are underlie at least some of the impairment in memory performance observed in depression.

1.9 THE SPECIFICITY OF MEMORY IMPAIRMENT IN DEPRESSION

There has been some interest in whether there is a pattern of memory impairment which is specific to depression and which, therefore, can be used to differentiate between patients suffering from depression and those with other psychiatric conditions.

The question of how to differentiate patients suffering from depression from those suffering from dementia has received particular attention. This is because cognitive impairment in depression can be so severe that it can be difficult to distinguish it from impairment due to dementia, particularly in elderly patients (Albert, 1984; Klerman and Davidson, 1984). The fact that early-stage dementia patients are likely to be depressed in response to their self-perceived impairments compounds the difficulties involved in reaching an appropriate differential diagnosis (Feinberg and Goodman, 1984). The implications of misdiagnosing a patient as demented rather than depressed can be severe as the former is a progressive incurable condition while the latter is treatable and the associated cognitive impairment is reversible. Much attention has, therefore, been directed to the syndrome of 'pseudodementia'.

1.9.1 'Pseudodementia'

In his comprehensive study of this syndrome Wells (1979) defined patients suffering from pseudodementia as patients showing classic signs of dementia (impairment of orientation, memory, judgment and intellectual functions such as comprehension, calculation and knowledge) but in whom cognitive dysfunction eventually disappears after resolution of the underlying psychiatric disorder. As Wells (1979) points out depression is not the only psychiatric condition associated with 'pseudodementia': four of his sample of pseudodemented patients had diagnoses other than depression, notably conversion reactions and schizophrenia. However, pseudodementia is particularly associated with depression

Several studies have looked at the extent of erroneous diagnoses of dementia in patients who were subsequently considered to have had pseudodementia. Notts and Fleminger (1975) found that only 45% of a group of 35 patients given a primary diagnosis of presenile dementia at St Guy's Hospital between 1950 and 1969 demonstrated progressive decline: nine per cent were thought to have had depression at the time they received their original diagnosis. Smith and Kiholi (1981) followed up 200 consecutive attenders with a provisional diagnosis of dementia referred to the Neuro-psychiatric Institute in Sydney. After extensive evaluations ten per cent were considered to have the pseudodementing syndrome. Ten were diagnosed as having a depressive illness and therefore received treatment for depression: they all responded to this and returned to normal levels of cognitive functioning. On the basis of these and other similar studies Feinberg and Goodman (1984) estimated that even in a group of well-diagnosed demented subjects between five and fifteen per cent would be found at follow-up to have had an affective illness only.

The results of depressed and demented patients on instruments commonly used to screen for altered brain function show why it is sometimes difficult for clinicians to distinguish between irreversible dementia and pseudodementia. McHugh and Folstein (1979) compared the scores on the MMSE (which covers orientation, memory, attention, language and construction) of a group of geriatric patients admitted for depression with that of a group of non-depressed patients with Alzheimer-type dementia. The results of the depressed group showed a decremental curve from normal to a few severely impaired individuals with scores at the level of the mean score of the demented group. It would be extremely difficult to differentiate the severely impaired depressed subjects from demented subjects on the basis of their scores on the MMSE.

Rabins, Merchant and Nestadt (1984) also found that it would be difficult to distinguish between individual impaired-depressed and demented patients on the basis of these scores, even though the mean score of a group of depressed patients with pseudodementia was significantly higher (less impaired) than that of a group of patients with irreversible dementia. Both studies suggest that memory impairment due to depression is confounded by the effects of ageing on memory,

making it particularly likely that cognitive deficits in elderly, as opposed to younger, depressed patients would reach levels comparable with those shown by demented patients.

A number of studies have attempted to distinguish memory impairments associated with depression from those associated with irreversible dementia. Weingartner and Silberman (1982) carried out a series of studies to investigate characteristics of memory failure in the two groups. They concluded that while depressed patients were able to make use of organisational and semantic relationships between words, provided the words were grouped in a way that made the relationships explicit, demented patients did not use organisational or semantic relationships as an aid in encoding, even when the relationships were obvious. This meant that even when demented and depressed patients obtained similar scores on the Wechsler Memory Scale (WMS) and a recall test using random words, demented patients were more impaired than depressed patients on a recall test with related words. The authors also concluded that depressed patients had intact access to semantic memory, while demented patients did not.

In a series of three studies Hart and his colleagues compared the performance of depressed and demented patients on a recognition memory test designed to investigate the rate of forgetting; a selective reminding procedure; and the Digit Symbols test combined with an incidental learning trial (Hart et al, 1987a; Hart et al, 1987b; Hart et al, 1987c). The depressed patients showed normal forgetting over time, while demented patients had a problem with the consolidation of material; they were less impaired than demented patients on the selective reminding procedure, and scored significantly higher on the incidental learning trial than the demented patients. The authors concluded that an incidental learning task may be particularly useful for differentiating between depressed and demented patients.

There is, therefore, some evidence that demented patients can be differentiated from depressed patients on the basis of their cognitive performance: according to Tariot and Weingartner (1986) tests of semantic memory, effortful and automatic processing would be particularly valuable in doing this as demented patients would be expected to be impaired on

all three, while depressed patients would only be impaired on tests of effortful processing. Less is known, however, about whether there is a pattern of cognitive functioning which is unique to depression and not shown by other psychiatric conditions.

1.9.2 Memory Impairments in Depression Compared to Those Found in Other Psychiatric Conditions

Miller (1975) concluded that his extensive review of psychological functioning in depression had found little evidence for impairments in functioning that were unique to depression: their performance on cognitive, motor and perceptual tasks tended to be either better or similar to the impaired performance of schizophrenics on such tasks. There has been little work in this area since that time.

In one of the few studies comparing schizophrenic and depressed patients, Taylor and Abrams (1983) used a battery of neuropsychological tests to differentiate between dominant and non-dominant hemispheric impairment. The results showed that schizophrenics had significantly more dominant hemisphere and global impairment than depressed patients, but that the two groups did not differ in the incidence of non-dominant errors. Similar results were obtained by Taylor and Abrams (1987), who found that only a subgroup of schizophrenics had dominant hemisphere impairment in addition to non-dominant impairment. Thus, it is difficult to reliably differentiate between depressed and schizophrenic patients on the basis of the pattern of deficit on tests of hemispheric function. The same conclusion was reached in an earlier study (Taylor, Greenspan and Abrams, 1979). Frame and Oltmanns (1982) also failed to find memory deficits in depressed patients which were not also shown by schizophrenic patients. Koh and Wolpert (1983) found no significant differences between the performance of unipolar and bipolar depressed patients and schizophrenics on the Sternberg short-term memory scanning procedure while Cutting (1979) found that there were no significant differences between acute schizophrenics and depressed patients on tests of verbal learning and pattern recognition memory, but that chronic schizophrenics were significantly more impaired on all tests.

There has been virtually no research on how the memory impairments found in depressed patients differ from those shown by anxious patients: the exception is a recent paper (Brands and Jolles, 1987) which compared the performance of depressed and anxious patients on the Sternberg short-term memory scanning procedure: depressed patients had significantly slower overall reaction times, and showed slower memory scanning on one task.

In summary, there is no conclusive evidence for deficits that are unique to depression and not also shown by schizophrenic patients. There is very little research comparing depressed patients with patients with anxiety disorders: the available evidence suggests that anxious patients may show less psychomotor retardation and be less impaired on short-term memory tasks.

1.9.3 Memory Impairments in Sub-Types of Depression

Miller (1975) reviewed the evidence for different patterns of memory impairment in different sub-types of depression. He concluded that 'one of the most striking findings to emerge from the review is that there are so few differences in the deficits manifested by different sub-types of depressives. In fact, the differences that have been found generally have been differences in the degree of impairment exhibited, rather than in the type of impairment'.

Few studies since this review have compared sub-types of depressed subjects. As indicated above, Koh and Wolpert (1983) did not find any significant differences between unipolar and bipolar depressed patients on measures of short-term memory scanning and overall reaction time: neither group had any acute psychotic disturbance at the time of testing. Brand and Jolles (1987) used similar memory tests and found that while unipolar patients were both slower overall and had short-term memory impairments compared to non-depressed controls, bipolar patients were not impaired. Robertson and Taylor (1985) found that unipolar depressed prisoners were significantly more impaired on a battery of memory tests than bipolar prisoners who were predominantly in the manic phase of their illness: there were no significant differences between unipolar psychotic and unipolar reactive depressed prisoners. Calev et al (1986)

compared depressed patients, euthymic patients who had a history of bipolar affective disorders, and normal controls on verbal and non-verbal memory tasks. Depressed patients were significantly impaired, but this was not true of the euthymic patients. Wolfe et al (1987) found that depressed bipolar patients were more impaired than unipolar patients on the Rey Auditory Verbal Learning Test: they suggest that this may have been because the bipolar group were more severely ill.

The conflicting results on differences between unipolar and bipolar patients are presumably due to differences in the bipolar patients used: some have been euthymic (Calev et al, 1986), some in a manic phase (Robertson and Taylor, 1985), whilst others have been depressed (Wolfe et al, 1987).

Fromm-Auch (1983) compared psychotic and neurotic depressed in-patients on a neuropsychological test battery. The psychotic patients had more abnormal test profiles, but the groups did not differ in the pattern of impairment. The authors concluded that the psychotic patients showed more impairment because they were the more severely ill group. Miller (1975) suggested that the degree of memory impairment shown was related to the severity of depression, not to the sub-type: the results of studies published since this review support this suggestion, but the available data is very limited.

Silberman et al (1985) compared the memory performance of sub-types of depression defined not by their diagnostic sub-type, but by the response of the depressed patient to the Dexamethosone Suppression Test (DST). They sugested that patients exhibiting abnormal DST responses may represent an endogenous or metabolic depression and might, therefore, be expected to show more memory impairment than those with less evidence of physiological dysfunction. The results were opposite to those expected: normal DST response was associated with increased memory impairment. The groups did not differ in the severity of depression. This study had small subject numbers and was intended to be preliminary. It does, however, raise the possibility that there may be differences in memory performance in sub-types of depression which can not be explained by the severity of depression.

1.9.4 Summary

In summary, although different patterns of memory impairment have been identifed in depressed patients and those with dementia, there is little information on how the memory performance of depressed patients differs from that shown by other psychiatric groups, or if and how sub-types of depression differ. The conclusions reached by Miller in 1975 still stand: there is no evidence for impairments which are specific to depression and not shown by other psychiatric groups, notably schizophrenics; and there is little evidence for differences between sub-types of depression which cannot be explained by differences in severity of depression.

1.10 SUMMARY OF LITERATURE ON MEMORY IN DEPRESSION

This review of memory in depression has shown that there is considerable evidence that depressed people are impaired on a variety of memory tasks, and that these impairments abate as the depression remits. There is some conflicting evidence which may be related to the type of depressed subjects used.

The majority of studies have used depressed psychiatric in-patients: in each case the depressed subjects were significantly impaired, and/or showed evidence of improvement in memory performance as they recovered from the depression (Sections 1.4 and 1.6). Only two out of six studies using depressed psychiatric out-patients, and three out of five using medical and geriatric in-patients, have found evidence of significant impairment. This suggests that memory impairment is most prevalent in depressed individuals who are having in-patient care, and may not be found in less severely ill depressed patients.

The issue of what types of depressed individuals show impairment has not, however, been resolved: for instance two studies (Ellis, Thomas and Rodriguez, 1984; Ellis et al, 1985) have found that students subjected to a depressed mood induction procedure were significantly impaired on verbal memory tests. As already noted (Section 1.4) it is difficult to see why these subjects were impaired while out-patients and medical patients with clinically significant levels of depression have not been. This may

be due to tighter experimental control in the former studies, or to differences in the type of memory test used. It is clear that more research is needed to investigate which depressed people will have memory impairments.

There is some evidence that the severity of memory impairment is related to the severity of depression (Section 1.5). However again there is conflicting evidence: only seven out of eighteen studies reporting a relationship between the severity of depression and performance found that the two were significantly related. This is surprising given the evidence that memory improves as depression abates, and the suggestion that more severely ill depressed patients, as assessed by the need for in-patient care, are most likely to have impaired memories. Reasons for the failure to find consistent results in this area have already been discussed (Section 1.5) and again it is clear that more research is needed to clarify this issue.

Turning to the characteristics of memory impairment in depression, there is evidence that while learning is impaired, forgetting or consolidation are not affected; that short-term memory scanning is intact but there are impairments in the encoding, decision-making or motor response stages of short-term memory; and that depressed subjects process information in inefficient ways, unless structure and organisation are clearly provided (Section 1.7). The extent of impairment will be affected by the hedonic tone of the material to be remembered. It has been shown that depressed subjects are particularly impaired on tasks requiring effortful processing, and therefore may show impairments on any memory task provided it is sufficiently complex and requires sufficient effort.

Related to this, there is some evidence suggesting that memory deficits in depression are caused by reduced motivation, arousal or attention: one way in which reduced motivation may affect performance is by causing psychomotor retardation (Section 1.8). Some studies have suggested that depressed subjects may not have impaired memories at all, but instead have conservative response criteria which mean that they do not respond even when they know an answer: other studies have not, however, supported this conclusion and the role of response selection in

the aetiology of memory deficits in depression remains unproven. The possibility that interference from intrusive thoughts reduces performance in depression has attracted very little research, although it was suggested as a cause of impairment by early researchers in the field. The final hypothesis put forward to account for impairment in depression is that it is caused by changes in laterality between the cerebral hemispheres: there is little conclusive evidence to support this.

Although it has been shown to be possible to differentiate between depression and dementia on the basis of the pattern of memory performance, there is little evidence for memory deficits which are specific to depression, or to a particular depressive sub-type. Miller suggested in 1975 that the extent of impairment shown was more related to the severity of the psychiatric illness than to the diagnostic group or sub-type of depression: there has been little progress in this area since then and the limited available evidence supports this suggestion.

In summary, it is clear that there are still many unanswered questions about the nature, aetiology and specificity of memory impairments in depression. In addition all the reviewed studies have been concerned with memory performance on experimental memory tests: there is no information on how this relates to the memory performance of depressed individuals in everyday life.

1.11 OUTLINE OF THESIS

As indicated above (Section 1.1) this thesis is concerned with three aspects of memory in unipolar clinically depressed subjects: their performance on laboratory memory tests; their reports of memory problems in everyday life and the relationship of these reports to performance on the laboratory tests; and the memory performance of depressed patients in an important everyday situation. It addresses the following questions:

1) Are clinically depressed psychiatric patients significantly impaired on a battery of memory tests compared to controls who are not depressed?

2) Do depressed psychiatric patients show the same pattern of memory impairment as anxious psychiatric patients, and do the two sub-types of depressed patient differ significantly from each other?

3) Is the severity of memory impairment related to the severity of depressed mood, to the severity of the frequently concomitant anxiety, or to neither?

4) Is there a significant relationship between the severity of impairment on the test battery, and the extent of memory problems reported by the subject in everyday life?

5) Do depressed subjects show significant memory impairments in an important everyday situation - the general practice consultation.?

The subjects and methods used in this thesis are described in detail in Chapters Two and Three.

METHODS: MATERIALS AND PROCEDURE

2.1 INTRODUCTION

Chapters Four to Seven of this thesis investigate the performance of depressed patients on a battery of laboratory memory tests, their reports of memory problems in their daily lives, and the relationship between the two. The data are derived from a battery of memory tests which was given to three groups of subjects: a group of non-psychotic depressed patients; an anxious (but non-depressed) group; and controls who were neither depressed or anxious. As the battery of tests, the experimental procedure and the subjects used are common to these chapters they are introduced in two preliminary chapters: this chapter is concerned with the memory tests used and the procedure; the next, Chapter Three, describes the subjects used.

2.2 MATERIALS

2.2.1 Memory Test Battery.

This comprised tests of registration and immediate memory, retention and forgetting, and retrieval. Memory for several different types of material was tested, including pictures (Picture Recognition test), numbers (Digit Span Forwards and Backwards), words (Free Recall test and Serial Learning test), and prose (Prose Passage Recall). The tests used are listed in Table 2.1, which also shows the type of material, the mode of recall, and the aspect of memory tested in each case.

Table 2.1(a) categorises the tests included in this test battery by type of material used and type of recall measured, while Table 2.1(b) indicates both the aspects of memory tested in this battery and those aspects which were not tested. These show that recognition was only tested using non-verbal material (Picture Recognition test) while cued and free recall were only tested using verbal material (words; Free Recall test, Serial Learning test and Paired Associate Learning). It will therefore be difficult to draw conclusions about the effects of depression on retrieval from this battery. If depressed patients are impaired on the free recall tests but not on the recognition test this may reflect a retrieval problem or may indicate that depressed patients have better memory for non-verbal than verbal memory. In order to explore the effects

of depression on retrieval the same type of material needs to be included in the free recall, cued recall and recognition tests: for instance Watts and Sharrock (1987) report a study in which memory for a prose passage was tested using all three types of recall. Ideally tests also need to be matched for difficulty in order to ensure that differences in performance on these tests are due to the type of recall rather than to the level of difficulty of the task (Calev and Erwin, 1985). The tests included in this test battery were not matched for difficulty nor did they test memory for the same type of material with all three types of recall. It will therefore be difficult to draw firm conclusions about the effects of depression on retrieval as different levels of impairment on the free recall, cued recall and recognition tests may reflect differences in type of material used or task difficulty rather than a differential effect of depression on different types of recall

As non-verbal and verbal memory were tested using different types of recall it will be difficult to draw conclusions from this battery about the relative effects of depression on verbal and non-verbal memory. Different methods were also used to look at the extent of forgetting of verbal and non-verbal memory: the verbal task (Serial Learning) was presented several times before a final trial on which recall was tested without the prior presentation of the target words while, in contrast, the target pictures for the Picture Recognition test were presented once only and then the rate of forgetting measured over several trials. Comparisons of the proportion of verbal and non-verbal material forgotten during the course of the testing session will therefore be difficult to interpret.

This memory test battery will not, therefore, provide conclusive information on whether depressed subjects have a retrieval deficit, or whether they have a differential deficit for verbal rather than non-verbal naterial (or vice versa). It will, however, show whether depressed and anxious subjects have difficulty learning new material and/or in retaining that information in memory. It will also indicate whether the learning deficit is most evident on unconnected words or on a structured prose passage.

TABLE 2.1 MEMORY TEST BATTERY

TEST	TYPE OF MATERIAL	TYPE OF RECALL	ASPECT OF MEMORY MEASURED			
			IMMEDIATE LEARNING	SPEED OF LEARNING	FORGETTING/ RETENTION	RETRIEVAL
Picture Recognition	Pictures	Recognition	Trial One		Trial Four compared with Trial One	Compared with Free recall tests
Free Recall	Words, visually presented	Free recall	via serial position curve			
Serial Learning	Words, visually presented	Free recall	Trial One	Trials Two to Four		
Prose Passage recall	Words, orally presented	Free recall	Passage One, immediate recall		Delayed recall versus immediate recall	
Paired Associate Learning	Words, orally presented	Cued recall	Trial One	Trials Two to Four	Trial Four versus Trial Five	Compared with free recall tests
Digit Span, Forwards and Backwards	Digits, orally presented	Free recall	Yes			

TABLE 2.1(a) TESTS CATEGORISED BY TYPE OF MATERIAL USED AND TYPE OF RECALL MEASURED

TYPE OF MATERIAL	TYPE OF RECALL		
	FREE RECALL	CUED RECALL	RECOGNITION
Words	Yes (Free Recall, Serial Learning)	Yes (Paired Associate)	No
Prose	Yes (Prose Passage recall)	No	No
Pictures	No	No	Yes (Picture Recognition)

TABLE 2.1(b) ASPECTS OF MEMORY MEASURED BY TEST BATTERY

TYPE OF MATERIAL	ASPECT OF MEMORY			
	IMMEDIATE LEARNING	SPEED OF LEARNING	FORGETTING/ RETENTION	RETRIEVAL
Words	Yes	Yes	Yes	Yes (cued v free recall)
Prose	Yes	No	No	No
Pictures	Yes	No	Yes	No

Picture Recognition test.

The materials for this were line drawings taken from the 'Charlie Brown' cartoon strips by Schultz, as featured in 'The Observer' newspaper. They star the character 'Snoopy' and therefore will subsequently be referred to as the 'Snoopy cartoons'. They were presented on slides using a Carousel projector set to show the slides automatically at the rate of

one every two seconds. Forty slides were shown in an initial presentation; these are referred to as the target slides. Four recognition trials followed: in each, ten of the target slides were presented intermingled with ten distractor slides (slides not included in the original 40). The subject's task was to say which of the slides s/he had seen before.

The subject was instructed to look carefully at the slides during the first presentation but was not told that s/he would be asked to recognise them later in the testing session. Before each of the recognition trials, s/he was told that s/he was going to be shown twenty Snoopy slides, ten of which s/he had seen previously in the initial presentation. If s/he recognised the slide s/he was asked to put a tick beside the number of the slide in the answer book provided. If s/he did not recognise it, s/he was to put a cross. S/he was asked to guess if unsure, and to make a response to each slide. The investigator called out the number of each slide as it was shown to prevent confusion.

There were four sets of slides for the recognition trials. Each consisted of ten distractor slides mixed with ten target slides. The order of slides within each set was the same for each subject, and each set was used once only. A random number list, generated on a microcomputer, was used to determine which set of slides to use for each recognition trial for each subject. This was to ensure that changes in recognition scores over the four trials were not confounded with differences in the memorability of different sets of slides.

The number of hits, misses, false positives and correct negatives was calculated for each subject on each recognition trial. The relationships between these scores, the status of the slide (distractor versus target) and the subject's response (seen before/not seen before) are shown in Table 2.2.

This recognition memory task used forced recognition so that a signal detection analysis of the data could be carried out. This is based on the hit rate (proportion of repeated items correctly identified) and the false positive rate (proportion of 'new' slides said to have been seen before). Such an analysis gives a more accurate impression of the subject's performance than do raw scores, because subjects could score

perfectly on this type of task by simply saying they have seen all the slides before.

Table 2.2. RELATIONSHIP BETWEEN STATUS OF SLIDE AND SUBJECT'S RESPONSE

Subject's response	Status of Slide	
	Target Slide	Distractor Slide
'Seen before'	HIT	FALSE POSITIVE
'Not seen before'	MISS	CORRECT NEGATIVE

Signal detection analysis gives a measure of the subject's response stategy (β) in addition to information about his/her ability to remember and recognise such material (d'). An example of its use comes from the study by Miller and Lewis (1977) referred to in Section 1.8.2. They were testing the hypothesis that elderly depressed patients perform as badly as demented patients on some memory tests because they adopt a conservative response stategy, rather than because their memory is really impaired. Their results supported this hypothesis: depressed patients and normal controls had similar d' levels and therefore similar basic memory capacity, but their β levels suggest that they demanded a higher level of subjective certainty before they responded than other subjects.

d' was calculated in the present study according to the formula:

$$d' = ZHit - ZFA$$

where ZHit is the standard score of the proportion of hits achieved out of the total possible, and ZFA is the standard score of the proportion of false alarms (McNicol, 1972). The d' value increases as the subject's ability to discriminate between distractor and target slides increases. β was calculated by dividing the ordinate of the standard score of the proportion of hits by the ordinate of the standard score of the proportion of false alarms (McNicol, 1972). Where β is less than one, the subject is biased towards saying that s/he has seen the slide before, and therefore makes more false positive errors. If β is more than one, the

subject is biased towards responding that s/he has not seen the slide before and therefore will make fewer false positive errors.

Neither d' or β can be calculated when the proportion of hits or false alarms is zero because the standard score of the proportion cannot be calculated in this situation.

Free Recall Test

Twenty words were used in this test. They were all taken from Thorndike and Lorge's list of 'A' frequency words (Thorndike and Lorge, 1944) (Table 2.3). Each word was printed in black on a white slide and shown to the subject using a Carousel projector at the rate of one every two seconds. The subject was asked to concentrate hard on each word. Once all 30 had been presented s/he was told to write down as many as s/he could remember in any order. The order of the presentation of the slides varied between subjects, according to random number lists generated on a micro-computer.

Table 2.3 WORDS USED IN THE FREE RECALL TEST

1. Harbour	2. Address	3. Governor	4. Giant	5. Uniform
6. Witness	7. Individual	8. Match	9. Cabin	10. Temple
11. League	12. Shell	13. Lamp	14. Factory	15. Expression
16. Element	17. Bond	18. Accident	19. Wealth	20. Dawn

This test has been shown to comprise two separate components, one suggesting a labile short-term memory component, and the other a more stable long-term one (Atkinson and Shriffin, 1968). When recall is immediate the last few items tend to be recalled first and best: this is the recency effect. In addition the first few items in the list are generally recalled well: this is the primacy effect. Together they produce the typical U shaped serial position curve.

This was initially taken as evidence that information is first registered in a limited capacity short-term memory store and needs to be rehearsed if it is to enter long-term memory (Atkinson and Shriffin, 1968). According to this view the first few items in a list are recalled well because they are rehearsed better than later items, and are therefore more likely to enter the long-term store. In contrast the last few items represent the content of the short-term store and need to be recalled immediately, or to be maintained by rehearsal. If recall is not immediate and rehearsal is prevented the recency effect disappears but performance on the earlier items in the list is unaffected (Postman and Philips, 1965).

Baddeley and Warrington (1970) gave amnesic patients a free recall task comprising a list of ten words which were recalled immediately or after a thirty second delay, during which time rehearsal was prevented by a counting task. The patients had a normal recency effect but performed badly after a delay. The authors concluded that these patients had very poor long-term memory but completely unimpaired short-term memory. This is compatible with the view that there are two distinct components of memory. However, the view of Atkinson and Shriffin that the short-term store is essential for input to, and retrieval from, long-term memory is challenged by the finding that some patients have an impaired short-term memory but a normal long-term memory. For instance K.F, who has damage to his left parieto-occuipital region, has been shown to have good long-term memory but a much reduced recency effect (Warrington, Logue and Pratt, 1971).

It is clear that the original explanation for the serial position curve is not sufficient, but the evidence remains that the recency and primacy effects are due to two different components of memory. For instance the recency effect is very robust provided recall is immediate, but recall of the earlier part of the list is very sensitive to factors such as the rate of presentation (Glanzer and Cunitz, 1966) and word frequency (Raymond, 1969). The free recall test is, therefore, useful in a battery of memory tests because it can be used to differentiate between these components.

The total score of this test makes up the variable 'Free Recall - total'. In addition the serial position curves are examined.

Serial Learning test.

A slide sequence of fifteen words was shown four times during the testing sessions (Table 2.4). The words were chosen in the same way as those in the Free Recall test, and were also presented at two second intervals. They were shown in the same order to each subject and on each trial. The subject was told that s/he would be shown fifteen slides with one word on each. Once all fifteen had been presented, s/he was to write down as many as s/he could remember. No mention was made at this stage of the fact that the words would be shown again. On the second and subsequent trials the subject was given similar instructions, except that s/he was told that the words were the same as before and s/he should write down all the words s/he could remember and not just the ones s/he failed to recall on previous trials.

Table 2.4 WORDS USED IN THE SERIAL LEARNING TEST

1. Editor	2. Operation	3. Sheet	4. Secretary	5. Wire
6. Trick	7. Handle	8. Clothing	9. Judgment	10. Flood
11. Noble	12. Diamond	13. Basket	14. Region	15. Plate

The first trial of this test is similiar to the free recall test and would be expected to show the serial position curve with both the first and last items being recalled well. The test is repeated to examine the speed at which subjects can learn new material.

This test results in two variables: Serial Learning - immediate recall (Trial One) and Serial Learning - speed of learning (mean on Trials Two to Four)

Paired Associate Learning test

This test was taken from the Wechsler Memory Scale (Wechsler, 1945). The material was pre-recorded and presented via a cassette recorder to eliminate variations in presentation between subjects. There were four trials, in each of which five pairs of related words (eg up, down) and five of unrelated words (eg crush, lark) were read at two second intervals. Immediate recall was then tested by giving the subject the first word of the pair. The subject was given five seconds to recall the other word of the pair, at which point a bleep sounded on the tape. If the subject was wrong, or did not respond in time, the recorder was stopped so that s/he could be corrected before the next word was presented. If s/he was right, the tape continued with the first word of the next pair. After the ten pairs of words had been presented and recalled, there was a ten second pause before the next trial began. Both the pairs and the recall words were in a different random order on each trial. The pairs of words used are given in Table 2.5.

This test was followed by other tests from the test battery and not less than fifteen minutes later recall of the pairs was tested again, but without each pair being presented beforehand. This was Trial Five.

Table 2.5 WORDS USED IN PAIRED ASSOCIATE TEST

1.	North	-	South	6.	Cabbage	-Pen
2.	Up	-	Down	7.	Crush	-Lark
3.	Fruit	-	Apple	8.	Baby	-Cries
4.	Rose	-	Flower	9.	School	-Grocery
5.	Metal	-	Iron	10.	Obey	-Inch

Before the trial began, the subject was instructed to listen carefully to the tape because s/he would be asked to remember the words that went together. Examples were given to ensure that s/he understood. Between each of the first four trials, the subject was told that s/he would hear

the same pairs again but in a different order; again his/her task was to remember the words that went together. Before the fifth trial, it was stressed that this time the pairs of words would not be presented before recall.

In contrast to the two verbal learning tests already described this test uses cued recall. This should benefit patients who have problems gaining access to information which is stored in their memory. For instance Hultsch (1975) tested memory for categorised word lists with free and cued recall and found that the elderly benefited more from cueing than did younger subjects. If this study finds that there is less difference between the scores of normal controls and depressed patients on this task than on the free recall tasks, it would suggest that the depressed patients have a retrieval deficit.

If the depressed patients have an impaired ability to retain material in the memory then this would be shown by the difference between scores on the fourth and fifth trials: the difference would be higher in the depressed than non-depressed subjects, reflecting the fact that material has been forgotten.

This test has been used with depressed patients in previous studies (for instance Stromgren (1977), Breslow, Kocsis and Belkin (1980), Kopleman (1986), Siegfried, Jansen and Pahnke (1984)). It is similar to the Inglis Paired Associate Tasks (Inglis, 1957) which have been used extensively to assess memory impairments in elderly patients. The hard (unrelated) pairs have been shown to differentiate particularly well between elderly patients clinically judged to be memory impaired and those with normal memories. They have the added advantage that performance on them is not related to I.Q. or age (Inglis, 1959). They have been recommended to clinicans interested in memory performance as the best short test of a patient's capabilities (Erickson and Scott, 1977).

A version of this test was used by Sunderland, Harris and Baddeley (1982) in a study of head-injured patients. They found that scores on the test correlated well both with the relatives' accounts of how often memory failures happened to the patient, and with a checklist kept by the patient of problems experienced over a period of time. Recall after a

delay was most strongly related; for instance a Pearson correlation coefficient of .47 was found between it and the relatives' questionnaire. (This was regarded as an important relationship because it is rare to find significant correlations between reports of memory performance and scores on memory tests. This is discussed in Chapter Seven.) They suggest that it must assess general aspects of verbal memory that are relied on in many everyday situations.

The first trial of this test is taken as a measure of immediate recall (Paired Associate - immediate recall). The mean of Trials Two to Four is used as a measure of speed of learning (Paired Associate - speed of learning). The final variable from this test is Paired Associate - forgetting, which is calculated by subtracting the scores from Trial Five from the scores of Trial Four.

Prose Passage Recall

Two short prose passages were used in this test. They were taken from the 'logical memory' subtest of the Wechsler Memory Scale, version 2 (Wechsler, 1945). The second passage was amended slightly to make it more suitable for British subjects and to update it: the place where a shell exploded was changed from France to Israel, and 'schoolhouse' was abbreviated to 'school' (Table 2.6). Both passages were recorded on tape, as were the instructions which asked the subject to listen carefully so that s/he could repeat what s/he had heard.

The first passage was played and the investigator then asked the subject to tell her everything s/he had heard, starting at the beginning. The responses were taped. The second passage was then played to him/her. This time s/he was not asked to recall it immediately. Instead, s/he was told s/he would be asked about it later in the testing session. At least fifteen minutes later the investigator returned to these passages and asked the subject to recall as much as s/he could remember about both passages.

The scoring system devised by Wechsler was used. Each story was divided into 23 units, each unit expressing a detail of the story. One

point was awarded for a unit correctly recalled, and half a point for partial recall.

The first prose passage is recalled immediately and again after a delay in order to investigate the forgetting of information which is known to have been learned initially. The second passage is only recalled after a delay. This is because the initial recall of the first passage may rehearse it and therefore enhance its later recall. If the second passage is recalled significantly less well than the first, it would suggest that the results for the first passage underestimate the amount of forgetting which is likely to occur when there is no opportunity for rehearsal. This test therefore produces four variables: Prose Passage One - immediate recall; Prose Passage One - delayed recall; Prose Passage One - forgetting (immediate minus delayed recall); and Prose Passage Two - delayed recall.

Table 2.6 PROSE PASSAGES

Passage one

Dogs/ are trained/ to find/ the wounded/ in wartime/. Police dogs/ are also trained/ to rescue/ drowning people/. Instead of running/ down to the water/ and striking out/ they are taught/ to make/ a flying leap/ by which they save/ many swimming strokes/ and valuable/ seconds of time/. The European sheep dog/ makes the best/ police/ dog/.

58 words, 23 units

Passage two

Many/ school/ children/ in Northern/ Israel/ were killed/ or fatally hurt/ and others/ seriously injured/ when a shell/ wrecked/ the school/ in their village/. The children/ were thrown/ down a hillside/ and across/ a ravine/ a long distance/ from the school/. Only two/ children/ escaped uninjured/.

45 words, 23 units

Memory tests which use single words can be criticised for being artificial and having little in common with memory in everyday life: people rarely learn lists of unconnected words. In contrast they do

remember stories and conversations. These have a definite structure (or schemata) which means that new information can be incorporated into an existing framework. Prose passages are therefore included in memory test batteries because they are more realistic than most of the tests used. Sunderland, Harris and Baddeley (1982) found that scores on a prose passage test correlated significantly with relatives' reports of memory failures in head injury patients. Not surprisingly the correlations were higher than those between scores on the more artificial Paired Associate test and the relatives' reports (Pearson correlation coefficient =.72 (immediate recall) and .63 (delayed recall) compared with .47 (delayed recall trial of Paired Associate test)).

The Prose Passage recall test has been used in several other studies of memory in depression (Breslow, Kocsis and Belkin, 1980; Kopelman, 1986; McAllister et al, 1987).

Digit Span

This test was also taken from the Wechsler Memory Scale (Wechsler, op.cit.).

A) Forwards

Four digits were read out by the investigator. The subject was asked to repeat them in the order they were given. A different group of four digits was then read out and again the subject was asked to repeat them. If s/he correctly repeated the four digits on at least one of the two trials, the investigator then read out five digits. This procedure was then repeated to a maximum of eight digits, with two trials for each number of digits. The subject's score was the maximum number correctly repeated. The groups of digits used are given in Table 2.7.

The number of digits which can be correctly repeated (memory span) was shown by Miller (1956) to be seven, plus or minus two. Memory span has been shown to be unimpaired in patients who have suffered bilateral damage to the temporal lobes and the hippocampus and who consequently have grossly impaired ability to learn new things. For instance the famous case H.M. has a normal digit span but cannot learn anything new

(Milner, 1970). Similarly, Drachman and Arbit (1966) found that amnesics with a damaged temporal lobe did not differ from controls in the length of their memory span but once this was exceeded they had extreme difficulty learning number sequences. However, the patient K.F. who has a good long-term memory as demonstrated by normal scores on free recall tests, has a digit span of less than three (Warrington, Logue and Pratt, 1971). These results are seen as evidence that only the short-term component of memory is involved in this task. However, there is some evidence that memory span for words, at least, may involve both short-term and long-term memory (Watkins, 1977). In contrast Baddeley and Hitch argue that memory span depends on the interaction of two components of working memory: the articulatory loop which can store about three items in serial order and the central executive (Baddeley and Hitch, 1974; Baddeley, 1981b).

The digit span test is often considered to be a measure of concentration. For instance Kear-Colwell (1973) factor-analysed Wechsler Memory scale scores derived from 250 people. Three factors accounted for 72% of the variance. These were identified as the learning and recall of complex novel material; attention and concentration; and orientation and information. The tests most heavily loaded on the attention and concentration factor were 'mental control' and the digit span tests, thus providing evidence that these tests should be regarded as measures of concentration. This is consistent with the view of Baddeley and Hitch that memory span provides a measure of the available capacity of working memory, and consequently of the attention allocated to the task (Baddeley and Hitch, 1974; Baddeley, 1981b).

B) Backwards

The subject was read three digits on the first trial. S/he was asked to repeat them but in reverse order. For example, if s/he was read "3,2,1" s/he was expected to reply "1,2,3". Several examples were given to ensure that the subject understood what s/he was supposed to do. The test followed the same pattern as 'Digit Span Forward', except that there was a maximum of seven digits. The score was the number of digits correctly repeated.

This test is identical to Digit Span Forwards except that it involves the manipulation of the digits. It therefore makes more demands on concentration and working memory.

Table 2.7 DIGIT SPAN

A) Forwards	Score	B) Backwards	Score
6-4-3-9	4	2-8-3	3
7-2-8-6	4	4-1-5	3
4-2-7-3-1	5	3-2-7-9	4
7-5-8-3-6	5	4-9-6-8	4
6-1-9-4-7-3	6	1-5-2-8-6	5
3-9-2-4-8-7	6	6-1-8-4-3	5
5-9-1-7-4-2-3	7	5-3-9-4-1-8	6
4-1-7-9-3-8-6	7	7-2-4-8-5-6	6
5-8-1-9-2-6-4-7	8	8-1-2-9-3-6-5	7
3-8-2-9-5-1-7-4	8	4-7-3-9-1-2-8	7

The memory test battery was concerned solely with memory for material given during the testing session. In contrast, past public events questionnaires were used to assess memory for events occurring in the years before the session and, in addition, self-rating memory questionnaires were given to see how the subjects rated their own memory performance. These tests are described in the next two sections.

2.2.2 Past Public Events Questionnaires

There were two parallel forms of these questionnaires. Both contained 40 questions about events in the news between 1966-81, with five questions about each two-year time period (Appendix A).

The questionnaires were developed by selecting several events from each year in this period from 'The Times End of Year Reviews'. The events

chosen were those which seemed to have been the most striking and significant at the time, regardless of later events. A questionnaire was produced which contained 118 questions about events over the fifteen year period. It was piloted on academic staff, students, clerical and technical staff of the University of Durham Psychology Department. Questions which were answered incorrectly by more than half the subjects were eliminated. In cases where more than ten questions from a two year period were left, the ten which were correctly answered most frequently were selected. In two time periods (1968-1969; 1975-76), only nine questions remained.

The remaining questions were again piloted. The subjects were from a variety of backgrounds and a range of occupations and were not connected with the university. As a result of the pilot several questions which were found to be difficult were reworded or changed to cover a different aspect of the same event. For instance the question 'who was the Communist Party secretary in Czechoslovakia whose appointment in January 1968 led to a series of social and economic reforms, which eventually resulted in the Russian invasion' was changed to 'Which Eastern European country was invaded by the Russians in 1968'. One question was removed completely, reducing the total number to eighty.

Two parallel versions of the questionnaire (versions A and B) were needed so that each subject could complete a free recall and a multi-choice version. The following criteria were therefore used to divide the questions into two groups:

1. There should be five questions from every two-year period in each questionnaire.
   This was achieved except in the two cases where there were only nine questions: four questions from 1968-1969 went into the first version of the questionnaire, and five into the second version. This was reversed for questions from 1975-1976.

2. The level of difficulty of the questions should be the same in the two versions.
   As the final selection of questions in both versions had a mean response rate in the pilot study of 84% and in both cases the

response rate to individual questions ranged from 58% to 100% this seems to have been achieved.

3. Given the above constraints, the number of questions about different types of news should be the same in the two versions.
   It was recognised that peoples' interest in, for example, sport, politics, foreign affairs, 'scandals' and the Royal Family varies considerably. It was therefore important that both versions of the questionnaire should cover a range of topics.

Multi-choice versions of each questionnaire were developed. Each question had four possible answers; one correct and three 'distractors'. These were chosen on the basis that they were plausible but incorrect responses to the question. For example the possible responses to the question 'what was introduced in Britain on February 15th, 1971' were: decimal currency (correct response), comprehensive schools, Value Added Tax, and credit cards. These were judged to be plausible responses to the question.

Each subject in the experiment completed the free recall version of one questionnaire, and the multi-choice version of the other. Random number tables were used to decide which questionnaire was given in which form. The subject was told that s/he had fifteen minutes in which to complete each version of the questionnaire and that s/he should not worry if s/he did not finish it in time. If s/he did not, the number of the question s/he reached in the fifteen minutes was recorded. Before completing the free recall version the subject was told to write down an answer even if s/he was not sure about it. The instructions for the multi-choice questionnaire asked the subject to guess even if s/he did not know the answer and to ring a response for each question.

Similar questionnaires were initially develped by Warrington and Silberstein (1970) in order to get quantitative data on the duration of retrograde amnesia in patients with memory loss. It was presumed that everyone is exposed to a greater or lesser extent through the mass media to a continuing series of public events and that these therefore provide a source of common experiences.

They do not provide a perfect way of measuring very long-term memories: firstly because it is impossible to be sure that the information was learned initially; and secondly because it is not possible to be certain that questions about events from different time periods are equally difficult. In addition studies of remote memory in normal subjects have suggested that people acquire some information about public events after they occur. For instance Botwinick and Storandt (1974) found that subjects in their thirties recalled more events from every time period between 1890 and 1960 than did subjects in their twenties. Some attempts have been made to overcome these problems, for instance by testing memory for television programmes which have a limited run of only one season (Squire and Slater, 1975). This has been done in the United States and would be more difficult in Britain because there are far fewer suitable programmes.

Despite these problems past public events questionnaires have been used extensively to shed light on causes of amnesia. For instance Squire, Slater and Chase (1975) found that after E.C.T. patients had retrograde amnesia for public events occurring in the few years prior to treatment. In a later study they found a persisting loss for events occurring in the few days before treatment, whilst the initial amnesia for events in the years before declined with time (Squire, Slater and Miller, 1981). They argue that these results are consistent with theories of amnesia which emphasise problems in the storage or consolidation of new material. In contrast a general deficit in memory retrieval has been postulated to account for the performance of patients with Korakoff's syndrome on such tests: they show extensive impairment over several decades and before the onset of the disorder (Warrington and Weiskrantz, 1973).

If depressed patients just have difficulty learning new material then they should perform as well as other subjects on this test. If, however, they have difficulty retrieving material already in memory then they would be expected to be impaired, particularly on the free recall version of the questionnaire.

2.2.3 Memory Complaints Questionnaires

These ask the subject to assess his/her own memory. Two questionnaires were used in this study. They cover slightly different aspects of cognitive functioning and differ in the type of rating the subject is asked to make: in one s/he is asked how often various cognitive 'slips' have happened to him/her over the past six months; in the other s/he is asked the extent to which his/her memory has deteriorated since the onset of depression (depressed patients), anxiety (anxious patients), or in the past year (subjects who were neither depressed or anxious). Copies of both questionnaires are included in Appendix B.

Cognitive Failures Questionnaire

This was developed by Broadbent and his colleagues (Broadbent et al, 1982). It asks about cognitive mistakes in daily life and covers 25 self-reported failures in perception, memory and motor functions. Broadbent states that these were selected from ones which either the experimenters or their acquaintances had experienced. It seems to have been designed on an intuitive basis without any theoretical reason for the inclusion of particular questions.

Although it is not specific to memory it does include a significant proportion of questions relevant to memory. These cover absent-mindedness, forgetting appointments and conversations, 'tip of tongue' experiences and the ability to learn new material. Examples include questions about how frequently the subject makes mistakes such as putting a spent match in his/her pocket and throwing the matchbox away, or forgetting people's names. In addition to questions about memory there are questions about perceptual slips (for example 'do you fail to notice signposts on the road?') and motor function mistakes (for example 'do you drop things?'). Questionnaires completed by a variety of subjects (including NHS laundry workers, student nurses and car factory employees) were factor-analysed and subjected to multi-dimensional scaling to see if there was any evidence of separate factors containing questions from these different areas. The results varied from group to group and there was no evidence for separate categories of perceptual, memory and action

failure. Thus subjects who reported memory slips were also likely to report perceptual and action slips.

Scores on the CFQ seem to be quite stable over time. For instance Broadbent reports that 73 student nurses followed for sixteen months through training had an initial-final product moment correlation of 0.54 (further details of this study are described by Parke, 1980). In another study he found a test-retest reliability of 0.85 (Broadbent, Broadbent and Jones, 1986). He also found that scores on the CFQ correlated with spouse's ratings of their partner's cognitive slips: taus of 0.31 (husband's and wife's ratings of wife's cognitive slips) and 0.36 (the reverse) were reported (Broadbent et al, 1982). He concludes from this that scores on the CFQ have some external importance. This is supported by the finding of Harris and Wilkins (1982) that subjects with high CFQ scores were late on a greater number of occasions than subjects with low scores in a a study in which subjects watched a film and had to hold a sheet up to a video camera at three or nine minute intervals.

Scores on the CFQ are not closely related to test intelligence or to educational level. For instance Weekes in a study in Edinburgh found correlations of -0.15 with the Mill Hill test and -0.15 with scores on the Progressive Matrices (Broadbent et al, 1982).

In the present study the subject was told that the questionnaire was about minor mistakes that everyone makes from time to time. S/he was asked to decide how often the mistakes listed had happened in the past six months and to circle the appropriate response. The options were 'very often', 'quite often', 'occasionally', 'very rarely' and 'never'. The options are scored from 0 (never) to 4 (very often): a high score therefore reflects the frequent occurrence of these cognitive slips.

Memory Complaints Questionnaire

This was a modified version of a questionnaire developed by Sunderland and Harris (The Everyday Memory Questionnaire, Harris and Sunderland, 1981; Sunderland, Harris and Gleave, 1984). This was designed to assess the severity and nature of memory problems experienced by closed head-injury patients and contains questions taken from an earlier

questionnaire, the Head Injury Questionnaire (Sunderland, Harris and Baddeley, 1983). This covers memory and concentrational difficulties which may occur in everyday life, such as forgetting appointments, conversations and names. Questions were included in the Everyday Memory Questionnaire if a problem was reported more frequently by patients with severe closed head injury than by control subjects; if relatives of these patients said it occurred more frequently than did relatives of the controls; and if a problem was mentioned frequently in interviews with these subjects but was not included in the original questionnaire. It therefore initially contained items chosen to be particulary sensitive to the memory problems of head-injury patients, although it was subsequently revised to be suitable for a wider range of subjects. It has been shown to have a test-retest reliability over a six month period for the total score of 0.78 (Spearman's rho; Harris and Sunderland, 1981).

In the original versions the subjects were asked to indicate how frequently they had experienced the various difficulties in the past six months. They used a nine point scale on which responses ranged from 'more than once a day' to 'not at all in the past six months'. Sunderland et al (1986) suggested that a more accurate self-assessment might be obtained if subjects were asked about changes in memory following head-injury, rather than how often difficulties occured. This is supported by the finding by Rabbitt (1982) that the difference between elderly subjects' estimation of how often these memory difficulties occurred now and their estimation of how often they occured when they were thirty was significantly correlated with performance on laboratory memory tests, whilst the estimations of current difficulties were not. Although Sunderland et al (1986) were unable to replicate these results, probably due to the low level of reported difficulties at age 30, it does suggest that people may be more accurate at assessing the degree of change than the frequency of memory failures. It therefore seems appropriate to ask subjects about the degree of self-perceived change when the interest is in changes in memory functioning, whether these changes are due to illness, injury or ageing.

In this study this questionnaire is used to assess depressed and anxious people's experiences of memory problems and is therefore concerned with changes in memory functioning resulting from these

disorders. The questionnaire was therefore modified to measure self-perceived changes in memory rather than the frequency of memory difficulties. The subjects were asked to rate their memory performance now compared with before the onset of the illness. A five point rating scale was used with responses which ranged from 'much less often' to 'much more often'. The highest scores were obtained by those subjects with the largest self-perceived deterioration in memory.

The subjects who were neither depressed or anxious presented a problem as they could not compare their memory now with before the onset of depression or anxiety because by definition they were not anxious or depressed. They were therefore asked to compare their memory performance now to a year ago. The same five point response scales were used. This was intended to give a measure of the variability in perceived memory performance over time.

Each subject was seen twice and one of the two self-rating memory questionnaires was given in each session. They were completed before the memory testing began so that scores would not be influenced by performance on these memory tests.

2.2.4 Self-rating of Depression and Anxiety Symptoms

In addition to the tests already described which were concerned with different aspects of memory the subjects also completed questionnaires designed to assess their mood. These are considered in this section.

The Irritability-Depression-Anxiety (I.D.A) Scale (Snaith et al, 1978) was used to assess how depressed and anxious the subjects were at the time of testing. (A copy of the scale is included in Appendix C).

This is designed to measure the symptoms of depression and anxiety. It is not a scale intended to measure the severity of a clinically diagnosed depression or a clinically diagnosed anxiety state. Such a scale would would obviously include symptoms specific to the disorder, but might also include other symptoms if they occur frequently in the disorder. For instance a depression scale of this kind would include anxiety symptoms because these frequently occur in depressed patients

(Section 6.1). The Beck Depression Inventory (Beck et al, 1961) is an example of this type of scale. It was intended that a high score on this should reflect the severity of depressive illness, not just the severity of symptoms of depression, and the questions were derived from clinical observations of the attitudes and symptoms displayed by depressed psychiatric patients. It includes symptoms normally regarded as specific to depression; for instance depressed mood, suicidal wishes and loss of appetite. However it also includes symptoms which would also be found in other psychiatric conditions, notably an anxiety state; for instance irritability and social withdrawal. Not surprisingly it has been shown to be associated with measures of anxiety (Meites, Lovallo and Pishkin, 1980).

In contrast the I.D.A was designed to measure two groups of symptoms which may occur to varying degrees in a variety of psychiatric conditions: a high score on the depression subset may occur in patients whose primary diagnosis is not depression but in whom symptoms of depression are a prominent feature of their illness. It therefore assesses the severity of the symptoms of depression and anxiety, rather than the severity of depressive illness or an anxiety state.

Measures of symptoms of depression and anxiety were required in this study for subjects with a variety of psychiatric conditions. The questionnaire used had to be as short and concise as possible to help overcome the problems of poor concentration which are inherent in research with depressed and anxious subjects. It also had to measure mood at the time of completion rather than more general personality traits, for example 'state' rather than 'trait' anxiety (Spielberger, Gorsuch and Lushene, 1970). The I.D.A satisfied these criteria: it measures symptoms of anxiety and depression as separate syndromes; it is short and easy to complete and it refers to the subject's present mood state.

The I.D.A consists of a depression subscale, similar to the Leeds Self-assessment of Depression General scale, and an anxiety subscale, similar to the Leeds Self-assessment of Anxiety General scale (Snaith, Bridges and Hamilton, 1976). In addition it contains a subscale intended to measure outwards- and inwards- directed irritability. Because of this,

one question from each of the Leeds scales has been omitted by the authors because it referred to irritability.

The I.D.A depression subscale has been shown to correlate highly with ratings by psychiatrists on the Hamilton Depression Rating Scale (r=0.75). The anxiety subscale has similarly been shown to be highly correlated with the Hamilton Anxiety Rating Scale (r=0.70; Snaith et al, 1978). Aylard et al (1987) investigated the validity of the I.D.A scale and found that the scores of hospital out-patients on the depression subscale correlated significantly (r=.72) with scores on the Montgomery-Asberg Depression Rating Scale, an interviewer administered scale; scores on the anxiety subscale correlated significantly (r=.51) with scores on the Clinical Anxiety Scale. Validity of the I.D.A. is further supported by an unpublished study referred to by Snaith (1982) on the prevalence of post-natal depression in general practice. Cut-off points for clinically significant mood disorder were taken as 4/5 on the depression subscale, and 7/8 on the anxiety subscale. The women were then interviewed using Goldberg et al's Standardised Psychiatric Interview (Goldberg et al, 1970) and few misclassifications were found: ten per cent of the depression scores above the cut-off point were false positives, as were twelve per cent on the anxiety subscale; there were no false negatives on either scale.

The depression and anxiety subscales each have five questions while the outwards- and inwards- directed irritability subscales have four questions each. The questions from the subscales are intermingled. Each item has four possible responses. For instance the statement 'I feel cheerful' (from the depression subscale) is followed by the options: 'Yes, definitely'; 'Yes, sometimes'; 'No, not much'; 'No, not at all'. The responses to some items are worded so that agreement with the statement indicates the presence of the symptom: in other cases this is reversed in order to overcome any possibility of a tendency to agree with the items affecting the results. The range of scores on each item is 0 to 3, with the higher scores always representing the most severe symptoms. Each subscale is scored separately.

Subjects completed the I.D.A at each testing session. S/he was told to read each item in turn and then underline the response which best

showed how s/he had been feeling over the last few days. S/he was also told that each question should be completed. The instructions were also written on the questionnaire. In some cases the patient reported that his/her mood had recently changed and that consequently s/he was unsure how to complete the questionnaire. S/he was told to refer to how s/he was feeling that day as the I.D.A was being used to measure current mood.

2.3 PROCEDURE

2.3.1 Location of Testing Sessions

The testing sessions with in-patients took place in hospital. However patients attending the Day Unit could chose whether they wanted the sessions to take place in the hospital or in their own homes. This was because it was envisaged that it would be difficult to find times for the testing which would not interfere with the patients' consultations or other treatment sessions. However it was recognised that some patients would not participate if it involved the experimenter visiting them at home, and therefore they were offered the choice. All the control subjects were tested in their own homes.

It is obviously more difficult to ensure a suitable quiet and undisturbed environment in either a hospital or in the subjects' own homes than in an experimental laboratory. However the vast majority of testing sessions in both homes and hospital took place without interr-uptions or excess noise. A projector, projector screen and tape-recorder were used at each session. These were set up in positions which ensured that the subject could see the screen and hear the tape clearly.

2.3.2 Content of Testing Sessions

The tests took at least two hours to complete. It was felt that severely depressed and anxious subjects would have difficulty concen-trating for this long and the tests were therefore divided between two sessions. The tests which were included in the sessions are listed in Table 2.9.

Table 2.9 CONTENT OF TESTING SESSIONS
Memory test battery
Picture Recognition test
Free Recall test
Serial Learning test
Paired Associate learning
Prose Passage recall
Digit Span: Forwards and Backwards
Past Public Events Questionnnaires
Past Events free recall questionnaire, versions A and B
Past Events multi-choice questionnaire, versions A and B
Self-Rating of Mood
I.D.A questionnaire
Memory Complaints Questionnaires
Cognitive Failures questionnaire
Memory Complaints questionnaire

The issue of whether to include an I.Q or vocabulary test to either match subjects on these variables or to control for their effects on memory statistically was considered at some length. It was decided not to use them for two reasons: firstly because depression itself may lead to a reduced IQ score, and secondly, as indicated above, that the number of tests these subjects could reasonably be asked to complete is limited by problems of concentration.

The example of H.M (Milner, 1968) demonstrates that intelligence does not depend solely on memory: following surgery for the relief of epilepsy

he was unable to learn anything new, but had a higher I.Q than before the operation. However, as Baddeley (1976) has noted, many of the subscales used in I.Q testing have a major memory component. For example completion of the Wechsler Adult Intelligence scale (WAIS; Wechsler, 1944) would involve working memory in mental arithmetric exercises; semantic memory in comprehension tests; short-term memory in digit-symbol substitution tests and visual memory in picture completion tests. Strong correlations would therefore be expected between memory test scores and intelligence, and this was found by Eysenck and Halstead (1945). Consequently if memory is affected by depression it is likely to lead to deflated scores on IQ tests compared with the subject's premorbid scores.

The effect of depression on I.Q scores was investigated by Donnelly et al (1982) who did not find significant changes in depressed people's W.A.I.S scores on remission of depression. However they did not assess the subjects' memories. It is therefore possible that they did not find any changes in IQ scores because these people did not have memory problems. Miller (1975) reviewed this area and concluded that no study had made the vital direct comparison of the depressives' premorbid IQ with their IQs during the depressive episode; this remains the case. The possibility remains that depression affects the memory and therefore leads to reduced IQ scores.

If this is so it would be misleading to compare the memory performance of depressed subjects with that of a group with comparable I.Qs in order to control for the effects of intelligence on memory. Given the strong correlation between memory test scores and intelligence (Eysenck and Halstead, 1945), such subjects would be expected to do less well on the memory tests than people with IQs comparable with the depressed subjects' premorbid scores. If there was no difference between the control and depressed subjects' memory performances it would actually mean that the depressed subjects were doing badly. Measuring IQ scores at the time of testing and matching subjects to controls who were not depressed or anxious on this basis is therefore not an adequate method of controlling for the effects of intelligence on memory. If pre-morbid IQ scores were available this would overcome this problem. In their absence, the only way to control to some extent for the effect of intelligence is

to use an indirect measure of it, such as the number of years of full-time education completed.

The number of tests which the psychiatric subjects could reasonably be asked to complete was limited by their ability to concentrate, and willingness to participate. A balance was needed between collecting every item of data which might be relevant to the study and ensuring that the number of tests did not make so many demands on the patients that they were unable to complete the sessions. As there were good reasons to doubt the value of I.Q scores in this context it was decided to omit measures of IQ in order to make time for other more useful tests.

The testing sessions were the same for the depressed, anxious and control subjects except that after completing these tests the depressed and anxious subjects were interviewed using a semi-structured psychiatric interview: the Present State Examination (Wing, Cooper and Sartorius, 1974). In most cases this took place at the end of the second testing session. However, a third meeting was arranged for this interview with just over one third of the depressed and anxious subjects. In three cases the P.S.E was carried out at a fourth meeting because the tests took three sessions; these subjects took a long time to complete the tests and those which were not completed in an hour were transferred to a third session.

The allocation of tests to the first or second session, and the order of tests within each session was decided randomly (using random number lists generated on a microcomputer) subject to the following constraints:

1) Neither session was to last more than one hour.

2) The I.D.A questionnaire was always given at the beginning of the session so that mood ratings would not be affected by performance on these tests.

3) One of the self-rating memory questionnaires (Cognitive Failures Questionnaire and Memory Complaints Questionnaire) was given immediately after the I.D.A at each session as it was also important that scores on these were not affected by performance on the memory

tests. Random numbers were used to decide which of the two self-rating questionnaires should be given at which testing session.

4) The first trial of the Picture Recognition test was always placed near the beginning of the session as other tests had to be interspersed between each of the four trials. However the allocation of this test to one of the two sessions was determined randomly.

5) The Paired Associate and Prose Passage recall tests both had delayed recall trials which had to be separated from the rest of the test by at least fifteen minutes.

On all but seven occasions the tests were given in the order determined randomly before the sessions. Practical problems with the equipment meant that some tests had to be transferred between the two sessions on three occasions. On another occasion the subject forgot his glasses so tests using the projector were postponed until the next session. Three subjects did not complete the tests within an hour so the remaining tests were transferred to a third session.

2.3.3 Conduct of Testing Sessions

At the beginning of the first testing session with each subject s/he was asked his/her date of birth, occupation and the age at which s/he had left full-time education. The control subjects were also screened for psychiatric disorder and asked about any episodes of psychiatric disorder (Section 3.5.2). It was then explained that the session was concerned with his/her memory. It was stressed that s/he should not worry if s/he found the tests difficult and that poor performance on them did not imply anything about the subject's intelligence. S/he was told that participation in the study was entirely voluntary and that s/he could stop the session at any point. The aim of this introduction to the session was to establish a good rapport between the investigator and subject: this was found to be particularly important with the depressed and anxious subjects who often needed continual reassurance throughout the sessions.

The testing session then began and followed a predetermined order, as explained above (Section 2.3.2). Each test was carefully explained to the subject to ensure that s/he fully understood what s/he had to do (the instructions for each test are given in Section 2.2). At the end of the session it was again stressed that the subject was free to withdraw from the study if s/he wanted to. However all the subjects agreed to take part in another session which was then arranged.

The second session took the same format as the first: after it had been stressed that participation was voluntary the tests were given in the predetermined order, each test being preceded by clear instructions. As explained above (Section 2.3.2) three subjects then had similar third sessions. The control subjects did not participate in the study further. Approximately two thirds of the psychiatric patients were interviewed using the P.S.E at the end of second session. The remainder were seen one more time and the psychiatric interview was carried out at this final session.

2.4 SUMMARY

Depressed subjects, anxious subjects, and subjects who were neither depressed or anxious were given a battery of memory tests designed to cover a range of aspects of memory, including immediate memory, retention and forgetting, and retrieval. In addition they completed past public events questionnaires, memory complaints questionnaires and self-rating mood scales.

The test battery consisted of the following tests: Picture Recognition; Free Recall; Serial Learning; Paired Associate Learning; Prose Passage recall; and Digit Span, Forwards and Backwards. The past public events questionnaires were used in both a free recall and multi-choice version. Two self-assesssment of memory questionnaires were included: the Cognitive Failures Questionnaire, which is concerned with how often cognitive slips happen, and the Memory Complaints Questionnaire, which asks about changes in self-perceived memory over time. The four scales making up the Irritability, Depression and Anxiety Scale (Snaith et al, 1978) were used to measure current mood state.

The tests were divided between usually two, and occasionally three, testing sessions, each of which lasted about an hour. The division of the tests between, and the order within, sessions was determined randomly, subject to specified constraints. The sessions took place either in the psychiatric hospital from which the depressed and anxious subjects were recruited, or in the subjects' own homes.

METHODS: SUBJECTS

3.1 INTRODUCTION

As described in the previous chapter, Chapters Four to Seven of this thesis are concerned with the performance of depressed subjects on laboratory memory tests, their reports of memory problems, and the relationship between the two. In each case the data are derived from a battery of memory tests given to depressed and anxious people, and to controls who were neither depressed or anxious. The battery of tests and the experimental procedure have been described in Chapter Two. The subjects used in this study are described in this chapter.

3.2 DEPRESSED AND ANXIOUS SUBJECTS: RECRUITMENT

Depressed and anxious subjects were needed for this study. They were recruited from the Day Unit and in-patient wards at a local psychiatric hospital (the County Hospital, Durham). All subjects were between the ages of eighteen and seventy. They were eligible for inclusion in the study if they suffered from 'neurotic' clinical depression, an anxiety neurosis or a mixture of the two. Patients were excluded if, in the judgment of their consultant psychiatrist, they had any psychotic symptoms, were alcoholics, had a history of severe head injury or showed signs of senile dementia.

Patients taking psychotropic medication or who had had electro-convulsive therapy (E.C.T) were not excluded for both practical and theoretical reasons. The practical reason was that problems were experienced in recruiting subjects and it was judged that eliminating these patients would have brought the research to a halt: the available subject pool would have been severely restricted as the vast majority of patients in the hospital were on medication.

Alternative sources of patients not on medication were considered: for instance asking general practitioners to refer patients who were consulting for the first time with symptoms of depression or anxiety either before they were prescribed medication or before it began to have an effect (tricyclic anti-depressants which are commonly prescribed

typically take several weeks to become effective (Harrison-Read, 1984)). Alternatively patients attending general practice surgeries could have been screened to identify depressed people not known to the doctor and therefore not being treated: it has been estimated that the 'average' G.P fails to identify at least five cases of depression each month (Sireling et al, 1985) and therefore this might have been a good source of subjects. However, these options were eliminated because of the investigator's experience of research in general practice (Chapter Eight): although the general practioners were very willing to take part in research the pressures of time during consultations meant that they frequently forgot to do the things they intended to do, for instance switching on the tape-recorder. There was no reason to suppose their participation would be more vigilant in another study. It was therefore decided that for practical reasons the only feasible option was to include in the study patients on psychotropic medication and who had had E.C.T.

The theoretical reason for including patients who were taking psychotropic drugs or who had had E.C.T was that to select out these patients would have left a sample very atypical of most depressed and anxious patients who come into contact with the medical profession. For instance Johnson (1973) found that most new cases of depression in general practice received psychotropic medication; a similar picture comes from the finding of Brown and Harris (1978) that four-fifths of definite cases of depression consulting their family doctor were given psychotropic medication. The prescribing rate for patients seeing a psychiatrist is likely to be at least as high. Memory problems experienced by patients not on psychotropic medication may not, therefore, be representative of the wider group of patients in which problems caused by depression and anxiety may be exacerbated by the effects of medication or E.C.T.

Patients on psychotropic medication, and who had had E.C.T were therefore included in this study for both practical and theoretical reasons. The effects of these factors were partialled out in the analysis using regression techniques to ensure that differences in the memory performance of depressed subjects, anxious subjects and controls who were neither depressed or anxious were not due to differences in medication rather than to differences in psychiatric status (Chapter Five).

Initially patients were referred to the investigator by one of the four consultant psychiatrists who agreed to help with the study, or by a clinical psychologist. This resulted in very few subjects being recruited, presumably because of many demands made on the psychiatrists' time. The procedure was therefore changed: both wards in the hospital and the Day Unit were visited weekly to check whether any suitable patients had come into the hospital in the past week. Their names were then passed on to the consultants who checked that they were suitable for inclusion in the study and gave their permission for them to be approached by the investigator. Patients were not asked to participate in the study unless their consultant had given permission.

The investigator then met the patient and explained the study and why they were being asked to help. It was stressed that participation was voluntary. If the patient agreed to take part s/he signed a consent form and arrangements were made for the first testing session. If they were in-patients the sessions took place in a quiet room on the ward. If they were attending the Day Unit they had the option of arranging the sessions at the unit or in their own homes. A total of 90 testing sessions were completed with the 34 depressed and anxious subjects. Forty-nine (54%) of these took place in the hospital.

Sixty-two names of possible subjects were obtained during the year long period of data collection: 31 from the Day Unit, 28 from the wards and two from the clinical psychologist. Ten patients were lost to the study because they were discharged before permission to approach them had been obtained from the psychiatrists. In addition eight patients suggested by the ward staff were not felt to be suitable for inclusion by the psychiatrists: they disagreed about the diagnosis or felt that the patient was under too much stress to cope with taking part in the study.

Five patients declined to take part in the study when it was explained to them by the investigator. Testing sessions were arranged with the remaining 38 patients. Two of these (both recruited from the Day Unit) were not willing to take part in the first session despite having previously agreed to do so. One patient started the session but felt unable to continue because of her anxieties about taking part. Two patients completed the first session and arranged a second one, which

they later cancelled because they did not want to participate further. Thus 34 patients successfully completed the study out of the 62 originally suggested for recruitment (Table 3.1).

These figures reflect the difficulties inherent in working with depressed and anxious people: novel events such as participation in a psychological experiment are likely to provoke anxiety and it is not therefore surprising that a few felt unable to take part. In addition depression is associated with a loss of energy and a feeling that everything is 'too much effort' (Section 1.8.1). This sense of exhaustion and apathy was probably connected with some of the refusals and helps to explain why some patients dropped out after the first session. The investigator tried to create a relaxed and supportive atmosphere and this seemed to be successful in most cases, but it is not surprising that some patients found taking part irksome or anxiety provoking.

Table 3.1 OUTCOME OF ATTEMPTS TO RECRUIT PSYCHIATRIC PATIENTS, BY PLACE OF REFERRAL

Outcome	Referred by: Day Unit (N=31)	Wards (N=28)	Psychologist (N=2)
Judged by psychiatrist to be unsuitable for inclusion	4	4	-
Discharged before permission from psychiatrist obtained	5	4	1
Refused	4	3	-
Failed to complete first session	-	1	-
Refused to take part in second session	-	2	-
Successfully completed all sessions	18	15	1

Unsuccessful attempts were made to set up contacts with another psychiatric hospital in an adjacent district in order to recruit more subjects. The main reason for the failure to establish links with this hospital seemed to be the reluctance of the clinical psychologists there

to give their active support at a time when they were heavily overladen with work: the support of their 'opposite number' at the participating hospital was essential in obtaining the psychiatrists' support for the study and without this it would have been very difficult to get it off the ground. The failure to set the study up at the second hospital without similar support highlights its importance.

3.3 DIAGNOSIS

3.3.1 Diagnosis in Psychiatric Research: General Issues.

One difficulty commonly experienced in research into psychiatric problems is knowing how to allocate subjects to diagnostic groups in a reliable way. Traditionally diagnoses have been reached by psychiatrists questioning their patients and matching the information obtained with one of the pictures of 'typical clinical patterns' they reputedly carry in their heads (Wing et al, 1978). It was presumed that because psychiatrists are highly trained they would make accurate diagnoses and that there would be a high level of agreement amongst them.

This is not necessarily the case: the pictures of 'typical clinical patterns' may vary greatly, as illustrated by the contrasting use of the term 'schizophrenia' in the United Kingdom and United States of America highlighted in the US-UK Diagnostic project (Cooper et al, 1972). A total of 500 admissions to a New York psychiatric hospital and to a London psychiatric hospital were interviewed by research psychiatrists following an agreed interview and given a 'project diagnosis' based on this information. In addition the 'hospital diagnosis' was obtained for each patient. According to the latter there were more schizophrenics and alcoholics in the Brooklyn hospital, and more psychotic depressives and people with personality disorders in London. When the project diagnoses were examined there were much smaller differences: there were still more depressives in London and alcoholics in New York but there were no differences between the two centres in the incidence of schizophrenia and other diagnoses. Thus the psychiatrists based in the two hospitals had differing pictures of a typical case of schizophrenia and applied the label to different patients.

This source of error may be reduced by reference to an agreed definition of disorders, such as the International Classification of Disease (I.C.D; World Health Organisation, 1978) or DSM-III (American Psychiatric Association, 1980). The latter is particularly useful as it contains detailed rules for deciding whether or not a patient 'fits' a category, rather than just a description of the typical symptoms of a member of the category. However diagnosis is still dependent on the psychiatrists' interviewing skills, the information they obtain and their interpretation of that information. Consequently it is not certain that two psychiatrists using the same terms to describe their patients, whether it is 'endogeneous depression' or 'personality disorder', are talking about the same type of condition, even when agreed definitions have been used.

This presents a problem for researchers. Comparisons between studies carried out at different centres are made very difficult when the criteria for diagnosis are unknown; replication of the studies is also impeded. For example there is some interest in whether different sub-types of depression have different patterns of cognitive impairment (Miller, 1975). The situation at present is unclear (Section 1.9.3) and it is likely to remain so for as long as there is no way of knowing whether or not one centre's 'endogeneous depression' is the same as another's 'primary depression'. Both the sub-type of depression involved, defined according to an agreed definition, and the method of reaching the diagnosis need to be stated. As long as some studies continue to state that, for instance, their sample of depressed patients consisted of patients 'judged by their psychiatrist to be suffering from depression' (Coughlan and Hollows, 1984) progress is likely to be slow.

The majority of recent studies of memory in depression have used an agreed definition of depression to categorise their subjects. For example some (eg Breslow, Kocsis and Belkin, 1981; Newman and Sweet, 1986) have used the Research Diagnostic Criteria (RDC, Spitzer, Endicott and Robins, 1978), whilst others (eg Abrams and Taylor, 1987; McAllister et al, 1987) have based their diagnoses on DSM-III (American Psychiatric Association, 1980). However, this does not remove all the sources of variability in diagnosis which make comparisons between studies difficult. For instance, there is still a great variety in the terms used, as there is likely to be

until there is more consensus on how depression should be sub-classified (Section 1.3). In addition even when agreed definitions are used psychiatrists still differ in the questions they ask and consequently in the information they obtain. Standardised measures of psychiatric conditions are needed so that diagnoses are not based solely on the investigator's clinical judgments about the questions to ask, how to interpret the information elicited and the diagnosis to apply.

Diagnosis was a problem in this study because four psychiatrists and a clinical psychologist were referring patients and it was not possible to arrange for all patients to be interviewed by an independent psychiatrist who could be asked to carefully examine and record how they reached their diagnosis. If the diagnosis of the referring consultant had been used there would have been no way of knowing how they reached their conclusions, how they applied diagnostic labels and how this compared with the useage of the same terms in, for example, the DSM-III (American Psychiatric Association, 1980). Comparisons with other studies would therefore have been greatly hindered.

Wing and his colleagues at the Institute of Psychiatry have been concerned with the adequate description of psychiatric patients in research for the past 25 years. They have developed the PSE-ID-CATEGO system which goes some way towards resolving the difficulties.

3.3.2 PSE-ID-CATEGO System

This was developed to provide a structured, classifiable and communicable description of mental state (Wing, Cooper and Sartorius, 1974). It consists of three stages; the Present State Examination, the Index of Definition and the Catego program.

The Present State Examination

Wing (1983) describes the Present State Examination (P.S.E) as a standardisation of the type of examination frequently used by psychiatrists. It is a semi-structured interview in which the interviewer follows a schedule containing questions about 140 items. The majority are concerned with psychiatric symptoms, defined in an accompanying glossary

of definitions. The aim is to establish whether the target symptoms are present or have been during the previous four weeks.

Clear definitions of each symptom are provided. For instance 'worry' is defined as 'a round of painful thought which cannot be interrupted by turning attention elsewhere and is out of proportion to the matter worried about'. The interviewer is free to ask other questions besides those specified and to return to ones already covered in order to get enough information to decide whether or not the symptom is present. Once s/he feels that s/he has got enough information s/he records the decision as to whether the symptom is absent (0), present to a moderate degree (1) or present to a severe degree (2). Instructions for making these ratings are provided. The only decisions which have to be made by the interviewer are whether or not the symptom is present and, if it is, how severe it is. The symptoms to be covered and their definitions are both provided. The interview results in a list of symptoms which are then grouped by the ID-CATEGO computer program into syndromes.

These can be used to provide a description of the syndrome profile of the patient, or group of patients. For instance Gath, Cooper and Day (1982) compared the frequency of the ten most frequent syndromes found in women awaiting a hysterectomy for menorrhagia of benign origin with their frequency in a general population survey (Wing, 1976). They found that all the syndromes were more frequent in the hysterectomy group than in the general population. They also present CATEGO classes for the subjects but argue that the syndrome profiles give more information about the type of disorder experienced. Cooper and Fairburn (1986) used the P.S.E. to investigate the characteristics of depressive symptoms in patients with Bulimia Nervosa. Although the total scores on the P.S.E were the same in this group and a group of depressed patients the syndrome profiles of the groups were significantly different: the patients with Bulimia had higher scores on the 'other features of depression' syndrome, and lower scores on 'special features of depression' (see Table 3.4). Syndrome profiles have also been used to show that depressed people from population surveys have a lower incidence of certain features of depression than do in- and out-patients: they have less guilt and retardation, and fewer delusions and suicidal ideas (Wing et al, 1978).

Initially the P.S.E was used exclusively by psychiatrists and used extensively with both in- and out- patients: for instance Wing (1976) reports findings from its use with two in-patient samples and a sample of out-patients. In addition it was used in two large scale projects: the US-UK Diagnostic Project, which is described above (Cooper et al, 1972), and the WHO International Pilot Study of Schizophrenia (WHO, 1973). More recently a shortened version of the interview has been developed which excludes sections relating to psychotic symptoms and has 58 obligatory items (Wing et al, 1977). Psychotic symptoms were excluded from this version (known as the screening version) because of their rarity in a community sample: it was shown that about one half of the items in the full P.S.E were concerned with delusions, hallucinations and abnormal behaviour during the interview and were rarely rated as present except in psychiatric patients.

Non-medical interviewers have been trained to use the screening version of the P.S.E. The training lasts a week and concentrates on interviewing and watching videotapes of psychiatric patients in order to teach psychiatric rather than everyday definitions of terms such as 'depression' and 'worry'. It also provides familiarity with symptoms not normally encountered by lay people.

The original reason for training people other than psychiatrists to use the P.S.E was to make it feasible to use it in large community surveys. Wing et al (1977) comment that 'we consider that non-medical interviewers are well able to use the P.S.E in community surveys etc after appropriate training and to obtain results comparable with those of psychiatrists'. They found a satisfactory degree of concordance between the interviewers' and psychiatrists' total ratings and syndrome scores, whether obtained by re-interviewing the same patient, or from audiotapes of the original interviews. For instance, the product-moment correlation coefficient between total scores obtained by a lay interviewer and a psychiatrist was 0.67 when the patient was re-interviewed, and 0.96 when audiotapes were used. This is of the same order as that found between psychiatrists using both an in-patient and an out-patient sample where the equivalent correlations were 0.80 (two interviews) and 0.97 (interview-audiotape) (Wing et al, 1967). In addition there was good agreement between a lay interviewer and psychiatrist rating the same

interview as to whether the disorder was above or below the threshold point for a psychiatric disorder (kappa=.89). Similar results have been obtained in two other studies (Rodgers and Mann, 1986; Sturt et al, 1981). There is, therefore, good reason to believe that this version of the P.S.E can be used reliably by trained interviewers. It has been used extensively in community surveys; for instance it was used by Brown and Harris (1978) in their famous study of depression in the Camberwell district of London, and, as described above, has been used to investigate the psychological impact of hysterectomy (Gath, Cooper and Day, 1982).

The Index of Definition

This is a method of determining the confidence with which it can be stated that a psychiatric disorder is present (Wing et al, 1978). The rules for determining this are clearly laid down in the ID-CATEGO computer program and take into account both the total P.S.E score and the type, severity and combination of symptoms which are present. They were developed on the basis of clinical experience and have the advantage that they are derived in precisely the same way for all individuals: once the interviewer has established which symptoms are present no further subjective judgements are necessary. Wing et al (1978) found a 90% agreement between the clinical judgement of research psychiatrists as to 'caseness' (defined as someone the psychiatrists would not be surprised to see in an out-patient clinic and would expect to benefit from treatment) and the Index of Definition (I.D) in a survey of eighteen to 65 year old women in South East London. The validity of the I.D is further supported by the findings that more in-patients than out-patients reached the threshold level: 99% versus 83% (Wing, 1976). In addition the number of depressed patients in general practice who reach this level is even lower: 82% of patients getting anti-depressant treatment and 71% of patients getting other treatment (Sireling et al, 1985).

The Index of Definition has eight levels. Level One is defined by the absence of any P.S.E symptoms: the total score is zero. Levels Two and Three are applied to cases where the total score is made up of ratings on non-specific neurotic symptoms (NSN) such as worrying and muscle tension. For Level Two the total score is between one and four, while for the next level it is between five and nine. The rules for defining Level

Four are more complex. It can be determined either by a total score from NSN symptoms of ten or more, or by the presence of one key symptom such as depressed mood or autonomic anxiety in the absence of other related symptoms. Most psychiatrists would not consider this sufficient to warrant a specific diagnosis although there remains the possibility that a disorder may develop. At this level there is insufficient information for the CATEGO program to attempt a clinical classification.

Level Five is known as the threshold level and provides a minimum basis for such a classification. It is determined by the presence of key affective symptoms in moderate forms, either with other related symptoms or with certain other important symptoms. For instance the combination of depressed mood and autonomic anxiety, or depressed mood and pathological guilt would be sufficient for Level Five, even though a total P.S.E score of ten had not been reached. Levels Six, Seven and Eight provide increasing degrees of certainty that the symptoms present can be classified into one of the conventional categories of psychiatric disorder. For instance in depression more 'key' symptoms such as psychomotor retardation would be present at these levels.

The threshold for the presence of a psychiatric disorder (Level Five) is set quite low. Wing (1980) states that it is below the level at which most British psychiatrists would make a definite diagnosis and that consequently there are likely to be more false positives than false negatives (Wing, 1983). This was deliberate as it was felt that missing cases in a community sample was of more importance than including a few non-cases by mistake.

One way of looking at the validity of definitions used in the I.D is to compare them with other criteria laid down for the diagnosis of, for instance, depression. Feighner et al (1972) suggested that for this diagnosis to be made, depressed mood should have been present for at least one month, together with five of eight other symptoms (the presence of four of these symptoms would give a probable diagnosis). Wing et al (1978) applied these criteria to their samples of in- and out- patients, all of whom had reached at least Level Five on the I.D. Sixteen out of 23 in-patient 'depressives' were 'definite' according to the Feighner criteria, one was probable. Seven out of fourteen out-patients were

'definite' and five 'probable'. The I.D is therefore more inclusive than the Feighner criteria. In cases where this is not desirable a higher I.D level can be taken as the level at which a definite classification is appropriate.

The rules used in the Index of Definition are essentially arbitary although based on clinical experience. Their great strength, however, is that they do not vary.

CATEGO Program

This is based on hierarchical principles commonly used in clinical diagnosis in which symptoms from conditions 'higher' up the 'ladder' preclude the diagnosis of a 'lower' condition. For instance Tyrer (1984) describes a hierarchy which has anxiety on the bottom rung, preceded by (in ascending order) phobias; depression; manic and paranoid psychosis; and with schizophrenia at the top. Classificatory rules similar to these have been specified precisely enough to be incorporated into a computer program (CATEGO) and applied to symptom ratings derived from the P.S.E (Wing, Cooper and Sartorius, 1974).

The CATEGO program consists of ten stages, the first eight of which are concerned with reducing the P.S.E symptoms to six descriptive categories. This involves sorting the symptoms into 'clusters' or 'syndromes' and then combining the syndromes in predetermined ways. At the ninth stage the patient is placed in an unique 'descriptive group' or CATEGO class. The final stage allows the incorporation of clinical data about past history and aetological factors where this is desired. The program operates at I.D levels Five and above to provide a standard clinical classification. As its rules are invariant, like those of the I.D, their application to the symptom profile produces a classification which is extremely useful for research purposes.

It is tempting to talk in terms of a 'CATEGO diagnosis' but Wing (1983) cautions against this on the basis that a true psychiatric diagnosis can only be made by trained clinicians making use of past history and exploring aetological factors etc; the CATEGO program is descriptive, it is not truly diagnostic. Despite this however, Wing

observes that the diagnostic significance of the CATEGO classes is shown by the fact that they have been found to be highly concordant with clinical diagnosis in many studies (Wing, 1983). For instance there was a marked degree of association between the project diagnosis of the WHO International Pilot Study of Schizophrenia (IPSS; WHO, 1973), which was based on the the I.C.D, and the CATEGO class with 82% of cases being concordantly classified. There was a similarly high concordance between the UK-US Diagnostic Project research diagnosis (Cooper et al, 1972) and CATEGO class (Wing et al, 1974).

These studies had very few cases of anxiety, which limits their usefulness in assessing the validity of the CATEGO classes in this area. However data from a study conducted in Benim, Nigeria by Binitie and reported by Wing et al (1974) which included a large number of anxious patients found a 79% concordance between the psychiatrists' diagnosis of anxiety and allocation to CATEGO class A. In contrast Dean, Surtees and Sashidharan (1983) compared diagnostic schemes in a community sample and found that the RDC and CATEGO differed substantially in their handling of cases of anxiety; for instance CATEGO only assigns a label of anxiety neurosis when autonomous symptoms are present whilst RDC does not require them to be present. The level of disagreement may also reflect the difficulty observed in several studies in distinguishing between cases of anxiety and depression in community samples (Goldberg et al, 1987; Tyrer et al, 1987). Nevertheless, although CATEGO may differ from other diagnostic systems in its handling of anxiety it is clear what criteria are used to assign patients to this category; this would not be the case if psychiatrists' diagnoses were used.

The CATEGO program sorts depressive disorders into three classes: Class D+, Class R and Class N. Anxiety neuroses are assigned to Class A. The subjects in the present study were all classifed into one of these categories. The chief symptoms, descriptions and ICD-8 categories corresponding to these classes (W.H.O, 1967) are given in Table 3.2.

Table 3.2 DESCRIPTIONS OF CATEGO CLASSES D+, R, N and A

Class D+, Depressive psychosis

ICD diagnosis 296.2 **(manic-depressive, depressed type)**

Chief symptoms:
depressed mood
depressive delusions or hallucinations

Both symptoms must be present in the absence of symptoms of Classes S+ or M+ (schizophrenic and manic psychosis).

Class R, Retarded depression

ICD diagnosis 296.2 **(manic-depressive, depressed type) or**
300.4 **(depressive neurosis)**

Chief symptoms:
depressed mood
retardation
guilt, self depreciation
agitation

The first of these symptoms must be present, together with one of the others, in the absence of depressive delusions or other psychotic symptoms.

Class N, Neurotic depression

ICD diagnosis 300.4 **(depressive neurosis)**

Chief symptoms:
depressed mood
anxiety

The first symptom must be present, in the absence of psychotic symptoms or symptoms characteristic of Class R.

Class A, Anxiety states

ICD diagnosis 300.0 **(anxiety neurosis)**

Chief symptoms:
subjective or observed anxiety
situational anxiety
specific anxiety

Symptoms of depression must not predominate; there must be no psychotic symptoms.

The P.S.E-I.D-CATEGO system is a useful research tool. The only stage at which subjective judgments are made is during the P.S.E interview as the interviewer has to decide whether or not symptoms have been present in the previous four weeks. All the symptoms are, however, carefully defined in an accompanying glossary and rules are given which are followed when making the necessary decisions. In addition the authors stress the need for training for the interviewers in order to promote reliability. Evidence presented above suggests that reliability is high, both when psychiatrists and trained interviewers make the ratings (Wing et al, 1977).

Once a symptom profile has been obtained no other clinical judgments are necessary. Predetermined rules contained in the I.D and CATEGO computer programs are used both to make the decision as to whether the symptoms are sufficient to warrant a specific diagnosis, and to classify the subject into a descriptive category or 'diagnostic' group. Other researchers may disagree with these rules but at least they are clearly laid out and applied in the same way to every case, thus making comparisons between studies much easier than would otherwise be the case. The widespread use of this system and its translation into Chinese and Egyptian, amongst other languages, indicates how valuable it has proved in promoting the reliability of the measurement of severity and the classification of psychiatric disorders in research.

3.3.3 Diagnosis in the Present Study

The psychiatrists who referred patients to this study were not asked to provide a clinical diagnosis and did not play any subsequent part in the diagnostic process. Instead the P.S.E-I.D-CATEGO was used to provide a reliable and standard description and classification of the depressed and anxious subjects. The investigator was trained at the Warneford Hospital, Oxford in 1982 to use the shortened version of the P.S.E, which omits the sections about psychotic symptoms (this was appropriate for this study because patients who were judged by the consultant psychiatrists to have any psychotic symptoms were excluded). The completed symptom profiles were analysed by the CATEGO program held by the Department of Psychiatry, University of Oxford.

Table 3.3 shows the number of patients in the study who fell into the CATEGO classes R (retarded depression), N (neurotic depression) and A (anxiety states). Each class is subdivided by I.D. levels. One patient reached I.D. Level Four, which indicates that her symptoms were such that most psychiatrists would not judge her to be a 'case'. Eight patients (27%) reached I.D. Level Five, the borderline level for 'caseness'; thirteen (43%) Level Six; seven (23%) Level Seven and one (3%) Level Eight. Ten patients (33%) were classifed as having retarded depression, thirteen (43%) neurotic depression and seven (21%) an anxiety state. Information on Catego class and I.D level was not available for four of the 34 patients in this study because P.S.E interviews were not completed with these subjects (In each case it was not possible to include the P.S.E interview in the second testing session because of time constraints. One patient did not want to participate further. Third sessions were arranged with the remaining three subjects: one patient failed to keep the appointment and would not arrange another one; the other two were discharged from the hospital unexpectedly.)

Table 3.3 CATEGO CLASS AND I.D LEVELS OF THE ANXIOUS AND DEPRESSED SUBJECTS

Index of Definition	CATEGO class		
	R Retarded depression	N Neurotic depression	A Anxiety State
4	0	0	1
5	1	4	3
6	2	8	3
7	6	1	0
8	1	0	0

Patients described by the CATEGO program as having retarded depression reached higher I.D. levels than the other subjects: 70% were at Level Seven or Eight, compared with eight per cent of those with neurotic depression and none of those with an anxiety state. The I.D. is a means of expressing the certainty with which the patient is allocated to the CATEGO class and this will depend on the severity of the symptoms. Consequently it is apparent that those patients with retarded depression had both more and more serious symptoms than patients in the other classes: they were therefore iller than the other patients.

The CATEGO program can be used to provide a description of the patients in each CATEGO class. Before it classifies patients it first groups the symptoms which were found to be present during the P.S.E interview into syndromes, using predetermined rules. Levels of certainty for the presence of each syndrome are then established such that the symbol ? means that the syndrome may be present, while + and ++ represent degrees of increasing certainty that the syndrome is present. Those syndromes which are present are then grouped together in predetermined ways in order to allocate subjects to one of the CATEGO classes.

The complete list of symptoms covered by the P.S.E is too cumbersome to be used for descriptive purposes, while the CATEGO classes do not give much information about the particular symptoms experienced: as the classes are primarily used to classify patients their definitions concentrate on the symptoms which differ between the classes, rather than those which may occur in any class. A list of the syndromes (a 'syndrome profile') provides a description of the subjects which falls between the detail of the list of symptoms and the summary provided by the CATEGO classes. Table 3.4 lists the syndromes which were found in the subjects in the present study, together with their full titles and the symptoms which make up the syndrome (full definitions of these syndromes can be found in the P.S.E manual (Wing, Cooper and Sartorius, 1974, pp 118-126)).

Table 3.4 P.S.E-I.D-CATEGO SYNDROMES

Label	Full title	Comprises:
SD	Depressed mood	Depressed mood; hopelessness, inefficient thinking; suicidal ideation
ED	Special features of depression	Self-depreciation; guilty ideas of reference; pathological guilt; dulled emotions
OD	Somatic features of depression	Morning depression; weight loss; early waking; loss of libido; premenstrual tension
LE	Lack of energy	Subjective anergia and retardation
IC	Loss of interest and concentration	as title
DE	Depersonalisation	Derealisation; depersonalisation
IR	Ideas of reference	as title, e.g. marked self-consciousness
ON	Obessional syndrome	Obsessional checking and repeating; obsessional cleanliness and similar rituals; obsessional ideas
GA	General Anxiety	Autonomic anxiety; panic attacks
SA	Situational Anxiety	Situational anxiety; phobias; avoidance of anxiety provoking situations
HY	Hysteria	Hypochondriasis
TE	Tension	Tension pains; muscular tensions; restlessness
WO	Worrying etc.	Worry; tiredness; nervous tension; brooding; delayed sleep
IT	Irritability	as title
SU	Social Unease	Social withdrawal; lack of self-confidence; anxiety in social situations

FIGURE 3.1

FREQUENCY OF PSE SYNDROMES IN THE DEPRESSED [RETARDED & NEUROTIC] AND ANXIOUS PATIENT GROUPS

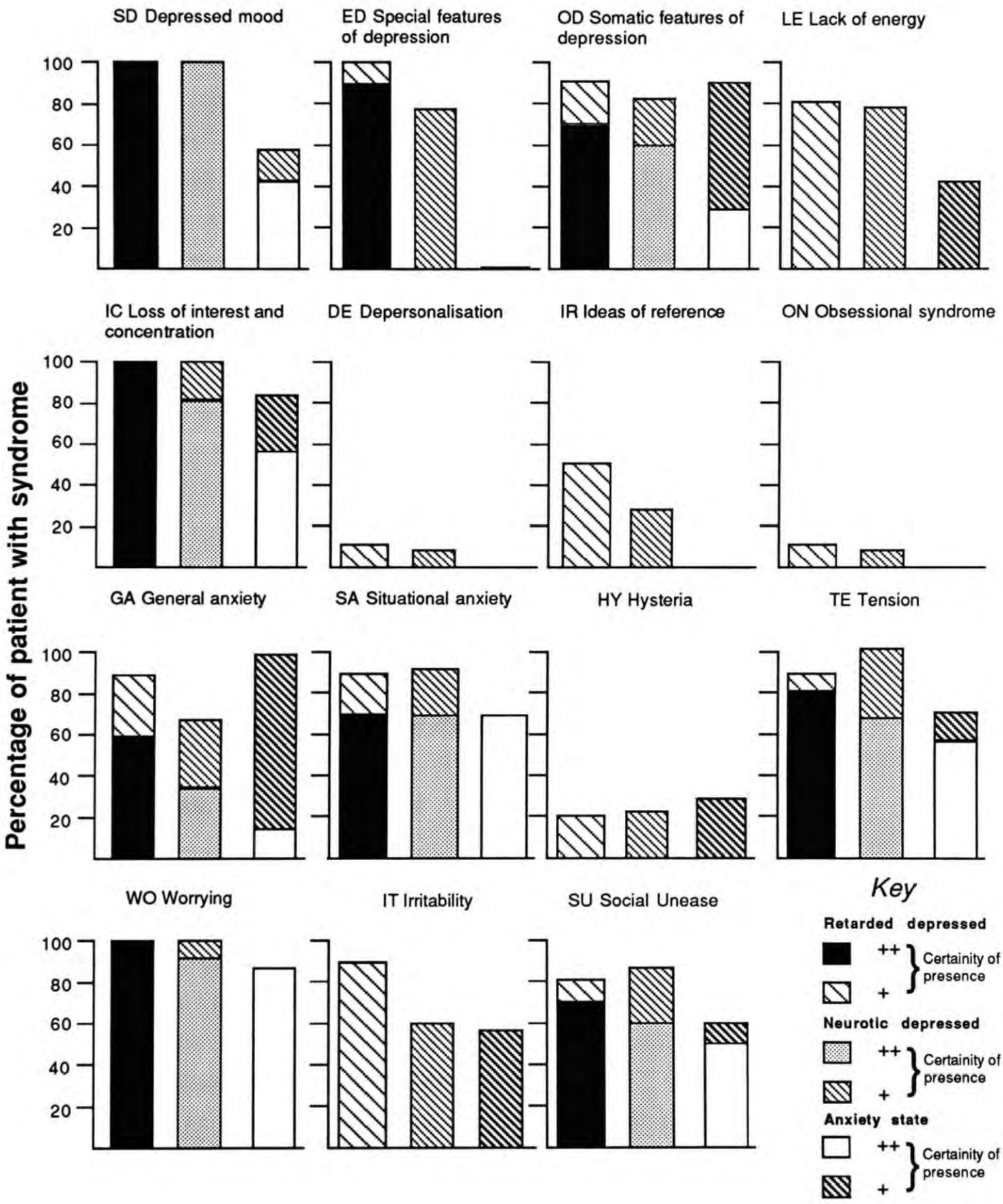


Figure 3.1 shows the percentage of patients in each CATEGO class (retarded depressed, neurotic depressed and anxiety states) who had either a + or a ++ rating on each syndrome. It is apparent that many syndromes were found in all three classes. The frequency of some did not differ much betweeen the groups, for instance 'somatic features of depression' (OD) and 'worrying' (WO). Others are present in each class but are more frequent in some than in others: 'depressed mood' (SD) was found in all three but was more common in retarded and neurotic depression than in the anxiety states. This was expected as the definition of Class A states that depression should not be a dominant symptom. A few syndromes seemed to differentiate between the classes, for instance 'special features of depression' (ED) did not occur at all in anxious patients, occurred only at the least severe level in those with neurotic depression but was very common at the most severe level in patients with retarded depression. This was consistent with the definition of this class which lists self-depreciation and guilt (two of the four symptoms in this syndrome) as characteristic of it: clear evidence of their presence would preclude classification into the other two classes.

It should be noted that depressed mood (SD), general anxiety (GA) and situational anxiety (SA) are all present to some extent in each class. Depressed mood is not exclusive to retarded and neurotic depression, nor is anxiety present only in the patients with an anxiety state.

3.4 CHARACTERISTICS OF DEPRESSED AND ANXIOUS SUBJECTS

3.4.1 Age, Social Class and Years of Education

Subjects were asked their age, the age at which they left full-time education and their occupation (or former occupation if they were male and not currently working, or that of their husband if they were female and not currently employed). They were allocated to a social class on the basis of their occupation. The procedure given in 'The 1971 Census - the Classification of Occupations' (O.P.C.S, 1970) was followed. This assigns people to social classes on the basis of the general standing within the community of the occupation concerned. Table 3.5 shows the social class distribution of the psychiatric subjects. It also shows the age distribution. The mean age was 43.8 years and the distribution ranged

from 23.9 to 68.4 years. The number of years the subjects had spent in full-time education was calculated from the age at which they left education. The distribution of this variable is also given in Table 3.5.

Table 3.5 CHARACTERISTICS OF DEPRESSED AND ANXIOUS SUBJECTS

Social Class Distribution of Anxious and Depressed Subjects	
Class	Number of subjects (%) (N=34)
1 Professional	0
2 Intermediate Occupations	6 (18%)
3N Skilled - non manual	10 (29%)
3M Skilled - manual	10 (29%)
4 Semi skilled	7 (21%)
5 Unskilled	1 (3%)
Age Distribution	
Age range	Number of subjects (%)
20-29	6 (18%)
30-39	9 (26%)
40-49	8 (24%)
50-59	7 (21%)
60-69	4 (12%)
Distribution of Years of Education	
Years of education	Number of subjects (%)
9	4 (12%)
10	19 (56%)
11	6 (18%)
12	4 (12%)
13	1 (3%)

3.4.2 Past History of Psychiatric Problems

The depressed and anxious subjects were asked whether they had had previous episodes of psychiatric troubles. Their accounts were not corroborated from records. Twenty-one of the 32 patients who gave information on their psychiatric history said they had had previous episodes of depression. Two said they had had almost continual problems with depression for twelve and 32 years respectively. Eleven patients had not had prevous psychiatric problems. The median length of the present episode was sixteen and a half months; the range four months to 32 years As this information relies on the patients' recollection it can only be regarded as approximate.

3.4.3 Psychotropic Medication

Information about prescribed psychotropic medication was obtained from the case notes of 29 of the participating patients. Three of these were not on any psychotropic medication. Seven types of medication were prescribed for the remaining patients. These were classified according to the system given by Silverstone and Turner (1982). The classes were M.A.O inhibitors; tricyclic anti-depressants; lithium; benzodiazepine drugs; major tranquillisers (neuroleptics); beta-adrenergic drugs and L-tryptophan. The numbers taking each type of medication, together with the mean, median and range of daily dosages are given in Table 3.6. Patients were seen on more than one occasion and in five cases the medication had been changed between the sessions. The figures given in Table 3.6 are the total number of patients prescibed each class of drug. Consequently the number of patients taking each type of drug at a specific testing session, or when completing a particular test, does not necessarily correspond with these figures. However only the scores of those patients who were taking a specific drug at the time a test was given are included in the analysis of the effects of these drugs on memory (Chapter Five).

The number of drugs prescribed varied from none in three cases to four in one case. Table 3.7 shows the number of drugs prescribed per patient at the first and second testing sessions. Three patients had a further session but the medication was not changed between the second and third sessions.

Table 3.6 PSYCHOTROPIC DRUG USE: THE NUMBER OF PATIENTS TAKING EACH TYPE, AND DETAILS OF DOSAGE.

Type of drug	Number taking drug	Mean dose (mg)	Median dose (mg)	Range (mg)
Benzodiazepines	21	12	11	.25 - 40
Tricyclic anti-depressants	14	132	150	75.0 - 200
Major tranquillisers	9	40	15	2.0 - 100
MAO inhibitors	4	41	45	15.0 - 60
L-tryptophan	4	1375	750	500.0 - 3000
Lithium	3	550	500	400.0 - 750
Beta-adrenergic drugs	4	150	60	60.0 - 240

Table 3.7 NUMBER OF PSYCHOTROPIC DRUGS PRESCRIBED PER PATIENT

Number of drugs prescribed	Testing session	
	One	Two
0	3	3
1	5	3
2	15	17
3	5	5
4	1	1
Median	2	2

3.4.4 Electro-Convulsive Treatment (E.C.T)

Seven patients received E.C.T in the twelve months prior to testing. One patient completed her final session two weeks before the first testing session, one three weeks before it, two at least four weeks before, two more than three months before and one patient between four and five months before. Two patients had had four sessions of E.C.T, one five sessions, two six sessions and two eight sessions. Further information on, for instance, the placement of the electrodes, was not available.

Three patients had had E.C.T more than a year before the study; two of these had had it in the past two years and the third had had it more than twelve years before. One patient in this group had also had it in the past year.

3.5 CONTROL SUBJECTS

In addition to the depressed and anxious subjects a group of people who were not depressed or anxious was also needed in this study. Their performance on tests of memory provide a standard against which to compare the performance of the depressed and anxious subjects. They will be referred to as the 'control' subjects.

3.5.1 Recruitment

The control subjects were recruited from the patients registered with a local general practice. This was judged to be the best way of getting subjects from the same population as the psychiatric subjects, all of whom would have been referred to the psychiatrists by their general practioners. Both groups of subjects were therefore drawn from the lists of patients registered with general practioners who refer patients to the participating psychiatric hospital.

3.5.2 Screening for Psychiatric Disorder

The '10 Questions' taken from the screening version of the P.S.E. (Cooper and MacKenzie, 1981) were used to screen the control subjects for psychiatric disorder. They were developed to identify people who were likely to get a zero or very low score on the P.S.E. and are a brief screening procedure which only takes about five minutes to complete. At least fifteen minutes is needed to complete the P.S.E. which can be very frustrating to both the interviewer and the subject if it is apparent very early in the interview that the subject has no psychiatric symptoms. The '10 Questions' is therefore used first to reduce the chances of this happening.

Each of the '10 Questions' covers a major section of the shortened P.S.E and consequently covers a large amount of information. Each question consists of two separate or related points and the subject scores one if either part is answered positively and two if both parts are. A positive score means that the interviewer judged a positive rating on the related questions on the full P.S.E. would be the most likely result if the full P.S.E. was used after subject's reply to the screening question. Cooper and MacKenzie (1981) tested these questions in a variety of population studies and on patients who were attending a psychiatric out-patient clinic. In each case they followed the '10 Questions' with the P.S.E. They concluded that very few 'cases' would be missed if a score of two on the '10 Questions' was used as a cut-off point for further interviewing with the P.S.E. A score of zero or one on the '10 Questions' when they are administered by a trained P.S.E interviewer is, in their opinion, a very effective predictor of a zero or low score on the P.S.E.

In this study a score of two was taken as a cut-off point for controls: if they scored more than this they could not be included in the control group unless they were also found to have a very low score on the complete P.S.E interview. In the event none of the controls scored more than two and consequently the P.S.E was not used with these subjects. Four patients had zero scores on the '10 Questions', five scored one and two scored two. They were all therefore judged to be free of psychiatric disturbance.

In addition to using the '10 Questions' to screen for current psychiatric problems the controls were asked whether they had ever had any problems with their 'nerves'. Six subjects said that they never had. The remaining subjects had all consulted their general practioner for problems with their 'nerves' or for difficulties with sleeping. None had ever been referred to the psychiatric services. The length of time which had elapsed between these difficulties and the present study ranged from six to twelve years, with a mean of 8.8. It was felt appropriate to include these subjects in the control group because of the length of time which had elapsed and because none had any signs of current psychiatric problems. None were taking any kind of psychotropic medication. It may well be that such experiences in the past make people more likely to agree to take part in studies on depression.

3.5.3 Characteristics of Control Subjects

Age, Social Class, and Years of Education

Like the depressed and anxious subjects the controls were asked their age, the age at which they left full-time education, their occupation (or former occupation if male and unemployed, or that of their husband if female and not currently employed). They were allocated to a social class on the basis of their occupation. Table 3.8 shows the social class distribution of the controls. The control group had a higher median social class than the depressed and anxious subjects: 3N rather than 3M. Almost 25% of the anxious and depressed subjects were in social classes 4 and 5, whilst none of the control ones were.

Table 3.8 also shows the age distribution. The mean age was 38.1 years and the distribution ranged from 23.9 to 60.3 years. Fifty-five percent of the controls and 45% of the depressed and anxious subjects were under forty; 18% of the controls and 32% of the depressed and anxious subjects were over fifty.

Table 3.8 CONTROL SUBJECT CHARACTERISTICS

Social Class Distribution of Control Subjects

Class	Number of subjects (%) (N=11)
1 Professional	1 (9.0%)
2 Intermediate Occupations	4 (36.4%)
3N Skilled - non manual	4 (36.4%)
3M Skilled - manual	2 (18.2%)
4 Semi-skilled	0 (0 %)
5 Unskilled	0 (0 %)

Age Distribution

Age range	Number of subjects (%)
20-29	4 (36.4%)
30-39	2 (18.2%)
40-49	3 (27.3%)
50-59	1 (9.1%)
60-69	1 (9.1%)

Distribution of years of education

Years of education	Number of subjects (%)
9	2(18.2%)
10	3(27.3%)
11	2(18.2%)
12	1(9.0%)
13	3(27.3%)

The controls were asked how many years of full-time education they had completed. The distribution of years of education is given in Table 3.8. The median length of time spent in full time education was eleven years, compared to ten years in the depressed and anxious subjects. This may be due to the lower average age of the controls which meant that

proportionally more of them would have had to stay at school until sixteen because of the raising of the school leaving age from fifteen to sixteen. However, as 27% of the controls and only three per cent of the anxious and depressed subjects had spent thirteen years in full time education it is likely that the control subjects were better educated.

3.6 SUMMARY

Thirty-four depressed and anxious patients were recruited from a local psychiatric hospital. They were suffering from clinical depression and/or anxiety states: none had psychotic symptoms. Practical difficulties were experienced in recruitment and the final numbers were lower than originally planned, although adequate for the purposes of this study. Eleven control subjects were recruited from patients registered with a local general practioner. Each subject was asked their age, social class and the number of years they had spent in full-time education.

A clinical diagnosis and description of the depressed and anxious subjects was obtained from the P.S.E.-I.D.-CATEGO system developed by Wing et al (1974). This provides a highly replicable and reliable psychiatric description of the patients. Information on past episodes of psychiatric disorder was obtained from the patients and details of prescribed psychotropic medication were taken from their case notes.

The '10 Questions' of the P.S.E, developed as a rapid predictor of zero or negative scores on the P.S.E, were used to ensure that the control subjects were free of psychiatric disorder. In addition they were asked about any past history of psychiatric problems. Although all the controls had low or zero scores on the '10 Questions', five had had minor problems in the past which had led to consultations with their general practioners. In each case this had been at least six years previously and it was therefore decided to include these people in the control group.

THE PERFORMANCE OF DEPRESSED, ANXIOUS AND CONTROL SUBJECTS ON A BATTERY OF MEMORY TESTS.

4.1 INTRODUCTION

This chapter is concerned with the performance of depressed, anxious and control subjects on the battery of memory tests described in Chapter Two (Section 2.2.1). Three questions are addressed:

1) Are clinically depressed patients impaired on the battery of tests compared to subjects who are neither depressed or anxious (control subjects)?

2) Are there significant differences between the performance of retarded depressed patients and neurotic depressed patients on these tests?

3) Does the performance of anxious psychiatric patients differ significantly from that of the depressed patients?

Each of these questions will now be considered in turn.

4.1.1 Are Clinically Depressed Patients Impaired on the Battery of Memory Tests Compared to Control Subjects?

As the literature review in Section 1.4 has shown, there is considerable evidence for memory impairments in depression. However, there is no agreement as to what form the deficits take and which groups of depressed people will be significantly impaired. It was therefore decided to look at a group of carefully described (Section 3.3.3) depressed psychiatric in- and day-patients to see whether such patients have memory impairments and, if so, what types of tasks they are impaired on.

The performance of the depressed patients on the battery of memory tests described in Section 2.2.1 is compared with that of controls who were neither depressed or anxious (Section 3.5). The tests used cover registration and immediate memory, retention and retrieval. Registration is measured using a digit span test, while immediate memory (or new

learning) is measured with a variety of tests which include the recall of unconnected words (Free Recall Test, Serial Learning Test) and the recall of a prose passage (Prose Passage Recall). Previous reports have suggested that new learning is impaired in depression (eg Breslow, Kocsis and Belkin, 1980; Cronholm and Ottosson, 1961; Frith et al, 1983; Steif et al, 1986; Sternberg and Jarvik, 1976). It is therefore anticipated that the depressed patients in this study will be impaired on the tests of new learning, especially those, like Free Recall and Serial Learning, which lack the structure and redundancy which would aid processing. In addition it is expected that they will also be impaired on the two tests which present information several times in order to measure the speed of learning (Paired Associate Learning and Serial Learning).

The battery also includes tests of the amount of information subjects forget between immediate and delayed recall. The evidence from previous studies (eg Cronholm and Ottosson, 1961; Sternberg and Jarvik, 1976) suggests that depressed patients are able to retain information once learned as well as controls, and it is therefore expected that the depressed and control subjects will not differ significantly on these tests.

Some studies have found that depressed subjects are impaired on both free recall and recognition memory tests (eg Calev and Erwin, 1985; Wolfe et al, 1987) whilst others have found them to be impaired on both free recall and cued recall tests (Kopelman, 1986; Watts and Sharrock, 1987). However it might be expected that, as depressed subjects have been shown to be most impaired on tests requiring effort for completion (Section 1.8.1), free recall tests would show most impairment because they are presumed to demand more effort for successful retrieval. Tests using free recall (Free Recall test, Serial Learning test), cued recall (Paired Associate Learning) and recognition (Picture Recognition test) were therefore included in the battery.

Questionnaires for past public events (Section 2.2.2) were also included in the battery in order to look at memory for events presumed to have happened before the onset of depression. If depressed subjects have difficulty retrieving information from memory then they would be expected to be impaired on these tests, and in particular on the free recall

version. If, however, they just have difficulty learning new material then they should do as well as control subjects on these tests. Similar tests were used by Squire, Slater and Chace (1975) to look at the effects of E.C.T on memory: before treatment depressed patients performed as well as controls, suggesting that the impairment in depression is restricted to learning new information. However, it has been suggested that depressed patients may show impairments on any sufficiently complex task regardless of the stage of memory involved (Cohen et al, 1982) and it is therefore possible that they will be impaired on these questionnaires.

β scores, a measure of response bias, were calculated from scores on the Picture Recognition test (Section 2.2.1). Miller and Lewis (1977) found evidence that elderly depressed subjects had conservative response criterion but other studies have failed to replicate this finding (Hilbert, Niederehe and Kahn, 1976; Watts et al, 1987). This test was therefore included in the battery in order to investigate whether depressed subjects have conservative response criteria.

In summary, this chapter investigated whether depressed in- and day-patients have impaired memories and, if so, what form the impairment takes. The performance of depressed and control subjects was compared on a battery of memory tests designed to investigate whether depressed subjects have difficulty learning new material, retaining information in memory and retrieving information once learned. As already noted, it is possible that if the tests are sufficiently complex the depressed patients would be impaired on all tests regardless of the stage of memory involved. In addition, β, a measure of response bias, was included to see if depressed subjects have conservative response criteria.

4.1.2 Are there Differences in the Performance of Retarded Depressed and Neurotic Depressed Patients?

Few studies have compared sub-types of depressed individuals to see if they show the same extent and types of memory impairment (Section 1.9.3). The conclusion reached by Miller (1975) that there is little evidence for differences between sub-types of depression which cannot be explained by differences in the severity of depression therefore still stands.

This chapter compares the performance of two groups of depressed patients, categorised according to the PSE-ID-CATEGO system (Section 3.2.2) as having either retarded depression or neurotic depression. The characteristics of these classes are given in Table 3.2. The retarded depressed group are characterised by symptoms such as retardation, agitation, guilt and self-depreciation which have been shown (Nelson and Charney, 1981) to be associated with the syndrome of severe depression labelled as 'endogenous' or 'melancholic' depression (Section 1.3.2). In addition they reached higher Index of Definition (I.D) levels (Wing, Cooper and Sartorious, 1974) and therefore had both more and more serious symptoms than neurotic depressed patients (Section 3.2.3). If memory impairments in depression are particularly associated with symptoms characteristic of endogenous depression such as retardation (Section 1.8.3), then the retarded depressed group would be expected to show the most impairments. This would also be true if the degree of impairment is related to the severity of the depression. It is also possible that the two groups of depressed patients will differ in the type, as well as the extent, of impairment. The pattern and extent of impairment shown by each group was therefore compared in this study.

4.1.3 Do Anxious Psychiatric Patients Show the Same Pattern of Impairment as Depressed Patients?

Miller (1975) noted that there was little evidence for memory impairments which were unique to depression and not shown by other psychiatric groups, notably schizophrenic patients. A few studies have compared memory in depression and schizophrenia since that time (Section 1.9.2) but there has been virtually no research on how memory in depression compares to that in another common psychiatric condition, anxiety. The exception is a study by Brands and Jolles (1987) which compared the performance of depressed and anxious patients on the Sternberg short-term memory scanning procedure and found that the depressed patients had significantly slower reaction times and showed slower memory scanning on one task.

The lack of research comparing memory in depressed and anxious patients is surprising given the evidence that anxiety often impairs memory (reviewed below) and the fact that it can be difficult to

distinguish between cases of depression and anxiety (Tyrer et al, 1987; Goldberg et al, 1987). If the two conditions are found to have different patterns of memory impairment this could help to distinguish between them. If, however, the pattern of impairment does not differ, this might lend weight to the argument that in many cases it is not meaningful to differentiate between depression and anxiety and that instead many patients could be correctly be assigned to a mixed anxiety-depression disorder (Goldberg et al, 1987).

There is evidence that anxiety does affect memory performance, and performance on other cognitive tests. For instance, Eysenck (1979) reported that eleven out of twelve studies showing a significant effect of state anxiety or situational stress on digit span performance found that anxiety impaired performance, while Stelmack et al (1984) found that state anxiety reduced recognition memory for pictures. Mueller (1979) argued that anxious subjects did not process information as deeply as other subjects, used less elaboration and were less flexible in utilising alternative memory strategies. He conducted a series of studies on free recall in anxiety and found that in six out of eleven studies anxiety significantly impaired retention (Eysenck, 1982). Trust and Oatley (1984) found that highly aroused, and presumabably highly anxious, expectant mothers at an ante-natal clinic remembered less information and advice about pregnancy than less anxious mothers-to-be. Bond, James and Lader (1974), conducted one of the few studies to use anxious psychiatric patients rather than students high in state or trait anxiety; they found that chronically anxious patients were significantly impaired on a variety of psychological measures. Eysenck (1982) reviewed the evidence for memory impairment in anxiety and concluded that anxiety typically impaired performance on most tasks.

Anxiety is not always associated with impairment however. For instance Mogg, Mathews and Weinman (1987) found no difference between clinically anxious patients and controls in the recall of adjectives or in d' scores calculated from a recognition memory task, while Arkin, Detchon and Maruyama (1982) found that students high in test anxiety performed significantly better than students with low levels of anxiety on an easy anagram task, but significantly less well than the low anxiety group on difficult tasks. The relationship between anxiety or arousal and

performance is described in the Yerkes-Dodson law (Yerkes and Dodson, 1908) which states that there is an inverted U-shaped function relating to arousal and performance, with intermediate levels of arousal being associated with highest levels of performance. In addition, it suggests that the optimal level of arousal decreases as the task difficulty increases. This describes the interaction between anxiety and task-difficulty found in the study by Arkin, Detchon and Maruyama (1982) and indicates that impairment would not be expected on all tasks. However it only describes the relationship between arousal or anxiety and performance, and does not explain it.

It has been suggested that anxiety consists of two components: emotionality, involving changes in physiological functioning and states of uneasiness and tension, and worry, the cognitive concern about the consequences of failure (Liebert and Morris, 1967; Morris, Brown and Halbert, 1977). There is evidence that poor performance is related to the worry component of anxiety. For instance, Morris and Liebert (1970) found that the correlation between worry scores and final exam grade in a group of students was negative and significant when emotionality was partialled out, whilst the relationship between emotionality and grades with worry partialled out was not significant. Deffenbacher (1978) argued that anxious subjects might be distracted by worry, or by heightened autonomic arousal (emotionality) or by competing response tendencies generated by the task. He asked students high or low in test anxiety to solve difficult anagrams under conditions of high or low stress, and found the worst performance in highly anxious students in the high stress condition. According to a post-test questionnaire this group experienced more interference than the other groups from emotionality, worry and the task, with worry having the largest effect. In a more recent study (Deffenbacher, 1986) students were asked to complete indices of worry, emotionality and task-generated interference immediately after a mid-term exam. Highly anxious subjects performed most poorly. When all three indices were regressed on performance only worry was significantly related to it; this indicated that worry was the most important source of interference from anxiety.

These studies suggest that performance is impaired in anxiety because of cognitive interference. This is supported by the finding of Arkin,

Detchon and Maruyama (1982) that the highly anxious subjects in their study reported experiencing considerably more cognitive interference than low anxiety subjects. Eysenck (1982) suggested that anxious subjects are dividing their attention between task-requirements and task-irrelevant cognitive activities such as worry and self-criticism. He presumed (Eysenck, 1979) that the part of the processing system most involved in the concurrent processing of task-relevant and irrelevant information is the limited capacity central processor and the articulatory loop of the working memory (Baddeley and Hitch, 1974). If this is the case it would have far reaching implications because, as Eysenck (1979) notes, the working memory is hypothesised to be critically involved in the processing and temporary holding of information; any reduction in its capacity resulting from task-irrelevant processing will affect the performance of a great variety of cognitive tasks. However, the degree of impairment shown will depend on the complexity of the task and the demands it therefore makes on working memory capacity.

Evidence that the capacity of working memory is reduced in anxiety comes from the finding, reported above, that anxiety typically reduces digit span. In addition Eysenck (1982) reports a study in which anxious subjects divided their attention between a main task, which varied in complexity, and a concurrent attention-demanding subsidiary task. High anxiety had a much greater detrimental effect on difficult tasks, presumably because they needed more cognitive capacity for successful completion. In another study, again reported by Eysenck (1982), subjects high or low in anxiety solved anagrams whilst counting backwards in threes (memory load condition), whilst rehearsing over-learned material (articulatory suppression) or whilst not performing an additional task. Anxiety produced a very large decrement in the memory-load condition, presumably because the attentional demands of the digit task when combined with the reduced capacity associated with anxiety meant that very few resources were available for the anagram task.

As already noted, there is evidence that the degree of impairment caused by anxiety will vary according to task difficulty. This is supported by the results of studies generated by Spence and Spence's (1966) theory of anxiety and performance. This theory stated that habit strength (measure of the strength of learning) multiplied by drive

produced excitatory potential. In a simple task, where there were no competing responses, anxiety (increased drive) would cause the correct response to be strengthened, more likely to exceed the response threshold, and therefore increase performance. On a more complex task, however, the correct response would have to be discriminated from other competing responses which may be stronger than the correct response; any increase in anxiety would therefore make competing responses even stronger and lead to a performance decrement. This theory led to studies on paired associate learning using pairs of words which differed in the number and strength of competing responses. Eysenck (1982) concluded that the available evidence showed that performance on more difficult pairs (those with strong competing responses) was impaired by anxiety, as hypothesised by Spence and Spence's theory. However, Eysenck argues that Spence and Spence's theory has limitations in that performance can only be predicted if the number and strength of all relevant responses is known which, as Eysenck points out, is rarely the case. In addition it only explains the effects of anxiety on retrieval, whilst there is evidence that anxiety also affects learning (eg Straughen and Dufort, 1969).

Eysenck (1982) therefore concluded that Spence and Spence's concept of intra-task response competition was not supported and that instead the results of the paired associate studies could be understood in terms of task-difficulty: learning pairs of words which had strong competing responses would make more demands on working-memory capacity and therefore would be impaired by high levels of anxiety which lead to a reduction in working memory capacity. In addition, he suggests that difficult tasks may be particularly impaired by high anxiety because they produce feelings of failure which lead to increased levels of anxiety.

The hypothesis that anxiety leads to a reduction in working memory capacity does not explain why anxiety has often been found to facilitate performance on easy tasks (eg Arkin, Detchon and Maruyama, 1982). However, Kahneman (1973, Section 1.8.1) suggested that one of the main determinants of the amount of effort expended by an individual is their evaluation of task demands. If anxious individuals have fewer processing capacities because of task-irrelevant processing, task demands may be greater for them. They may therefore attempt to compensate for reduced

capacity by expending more effort and consequently increasing attentional resources. Eysenck (1979) suggested that processing effectiveness, the relationship between the quality of performance and the effort invested in it, will always be reduced in anxiety on any task making demands on working memory. However, the extent to which anxiety affects processing efficiency (a measure of the quality of performance) will depend on the extent to which highly anxious subjects compensate for reduced processing effectiveness by enhanced effort.

Evidence that anxious subjects are more motivated and therefore expend more effort comes from a study in which the performance of individuals high and low in trait anxiety was compared on a letter transformation task. This was performed in the presence or absence of monetary incentive for superior performance (Eysenck, 1985). Anxiety only impaired performance on the more complex version of the task, while motivation in the form of monetary incentive improved the performance of low-anxiety subjects but had no effect on high-anxiety subjects. Presumably this was because the anxious subjects were trying to compensate for the adverse effects of anxiety by increasing effort and consequently the amount of processing capacity allocated to the task; they therefore were unable to further increase processing capacity when offered the monetary incentive.

Eysenck (1982) suggests that increased anxiety will not always lead to an increase in effort. For instance, one of the reasons why anxious individuals are presumed to expend more effort on tasks is because they believe that successfully completing the task will lead to a reduction in anxiety. If, therefore, they believe that they are unlikely to alleviate their anxiety by successfully completing the task they are not likely to invest a lot of effort in it. In addition, if the chances of successfully completing the task are perceived to be low, subjects are unlikely to be motivated to put much effort into it (Revelle and Micheals, 1976). Eysenck also suggests that anxiety should lead to increased effort if the source of anxiety is intrinsic to the task, for instance caused by fears of failure, but that there is no reason to suppose that anxiety will be associated with increased effort if the cause of anxiety is unrelated to the task.

It is difficult to predict whether or not clinically anxious patients will be impaired on the battery of memory tests used in this study. There have been few studies of memory in such patients and the available evidence is contradictory; Bond, James and Lader (1974) found impairment on psychological tests whilst Mogg, Mathews and Weinman (1987) found no evidence of memory impairment in similar patients. According to Eysenck (1979) high levels of anxiety at the time of testing would lead to reduced processing capacity, but whether or not this resulted in impairment would depend both on the level of task-difficulty and on whether the subjects increase the amount of effort they put into the task in an attempt to compensate for the reduction of processing capacity. If they do not expend more effort they might be expected to perform at similar levels to the depressed patients who have also been hypothesised to have reduced processing capacity and to expend little effort on the tasks (Hasher and Zacks, 1979; Section 1.8.1). This chapter therefore investigates whether or not anxious patients show the same pattern of performance on these tests as depressed patients, or whether they are able to overcome the hypothesised reduction in processing capacity and therefore perform as well as the control subjects.

4.1.4 Summary

This chapter compares the performance of depressed patients on a battery of memory tests to that of control subjects who were neither depressed or anxious; it compares the performance of two sub-types of depressed patients, retarded depressed and neurotic depressed; and, finally, it investigates the performance of anxious psychiatric patients on the test battery and compares it to that of both the depressed patients and the control subjects.

4.2 METHOD

This section gives a brief outline of the subjects and materials used in this study; both have been described in detail in the preceding chapters (materials and procedure, Chapter Two; subjects, Chapter Three).

4.2.1 Memory Test Battery

Details of these tests including their development, the type of material used and mode of recall are given in Sections 2.2.1 and 2.2.2. Some of these tests resulted in more than one variable as they were designed to measure more than one component of memory. The variables measuring immediate memory and the speed of learning new material are listed in Table 4.1, together with information on how they were derived from the original eight tests. Similarly Table 4.2 lists the variables measuring the retention or forgetting of information and the retrieval of information already in memory. In addition β, a measure of response bias rather than memory per se, was calculated fron scores on the Picture Recognition test: scores for Trials One and Four are given, together with the variable 'β - change' which was calculated by subtracting the score from Trial Four from that on Trial One.

4.2.2 Subjects

The subjects used in this study are described in Chapter Three. They were divided into four groups on the basis of their psychiatric condition (or lack of one): retarded depression; neurotic depression; anxiety state; and controls who were not anxious or depressed. The numbers in each group are given in Table 4.3, together with their mean age and mean years of education. Table 4.4 shows the social class distributions. The groups did not differ in their mean age ($F(3,37)=0.65$, $p>0.05$) or in the number of years spent in full-time education ($F(3,37)=0.99$, $p>0.05$). In addition there was no significant difference between the groups in the proportion having a non-manual (Classes 1, 2 and 3N) versus manual (Classes 3M, 4 and 5) occupation ($\chi^2=3.9$, df=3, NS).

Table 4.1 VARIABLES MEASURING REGISTRATION, IMMEDIATE MEMORY, AND SPEED OF LEARNING NEW INFORMATION

Variable	Test from which variable derived
Registration	
Digit Span Forwards	Digit Span Forwards.
Immediate memory	
d' - Trial One	Picture Recognition test, Trial One.
Free Recall - total	Total score, Free Recall test.
Serial Learning - immediate recall	Trial One, Serial Learning test.
Paired Associate - immediate recall	Trial One, Paired Associate Learning test.
Prose Passage One - immediate recall	Passage One, immediate recall, Prose Passage recall.
Digit Span Backwards	Digit Span Backwards
Speed of learning	
Serial Learning - Trial Two; Serial Learning - Trial Three; Serial Learning - Trial Four.	Mean on Trial Two; mean on Trial Three; mean on Trial Four, Serial Learning test.
Serial Learning - speed of learning	Mean score on Trials Two to Four, Serial Learning test.
Paired Associate - Trial Two; Paired Associate - Trial Three; Paired Associate - Trial Four	Mean on Trial Two; mean on Trial Three; mean on Trial Four, Paired Associate Learning test.
Paired Associate - speed of learning	Mean score on Trials Two to Four, Paired Associate Learning test.

Table 4.2 VARIABLES MEASURING RETENTION OR FORGETTING OF INFORMATION, AND THE RETRIEVAL OF INFORMATION ALREADY IN MEMORY

Variable	Tests from which variables derived
Retention/forgetting	
d' - forgetting	Difference between Trial One and Trial Four, Picture Recognition test.
Prose Passage One - forgetting	Difference between delayed and immediate recall, Passage One, Prose Passage recall.
Prose Passage Two - delayed recall	Passage Two, Prose Passage recall
Prose Passage One - delayed recall	Delayed recall of Passage One, Prose Passage recall
Paired Associate - forgetting	Difference between Trial Four and Trial Five, Paired Associate Learning test.
Paired Associate - Trial Five	score on Trial Five, Paired Associate Learning test.
Memory for past public events	
Past Events free recall questionnaire	Total score on this questionnaire
Past Events multi-choice questionnaire	Total score on this questionnaire

Table 4.3 MEAN AGE AND MEAN NUMBER OF YEARS OF FULL TIME EDUCATION (YEARS AND DECIMAL YEARS) OF THE FOUR SUBJECT GROUPS.

Subject group	N	Age		Years of education	
		Mean	(SD)	Mean	(SD)
Retarded depression	10	38.9	(9.2)	10.3	(0.9)
Neurotic depression	13	42.5	(15.9)	10.5	(1.1)
Anxiety state	7	45.4	(12.1)	10.1	(0.9)
Control subjects	11	36.6	(12.4)	11.0	(1.6)

Table 4.4 SOCIAL CLASS DISTRIBUTION

Subject group	N	Social class 1	2	3N	3M	4	5
Retarded depression	10	0	2	3	2	3	0
Neurotic depression	13	0	3	4	2	3	1
Anxiety state	7	0	1	1	4	1	0
Control subjects	11	1	4	4	2	0	0

4.2.3 Analysis

One-way analysis of variance was used on most of the variables to test the null hypothesis that there is no significant difference between the scores of the four subject groups on the memory tests. A non-parametric equivalent, the Kruskal-Wallis test, was used in cases where the variance differed significantly between the groups.

This study was designed to investigate whether the performance of the two groups of depressed patients differed from that of the control subjects; whether the performance of retarded depressed patients differed significantly from that of neurotic depressed patients, and whether that of the anxious patients differed from that of the depressed patients and the control subjects. Multiple comparisons were therefore used following statistically significant one-way analyses to compare the performance of each group to that of each other group. As there were unequal group sizes Gabriel's test for multiple comparisons was used (Kendall and Stuart, 1968). This has been designed to give significant levels which take into account the fact that the comparisons are not independent. It is very similar to Tukey's Honestly Significant Difference test, which can only be used with equal sized groups.

It was not appropriate to use Gabriel's test when the non-parametric Kruskal-Wallis test had been used initially. Instead, multiple comparisons were carried out using the Mann-Whitney U test: when there are two samples this is identical to the Kruskal-Wallis test. Amended

significance levels were used because the individual levels for each comparison do not take into account the fact that several comparisons are being made. Following the procedure recommended by Leach (p161, 1979) it was decided that, in order to get a true overall significance level of 0.05, each comparison should have a significance level of 0.05/6 (the number of comparisons). This meant that the comparison had to have a probability level of less than 0.008 to be accepted as significant at the five per cent level. Similarly the comparisons had to have a probability of less than 0.017 (0.1/6) to be significant at the ten per cent level. As this method is quite conservative it was decided to accept a significance level of 0.1 as indicating that there was a true difference between the groups.

In two cases the same test was given several times in order to look at the speed of learning of new material (Serial Learning test and Paired Associate Learning test). Two-way analysis of variance with repeated measures on one factor (test trials) was used on this data to test the null hypothesis that the four subject groups did not differ significantly; that performance did not change over the four trials of the tests; and that there was no interaction between group and trial.

The Picture Recognition test, from which d' and β were calculated, was given several times in order to assess the rate of forgetting. As d' and β cannot be calculated when the probability of a hit or false positive is zero (Section 2.2.1), there was missing data for different subjects on different trials. If a two-way analysis of variance had been used on this data a third of the cases would have been excluded due to missing data. It was therefore decided that as performance was expected to decline over the four trials it would be appropriate to fit a regression line to the d' data points for each case and to compare the regression coefficients for each group. This gave a measure of change over time which made the most efficient use of the available data. However, there was no reason to presume that β levels would decline over time and therefore a regression line was not fitted to this data.

4.3 RESULTS

The results for tests of registration and immediate memory are given first, followed by those for speed of learning, retention, memory for past public events and, finally, response bias (β). Within each section the results of the one-way anova or Kruskal-Wallis test of differences between the four subject groups are presented first. If these showed significant differences between the groups multiple comparisons were made between the four groups. The comparison of the depressed and control subjects is presented first, followed by the comparison of two groups of depressed patients, and finally the results of the comparisons of the anxious patients with both the depressed and control subjects.

4.3.1 Registration and Immediate Memory

There was no significant difference between the groups on the test of registration: the Digit Span Forwards test ($F(3,35)=1.23$, $p>0.05$, Figure 4.1). The differences between the groups on the variables measuring immediate memory were statistically significant (d'- Trial One, $F(3,32)=6.84$, $p<0.01$, Figure 4.2; Free Recall - total, $F(3,37)=4.36$, $p<0.01$, Figure 4.3; Serial Learning - immediate recall, $F(3,36)=5.76$, $p<0.01$, Figure 4.4; Prose Passage One - immediate recall, $F(3,34)=3.38$, $p<0.05$, Figure 4.5; Paired Associate - immediate recall, $F(3,37)=2.98$, $p<0.05$, Figure 4.6; Digit Span Backwards $F(3,35)=3.16$, $p<0.05$, Figure 4.7).

Gabriel's test for multiple comparisons was then used to compare every group with every other group on each of the six variables which had shown a significant difference between groups. The retarded depressed subjects differed from the control subjects at the one per cent level on Free Recall - total and d' - Trial One, and at the five per cent level on the remaining four variables: in each case they scored less than the controls. The neurotic depressed subjects differed significantly from the controls at the one per cent level on the Free Recall - total and Serial Learning - immediate recall variables, and at the five per cent level on d' - Trial One. They did not differ significantly from the controls on the remaining three variables, although the differences on these variables were in the expected direction with the depressed subjects recalling less than the controls.

The retarded depressed subjects were, therefore, clearly impaired on all six variables measuring immediate memory. The less severely ill neurotic depressed subjects were significantly impaired on three of the variables. The memory impairments shown by these depressed subjects are central to this thesis: the next three chapters explore aspects of the impairment, including its relationship to self-rated depression and anxiety levels (Chapter Six) and to the subjects' reports of memory problems (Chapter Seven).

The two depressed groups differed significantly on only one of the six variables, d' - Trial One, with the retarded depressed group getting significantly lower scores. Thus on three of the variables (Prose Passage One - immediate recall, Paired Associate - immediate recall and Digit Span Backwards) the two depressed groups did not differ significantly despite the fact that the scores of the retarded depressed group differed significantly from those of the controls while those of the neurotic depressed group did not.

The anxious group achieved higher scores than the depressed subjects and lower scores than the control group on all six variables. They did not differ significantly from the control subjects on any of the variables, although there was a trend for them to do so on Serial Learning - immediate recall and Digit Span Backwards ($p<0.10$, >0.05). They scored significantly higher than the retarded depressed subjects on d' - Trial One ($p<0.05$) and there was a trend for them to do so on Free recall - total ($p<.01$, >0.05). There was a similar trend towards a significant difference on this variable between the neurotic depressed and anxious subjects. Thus, despite the consistent pattern of scores, the difference between the anxious subjects and the other groups only reached statistical significance on one variable. This is probably due to the fact that there were only seven subjects in the anxious group. It is therefore not clear whether the anxious patients have a similar pattern of impairment to the depressed patients, although in a less severe form, or whether they are in fact performing comparably to the control subjects.

FIGURE 4.1

SCORES OF FOUR SUBJECT GROUPS ON DIGIT SPAN FORWARDS (MEAN±SD)

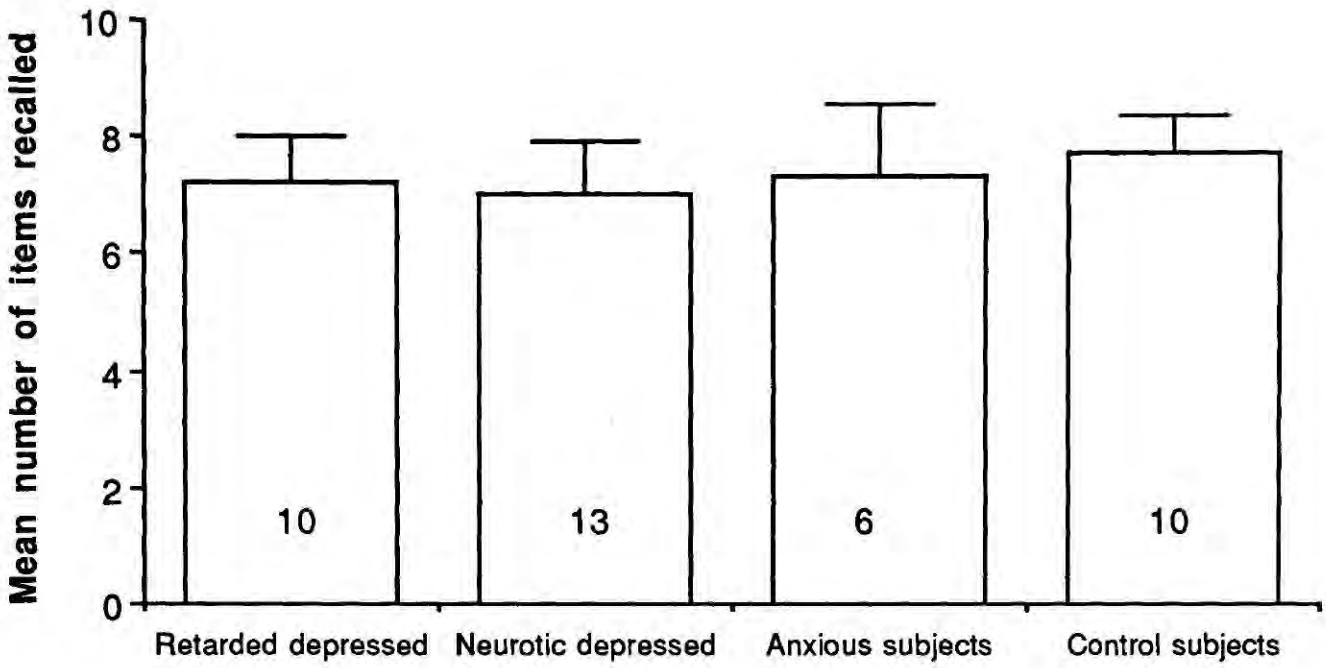


FIGURE 4.2

SCORES OF THE FOUR SUBJECT GROUPS ON d' TRIAL ONE (MEAN±SD)

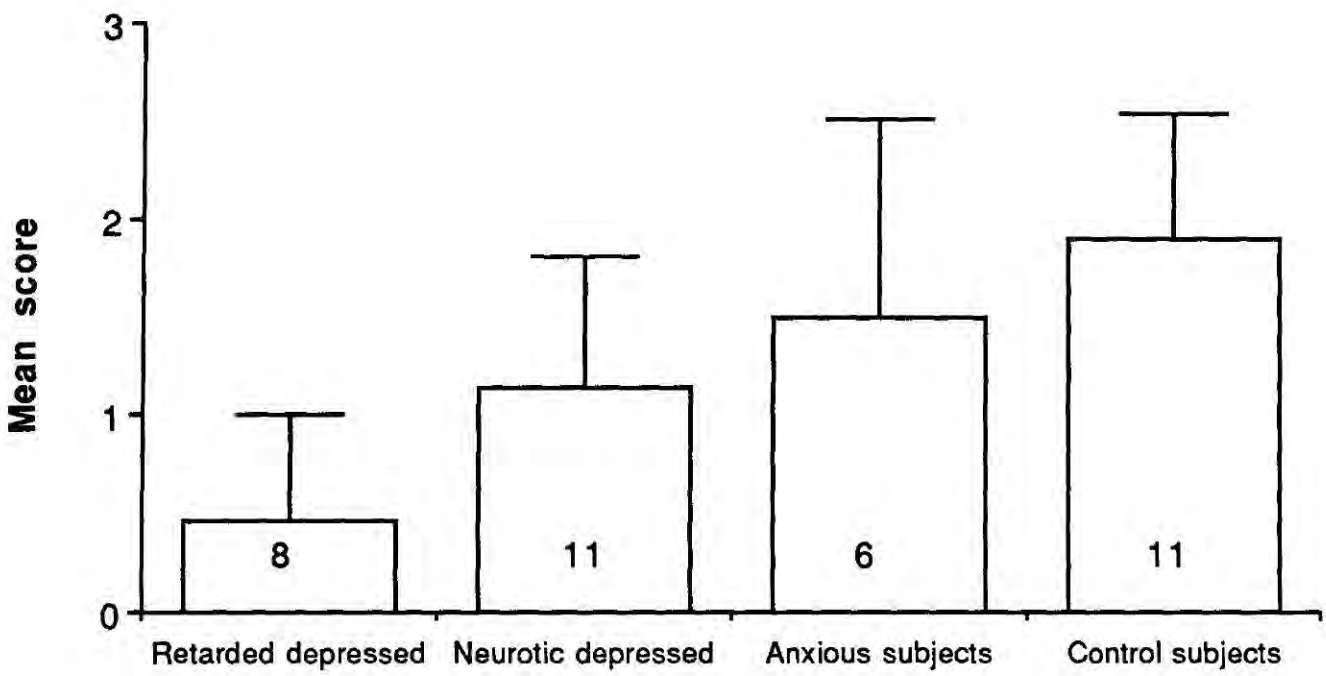


FIGURE 4.3

SCORES OF THE FOUR SUBJECT GROUPS ON FREE RECALL-TOTAL (MEAN±SD)

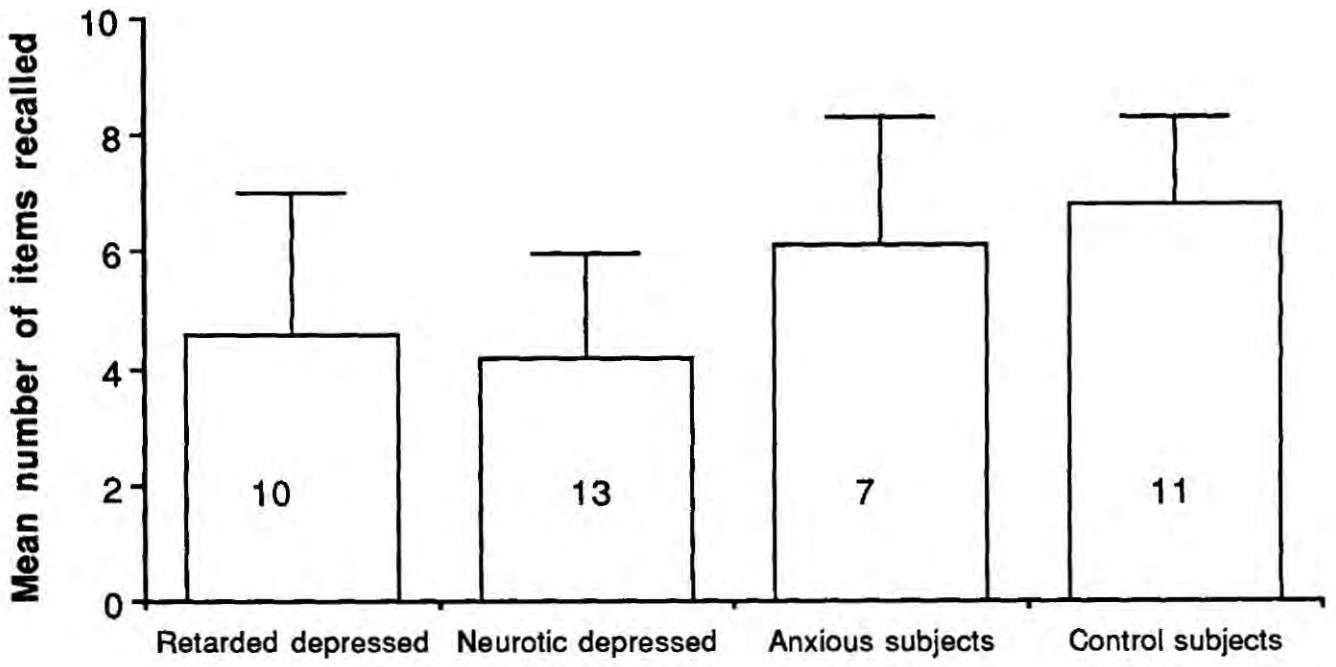


FIGURE 4.4

SCORES OF THE FOUR SUBJECT GROUPS ON SERIAL LEARNING-IMMEDIATE RECALL (MEAN±SD)

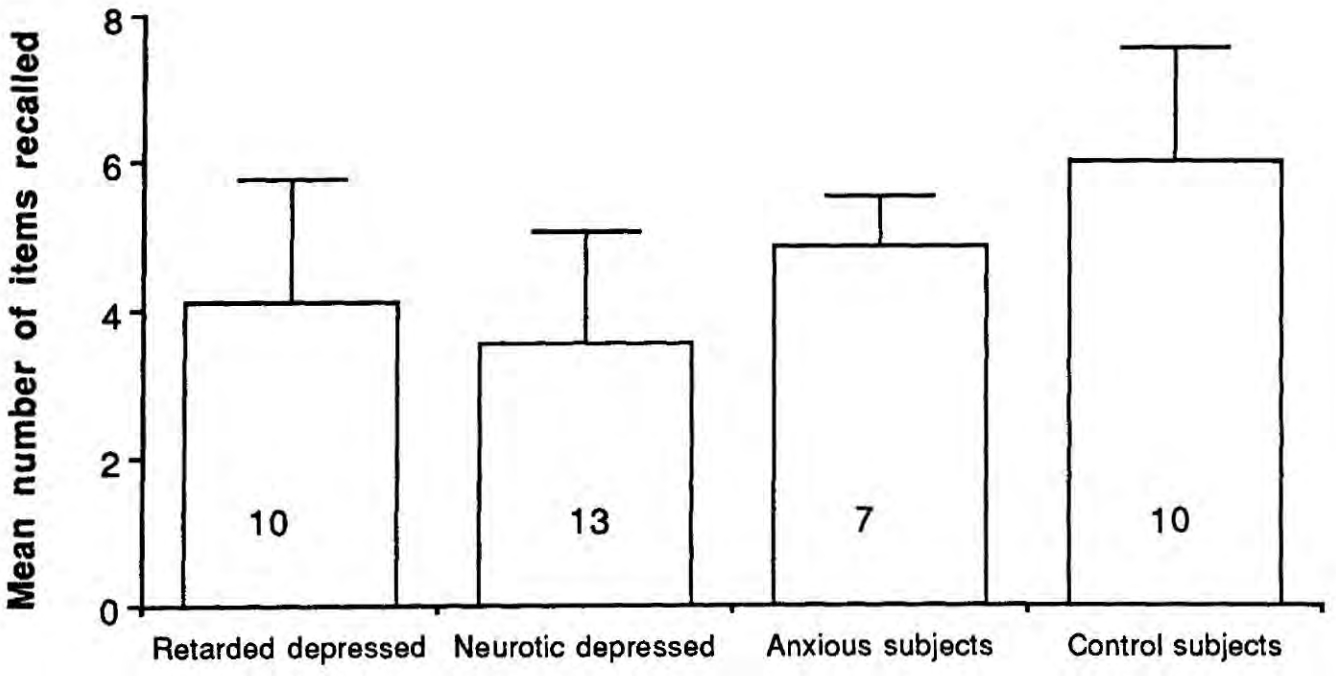


FIGURE 4.5

SCORES OF THE FOUR SUBJECT GROUPS ON PROSE PASSAGE ONE-IMMEDIATE RECALL (MEAN±SD)

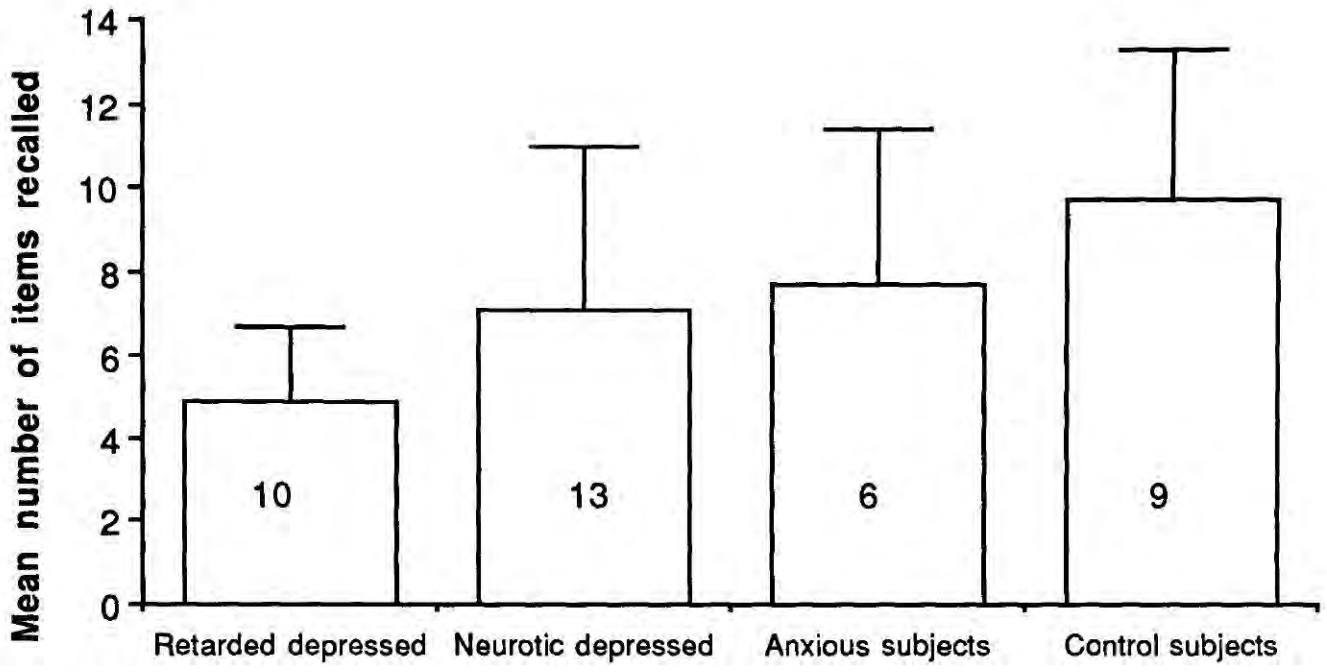


FIGURE 4.6

SCORES OF THE FOUR SUBJECT GROUPS ON PAIRED ASSOCIATE-IMMEDIATE RECALL (MEAN±SD)

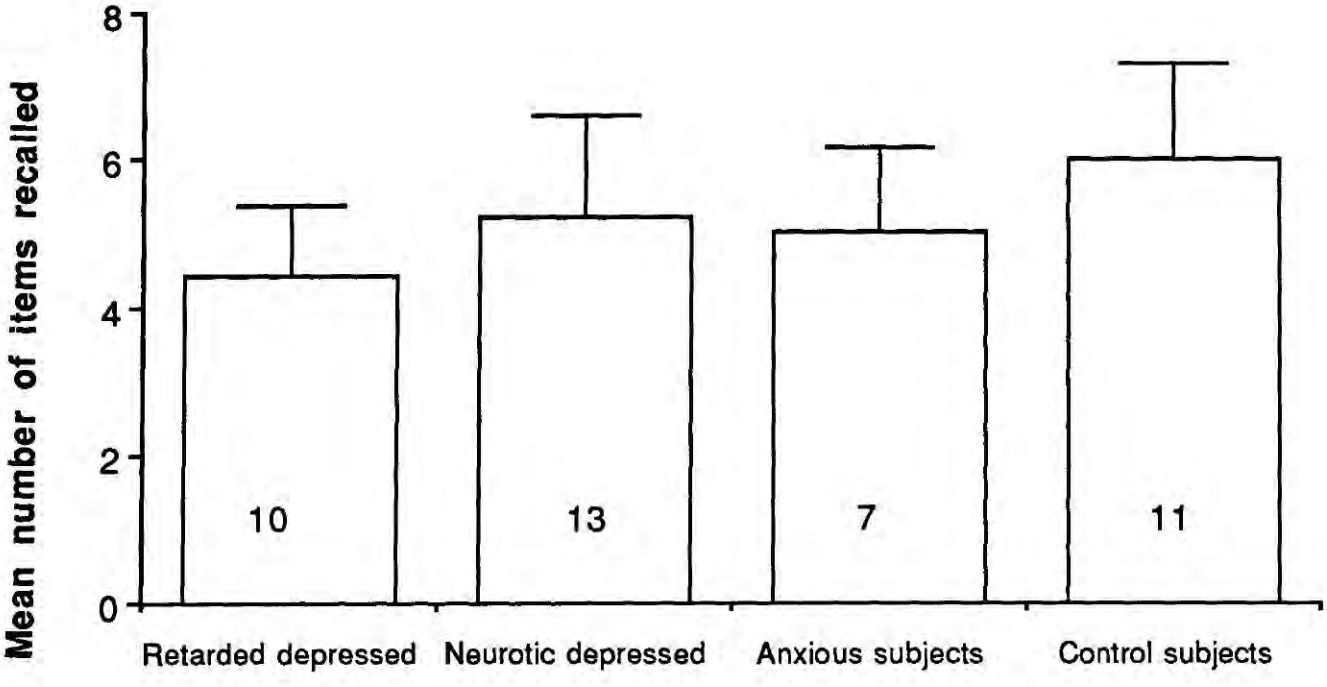


FIGURE 4.7

SCORES OF THE FOUR SUBJECT GROUPS ON DIGIT SPAN BACKWARDS (MEAN±SD)

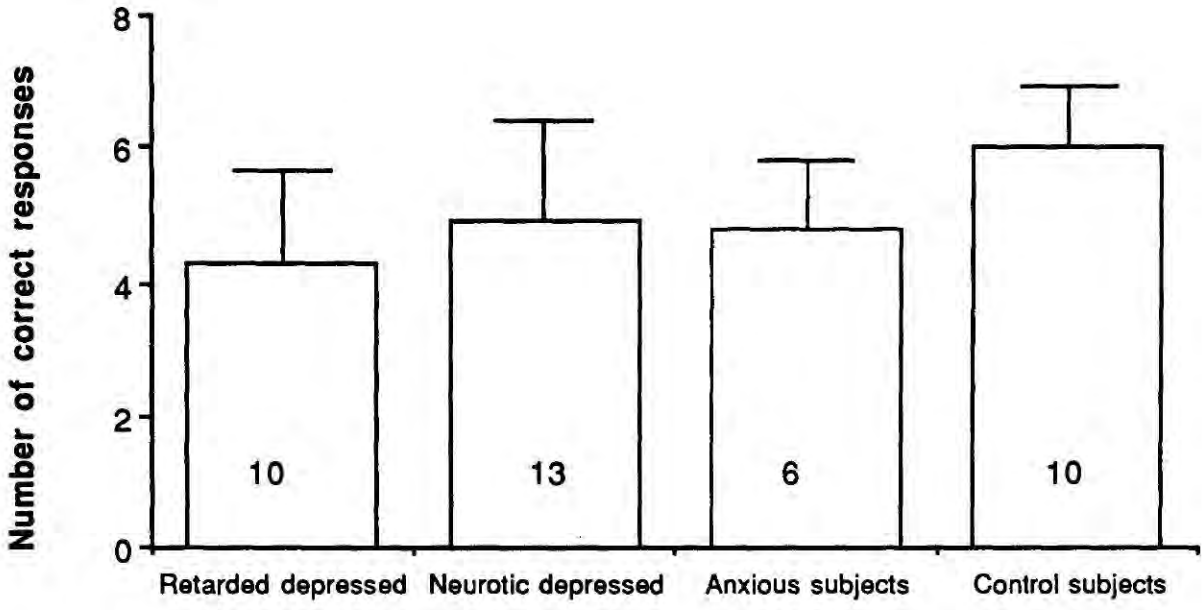
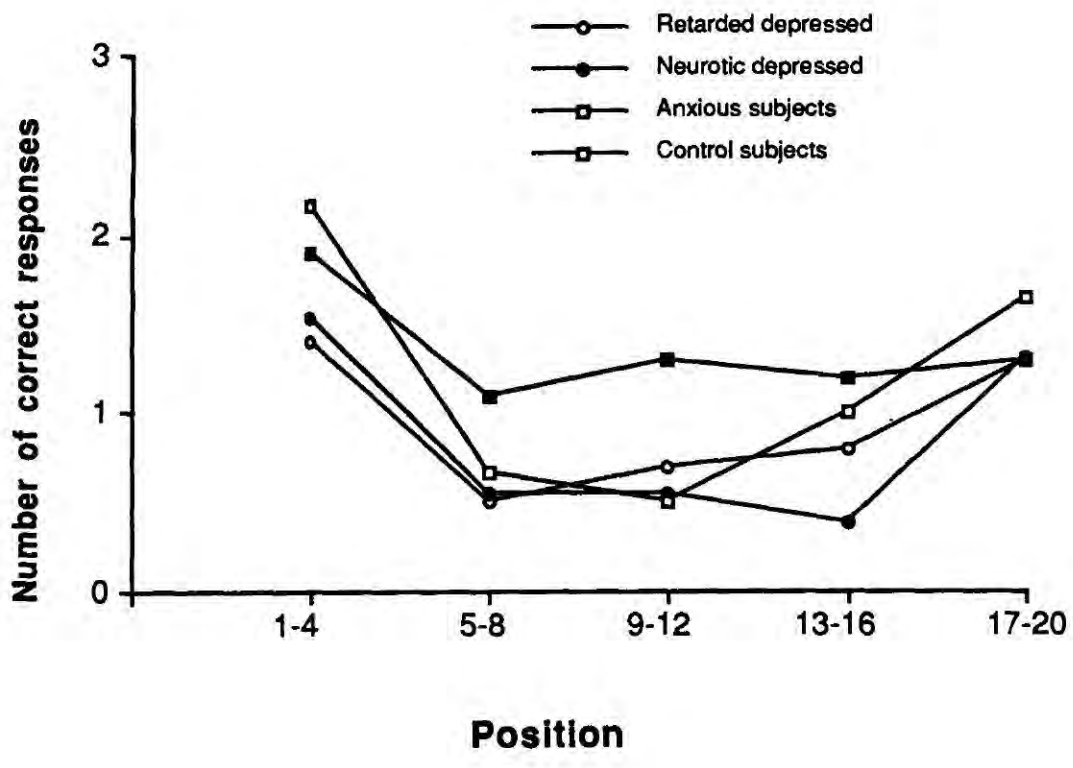


FIGURE 4.8

SERIAL POSITION CURVE FOR FREE RECALL-TOTAL DATA



In addition to looking at total scores on the Free Recall test the serial position curves of the four groups were examined (Figure 4.8) to see whether they showed the expected pattern of enhanced recall of the first few and last few items. This would supposedly be due to the first few items having been rehearsed most and entering the long-term component of memory (the primacy effect), and the last few items being recalled from the short-term memory store (the recency effect; Section 2.2.1). It can be seen in Figure 4.8 that the only group to deviate from the expected pattern was the control subjects who showed enhanced recall of the first four items but not of the last four items. In contrast, both depressed groups and the anxious subjects show the recency and primacy effects.

Table 4.5 shows the summary table of the two-way analysis of variance with repeated measures on one factor (the serial position of the recalled words) carried out on the serial position data. There was a significant effect of group and of serial position, but no interaction between the two. Thus there is no difference between the groups in the pattern of results, although the depressed groups appear impaired overall. This indicates that there is no specific impairment in depression restricted either to the long-term or short-term component of memory, but a more general deficit.

In order to compare three modes of recall (recognition, cued and free recall) the standard scores on d' (derived from the Picture Recognition test), Free Recall - total, and Paired Associate - Trial One were calculated. Table 4.6 shows the summary table of the two-way analysis of variance with repeated measure on one factor (mode of recall) using these data. There was an overall effect of group but no interaction between group and mode of recall, showing that the relationship between the groups did not differ over the three tests. The depressed patients were therefore not relatively more impaired on the free recall test than on the others.

Table 4.5 TWO-WAY ANALYSIS OF VARIANCE OF THE NUMBER OF ITEMS CORRECTLY RECALLED ON THE FREE RECALL TEST AS A FUNCTION OF GROUP MEMBERSHIP AND SERIAL POSITION.

Source	SS	df	MS	F	p
Between subjects					
Group membership	7.3	3	2.4	3.3	<0.05
Residual	25.8	35	0.7		
Within subjects					
Position	28.4	4	7.1	8.1	<0.001
Position x Group	6.3	12	0.5	0.6	NS
Residual	122.4	140	0.8		

Table 4.6 TWO-WAY ANALYSIS OF VARIANCE OF THE STANDARDISED SCORES ON MEMORY TESTS AS A FUNCTION OF GROUP MEMBERSHIP AND MODE OF RECALL.

Source	SS	df	MS	F	p
Between subjects					
Group membership	21.4	3	7.1	6.6	<0.01
Residual	34.6	32	1.1		
Within subjects					
Mode of recall	0.1	2	0.07	0.12	NS
Mode x Group	4.0	6	0.67	1.13	NS
Residual	38.2	64	0.59		

4.3.2 Speed of Learning

Serial Learning test

The Serial Learning and the Paired Associate tests were each presented and recalled four times (the Paired Associate test was also recalled a fifth time without the pairs of words being presented first). If, as indicated by the results presented in the previous section, the depressed subjects had difficulty learning new information they might be expected to recall less information than the controls on each of the four trials. Analysis of variance was therefore used to investigate whether scores on these tests varied over the four trials, and whether the performance of the four subject groups differed on these tests. In addition the interaction between group membership and trial was also examined: the depressed subjects may be able to overcome their initial difficulty in learning the material and perform as well as the control subjects on later trials, or alternatively they may find it difficult to concentrate on the task for the time required and consequently do less well in comparison with the control subjects on the last trials than on the first trial.

The scores of the four subject groups on the four trials of the Serial Learning test are given in Figure 4.9. Table 4.7 gives the summary table of the two-way analysis of variance with repeated measures on one factor (test trials). Scores differed significantly between the four groups and over the four trials; in addition there was an interaction between these two factors: the relationship between the scores of the different groups was not the same over all trials.

As indicated above (Section 4.3.1), the anxious subjects were not significantly different from either the depressed subjects or the control subjects on the first trial of this test. On the second trial the scores of the anxious and control subjects substantially increased whilst those of the two depressed groups increased only modestly. There was very little difference between the scores of the control and anxious subjects, or between those of the retarded depressed and neurotic depressed subjects; however there was a large difference between the scores of the control and anxious subjects on one hand, and the retarded and neurotic

control and anxious subjects on one hand, and the retarded and neurotic depressed subjects on the other. This pattern of results continued over the remaining two trials, except that the gap between the retarded and neurotic depressed subjects widened, with the latter group scoring more than the former.

The mean scores over the second to fourth trials were calculated (Serial learning - speed of learning); a one-way analysis of variance followed by Gabriel's multiple comparison test was then used to see whether the differences between the groups were statistically significant (Figure 4.10). As expected there was a significant overall difference between the groups ($F(3,36)=9.8$, $p<0.001$). The scores of the control subjects differed significantly from those of both the retarded and neurotic depressed groups at the five per cent level, showing that the depressed patients learnt new material more slowly than the controls and did not overcome the deficit shown on the first trial. There were no significant differences between the scores of the two depressed groups. The scores of the anxious subjects differed significantly from those of the depressed patients and were not significantly different from those of the controls.

Table 4.7 **TWO-WAY ANALYSIS OF VARIANCE OF THE NUMBER OF ITEMS CORRECTLY RECALLED ON THE SERIAL LEARNING TEST AS A FUNCTION OF GROUP MEMBERSHIP AND TRIAL.**

Source	SS	df	MS	F	p
Between subjects					
Group membership	410.9	3	136.9	8.7	<0.01
Residual	536.6	34	15.8		
Within subjects					
Trial	622.3	3	207.2	130.7	<0.01
Trial x Group	66.9	9	7.4	4.7	<0.01
Residual	161.8	102	1.6		

FIGURE 4.9

MEAN SCORES OF THE FOUR SUBJECT GROUPS ON THE FOUR TRIALS OF THE SERIAL LEARNING TEST

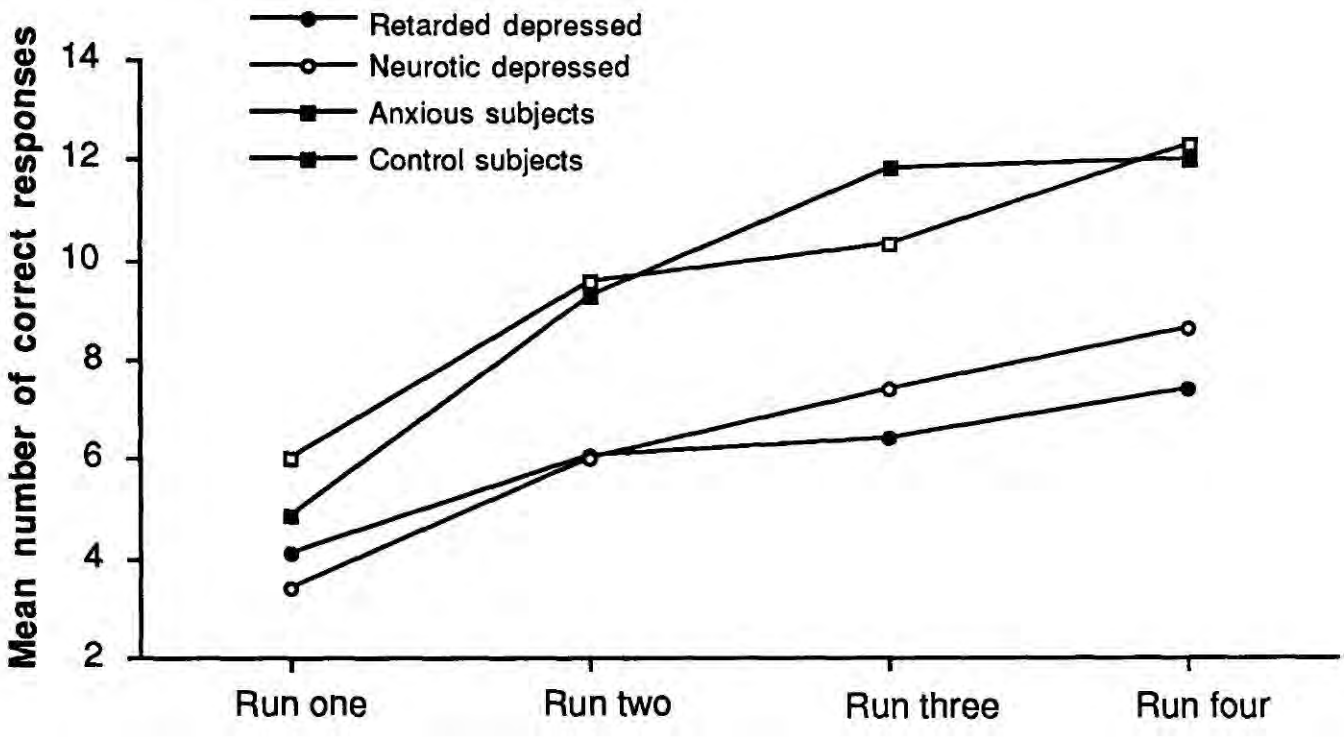
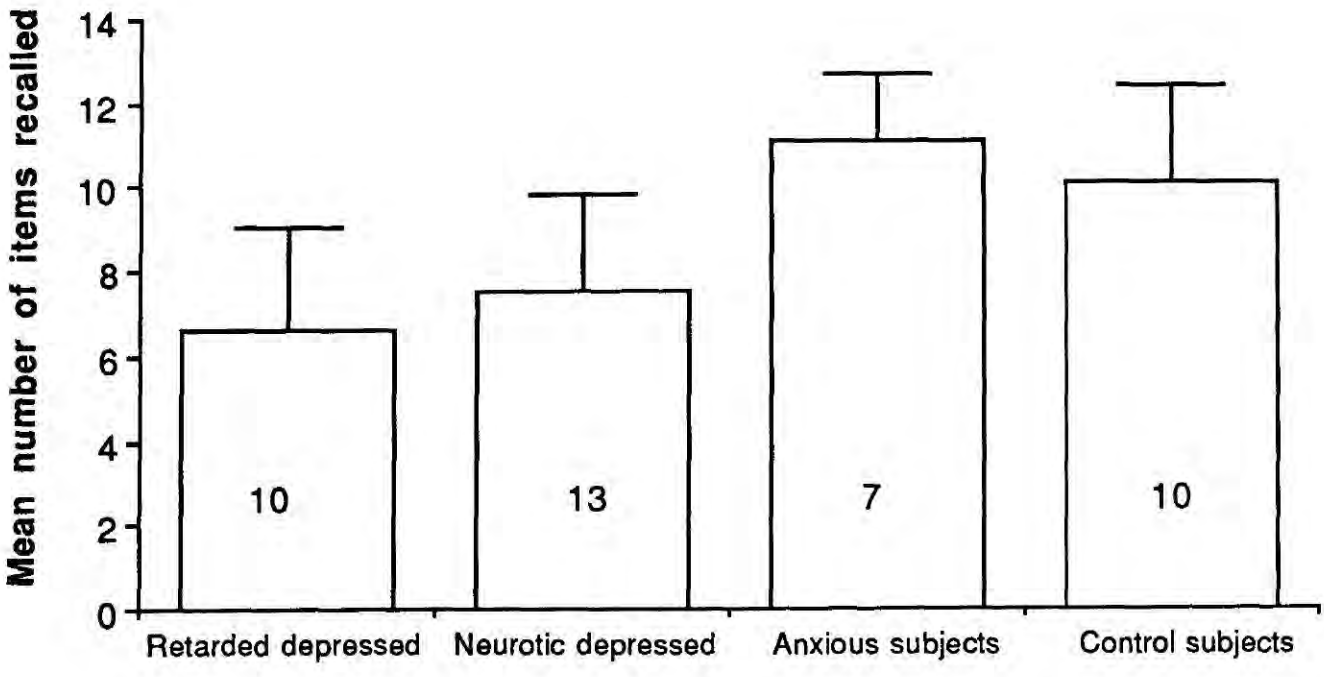


FIGURE 4.10

SCORES OF THE FOUR GROUPS ON SERIAL LEARNING-SPEED OF LEARNING (MEAN±SD)



Paired Associate test

Table 4.8 gives the summary table for the two-way analysis of variance using the data from the first four trials of the Paired Associate test (the fifth trial is excluded because it measured recall after a delay). The data are illustrated in Figure 4.11. There was a highly significant effect of group: the scores overall differed significantly between the four groups. There was also a significant effect of trial: scores differed between the four trials. In this case, however, there was no interaction between the two. This may be because there was a ceiling effect on this test: by the fourth trial three out of seven anxious subjects, five out of thirteen neurotic depressed and eight out of eleven controls were getting the maximum score of ten. These subjects may have increased their scores substantially if it had been possible to do so.

The mean scores over trials two to four were calculated (Paired Associate - speed of learning; Figure 4.12) and, as on the Serial Learning test, the scores of the four groups differed significantly ($F(3,37)=9.8$, $p<0.01$). Multiple comparisons showed that the control subjects differed significantly from both the retarded and neurotic depressed subjects at the five per cent level. In contrast to the Serial Learning test the neurotic depressed and retarded depressed groups differed significantly at the five per cent level, with the neurotic depressed subjects achieving higher scores. The anxious subjects differed significantly from the retarded depressed subjects on this test, but not from the less impaired neurotic depressed subjects. They did not differ from the control subjects.

The results from the two tests of the speed of learning showed that the depressed patients were impaired on both tests. There was a suggestion on the Serial Learning test that patients in the neurotic depressed group were able to learn the information more quickly than the retarded depressed group: this was clearly the case on the Paired Associate test. The anxious subjects were not impaired on these tests: they did not differ significantly from the controls but did differ from the retarded depressed group on both tests and from the neurotic depressed on the Serial Learning test.

FIGURE 4.11

MEAN SCORES OF THE FOUR SUBJECT GROUPS ON THE FIRST FOUR TRIALS OF THE PAIRED ASSOCIATE TEST

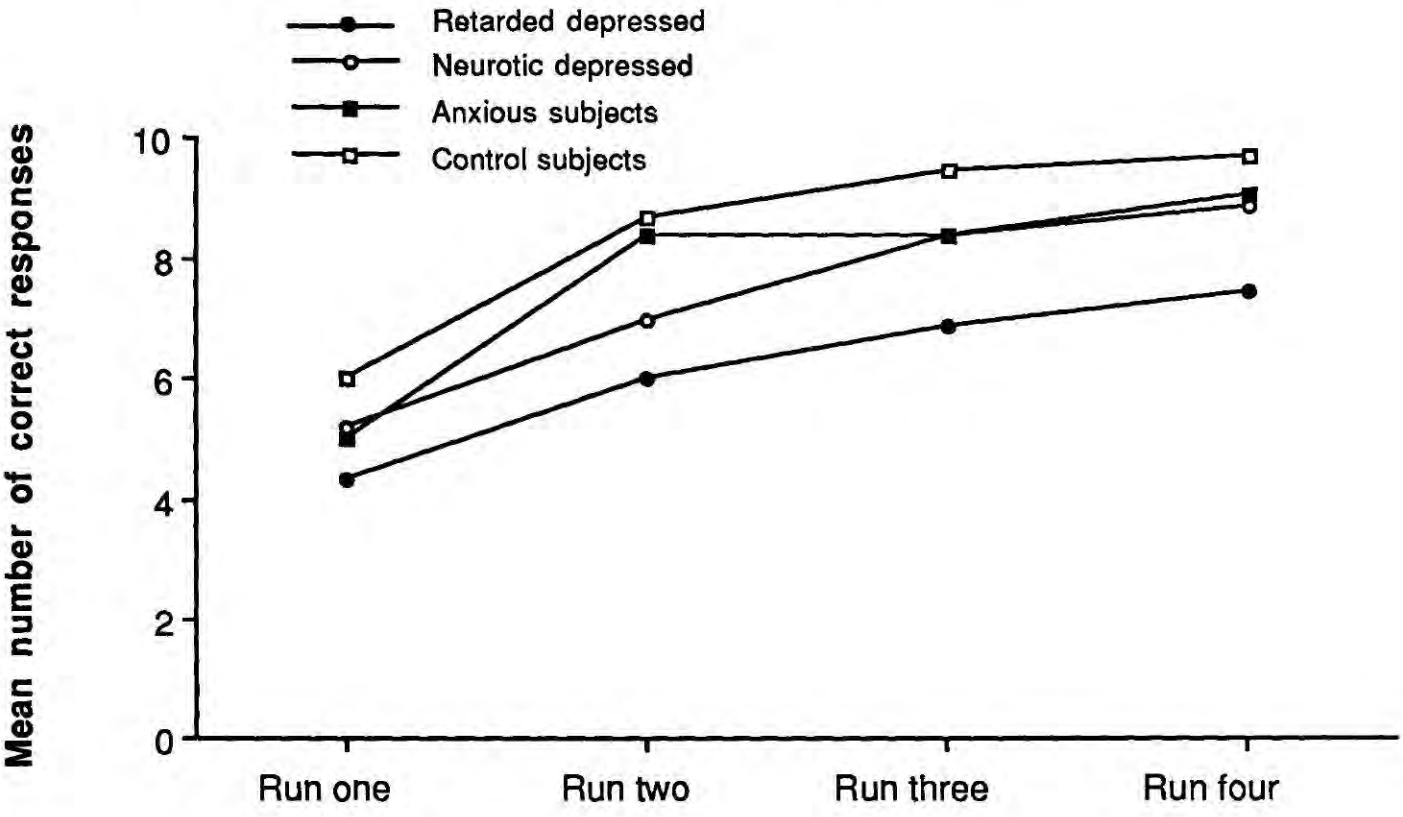


FIGURE 4.12

SCORES OF THE FOUR GROUPS ON PAIRED ASSOCIATE-SPEED OF LEARNING (MEAN±SD)

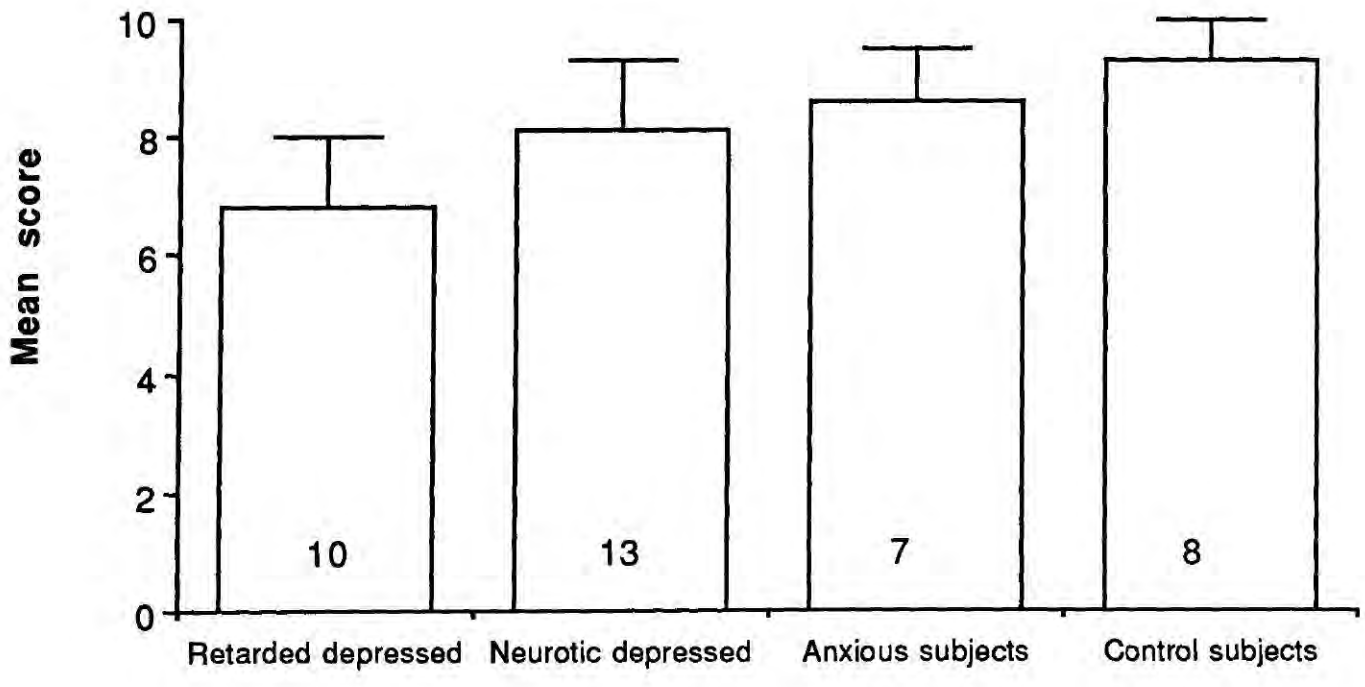


Table 4.8 TWO-WAY ANALYSIS OF VARIANCE OF THE NUMBER OF ITEMS CORRECTLY RECALLED ON THE PAIRED ASSOCIATE TEST AS A FUNCTION OF GROUP MEMBERSHIP AND TRIAL

Source	SS	df	MS	F	p
Between subjects					
Group membership	69.7	3	23.2	4.5	<0.01
Residual	189.3	37	5.1		
Within subjects					
Trial	263.9	3	87.9	56.2	<0.01
Trial x Group	20.6	9	2.3	1.5	NS
Residual	173.8	111	1.6		

4.3.3 Retention

Several of the tests in the test battery contained a trial in which subjects were asked to recall information which had been presented to them earlier in the testing session. This was to investigate whether the four subject groups differed in their ability to retain information in memory or, to put it another way, whether they differed in the amount of information they forgot over time.

Prose Passage tests

Prose Passage One was recalled immediately after it was read to the subject and again later on in the testing session. Prose Passage Two was just recalled after a delay, because it was felt that the initial recall of Passage One might rehearse the passage and result in the subjects recalling more information after the delay than they would otherwise. The scores on Prose Passage One - delayed recall are shown in Figure 4.13 and those on Passage Two - delayed recall in Figure 4.14. Figure 4.15 gives the scores on the variable 'Prose Passage One - forgetting' which was calculated by subtracting the number of items recalled after the delay from the number originally recalled: this therefore consists of the

number of items originally recalled which were forgotten before the delayed recall.

The Bartlett Box test for the equality of variance in the four groups showed that the variances on Prose Passage One - forgetting differed significantly between the groups ($F=2.7$, $p<0.05$) and therefore the Kruskal-Wallis test was used. There were no significant differences between the groups on this test ($N=37$, $\chi^{2}=2.45$, $p>0.05$): the depressed patients therefore did not differ significantly from the control or anxious subjects in their ability to retain information in memory.

There was a statistically significant difference between the scores of the four groups on both Prose Passage One - delayed recall and Prose Passage Two - delayed recall (Passage One - delayed recall, $F(3,34)=3.93$, $p<0.05$; Passage Two - delayed recall, $F(3,35)=3.15$, $p<0.05$). As the groups did not differ in the amount of information forgotten, the differences on these variables presumably result from differences in the amount originally learned.

Multiple comparisons, using Gabriel's test, were then made between the scores of each group on Prose Passage One - delayed recall and Prose Passage Two - delayed recall. The retarded depressed subjects differed significantly from the control subjects at the five per cent level on the former variable, and at the one per cent level on the latter, whilst the neurotic depressed subjects did not differ significantly from the controls on either variable. The two depressed groups did not differ significantly from each other and the anxious subjects did not differ significantly from either of the two groups of depressed subjects, or from the control subjects.

FIGURE 4.13

SCORES OF THE FOUR GROUPS ON PROSE PASSAGE ONE-DELAYED RECALL

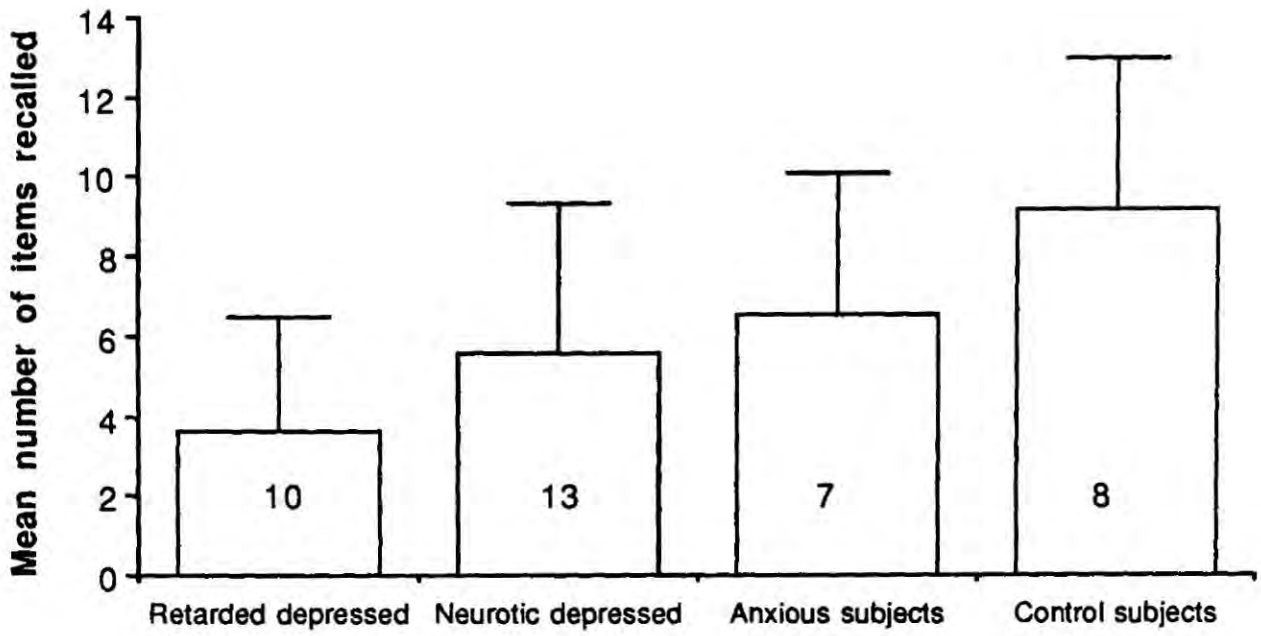


FIGURE 4.14

SCORES OF THE FOUR GROUPS ON PROSE PASSAGE TWO-DELAYED RECALL (MEAN±SD)

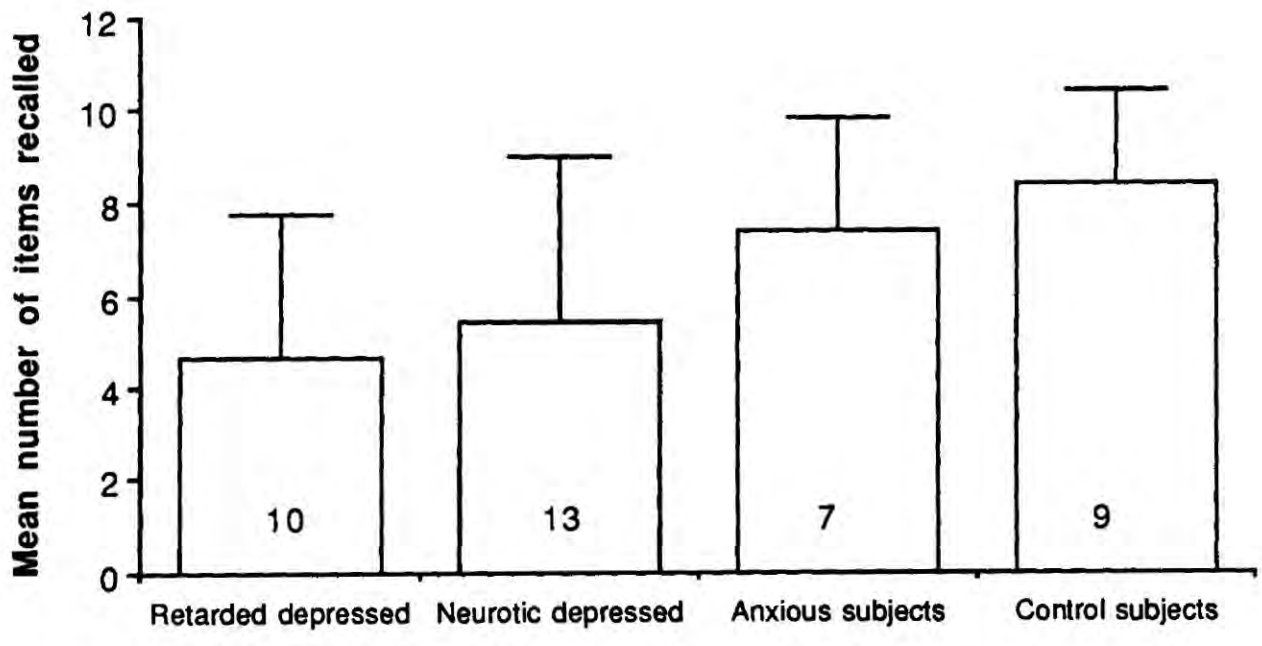


FIGURE 4.15

SCORES OF THE FOUR GROUPS ON PROSE PASSAGE ONE-FORGETTING (MEDIAN + INTERQUARTILE RANGE)

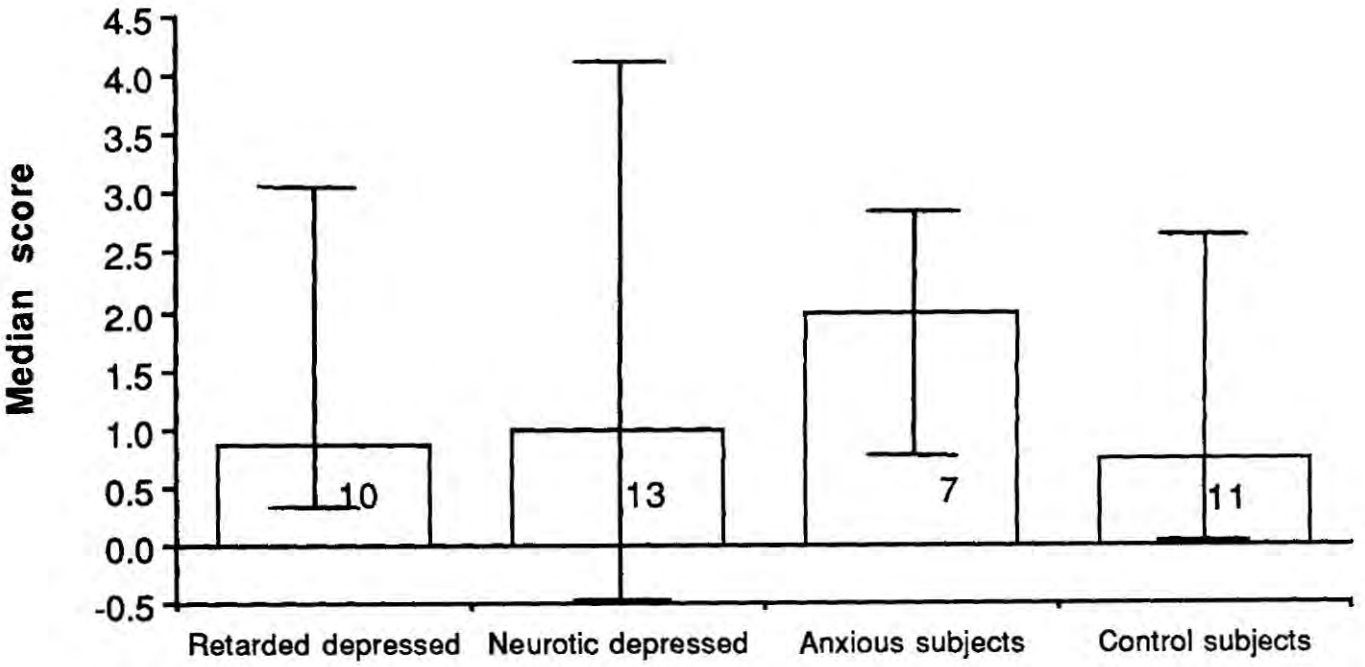


FIGURE 4.16

SCORES OF THE FOUR GROUPS ON PAIRED ASSOCIATE-FORGETTING (MEDIAN + INTERQUARTILE RANGE)

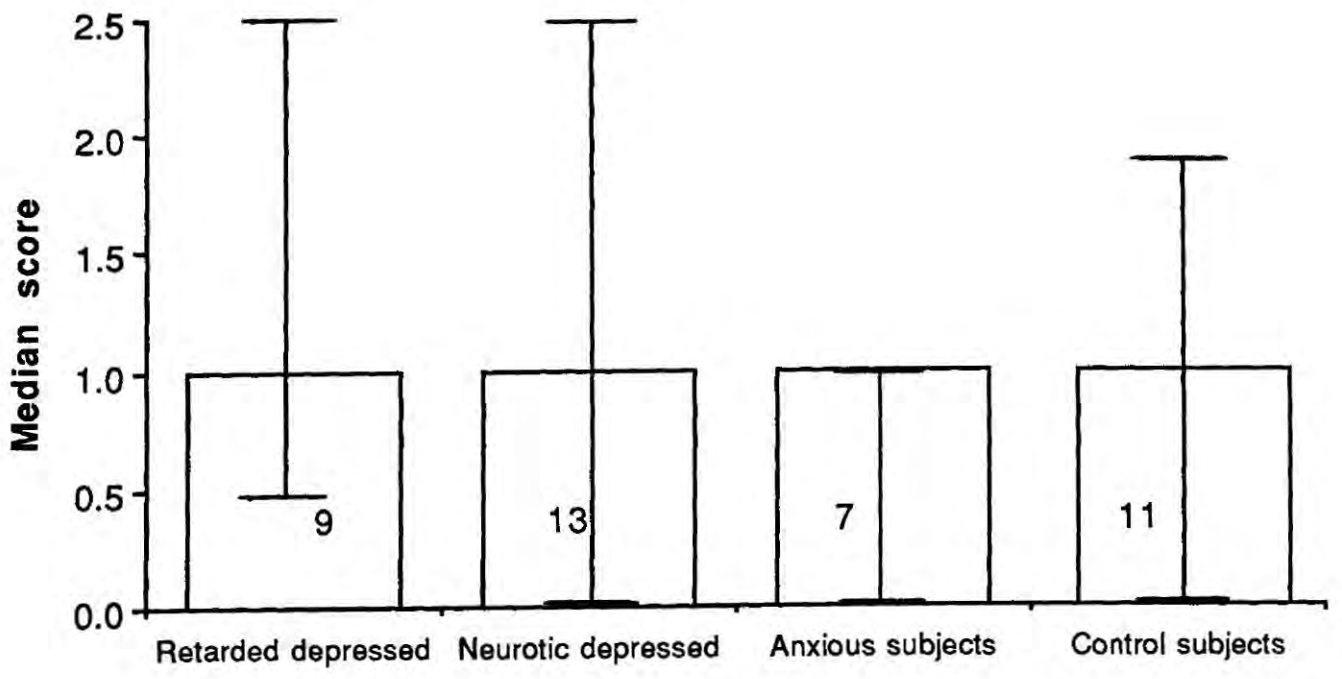
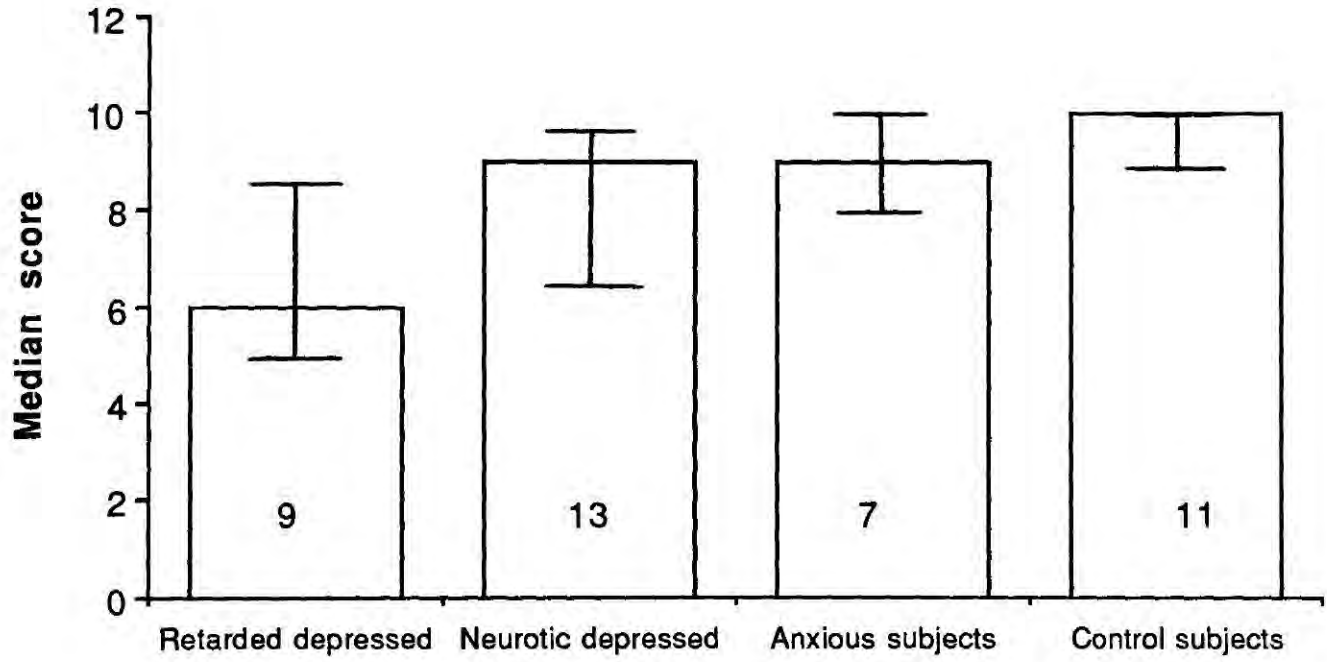


FIGURE 4.17

SCORES OF THE FOUR GROUPS ON PAIRED ASSOCIATE-TRIAL FIVE (MEDIAN)



Paired Associate test

As indicated above, the Paired Associate test was recalled five times, the final time after a delay and without the pairs being presented immediately beforehand. The variable 'Paired Associate - forgetting' was calculated by subtracting scores on Trial Five from those on Trial Four. It is illustrated in Figure 4.16 and the scores on Paired Associate - Trial Five are shown in Figure 4.17.

Since the variances differed significantly between the groups on both variables (Paired Associate - Trial Five, $F=2.89$, $p<0.05$; Paired Associate - forgetting, $F=5.59$, $p<0.05$) the Kruskal-Wallis test was used. There was a significant difference between the groups on Paired Associate - Trial Five ($N=40$, $\chi^2=14.3$, $p<0.001$), but not on Paired Associate - forgetting ($N=40$, $\chi^2=5.08$, $p>0.05$). Again, there is no evidence that the depressed patients differed from the anxious or control groups in the amount of information they forget over time.

Multiple comparisons were used to identify significant differences between groups on Paired Associate - Trial Five. The Mann-Whitney U test was used with amended significance levels to take account of the fact that several comparisons are being made (Section 4.2.3). The retarded depressed and neurotic depressed subjects both differed significantly from the control subjects, the former at the five per cent level and the latter at the ten percent. The two depressed groups did not differ significantly. The anxious subjects differed significantly from the Retarded depressed group ($p<0.1$), but not from the neurotic depressed patients or the control subjects.

Picture Recognition test (d')

Finally, the forgetting of material was investigated on the Picture Recognition test: the scores on this test were used to calculate d', a measure of the sensitivity of memory (Section 2.2.1). Like the Paired Associate test it consisted of several trials but in contrast to that test the material was presented only once. Consequently Figure 4.18, which shows d' scores over all four trials shows the rate at which memory for the pictures declined.

It can be seen that the scores of the control subjects declined sharply between the first and second trials, and between the third and fourth; their final score was lower than that of the other groups even though it was higher to begin with. In contrast the retarded depressed subjects showed a slight decline over the first three trials and then increased their score on the final trial. The scores of the neurotic depressed and anxious subjects both declined between the first and second trials. The neurotic depressed subjects' scores then declined further between the second and third trials, while the anxious subjects increased their scores slightly; both groups showed little change between the last two trials.

Figure 4.19 shows the mean regression coefficents for the relationship between d' scores on Trials One to Four. The coefficents for the anxious and retarded depressed subjects were small but positive, suggesting that their scores increased over the four trials. In contrast, those for the controls and neurotic depressed were both negative; the scores declined over time. The differences between the groups were statistically significant ($F(3,33)=6.9$, $p<0.001$). Gabriel's multiple comparisons showed that the control subjects differed from the retarded depressed subjects at the one per cent level but did not differ significantly from the neurotic depressed subjects. The neurotic depressed subjects differed from the retarded depressed subjects at the one per cent level. The anxious subjects differed from the neurotic depressed subjects at the five per cent level and the controls at the one per cent level. There is, therefore, no evidence that the depressed or anxious subjects forgot more information than the controls over the four trials of this test. Instead, the control subjects showed the biggest decline in scores, whilst the anxious and retarded subjects actually showed a small increase.

FIGURE 4.18

MEAN d' SCORES OF THE FOUR SUBJECT GROUPS ON THE FOUR TRIALS OF THE PICTURE RECOGNITION TEST

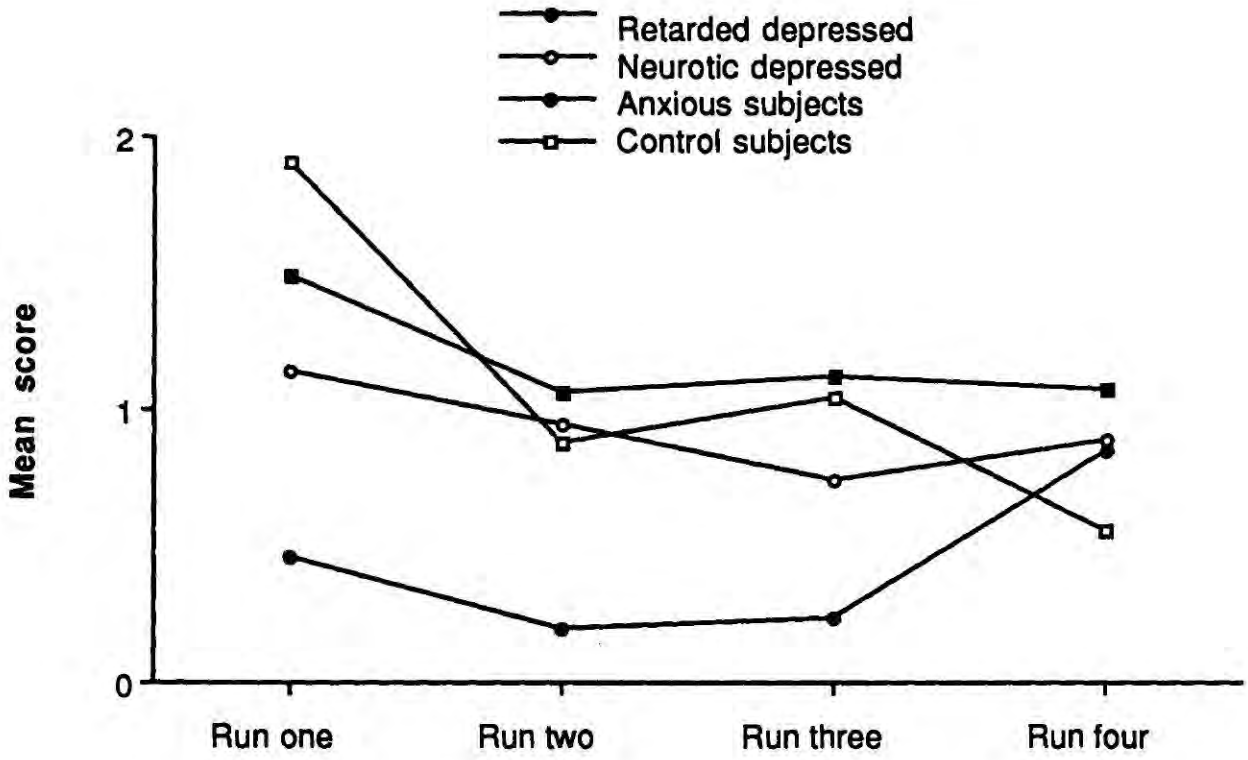
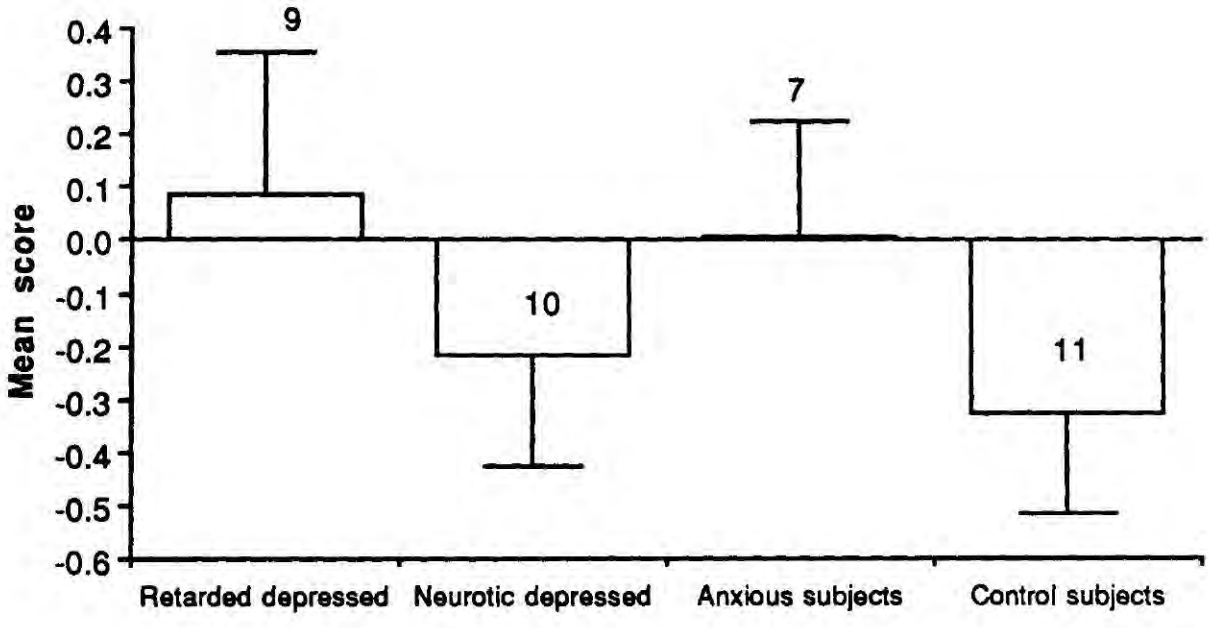


FIGURE 4.19

CHANGES IN d' SCORES OVER THE FOUR TRIALS OF THE PICTURE RECOGNITION TEST:REGRESSION COEFFICIENTS (MEAN±SD)



To summarise, there were no significant differences between the groups on two of the variables measuring the amount of information forgotten between inital and delayed recall: Prose passage One - forgetting amd Paired Associate - forgetting. There is, therefore, no evidence that the depressed or anxious patients differed from the controls in their ability to retain information in memory. The significant differences between the groups on Prose Passage One - delayed, Prose Passage Two - delayed and Paired Associate - Trial Five were therefore presumably due to the impairments in immediate learning reported above (Section 4.3.1) and showed the same pattern of results as the immediate learning variables. The significant differences between the groups in the rate of forgetting on the Picture Recognition test (d') were in the opposite direction to that hypothesised: the control subjects showed the greatest decline in memory whilst the scores of the retarded depressed and anxious subjects actually increased.

These results do not take into account the fact there were significant differences between the groups in the amount recalled on the first trial of the Prose Passage recall, Paired Associate and Picture Recognition tests (Section 4.3.1). The fact that the depressed subjects recalled less information initially may have obscured a real difference between the groups in their ability to retain information: the depressed subjects may not have forgotten more information than the other subjects because there was no more information to forget, not because they forgot the information at the same rate as the control subjects. This possibility needs to be borne in mind when interpreting these results, but seems unlikely as it is clear that most depressed patients could have forgotten more information than they did: only two out of thirteen neurotic depressed and two out of ten retarded depressed patients scored zero on Prose Passage One - delayed and none of the depressed patients did so on Paired Associate - Trial Five.

4.3.4 Memory for Past Public Events.

Figures 4.20 and 4.21 show the scores of the four groups on the two questionnaires measuring very long-term memory: Past Events free recall questionnaire and Past Events multi-choice questionnaire. There were significant differences between the groups on the Past Event multi-choice questionnaire ($F(3,35)=3.26$, $p<0.05$), whilst the differences on the Past Event free recall questionnaire approached significance ($F(3,37)=2.85$, $p=0.05$). Individual comparisons were then made. The retarded and neurotic depressed subjects both differed significantly from the controls on the multi-choice questionnaire ($p<0.05$) and there was a trend for them to do so on the free recall test ($p<0.1$, >0.05). The two groups of depressed patients did not differ significantly on either test, while the anxious subjects did not differ significantly from the depressed or the control subjects.

Depressed subjects may have had impaired memory for past public events because they were depressed at the time the event happened and therefore failed to encode it, rather than because they had difficulty retrieving the information due to current depression. This possibility was examined by correlating the length of the present episode of depression (Section 3.4.2) with scores on the past public events questionnaires. The correlations were not statistically significant (Past Events free recall questionnaire, $r=-27$, $N=23$, $p>0.05$; Past Events multi-choice questionnaire, $r=-.21$, $N=23$, $p>0.05$). The results do not, therefore support the hypothesis that the deficits on these questionnaires were due to encoding difficulties at the time the event happened rather than to current retrieval difficulties. However, the possibility cannot be entirely rejected because many subjects had had previous episodes of depression which may have occured in the fifteeen years covered by the questionnaires and therefore might have affected initial encoding: information on past episodes was collected from the patients rather than hospital notes and was therefore insufficiently precise to permit further analysis.

The subjects were given a maximum of fifteen minutes to complete each questionnaire. As depressed people frequently move and react more slowly than normal (Section 1.8.3) they may have scored less than the controls because they could not complete as much of the questionnaire in the time allowed. There were no statistically significant differences between the groups in the time taken to complete the free recall questionnaire ($F(3,37)=1.02$, $p>0.05$; Figure 4.22). However there were significant differences in the time taken to complete the multi-choice

FIGURE 4.20

SCORES OF THE FOUR GROUPS ON THE PAST EVENTS FREE RECALL QUESTIONNAIRE (MEAN±SD)

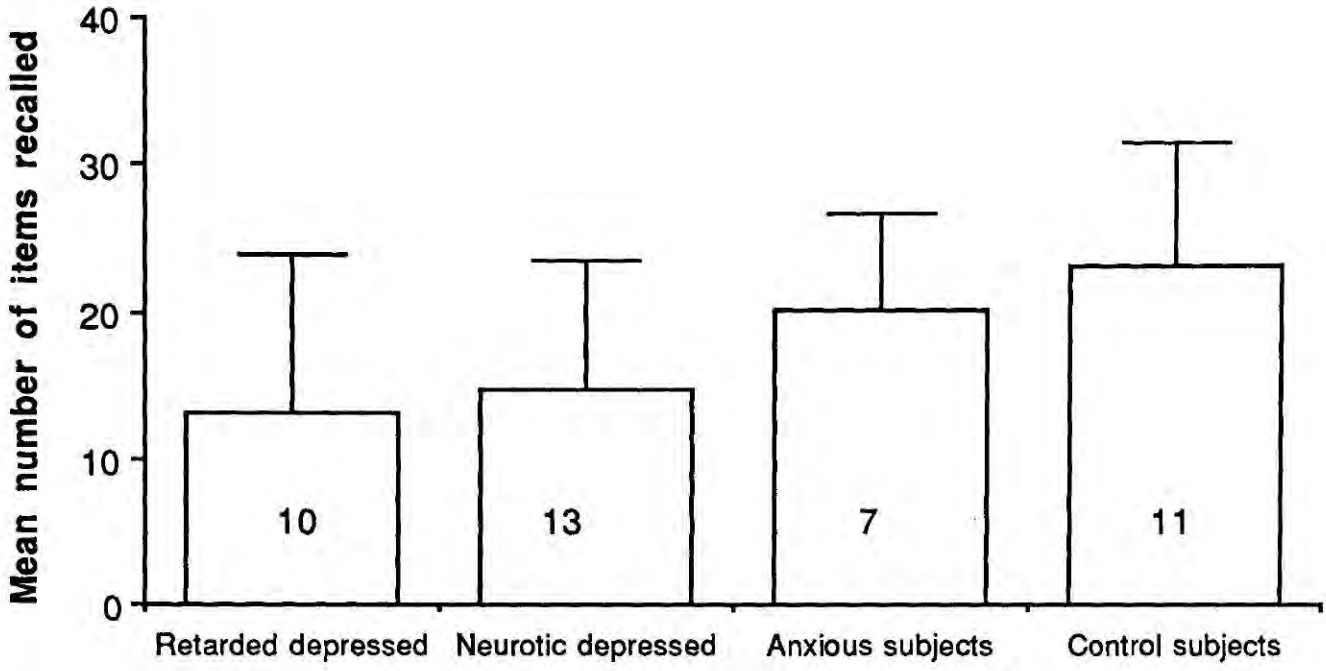


FIGURE 4.21

SCORES OF THE FOUR GROUPS ON THE PAST EVENTS MULTI-CHOICE QUESTIONNAIRE (MEAN±SD)

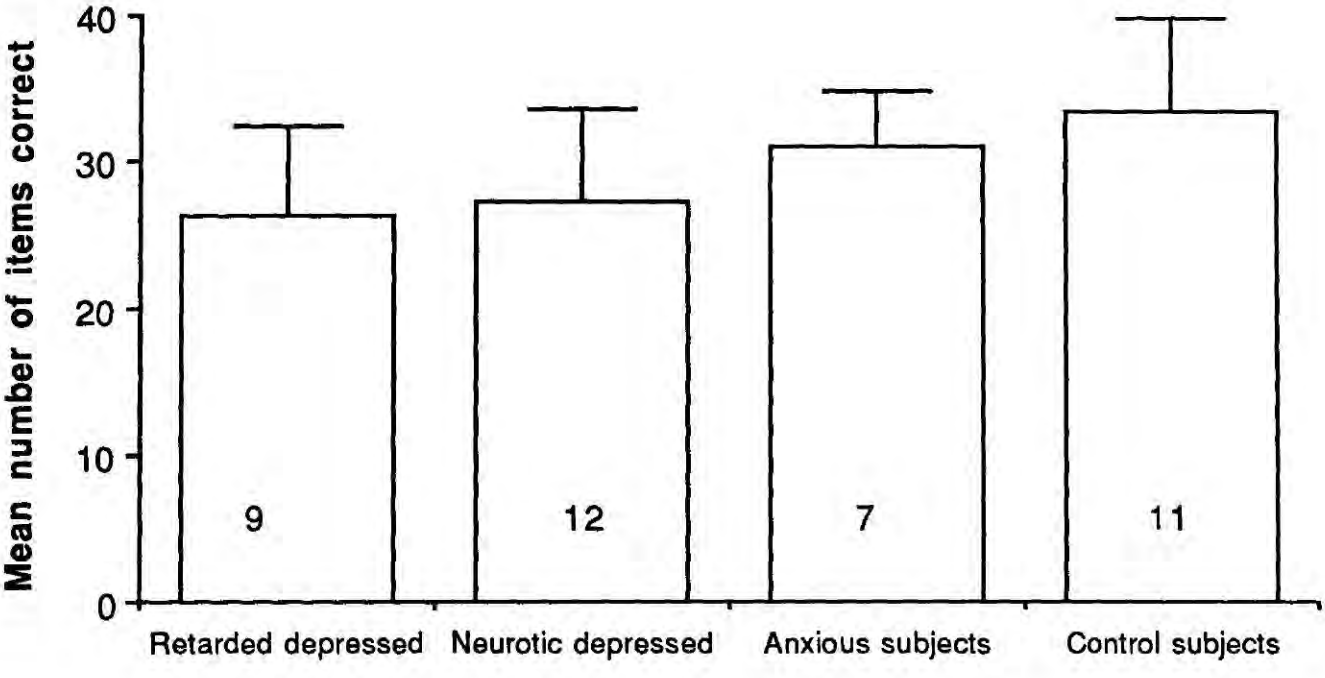


FIGURE 4.22

TIME TAKEN TO COMPLETE THE PAST EVENTS FREE RECALL QUESTIONNAIRE (MEAN±SD)

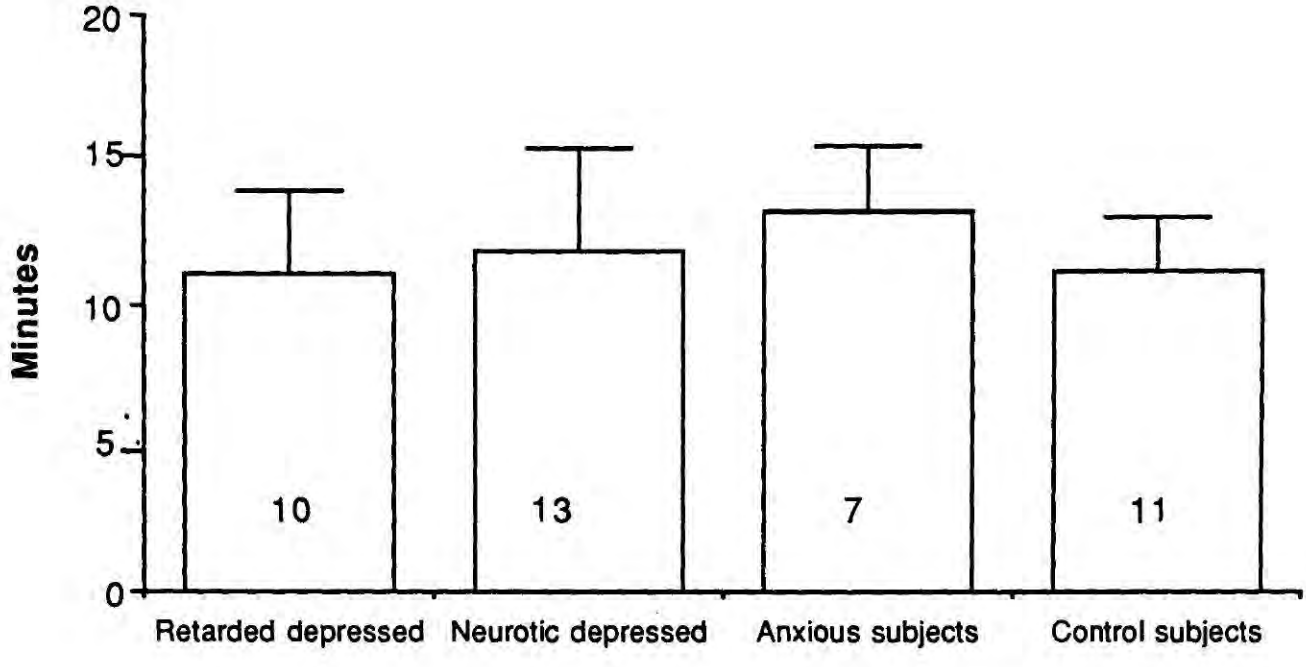
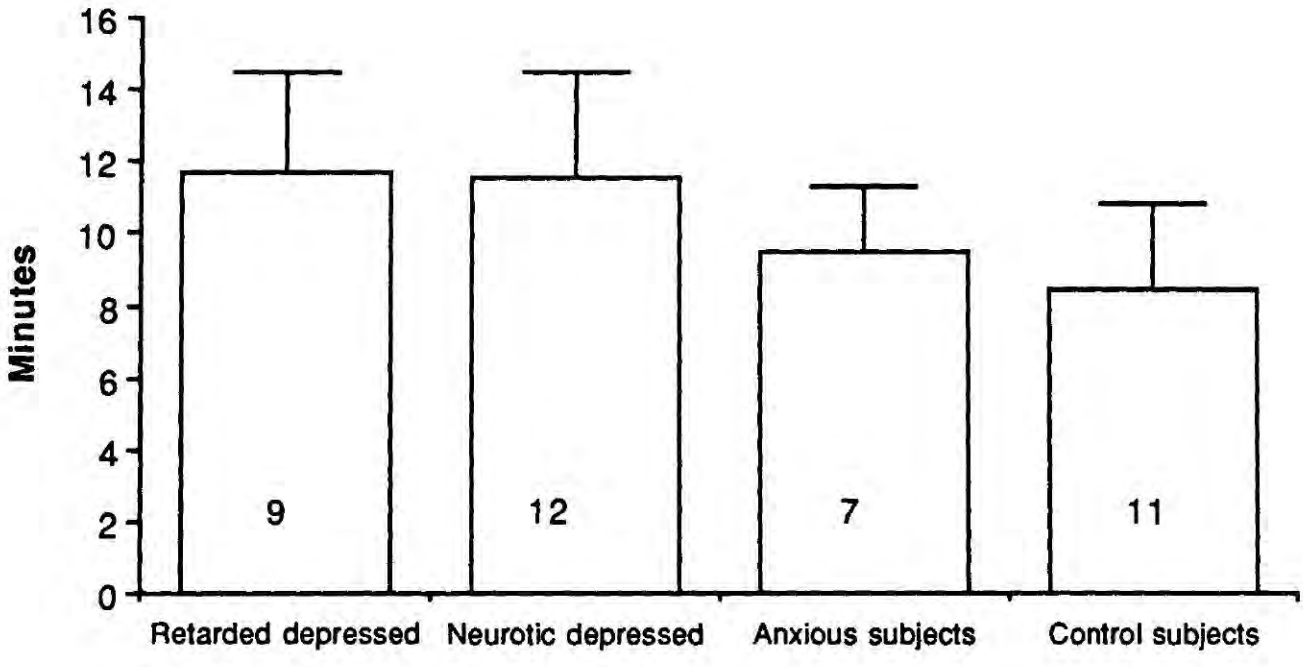


FIGURE 4.23

TIME TAKEN TO COMPLETE THE PAST EVENTS MULTI-CHOICE QUESTIONNAIRE (MEAN±SD)



questionnaire ($F(3,35)=3.87$, $p<0.05$). Multiple comparisons showed that the retarded depressed and neurotic depressed subjects took significantly longer than the control subjects ($p<0.05$; Figure 4.23).

Regression analyses were then used to see if there was a significant difference in recall between the groups on the questionnaires when the time taken to complete them was taken into consideration.

First, the results for the multi-choice questionnaire are considered. When time taken to complete the questionnaire was entered into the equation it explained 12% of the variance in recall which was significant ($F(1,37)=4.9$, $p<0.05$): the longer the subjects took, the less they recalled Subject group (Group), coded as three dummy variables, was then added. This caused an increase in R^2 of .12 which was not significant (F change=1.74; NS). The order in which the variables entered the equation was then reversed. When Group was entered first it explained 22% of the variance; this was significant ($F(3,35)=3.3$, $p<0.05$). Time taken to complete the questionnaire was then added to the equation causing an increase in R^2 of only .01: this was not significant (F change=.7, NS). Although neither variable caused a significant increase in the amount of variance explained when added to the other variable, the amount explained by Group when added to time was clearly much larger than that explained by time when added to Group. It can therefore be concluded that the significant differences observed between the groups were not due to differences in the time taken to complete the questionnaire.

The amount of variance in performance on the free recall questionnaire explained by time taken to complete the questionnaire was significant ($R^2=.1$; $F(1,39)=4.2$, $p<0.05$). When Group was added to this it caused a significant increase in R^2 (R^2 change=.19, F change=3.1; $p<0.05$). When the order in which the variables were entered into the equation was reversed, Group again explained a significant amount of the variance ($r^2=.19$; $F(3,37)=2.8$, $p<0.05$). Time taken to complete the questionnaire caused a significant increase in R^2 of .1 (F change=4.9; $p<0.05$): the longer the subjects took the more they recalled. It is clear that differences in the time taken to complete this questionnaire did not account for the marginal differences in recall between the groups.

FIGURE 4.24

NUMBER OF QUESTIONS INCORRECTLY ANSWERED ON THE PAST EVENTS FREE RECALL QUESTIONNAIRE (MEAN±SD)

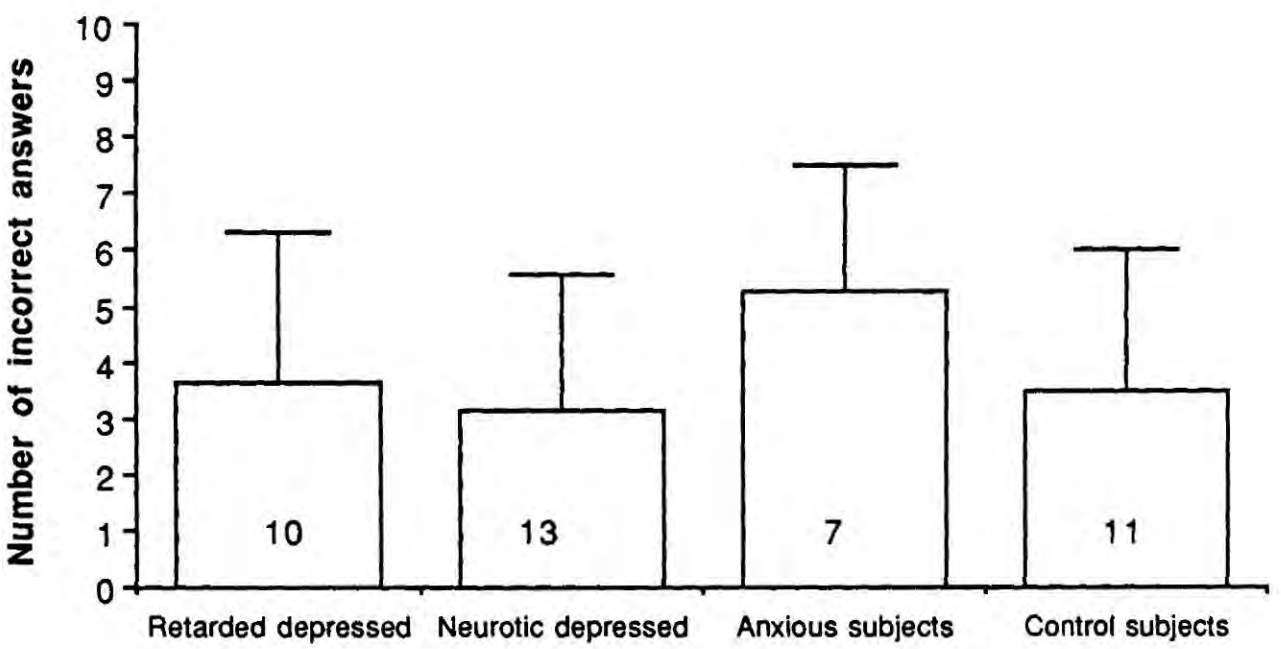
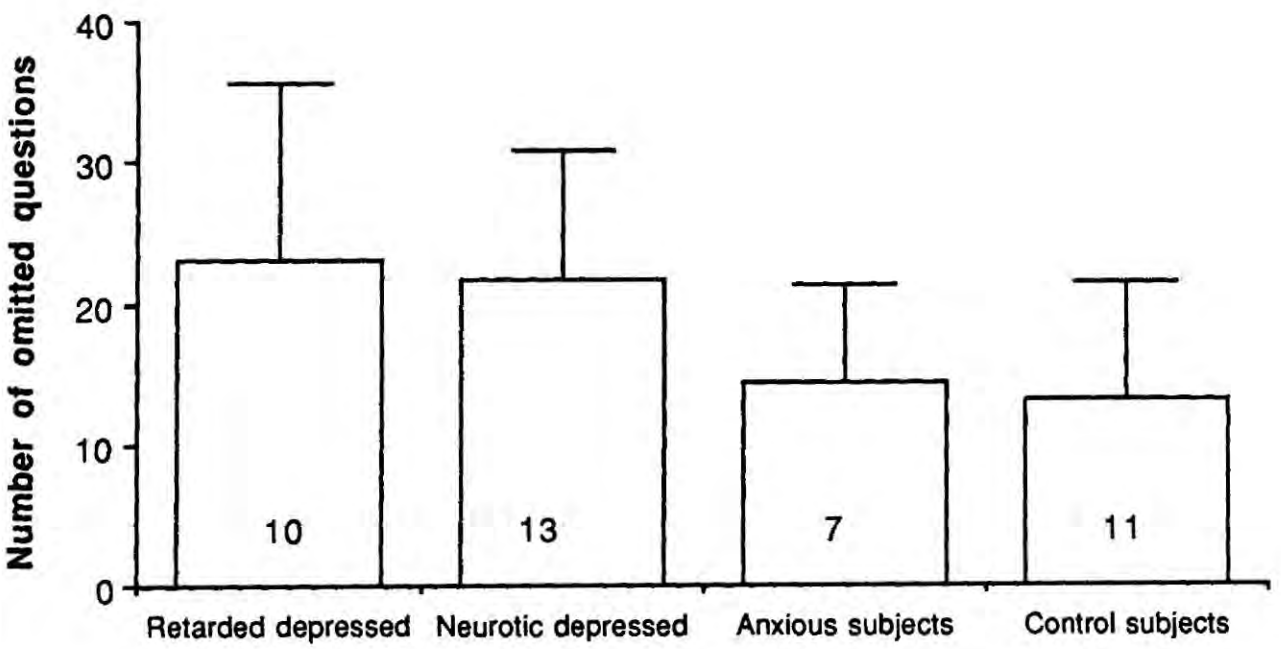


FIGURE 4.25

NUMBER OF QUESTIONS OMITTED ON THE PAST EVENTS FREE RECALL QUESTIONNAIRE (MEAN±SD)



Subjects were instructed to complete all the questions on the multi-choice questionnaire; all except three subjects did so. Low scores on this questionnaire therefore show that the subjects gave wrong answers. However subjects could score low on the free recall questionnaire either by getting questions wrong or by not attempting some questions at all. Figure 4.24 shows the number of questions each group answered wrong, and Figure 4.25 the number each group omitted. There were no significant differences between the groups on the former variable ($F(3,37)=1.19$, $p>0.05$). However the differences in the number of questions omitted by each group approached significance ($F(3,37) =2.76$, $p=0.05$). Individual comparisons showed there was a trend for the retarded and neurotic depressed subjects to omit more answers than the controls ($P<0.1$, >0.05).

In summary there is a trend for the depressed subjects, both retarded and neurotic, to achieve significantly lower scores than the controls on the free recall version of the questionnaire. This was not due to the depressed subjects taking longer to complete the questionnaire and therefore being unable to complete it in the time allowed. There were no significant differences between the groups in the number of questions the subjects answered wrong, but there was a trend for depressed subjects to omit significantly more answers than the controls. The two depressed groups did not differ significantly, and the anxious subjects did not differ significantly from either the depressed or the control subjects on any of these variables.

There were statistically significant differences between the groups on the multi-choice questionnaire with both groups of depressed subjects scoring significantly lower than the controls. Again this could not be accounted for by depressed subjects being slower to complete the questionnaire. Once more there were no statistically significant differences between the two groups of depressed patients, or between the anxious subjects and either the depressed or the control subjects.

Thus the two groups of depressed subjects performed less well than controls on two questionnaires measuring memory for past public events. The retarded and neurotic depressed patients did not differ significantly. The anxious subjects did not differ significantly from the controls, nor did they differ significantly from the depressed subjects.

4.3.5 Response Bias (β)

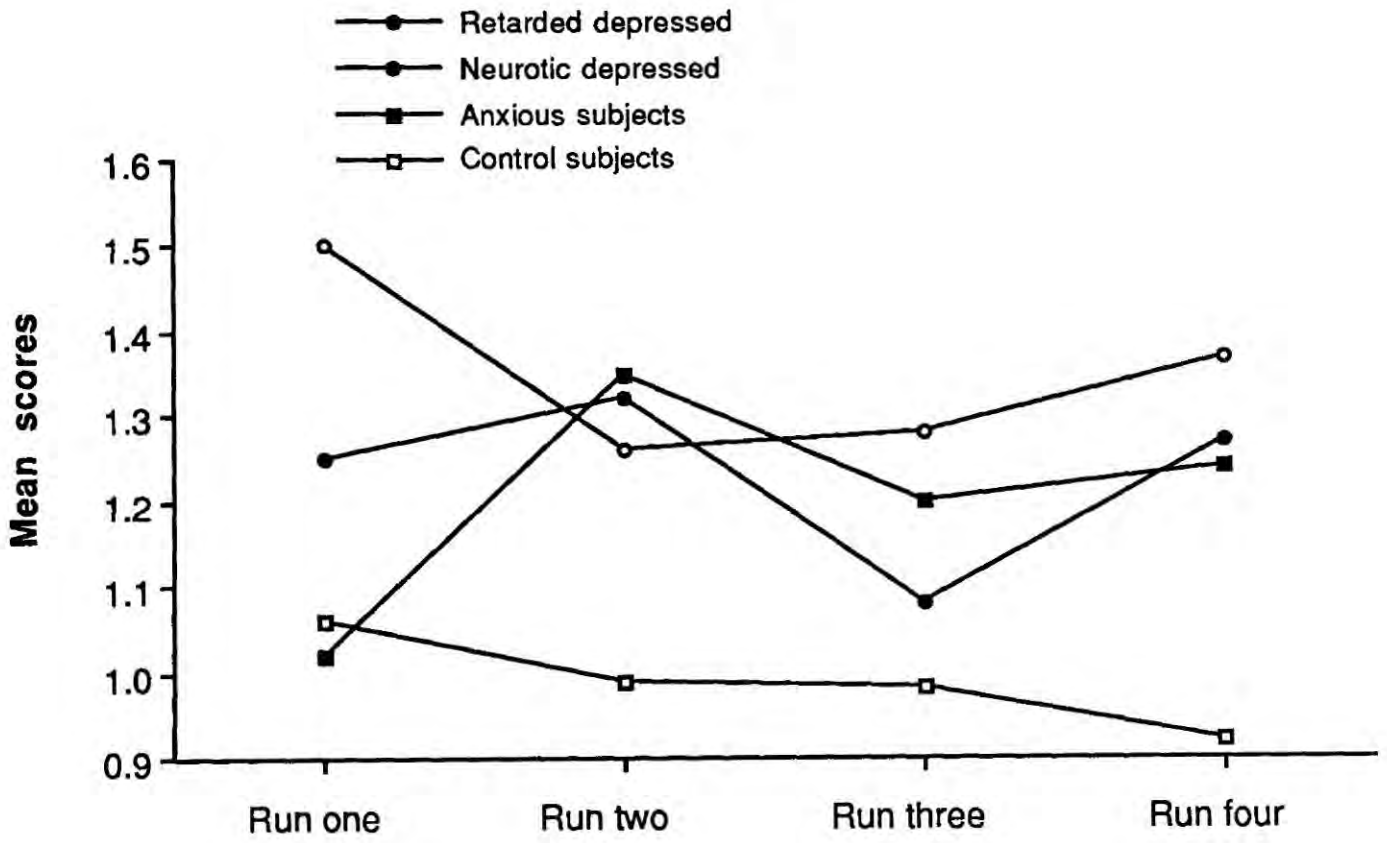
A measure of the subjects' response bias, β, was calculated from the scores on the forced-choice Picture Recognition test (Section 2.2.1). If β is less than one the subject is biased towards saying that s/he has seen the slide before; this will result in false positive errors. If β is more than one the subject is biased towards saying that they have not seen the slide before; this will result in more misses and fewer false positive responses.

The scores of the four groups over the four trials of the test are illustrated in Figure 4.26. β cannot be calculated when the probability of a hit or a false positive is zero, as explained above (Section 4.2.3). This means a two-way analysis of variance on this data is inappropriate because a considerable number of cases would be excluded due to missing data. In this case it was also not appropriate to fit regression lines as it was not hypothesised that β scores would decline in a linear manner over the four trials. Instead the results of the one-way analyses on β - Trial One; β - Trial Four; and the difference between the two (β - change) are presented.

There were no significant differences between the groups on β - Trial One ($F(3,30)=1.37$, $p>0.05$); on β - Trial Four ($F3,35)=2.09$, $p>0.05$); or on β - change ($F3,28)=.59$, $p>0.05$). There is, therefore, no evidence that the four groups differed in the criterion they set for responding to a slide: no group was more biased than the others towards saying that they had (or had not) seen the slide before. On Trial One all four groups had β values of above one; by Trial Four β for the control group was slightly below one whilst β for the other groups remained above it. These results suggest that the subjects were mainly biased towards negative responses and had adopted cautious criterion. However the β values in most cases were close to one and therefore it must be concluded that there is no evidence of a substantial bias in these subjects.

FIGURE 4.26

MEAN β SCORES OF THE FOUR SUBJECT GROUPS ON THE FOUR TRIALS OF THE PICTURE RECOGNITION TEST (MEAN±SD)



4.4 DISCUSSION

4.4.1 The Performance of Depressed Patients Compared to that of the Control Subjects.

As noted in Section 4.1.1, despite evidence that some depressed individuals have impaired memories there is little information about which groups of depressed subjects will experience impairment and what form this will take. This chapter therefore investigated whether retarded and neurotic depressed psychiatric patients, classified according to the PSE-ID-CATEGO system (Section 3.2.2), had impaired memories and, if so, what form the deficits took. The results are discussed in this section.

Registration and Immediate Memory

The depressed subjects were not impaired on Digit Span Forwards, which was included in the battery as a measure of registration (see Section 2.2.1) and which Baddeley and Hitch (1974) argue involves the use of both the articulatory loop and the central executive components of working memory. These results suggest that the depressed patients do not differ from control subjects in their ability to attend to or concentrate on material, at least as long as the amount of information does not exceed eight items, the maximum in this test. The results for Digit Span Backwards, which demands the manipulation of figures rather than simply their recall in a specified order, indicate that depressed patients may be impaired when the demands on working memory are greater. This would be consistent with the view of Hasher and Zacks (1979) that processing capacity is reduced in depression (Section 1.8.1).

Both groups of depressed subjects were impaired on the tasks requiring the immediate free recall of unconnected words (Free Recall - total; Serial Learning - immediate recall). They were also both impaired on d' - Trial One, a measure of pure memory capacity derived from the Picture Recognition test. Watts et al (1987) also found significantly lower d' scores in depressed patients than in control subjects, this time using a verbal recognition memory test. They criticised a previous study which had not found this effect (Miller and Lewis, 1977) for using geometric material which Watts et al (1987) suggested might be less

sensitive to deficits in depression than verbal material. The results of the present study suggest that this may not be the case and that therefore this explanation for the negative results obtained in the study by Miller and Lewis is inadequate. However, it may be that the materials used in these studies were not of comparable levels of difficulty and that as impairment in depression may be related to the difficulty of the task (Section 1.8.1) this accounts for the discrepancies between them.

Support for the suggestion that the degree of impairment found in depression is related to task difficulty (Ellis, 1985) comes from the finding that the neurotic depressed patients were not impaired on Paired Associate - immediate recall or on Prose Passage One - immediate recall. The paired associate task involved cued recall and consisted largely of over-learned pairs such as Metal - Iron, while the prose passages had a definite structure with inbuilt redundancy (Section 2.2.1). There are, therefore, reasons for believing that both tasks would be easier than the tests involving the recall of unconnected words which were impaired in both groups. As the neurotic depressed patients were less severely depressed and had fewer symptoms indicative of endogenous depression (Section 4.1.2) these results suggest an interaction between the severity of depression and the level of difficulty of the material such that impairments will most consistently be found on difficult tasks in severely depressed patients.

If the extent of impairment is related to the difficulty of the task it would be anticipated that performance on the recognition and cued recall tests would be less affected than performance on the free recall tests, as the former are presumed to demand less effort for completion than the latter. However, there was no significant interaction between mode of recall (recognition (d' - Trial One), free recall (Free Recall - total) and cued recall (Paired Associate - immediate recall)) and subject group when the test scores were standardised. If the depressed patients were more impaired on the free recall test than on the others the interaction between mode of recall and group would have been expected to be significant. Other factors may determine how difficult a task is in addition to the manner of recall and the tests may not in fact have differed much in the amount of effort they needed for completion. There may have been characteristics of the recognition memory test used in this

study which made it particularly difficult; for instance the 'Snoopy' cartoons may have been insufficiently distinct from one another. It would, therefore, be premature to conclude from these results that depressed patients do not have particular problems with difficult tasks.

The serial position curves derived from the Free Recall test showed both a primacy and a recency effect in the data for the depressed patients. There was no significant interaction between subject group and serial position of recalled words, as would have been expected if the depressed patients were impaired solely in the short-term or in the long-term component of memory. The depressed patients recalled as many of the final four words as the control subjects suggesting that their ability to retrieve information from short-term memory was unimpaired. However, the control subjects were the only group not to show a recency effect, presumably because they did not adopt the strategy of recalling these words first. It is therefore difficult to interpret the finding that the depressed patients did not differ from the controls in their recall of this final group of words. It may be that, as the results for Digit Span Forwards suggest, depressed patients are able to recall limited amounts of information from the working memory as well as controls but, because of the hypothesised reduction in processing capacity, show impairment either when the amount to be remembered is larger or when insufficient capacity remains to process the information adequately. However this remains a hypothesis.

In summary, the retarded depressed patients were impaired on all six variables measuring immediate memory or new learning while the less severely depressed neurotic group were significantly impaired on only three. It is argued that these included the most difficult tasks and that the degree of impairment shown in depression may be a function both of the severity of depression and the level of difficulty of the task. This is consistent with other research on the relationship between effort and performance in depression (Section 1.8.1). The serial position curves show that the impairment is not restricted either to the short-term or long-term component of memory, but is more generalised. Again, this is consistent with the view that impairment may be found at any stage of processing providing it demands sufficient effort (eg Cohen et al, 1982; Glass et al, 1981). There is some evidence from the serial position

curves and from the results for Digit Span Forwards to support the notion that impairment is related to how difficult the task is and the demands it makes on processing capacity: depressed patients may do as well as control subjects, provided the amount of information involved is limited and does not require effortful processing.

Speed of Learning

Both the retarded and neurotic depressed patients showed an impaired ability to learn new material even when it was presented several times. In contrast, Henry, Weingartner and Murphy (1973) found that bipolar and unipolar depressed patients were not impaired on the first trial of a similar test, but were on the remaining trials. They argued that this could be best interpreted as an inability to shift information from short-term to long-term memory. The present results show that the depressed patients were impaired on all trials, including the first, and therefore do not have the type of deficit proposed by Henry, Weingartner and Murphy (1973). Other studies have also failed to replicate these results (Section 1.7.1) and Johnson and Magaro (1987) have argued that this may be because Henry, Weingartner and Murphy (1973) included bipolar patients while other studies (including this one) have not.

Retention/Forgetting

There were significant differences between the retarded depressed and control subjects on two variables measuring recall after a delay: Prose Passage One - delayed recall and Prose Passage Two - delayed recall. The neurotic depressed were not significantly different from the controls, but this was also true on the first trial of the Prose Passage test. These results presumably reflect initial differences in the amount learned, because the results for the variables measuring the amount of information forgotten after a delay (Prose Passage One - forgetting, Paired Associate - forgetting) are consistent with those found in earlier studies (Cronholm and Ottosson, 1961; Sternberg and Jarvik, 1976; Steif et al, 1986): depressed patients do not differ significantly from controls in their ability to retain information once learnt. These results may be misleading because the depressed patients learned less information in the first place and therefore had less to forget but, as argued in Section

4.3.3, in all but four cases patients did not score zero on these tests and therefore could have forgotten more information than they did.

The pattern of d' scores over the four trials of the Picture Recognition test was unexpected in that the control subjects and neurotic depressed showed the greatest decline in memory while the retarded depressed showed a small increase. It is unclear why such a pattern was found but it can be concluded that again the depressed patients did not forget information more quickly that the controls.

Memory for Past Public Events

The depressed patients were significantly impaired on the multi-choice versions of these questionnaires, even when the time taken to complete it was controlled for. There was a trend for scores on the free recall questionnaires to differ between the depressed and control groups. This was unexpected as it had been hypothesised that because deficits on these tests were likely to be due to difficulty in retrieving information from memory they would be most apparent on the free recall version. The depressed patients may have been particularly impaired on the multi-choice questionnaire because they were unable to concentrate on the answers long enough to distinguish the correct answer from the distractor answers.

The important thing to note about these results, however, is that they suggest that the memory deficit in depression is not confined to learning new material but may also affect material already in memory. The results of the variables measuring the amount of information forgotten after a delay found that, at least over a short time period, depressed patients did not forget information more quickly that the controls. If this is the case, then the deficit observed here is presumably due to retrieval problems despite the fact that the multi-choice version is most affected.

There are, however, two other possibilities. The first is that the material was not learned in the first place: this is a difficulty with this type of questionnaire and this possibility cannot be completely eliminated especially with depressed subjects as earlier episodes of depression may have affected initial encoding. The second is that the depressed patients' memory for this

material was unaffected but that they were less motivated to complete the questionnaires and did not exert sufficient effort. This is supported by the finding that the depressed patients tended to omit more questions than controls on the free recall test. As already noted, it may also explain why they were particularly impaired on the multi-choice version, which may have required more concentration and effort for successful completion than the free recall version. This would be consistent with the suggestion that performance on any task may be impaired in depression providing it is sufficiently complex (Cohen et al, 1982; Glass et al, 1981; Section 1.8.1).

Response Bias

β, a measure of response bias, was calculated from scores on the Picture Recognition test (Section 2.2.1). The depressed and control groups did not differ on β scores for the first trial or the fourth trial. All the groups were, if anything, slightly biased towards being cautious. The lack of evidence for a response bias in these depressed patients conflicts with the findings of Miller and Lewis (1977), but is consistent with results obtained from a number of other studies (Hilbert, Niederehe and Kahn, 1976; Wolfe et al, 1987; Watts et al, 1987) which have also found no evidence for a conservative response bias in depression. This suggests that the lack of motivation postulated to be a cause of the poor performance of depressed patients (Section 1.8.1) does not lead to subjects retrieving information and then not producing it as a response, but must manifest itself at an earlier stage of memory: it may, for instance, affect the amount of processing carried out initially and therefore the likelihood of material entering memory; or it may affect the types of search strategies used to locate material in memory.

Summary

These results support the pattern of impairment found in previous studies in that new learning is impaired whilst depressed patients do not differ from controls in their ability to retain information once learnt. Impairment was shown on all tests of immediate memory by the most severely depressed group, but only on what were presumed to be the more difficult tests by the neurotic depressed group. Both groups were

impaired on questionnaires covering events which happened before the onset of depression: this suggests that impairment in depression is not restricted to new learning. It is hypothesised that this, as well as the pattern of impairment shown by the two groups on the immediate learning tests, are consistent with the view that impairment may be found in depression on any sufficiently complex task and that it results from a lack of motivation and effort. There was no evidence that the depressed and control groups differed in the extent of response bias shown and therefore it is suggested that the lack of motivation does not mean that the subjects have a conservative response criteria; instead it is suggested that reduced levels of motivation affect the processing of information or the type of search strategies used to access information in memory.

4.4.2 Differences in the Performance of the Retarded Depressed and Neurotic Depressed patients

As already noted the more severely depressed retarded depressed group were impaired on all six immediate memory variables, whilst the neurotic group were impaired on what were hypothesised to be the most difficult three. The performance of the two depressed groups differed significantly on only one test: d' - Trial One. Both groups were impaired on this but the retarded depressed group were clearly more impaired than the neurotic group. Thus the two groups of depressed patients did not differ significantly on the three tests where the retarded patients were significantly impaired but the neurotic patients were not. The scores of the neurotic patients were intermediate between those of the retarded depressed and control subjects but not significantly different from either. It is possible, therefore, that they had some degree of impairment on these tests, although the size of the impairment is clearly larger in the retarded depressed group.

Both groups of depressed patients were significantly impaired on both variables measuring the speed of learning. The neurotic depressed patients scored significantly higher than retarded depressed patients on Paired Associate Learning - speed of learning. The two groups both, therefore, showed some impairment in the speed at which they could learn

new information and this was particularly severe in the retarded depressed group.

Both depressed groups were able to retain information once learned as well as the control subjects, and they did not differ significantly on the variables measuring the amount of information forgotten after a delay. They also did not differ significantly on the memory for past public events questionnaires and were both impaired on the multi-choice version. Finally, they did not differ on β, the measure of response bias, and neither differed significantly from the control subjects.

The two groups of depressed patients showed the same pattern of impairment on the battery of memory tests: they were impaired on tests of immediate memory, speed of learning and the multi-choice past public events questionnaires, but were not impaired on variables measuring the amount of information forgotten after a delay or on a measure of response bias. The neurotic depressed patients were not significantly impaired on three of the immediate memory variables on which the retarded depressed did show impairment. It is hypothesised that these were the easier tests and that, as proposed above (Section 4.4.1), whether or not impairments are found in depressed patients will depend on both the severity of the depression and the level of difficulty of the task. Alternatively, it may be that what is important is not the overall severity of the depression but the presence of some key symptoms, such as those indicative of endogenous depression. As the two depressed groups in this study differed both in severity and symptom pattern it is not possible to distinguish between these hypotheses.

4.4.3 The Performance of Anxious Patients Compared to that of the Depressed Patients

The anxious patients scored higher than the depressed patients on all six variables measuring immediate memory. However, they were significantly different from the retarded depressed patients on only one variable, d' - Trial One, and did not differ significantly from the neurotic depressed patients on any variable. They scored lower than the control subjects which, together with the fact that there was only one significant difference between the scores of the anxious and depressed

patients, may suggest that the anxious patients were impaired on these variables. However, their scores did not differ significantly from those of the control subjects on any variable. These results are presumably a consequence of the small number of subjects in the anxious group. It is possible that the anxious patients were impaired on these variables measuring immediate memory but the evidence is inconclusive.

It is clear that the anxious patients did not have the same impairment on the speed of learning tests as the depressed patients. As Figures 4.9 and 4.11 show, the scores of the anxious patients were near those of the depressed patients on Trial One, but on Trial Two they performed at nearly the same level as the control subjects. The scores of the anxious patients on Serial learning - speed of learning were significantly higher than those of the depressed patients. Thus it is clear that the anxious patients were able to overcome any impairments they had on the first trials of these tests. This may be because they exerted more effort on the second trial after having assessed the resources required on the first trial. This is consistent with the view of Eysenck (1979) that working memory capacity is reduced in anxiety but that they overcome the effects by exerting more effort.

Like the depressed patients, there was no evidence that the anxious patients differed from the controls in their ability to retain information in memory. The pattern of results on the past public events questionnaires was similar to that found on the immediate memory variables: the scores of the anxious patients were intermediate between those of the depressed and control subjects but significantly different from neither. It is therefore unclear whether or not they were impaired on these tests. The results on the measure of response bias are clearer: as with the depressed patients, the anxious patients did not differ from the controls on these variables.

Few conclusions can be drawn about the performance of the anxious patients in relationship to that of the depressed patients due to the small number of anxious subjects. Like the depressed patients, they were not impaired on the variables measuring the amount of information forgotten before delayed recall, or on the measures of response bias. However, their scores were intermediate between those of depressed

patients and controls on the measures of immediate memory and on the two past public events questionnaires, but significantly different from neither. Further research is therefore required to ascertain whether or not the anxious patients are impaired on these measures. The results for the variables measuring speed of learning are more conclusive: the anxious patients performed significantly better than the depressed patients and therefore did not show the same pattern of impairment.

4.4.4 Summary

The results presented in this chapter show that depressed patients are impaired on measures of new learning, speed of learning and memory for past public events. They do not differ from control subjects in their ability to retain information in memory, nor in their response criteria. The two groups of depressed patients, retarded depressed and neurotic depressed, do not differ in the pattern of impairment shown but the less severely depressed neurotic group were only impaired on what were presumed to be the more difficult tests. It is hypothesised that these results support the suggestion that whether or not impairments are found in depression will depend on both the level of difficulty of the task (Section 1.1.8) and the severity of depression.

The results for the anxious patients were ambivalent, presumably due to the small numbers in this group. They suggest that the anxious patients may be impaired on tests of immediate memory amd memory for past public events and this possibility needs further exploration. However, it is clear that even if they are impaired on the first presentation of material, they are able to overcome the impairment when material is presented several times, possibly, as Eysenck (1979) suggests, by increasing the amount of effort they put into the task.

The four subject groups were not matched on any variable in this study; practical difficulties were experienced in recruiting subjects and this made any attempts at matching impracticable. It could therefore be argued that the differences observed between these groups were not due to differences in psychiatric status but to differences in, for instance, age, social class and educational level. However, there were no significant differenoes between the groups in mean age, social class or years of

education (Section 4.2.2). Despite the lack of matching, therefore, the groups do not differ on these variables. It therefore seems unlikely that the results reported in this chapter were a consequence of these variables rather than of the psychiatric status of the subjects.

However, before going on to explore the relative effects of depression and anxiety on the memory of these subjects it is necessary to ensure that the differences observed between the subject groups in this chapter are really due to their psychiatric condition and not to the psychotropic medication which the depressed and anxious patients were receiving, or to a history of E.C.T (Section 3.2). The possibility that the group differences are artifacts resulting from the treatment the subjects received is therefore examined in the next chapter (Chapter Five).

DO DEPRESSED, ANXIOUS AND CONTROL SUBJECTS STILL SHOW DIFFERENCES IN MEMORY WHEN THE EFFECTS OF PSYCHOTROPIC MEDICATION AND E.C.T. ARE CONTROLLED FOR?

5.1 INTRODUCTION

In Chapter Four, results were presented which showed that depressed patients were impaired on tests of immediate memory, speed of learning and memory for past public events but not on tests of the amount of information forgotten after a delay or on a measure of response bias. In addition, retarded depressed and neurotic depressed patients were shown to have the same pattern of memory impairment, but in the less depressed group of neurotic depressed patients impairment only occurred on what were hypothesised to be the more difficult tests. Anxious patients were shown not to be impaired on tests of the speed of learning, the retention of information and response bias whilst their results on tests of immediate learning and memory for past public events were ambivalent.

Before conclusions are drawn from these results about memory in depression and anxiety it is important to note that it is possible that the observed memory deficits were not due to the subjects' psychiatric condition but to the treatment they were receiving. As outlined in Section 3.2, patients taking psychotropic medication or who had had electro-convulsive therapy (E.C.T) were not excluded from the study for both practical and theoretical reasons: to do this would have made it very difficult to recruit subjects, whilst patients not on psychotropic medication would have been atypical of most depressed and anxious people in contact with the medical profession, the majority of whom are likely to be on some sort of medication (Johnson, 1973). The effects of these treatments need to be taken into consideration before attributing the memory deficits to the psychiatric disorders themselves as there is evidence that E.C.T and some types of psychotropic medication can impair memory.

E.C.T has been shown to have pronounced effects on concentration, short-term memory and learning in the first few days after treatment, although there is less evidence for long-term effects (Frith et al, 1983, 1987; Heaton and Crowley, 1981; Taylor et al, 1982). Curran (1986)

reviewed over 90 studies on the effects of benzodiazepines on memory and concluded that anterograde amnesia was very common; this conclusion was mainly based on studies in normal volunteers and there is little information on how they affect memory in patient populations. Thompson and Trimble (1982) reviewed the effects of non monoamine-oxidase inhibiting (MAOI) anti-depressants on memory and found that the evidence from studies on normal volunteers conflicted with the results of studies using depressed patients: the former typically found that tricyclic anti-depressants impaired memory, whilst the latter tended to find beneficial effects, presumably because depression was alleviated by treatment. Heaton and Crowley (1981), in their extensive review of the psychological effects of psychiatric somatic treatments, found evidence that neuroleptic drugs can produce memory deficits early on in treatment, although there is some evidence to contradict this (Killian et al, 1984). The evidence for the effects of lithium on memory is also equivocal and Anath, Ghadirian and Englesmann (1987) concluded that there is no convincing proof that it causes memory problems.

There is, therefore, reason to believe that E.C.T and benzodiazepines can impair memory, and some evidence that tricyclic antidepressants and neuroleptics may do so. It is therefore possible that the memory differences between the subject groups reported in Chapter Four were a consequence of these, rather than depression or anxiety. This chapter therefore considers whether there are still significant differences in performance on the battery of memory tests between the four groups of subjects (control, retarded depressed, neurotic depressed and anxious) once the effects on memory of the three most common groups of psychotropic medication (tricyclic anti-depressants; benzodiazepines; neuroleptics) and E.C.T. are controlled for statistically.

5.2 METHOD

5.2.1 Materials

The materials used in this study have already been described in detail (Chapter Two) and are outlined in Section 4.2. This chapter is concerned solely with those variables which were found in the preceding

chapter (Chapter Four) to differ significantly between the subject groups. This therefore excludes the test of registration (Digit Span Forward); variables measuring the amount forgotten after a delay (Prose Passage One - forgetting, Paired Associate - forgetting and d' - forgetting); the free recall version of the past public events questionnaire and the measure of response bias (β).

5.2.2 Subjects

The subjects used in this study are described in detail in Chapter Four: this section will therefore concentrate on their use of psychotropic medication and histories of E.C.T.

Psychotropic Medication

One subject in the neurotic depressed group was excluded from this study because it had not been possible to obtain information on his psychotropic medication. Three of the remaining 29 patients included in this study were not taking any type of psychotropic medication at the time of testing. As described in Section 3.4.3 the remaining 26 patients were prescribed a total of seven different types of psychotropic medication, classified according to the system used by Silverstone and Turner (1982). These are listed in Table 5.1, together with the number of subjects in each psychiatric group (retarded depressed, neurotic depressed and anxious) who were taking each type during at least one of the testing sessions. (The mean, median and range of daily doses are given in Table 3.6). The medication received by individual subjects varied between testing sessions: only the scores of subjects who were taking a drug at the time a test was given are included in the analysis of the effects of that drug on performance on that test.

This chapter concentrates on three types of medication: tricyclic anti-depressants; benzodiazepines and neuroleptics (major tranquillisers). These were the most frequently prescribed classes of drug: none of the remainder were prescribed often enough to be included in the analysis.

Electro-convulsive Therapy (E.C.T)

Six subjects in this study had had E.C.T in the year prior to testing, and the time between E.C.T and testing ranged from two weeks to five months. Five of the retarded depressed (50%), one of the neurotic depressed (7%) and none of the anxious subjects had had E.C.T in this period. Two of the neurotic depressed patients had had E.C.T more than a year previously, one of whom had also been given it in the previous twelve months.

Table 5.1 NUMBER OF SUBJECTS TAKING PSYCHOTROPIC MEDICATION AT AT LEAST ONE TESTING SESSION.

Class of drug	Number prescribed it		
	Retarded depressed (N=10)	Neurotic depressed (N=12)	Anxious (N=7)
Benzodiazepines	8	8	4
Tricyclics	3	8	3
Neuroleptics	5	3	1
M.A.O inhibitors	2	1	1
L-tryptophan	2	2	0
Lithium	2	1	0
Beta-adrenergic drugs	2	1	0

5.2.3 Analysis

Regression analysis was used to see if there were significant differences in recall between the four groups (retarded depressed, neurotic depressed, anxious and controls) when the effects of the three most frequently prescribed psychotropic drugs (tricyclics, benzodiazepines and neuroleptics) and the effect of having E.C.T in the year before testing were controlled for. This analysis is equivalent to the one-way

analyses of variance reported in the preceding chapter, except that the effects of the drugs and E.C.T were controlled for statistically.

Group membership was entered into the regression equation as three dummy variables, where a score of 1 on the variable D1 represented the anxious subjects, on D2 represented the retarded depressed and on D3 the neurotic depressed. The control group, coded as 0 on all three, made up the reference group. This means that the partial regression coefficients for each of the three dummy variables express the relationship between the group represented by the variable and the reference group. The statistical significance of the coefficient is therefore the significance of the relationship between the scores of that group and the control subjects after the effects of the psychotropic medication and E.C.T have been taken into account: the regression coefficents are therefore equivalent to the individual comparisons of the control subjects with the depressed and anxious subjects reported in Chapter Four, except that the effects of treatment are controlled for statistically.

In each equation the overall increase in variance explained by the treatment variables (tricyclics, benzodiazepines, neuroleptics, E.C.T) is considered, rather than the effect of individual predictors. This is because the focus of this chapter is on whether there are significant differences between groups when the treatment variables are taken into consideration, rather than on the role of the treatment variables themselves.

5.3 RESULTS

5.3.1 Immediate Memory

For each variable, Group was entered into the equation by itself as three dummy variables, where a score of 1 on the variable D1 represented the anxious subjects, on D2 represented the retarded depressed and on D3 the neurotic depressed. The control group, coded 0 on all three, made up the reference group. This is the same as the one-way analysis of variance reported in Section 4.3.1, except that one case is excluded from the present analysis because no information on psychotropic drug use was

available for the patient. The three classes of psychotropic medication and E.C.T (the treatment variables) were then added to the equation to see if they significantly increased the amount of variance (R^2) explained by the equation. They were then entered into an equation individually to see if they explained a significant amount of variance by themselves. Finally, and most importantly, Group was added to this equation: if performance still differed significantly between the groups when the effects of the treatment variables were controlled for then the addition of Group would cause a significant increase in the amount of variance explained.

As Table 5.2 shows, Group explained a significant amount of the variance in all six variables measuring immediate memory when placed alone in the equation: this was expected, as only those variables where a significant effect of Group was found initially (Section 4.3.1) were included in this analysis. On three of the variables, d' - Trial One, Free Recall - total and Serial Learning - immediate recall, Group caused a significant increase in variance when added to the regression equation containing the treatment variables. This shows that the significant differences between the subject groups found in the preceding chapter were not, in these cases at least, caused solely by the treatment patients were receiving rather than by their psychiatric condition.

Group did not cause a significant increase in the amount of variance explained in Prose Passage One - immediate recall when added to the equation containing the treatment variables: the significant difference between the subject groups reported in Chapter Four therefore disappeared when the effects of the treatment variables are controlled for. The treatment variables did not significantly increase the variance when added to the equation containing Group, although they had explained a significant amount of the variance when in the regression equation by themselves. These results indicate that it is not possible to distinguish between the effects of Group and the effects of the treatment variables on this variable.

On both Paired Associate - immediate recall and Digit Span Backwards, neither Group nor the treatment variables explained a significant amount of variance when added to the equation containing the other variable(s). Again, this could indicate that it is not possible to

distinguish between the effects of Group and the treatment variables. However, the treatment variables did not explain a significant amount of the variance on either of these variables when entered in an equation by itself. It is therefore concluded that as the treatment variables were not significantly related to performance the differences observed between the groups (Section 4.3.1) were due to the psychiatric conditions, not to the treatment variables.

Table 5.3 gives the standardised partial regression coefficents for the dummy variables from the regression equation containing both Group and the treatment variables. As explained above (Section 5.2.3), these express the relationship between the subject group represented by the dummy variable and the control subjects with the effects of the treatment variables partialled out. The retarded depressed patients differed significantly from the controls on five of the six immediate memory variables; the exception was Prose Passage One - immediate recall. The neurotic depressed patients were significantly different from the controls on the three variables on which they were shown to significantly impaired in the previous chapter: d' - Trial One, Free Recall - total, and Serial Learning - immediate recall. The anxious patients did not differ significantly from the control subjects on any of these variables, as was the case when the effects of the treatment variables were not partialled out (Section 4.3.1). However, on three of these variables (d' - Trial One, Paired Associate - Trial One, Digit Span Backwards) there was a trend for them to do so ($p>0.05$, <0.1). This suggests that they may have been impaired on these tests.

Controlling for the effects of the psychotropic drugs and E.C.T therefore has not changed the overall pattern of results obtained in Chapter Four: the retarded depressed patients are impaired on all but one of the immediate learning variables, the neurotic depressed are impaired on what are presumed to be the most difficult three, and there is no conclusive evidence for significant impairment in the anxious patients. The relationship between the regression coefficients of the depressed and anxious patients is the same as that found between their raw scores: the retarded depressed patients scored lowest, followed by the neurotic depressed patients with the scores of the anxious patients lying between those of the depressed and control subjects.

TABLE 5.2 IMMEDIATE MEMORY AND SPEED OF LEARNING: VARIANCE EXPLAINED (R^2) WHEN 'GROUP' ENTERS THE REGRESSION EQUATION BEFORE THE TREATMENT VARIABLES, AND WHEN THE ORDER IS REVERSED.

VARIABLE	R^2 EXPLAINED BY GROUP + TREATMENT VARIABLES		R^2 EXPLAINED BY TREATMENT VARIABLES + GROUP		RESIDUAL D.F.
d' - Trial One	.39	**	.24		27
	.45	*	.45	*	
Free Recall - total	.26	**	.04		32
	.31		.31	*	
Serial Learning - immediate recall	.32	**	.27	*	31
	.47		.47	**	
Prose Passage One - immediate recall	.24	*	.31	**	29
	.39		.39		
Paired Associate - immediate recall	.20	*	.12		32
	.30		.30		
Digit Span Backwards	.22	*	.12		30
	.27		.27		
Serial Learning - speed of learning	.45	**	.18		31
	.50		.50	**	
Paired Associate - speed of learning	.45	**	.26	*	32
	.58		.58	**	

Significance of change in R^2, * $p<0.05$ ** $p<0.01$

TABLE 5.3 IMMEDIATE MEMORY AND SPEED OF LEARNING: STANDARDISED PARTIAL REGRESSION COEFFICIENTS (B) AND t VALUES OF THE DEPRESSED AND ANXIOUS PATIENT GROUPS, WITH THE EFFECTS OF THE TREATMENT VARIABLES PARTIALLED OUT.

	RETARDED DEPRESSION			NEUROTIC DEPRESSION			ANXIETY STATE	
	B	t		B	t		B	t
d' - Trial One	-1.00	-3.42	**	-0.68	-2.79	**	-0.32	-1.74
Free Recall - total	-0.77	-2.83	**	-0.77	-3.27	**	-0.27	-1.39
Serial Learning - immediate recall	-0.70	-3.07	**	-0.63	-2.89	**	-0.22	-1.27
Prose Passage One - immediate recall	-0.50	-1.92		-0.39	-1.60		-0.17	-0.89
Paired Associate - immediate recall	-0.72	-2.72	*	-0.28	-1.19		-0.33	-1.72
Digit Span Backwards	-0.65	-2.33	*	-0.43	-1.64		-0.36	-1.82
Serial learning - speed of learning	-1.02	-4.95	**	-0.50	-2.36	*	0.02	0.14
Paired Associate - speed of learning	-0.77	-3.41	**	-0.57	-3.11	**	-0.34	-2.25

* $p<0.05$ ** $p<0.01$

5.3.2 Speed of Learning

When entered into the regression equation alone, Group explained a significant amount of the variance in both Paired Associate - speed of learning and Serial Learning - speed of learning variables (Table 5.2). When Group was added to the regression equation containing the treatment variables it caused a significant increase in the variance explained in both variables. Thus, the significant differences observed between the subject groups on these variables (Section 4.3.2) were not due to the treatment the subjects were receiving.

Both the retarded depressed and neurotic depressed patients differed significantly from the controls on these variables when the effects of the treatment variables were partialled out, as they had done in the initial analysis (Table 5.3). However, the anxious patients differed from the controls on Paired Associate - speed of learning which was not the case originally. This result suggests that although anxious patients were not as impaired as depressed patients on these variables (Section 4.3.2) they may have had some degree of impairment on them.

5.3.3 Retention

Group alone explained a significant amount of the variance in Prose Passage One - delayed recall (Table 5.4). However it did not significantly increase the amount of variance explained when added to the equation containing the treatment variables. This suggests that the significant differences between the groups observed in the original analysis (Section 4.3.3) were due to treatment the subjects were receiving rather than their psychiatric condition. However, the treatment variables did not explain a significant amount of variance when added to the equation containing Group, although there was a trend for them to do so when in the equation by themselves. As neither Group or the treatment variables have a significant effect on recall when the effects of the other were controlled for, something common to both variables was affecting performance on this test.

The results for Prose Passage Two - delayed recall are somewhat clearer (Table 5.4). Group explained a significant amount of variance when

placed in an equation by itself, and it also significantly increased the amount of variance explained when added to the equation containing the treatment variables. This indicates that the differences between the groups were not due to the subjects' treatment rather than to their psychiatric condition.

As in other analyses the data for Paired Associate - Trial Five were examined graphically before formal analysis was undertaken. In this case the variance of the scores was clearly correlated with their mean. The scores were therefore squared to overcome the heterogeneity of variance. Group explained a significant amount of variance when placed alone in the equation and significantly increased the amount of variance explained when added to the equation containing the treatment variables (Table 5.4). Again, this indicates that the differences between the groups reported in Section 4.3.3 were not solely due to the treatment the subjects were receiving, but also to their psychiatric condition.

Table 5.5 shows that the retarded depressed patients differed significantly from the controls on all three of the variables in this section, as they had done in the original analysis. The anxious patients also showed the same pattern of results as in the original analysis: they were not significantly impaired in relation to the controls on any of these variables. The neurotic depressed patients were significantly different from the controls on Paired Associate - Trial Five (again as in the original analysis) and they were also significantly different from the controls on Prose Passage Two - delayed recall. This was not originally the case.

TABLE 5.4 RETENTION AND PAST PUBLIC EVENTS: THE AMOUNT OF VARIANCE EXPLAINED (R^2) WHEN 'GROUP' ENTERS THE REGRESSION EQUATION BEFORE THE TREATMENT VARIABLES, AND WHEN THE ORDER IS REVERSED.

VARIABLE	R^2 EXPLAINED BY: GROUP + TREATMENT VARIABLES		R^2 EXPLAINED BY: TREATMENT VARIABLES + GROUP		RESIDUAL D.F.
Prose Passage One - delayed recall	.25 .36	*	.24 .36		29
Prose Passage Two - delayed recall	.21 .41	*	.19 .41	*	30
Paired Associate - Trial Five[a]	.40 .54	**	.37 .54	** *	31
Past Events multi-choice questionnaire	.22 .38	*	.20 .38	*	30

[a] This variable was squared to overcome heterogenity of variance

Significance of change in R^2, * $p<0.05$ ** $p<0.01$

TABLE 5.5 RETENTION AND PAST PUBLIC EVENTS: STANDARDISED PARTIAL REGRESSION COEFFICIENTS (B) AND t VALUES OF THE DEPRESSED AND ANXIOUS PATIENT GROUPS, WITH THE EFFECTS OF THE TREATMENT VARIABLES PARTIALLED OUT.

	RETARDED DEPRESSION			NEUROTIC DEPRESSION			ANXIETY STATE	
	B	t		B	t		B	t
Prose Passage One - delayed recall	-0.62	-2.32	*	-0.48	-1.91		-0.31	-1.48
Prose Passage Two - delayed recall	-0.76	-3.06	**	-0.70	-2.98	**	-0.30	-1.59
Paired Associate - Trial Five[a]	-0.75	-3.30	**	-0.47	-2.41	*	-0.25	-1.58
Past Events multi-choice questionnaire	-0.56	-2.35	*	-0.62	-2.80	**	-0.23	-1.25

[a] This variable was squared to overcome heterogeneity of variance

* $p<0.05$ ** $p<0.01$

5.3.4 Memory for Past Public Events

As expected, Group explained a significant amount of the variance in the Past Events multi-choice questionnaire when placed in the regression equation by itself (Table 5.4). It significantly increased the amount of variance explained when added to the equation containing the treatment variables. There is therefore no evidence that the differences in the group scores on this questionnaire were due to the treatment the subjects were receiving, rather than to group membership.

Table 5.5 shows that, as in the original analysis (Section 4.3.4) the scores of retarded depressed and neurotic depressed patients were significantly impaired compared to those of the control subjects. The significance of the partial regression coefficent for the anxious group approached significance ($p=0.06$), which lends support to the argument that the anxious patients may have been impaired on this test.

5.4 DISCUSSION

This chapter is concerned with the possibility that the significant differences in memory between the four subject groups (control, retarded depressed, neurotic depressed and anxious) reported in Chapter Four were not due to the subjects' psychiatric conditions but to the treatment they were receiving. Regression analysis was therefore used to see whether there were still significant differences between the groups when the effects on memory of the patients' psychotropic medication and E.C.T were taken into account.

5.4.1 Immediate Memory

Once the effects of the treatment variables (tricyclic anti-depressants; benzodiazepines; neuroleptics; E.C.T) were controlled for there were still statistically significant differences between the four groups on three of the six variables measuring immediate memory; d' - Trial One, Free recall - total, and Serial learning - immediate recall.

On Paired Associate - immediate recall and Digit Span Backwards, the amount of variance explained by subject group was not significant when the variables coding group (Group) were added to the regression equation containing the treatment variables. However, there was no evidence that the treatment variables affected performance on these variables and so it cannot be argued that the observed differences between the groups (Section 4.3.1) were due to the treatment the subjects were receiving. The effect of entering the treatment variables in the regression equation before Group was that any variance shared by the treatment variables and Group was allocated to the treatment variables; this reduced the variance allocated to Group and the likelihood of it being statistically significant. As the amount of variance explained by Group by itself was quite small the effect of entering it after the treatment variables was to reduce the amount of variance attributed to it below the level of significance. It is not surprising that there was only a weak effect of Group on these variables, since only the retarded depressed and control groups differed on them initially (Section 4.3.1). It is, however, clear that the differences between these groups were not due to psychotropic medication and E.C.T.

On Prose passage One - immediate recall the amount of variance explained by Group was not significant when it was added to the regression equation containing the treatment variables. In contrast to Paired Associate - immediate recall and Digit Span Backwards, the treatment variables were significant predictors of performance when entered in the equation by themselves, although they were not significantly related to performance when added to the equation containing Group. The treatment variables may have been affecting performance on this test rather than Group, but they did not explain a significant amount of the variance when added after Group because shared variance was allocated to Group. Alternatively these results may indicate that it is not possible to distinguish between the effects of the treatment variables and Group on this variable. It must be concluded that the differences between the groups found on this variable initially (Section 4.3.1) were at least as likely to have been due to the effects of psychotropic medication and E.C.T as to the psychiatric conditions of the patients. This may have been because this test differed from the other tests of immediate memory; the material was structured and meaningful and

as outlined in Section 4.4 this would have made it easier to recall. Consequently it would be less likely to be affected by a depression- or anxiety- related impairment in memory.

When the patient groups were compared to the control subjects it was found that the retarded depressed patients were impaired on five of the six variables measuring immediate memory: the exception was Prose Passage One - immediate recall. The neurotic depressed group were significantly impaired on what were presumed to be the three more difficult tests (d' - Trial One, Serial Learning - immediate recall and Free Recall - total), as they had been in the original analysis. The anxious patients were not significantly different from the controls on any of the six tests, although there was a trend ($p>0.05$, <0.1) for them to be so on d' - Trial One, Paired Associate - immediate recall and Digit Span Backwards. This suggests that significant differences may have been found if there had been more anxious subjects and lends weight to the conclusion drawn above (Section 4.4) that the anxious patients may have been impaired on these tests of immediate memory.

Differences between the two groups of depressed patients, and between the depressed and anxious patients, were not investigated in this chapter. However the conclusions drawn in Chapter Four about overall differences between the subjects groups have only been substantially altered on one of these six variables following the partialling out of the effects of the treatment variables. There is, therefore, little reason to suppose that the conclusions reached in Chapter Four about the differences between the two depressed groups, and between the depressed and anxious groups, would have been affected much by the results of an analysis controlling for the treatment variables.

In summary, the results on tests of immediate memory were little affected by controlling for the effects of psychotropic drug use and E.C.T on memory. The impairments found on Prose Passage One - immediate recall may have been due to the treatment the patients were receiving rather than to their psychiatric condition. With this exception, the original conclusion that the retarded depressed patients were impaired on all the tests, while the neurotic depressed were impaired on the more difficult

three, still stand. In addition, the results for the anxious patients were again inconclusive.

5.4.2 Speed of Learning

On both variables measuring the speed at which subjects learned words presented over several trials (Serial learning - speed of learning and Paired Associate - speed of learning) it was clear that the four groups continued to differ significantly once the effects of the treatment variables were controlled for. The differences found between the groups in Section 4.3.2 were clearly the result of the subjects' psychiatric condition and not just the result of the treatment they were receiving.

When the depressed and anxious groups were compared to the control subjects the depressed patients were again found to be significantly impaired. In contrast to the original analysis (Section 4.3.2), the anxious patients scored significantly lower than the controls on Paired Associate - speed of learning. This again indicated that they may have had difficulty learning new material although, as their scores were significantly higher than those of the retarded depressed patients (Section 4.3.2), they had less difficulty than the very depressed patients.

5.4.3 Retention

These variables might be more appropriately labelled 'recall after a delay' as the variables measuring the retention of material (or the amount forgotten after a delay) were not included in this chapter because they did not differ significantly between the subject groups (Section 4.3.3).

On two of the three variables measuring the recall of material after a delay (Prose passage Two - delayed recall and Paired Associate - Trial Five) there were still significant differences between the groups once the the effects of the treatment variables were controlled for.

The results for the third variable in this section (Prose Passage One - delayed recall) were less clear-cut. Group did not significantly increase the amount of explained variance when added to the regression

equation containing the treatment variables. However, the treatment variables were not significantly related to recall when entered into the equation without Group, although there was a trend for them to be so. They did not increase the amount of variance explained when entered into the equation containing Group. They were, therefore, unlikely to be the sole cause of the effects on memory found in Section 4.3.3. It was concluded that Group did not significantly increase the amount of variance explained by the treatment variables because, as already noted, the prose passage recall tests were relatively easy and not greatly affected by depression. Group was consequently not strongly related to performance on this test, and therefore no longer a statistically significant predictor of performance when shared variance was attributed to the treatment variables. The results for this variable, when combined with those for the immediate recall of this prose passage, suggest that the treatment variables did affect performance and that in addition depression and/or anxiety may have had a limited effect. The latter conclusion is supported by the fact that scores on Prose Passage Two, a very similar test, were significantly affected by group membership. It is further supported by the finding, reported below, that the retarded depressed patients were significantly different from the controls on Prose Passage One - delayed recall after the effects of the treatment variables were partialled out.

When the depressed and anxious groups were compared to the control group the retarded depressed patients were significantly impaired on all three variables, as they were in the original analysis (Section 4.4.1). The neurotic depressed patients were significantly impaired on Paired Associate - Trial Five and they were also impaired on Prose Passage Two - delayed recall, which was not the case originally.

Overall, therefore, controlling for the effects of psychotropic medication and E.C.T has not altered the conclusions drawn in Section 4.3.3: the retarded depressed patients are impaired on all three variables measuring recall after a delay; the neurotic depressed patients are impaired on two of them, while there is no evidence that the anxious patients were impaired on these variables.

5.4.4 Memory for Past Public Events

The four groups still differed significantly on the Past Events multi-choice questionnaire when the effects of psychotropic medication and E.C.T were controlled for statistically. In this case the differences between the groups reported in Section 4.3.4 were due to the subjects' psychiatric condition, rather than solely to the treatment they were receiving.

As before, both groups of depressed patients differed significantly from the controls on this test. There was a trend for the anxious patients to do so. This supports the conclusion drawn in Section 4.4.3 that the anxious patients may have been impaired on this test but that the results are inconclusive because of the small number of anxious patients.

5.4.5 Summary

In conclusion it is clear that, with the possible exception of one of the easier tests (Prose Passage One - immediate recall), the significant differences in performance of the four subject groups reported in Chapter Four were still evident when the effects on memory of treatment received by the subjects were controlled for statistically. They were not therefore solely a consequence of the psychotropic medication and E.C.T the subjects had received, although these did affect performance on some of the tests.

When the depressed and anxious patient groups were compared to the control subjects it was found that, as in the original analysis, the retarded depressed patients were impaired on tests of immediate learning, speed of learning, recall after a delay and memory for past public events. The neurotic depressed patients were significantly impaired on what were judged as the more difficult of these tests. The anxious patients showed a trend towards impairment on some of the tests of immediate learning and were impaired on one of the tests of speed of learning, although not as severely as the retarded depressed group. They also tended to be impaired on the memory for past public events questionnaire. These results support the conclusion reached in Chapter Four that the

possibility that anxious patients were impaired on these tests cannot be ruled out, although the present results are equivocal.

Having established that the differences between the four groups are not solely caused by differences in treatment it is appropriate to to look for explanations for the memory impairment based upon aspects of the psychiatric conditions themselves. In Chapter Six the relative effects of self-rated depression and anxiety on memory in these subjects are explored.

MEMORY IMPAIRMENT IN DEPRESSED AND ANXIOUS SUBJECTS: RELATED TO DEPRESSION, ANXIETY OR BOTH?

6.1 INTRODUCTION

The results of the previous two chapters (Chapters Four and Five) have shown that depressed patients are impaired on tests of new learning, speed of learning and memory for past public events. This is true even when the effects of psychotropic treatments and E.C.T are controlled for statistically. The impairments are particularly evident in the retarded depressed group, while the less severely ill neurotic depressed group are only impaired on what are judged to be the more difficult tests. The evidence from the anxious patients is equivocal and the possibility that they are impaired on the same tests as the depressed patients, although to a lesser extent, cannot be ruled out. These results suggest that memory is impaired by depression, and possibly by anxiety.

However, it may be more appropriate to conclude that memory is impaired *in* depression rather than *by* it, and similarly that memory may be impaired *in* anxiety, rather than *by* it. It can be hypothesised that the impairment found in depressed patients is really due to anxiety, or that alternatively the putative impairment in anxiety is really due to depression. This is because, as Stravrakaki and Vargo (1986) have noted, there is a considerable overlapping of symptomatology between the two disorders.

There is evidence, for example, that clinically depressed individuals have high levels of anxiety symptoms. Fawcett and Kravitz (1983) used the Schedule for Affective Disorders and Schizophrenia (SADS; Spitzer and Endicott, 1977) to assess 200 patients with a Research Diagnostic Criteria (RDC, Spitzer, Endicott and Robins, 1977) diagnosis of major depression. They found that 72% of the sample had at least moderate levels of worry, 42% had moderate or severe somatic anxiety, whilst 62% had moderate or severe psychic anxiety. Prusoff and Klerman (1974) used discriminant analysis to get a separation of depressed and anxious out-patients and found that the depressed patients had a significantly higher score on the anxiety factor than the anxious group. Foulds and Bedford (1976) found that 86% of psychiatric patients scoring above the cut-off

point for depression on the 'Delusions, Symptoms, States Inventory' (DSSI) also scored above the cut-off point for anxiety. Uhlenhuth et al (1983) found that 65% of subjects recruited during a nationwide survey, and allocated to the diagnostic group of major depression, had high somatic anxiety scores, while 87% had high anxious mood scores.

Bramley et al (1988) used patients diagnosed either as being clinically depressed, or as having a generalised anxiety disorder, to investigate the ability of rating scales to differentiate between the disorders of depression and anxiety. They found that although scores on depression self-rating scales did not correlate significantly with a criterion measure of anxiety (the Clinical Anxiety Scale) anxiety self-rating scales were significantly correlated with the criterion measure of depression (the Montgomery Asberg Depression Rating Scale). They concluded that this was because psychiatrists tend to overlook mild degrees of anxiety when diagnosing depression, and that if both are present depression is usually given precedence. Thus depressed patients are likely to also be anxious, while the reverse (according to Bramley et al, 1988) is not true.

There is, however, some evidence that anxious patients have symptoms of depression. Steer et al (1986) compared 100 depressed patients with 35 patients with generalised anxiety disorders and found that the mean Beck Depression Inventory (BDI) score of the anxious patients was in the mildly depressed range (14.4); similar results were obtained by Mathew, Swihart and Weinman (1982) in anxious patients specially selected because they did not have 'significant depressive affect'. Foulds and Bedford (1976) found that 83% of those scoring above the cut-off for anxiety on the DSSI were also above it for depression, while Uhlenhuth et al (1983) found that half of the anxious subjects in their study (described above) had high scores on a depressed mood factor. Barlow et al (1986) made DSM-III (American Psychiatric Association, 1980) diagnoses for patients attending an anxiety clinic without reference to the usual exclusionary systems: additional diagnoses were allowed if they were judged to be independent of the primary diagnosis. They found that major depression was given as an additional diagnosis in 39% of agoraphobics, 19% of patients with social phobias, 35% of those with panic disorders, 17% of those with generalised anxiety and 67% of those with an obsessive

compulsive diagnosis. Dealy et al (1981) found that a third of subjects recruited through newspaper adverts to take part in a drug study of anxiety had a secondary major affective disorder in addition to fulfilling the DSM-III criteria for a generalised anxiety or panic disorder. There is, therefore, considerable evidence for the co-existence of symptoms of depression and anxiety in individuals whose symptoms are predominantly those of anxiety.

Given the evidence that depressed patients are also likely to be anxious, and that anxious patients often have symptoms of depression, it is not surprising that significant correlations have been found between scores on depression rating scales and those on anxiety rating scales. Meites, Lovallo and Pishkin (1980) compared the scores of 170 undergraduates on the BDI and Zung depression scale with scores on the Taylor Manifest Anxiety scale and the neuroticism part of the Eysenck Personality Scale. All four were highly correlated, which led the authors to conclude that they were measuring a single personality factor. Dent and Salkovskis (1986) found that scores on the BDI of 243 subjects, most of whom were students, correlated highly with both the severity of anxiety symptoms (r=.61) and the frequency (r=0.63) as assessed by the Beck Anxiety Check List. Similar results have been found in patient samples. For instance Johnstone et al (1980) found correlations between the Leeds Depression and Anxiety subscales of 0.45 in the first week of a drug trial using neurotic anxious and depressed out-patients; and 0.67 in the fifth week of the trial. Significant correlations were also found between scores on the observer-rated Hamilton Depression Rating Scale and on the Hamilton Anxiety Rating Scale (week one, r=0.53; week five, r=0.77), indicating that the strong relationship in other studies does not just reflect the inability of patients to differentiate between anxiety and depression (Leff, 1978). Bramley et al (1988) found that all the self-rated anxiety and depression scales used in their study (see above) were highly correlated. A correlation of .48, for example, was found between the depression and anxiety subscales of the Irritability-Depression-Anxiety scale.

The fact that there is so much overlapping symptomatology between depression and anxiety has led to considerable debate about their relationship. Some researchers and clinicans have taken a unitary

approach and argued that depression and anxiety are variants of the same disorder and differ only quantitatively (eg. Gersh and Fowles, 1979; Johnstone et al, 1980; Kendell, 1974; Tyrer et al, 1987). Others argue for a pluralistic model in which there are two distinct disorders which differ qualitatively (eg Mountjoy and Roth, 1982; Prusoff and Klerman, 1974; Roth et al, 1972). It has also been suggested that patients with co-existing depression and anxiety have a different disorder to either anxiety or depression alone (eg. Clancey et al, 1978; Van-Valkenburg et al, 1984). Stravrakaki and Vargo (1986) reviewed the literature on the relationship between anxiety and depression and concluded that although clinically they often can be classified as different disorders in view of the contradictory research findings there is as yet 'no unequivocal solution to the problem of how anxiety and depression are related'. A similar conclusion was recently reached by Eaton and Ritter (1988) who used field survey data to explore the pattern of relationships of anxiety and depression to socio-demographic factors, prior psychopathology and Life Events, and found that none of these distinguished between the two syndromes.

Whatever view is taken of the relationship between depression and anxiety, it is clear that they frequently co-exist and that consequently individuals with high levels of one are also likely to have symptoms of the other. This means that levels of anxiety need to be taken into consideration when assessing the effects of depression on memory, and vice versa. However, this is rarely done. As Strack et al (1985) have noted 'the field now has relatively distinct theoretical and empirical literatures based around the selection of subjects on depression, anxiety and self-esteem measures'. This overlooks the fact that a subject high on one is also likely to be high on the others, and that a model such as that developed by Strack et al (1985) to account for the effects of depression on performance could equally be viewed as a model of the effects of anxiety, even though anxiety played no part in the selection of the subjects used to test the model.

Zarantonello et al (1984) have also pointed out the need for researchers to use measures of both anxiety and depression when investigating psychological performance in these conditions as this will lead to a 'greater specificity of prediction and results'. They

investigated whether any differential effects of anxiety and depression could be discerned in the anagram performance, ratings of cognitive interference and subjective evaluation of performance of students selected on the basis of their scores on the BDI and/or State-Trait Anxiety Inventory. Students high in both depression and anxiety and those with high anxiety scores were impaired on the anagram task, rated themselves as experiencing more cognitive interference and were more negative in their evaluations of performance than students low in depression and anxiety. The results of a Quasi-F procedure indicated that these results were significantly related to the anxiety factor common to both the depressed-anxious and anxious groups.

Watts and Sharrock (1985) tried to determine whether depression or anxiety was the main determinant of concentrational lapses during a reading task in clinically depressed patients. Anxiety correlated at 0.46 with the number of lapses, but was no longer significantly related to it when either the severity of depression and the endogeneity of depression were partialled out. Both the severity of depression and endogeneity were initially significantly correlated with concentration (r=.46 and .51): the severity of depression was no longer significantly related once the effects of anxiety were partialled out, although endogeneity was related. They concluded that it can only be safely concluded that all three variables operate together in identifying a symptom dimension which is associated with concentrational problems.

Considering the effects of both anxiety and depression on performance is likely to lead to insights into the causes of memory impairment in depressed and anxious individuals. It may, for instance, become clear that the impairments in the two groups have the same cause and are related to something common to both anxiety and depression. For instance, Zarantonello et al (1984) suggested that cognitive interference due to a negative evaluation of self and performance may be to blame because such interference has been postulated in both anxious (Eysenck, 1979; Mahoney, 1980) and depressed (Beck, 1967) individuals. It may, however, be that impairments are most closely related to symptoms of depression, or to symptoms of anxiety. This would influence the types of explanations put forward to account for it.

The two studies which have looked at the relative effects of depression and anxiety on cognition have been concerned with problem solving (Zarantonello et al, 1984) and concentration problems (Watts and Sharrock, 1985) respectively. There have apparently been no studies which have tried to differentiate between the effects of anxiety and depression on the memory of subjects diagnosed as being depressed or anxious. This chapter therefore looks at whether the memory deficits observed in Chapters Four and Five are more closely related to depression or anxiety.

The CATEGO syndromes of depression and anxiety both occurred in each of the three diagnostic groups in this study (Section 3.3.3): general anxiety was found in 90% of the retarded depressed, 69% of the neurotic depressed and all the anxious subjects, while depressed mood was found in 66% of the anxious and all the depressed subjects. They therefore provide a suitable subject group for the exploration of the relative effects of anxiety and depression. The control subjects were also included in the analysis as this gives the wide range of scores on depression and anxiety rating scales necessary to adequately explore the relationship between anxiety, depression and performance. Regression analysis was used to investigate this relationship on the memory tests used in the earlier chapters (Chapters Four and Five).

6.2 METHOD

This section will give a brief description of the subjects, materials and methods used in this study. Full details are given in Chapters Two and Three. As in the previous chapter (Chapter Five) the regression analysis was restricted to those variables which differed significantly between the subject groups (Chapter Four) and consequently where anxiety and depression might be expected to be related to memory performance.

6.2.1 Subjects

The subjects used in this analysis were the same as those used in Chapters Four and Five, with the addition of four subjects excluded from the preceding analysis because they had not completed a Present State Examination and therefore could not be categorised into a Catego class

(Wing, Cooper and Sartorius, 1974). They were included because this analysis does not require the subjects to be categorised into Catego classes.

Subjects were treated as one group, which gave a range of scores on the depression and anxiety scales from very low (control subjects) to high (the most severely depressed and anxious subjects). This allowed the correlation of these with performance to be assessed over a wide range of scores.

6.2.2 Materials

The Irritability-Depression-Anxiety (I.D.A) Scale (Snaith et al, 1978) was used to assess how depressed and anxious the subjects were at the time of testing. This is designed to measure depression and anxiety as separate, although often correlated, syndromes which may occur to varying degrees in people with a variety of diagnoses. It is described in detail in Section 2.2.4.

The I.D.A was completed at each testing session. However, the scores for the same subjects at different testing sessions were quite highly correlated (depression subscale, $N=45$, $r=.81$, $p<0.01$; anxiety subscale, $N=45$, $r=.76$, $p<0.01$). Their mean scores on each subscale were therefore calculated and used as predictor variables. Scores on the depression subscale ranged from 2 to 12.5 with a mean of 7.3, whilst those on the anxiety subscale ranged from 2 to 14.5 with a mean of 9.0. The two scales were significantly correlated ($r=.62$, $N=45$, $p<0.01$).

The control subjects were selected because they did not have any form of psychiatric disorder, as assessed by the Present State Examination '10 Questions' (Cooper and Mackenzie, 1981). It would therefore be expected that their depression and anxiety scores would be significantly lower than those of the depressed and anxious groups. Table 6.1 gives the mean scores of the groups on the two subscales. As the Bartlett Box test for the equality of variance showed there were significant differences in variance between the groups on the anxiety subscale and a trend for significant differences on the depression subscale (depression subscale, $F=2.3$, $p>0.05$, <0.1; anxiety subscale, $F=3.2$, $p<0.05$) the Kruskal-Wallis

test was used to see if there were significant differences between the groups on these variables.

Table 6.1 SCORES OF THE RETARDED DEPRESSED, NEUROTIC DEPRESSED, ANXIOUS AND CONTROL SUBJECTS ON THE DEPRESSION AND ANXIETY SUBSCALES OF THE I.D.A (Mean and S.D).

Group	N	Anxiety subscale	Depression subscale
Anxious	7	9.6 (4.3)	6.5 (2.8)
Retarded depressed	10	10.4 (1.6)	9.7 (1.6)
Neurotic depressed	13	10.0 (2.8)	8.5 (2.5)
Controls	11	5.9 (1.8)	3.5 (1.2)

There were significant differences between the groups on both subscales (anxiety subscale, N=41, χ^2=15.0, p<0.01; depression subscale, N=41, χ^2=24.2, p<0.0001). Multiple comparisons were then made using the Mann-Whitney U test (Section 4.2.3) to test whether the three depressed and anxious groups taken together differed significantly from the control subjects. They did so on both subscales (depression subscale, U=12, U'=78, p<0.0001); anxiety subscale, U=35, U'=101, p<0.0001) (Table 6.1). This supports the validity of the depression and anxiety subscales: groups selected because they were diagnosed as having depression or anxiety differ as expected from those selected because they did not have these conditions.

The control subjects were not entirely free from symptoms of anxiety and depression. This was expected as although the scales measure symptoms which are more severe in people who have developed clinically significant disorders, they also occur to a lesser degree in many people whom psychiatrists would not consider to have a psychiatric condition. Snaith et al (1978) suggests that scores of 4 to 6 on the depression scale and 6 to 8 on the anxiety subscale fall into a borderline zone, above which psychiatrists were likely to consider someone to be

clinically ill, and below which they would regard the subject as normal. The mean score of the controls on the depression scale was 3.5, and on the anxiety scale was 5.9. In both cases the mean scores of subjects selected because they were found to be free of a clinically significant disorder on the '10 Questions' (Cooper and Mackenzie, 1981) fall within the normal ranges on these subscales, whilst those of the clinically depressed and anxious subjects are above the cut-off points for psychiatric disorder. These results support the use of these scales as predictor variables in this study.

6.2.3 Analysis

Regression analysis was used to explore the relationship between depression, anxiety and memory impairment. It was carried out in a pre-determined order for each dependent variable. First, anxiety was entered into an equation by itself and depression was added to it. Depression was then entered in an equation by itself, and anxiety added to it.

As anxiety and depression are correlated if one of them predicts performance, the other will also when used alone in the equation. However, when added to the equation containing the first variable it would not cause a significant increase in the amount of variance explained, as measured by an increase in R^2: the shared variance would be attributed to the variable already in the equation, and since the other variable has no independent role it would not increase the predictive power of the equation. However, if neither variable increased R^2 when added to the equation containing the other, but both did separately, it would suggest that something common to both variables was related to performance.

6.3 RESULTS

Table 6.2 gives the amount of variance explained in scores on the Immediate Memory and Speed of Learning variables at each stage of the regression procedure and the statistical significance of the increase in R^2 caused by the predictor variables.

It can be seen that on two of the six immediate memory variables (Free Recall - total, Serial learning - immediate recall) both depression

and anxiety explained a significant amount of variance when entered into the regression equation by themselves. However, the increase in explained variance caused by adding anxiety to the equation containing depression was not statistically significant; this was also true when depression was added to the equation containing anxiety. It must therefore be concluded that something common to both variables was related to recall on these tests. The scores on d' - Trial One showed the same pattern, with little difference between the amount of variance explained by depression and anxiety when alone in the equation (.06 versus .07). However in this case neither depression nor anxiety was significantly related to performance at any stage.

Depression explained a significant amount of the variance in Digit Span Backwards when alone in the equation, whilst anxiety did not. Adding depression to the regression containing anxiety caused a significant increase in the amount of variance explained but the reverse was not true. Depression was therefore associated with deficits on this test, not anxiety. There was a trend for depression to explain a significant amount of the variance in scores on Paired Associate - immediate recall both when entered alone in the equation and when added to the equation containing anxiety ($p>0.05$, <0.1). The results on Prose Passage One - immediate recall showed the same pattern of a very low R^2 associated with anxiety by itself (.02) compared to that explained by depression alone (.08) but the contribution of depression did not reach significance on this test. It is concluded that depression was probably the main determinant of performance on these tests, although it should be noted that the evidence is only suggestive.

The results on the two variables measuring speed of learning new material (Serial Learning - speed of learning, Paired Associate - speed of learning) were clearer. Depression explained a significant amount of the variance in both variables when placed in the regression equation by itself whilst anxiety was not significantly related to performance on these tests. Depression also caused a significant increase in the amount of variance explained when added to the equation containing anxiety whilst the reverse was not true. Depression was therefore the main determinant of performance on these variables.

TABLE 6.2 IMMEDIATE MEMORY AND SPEED OF LEARNING: THE AMOUNT OF VARIANCE EXPLAINED (R^2) WHEN DEPRESSION ENTERS THE REGRESSION EQUATION BEFORE ANXIETY, AND WHEN THE ORDER IS REVERSED.

VARIABLE	R^2 EXPLAINED BY: DEPRESSION + ANXIETY		R^2 EXPLAINED BY: ANXIETY + DEPRESSION		RESIDUAL D.F
d' - Trial One	.06 .08		.07 .08		37
Free Recall - total	.17 .18	*	.13 .18	*	42
Serial Learning - immediate recall	.16 .22	**	.19 .22	**	41
Prose Passage One - immediate recall	.08 .09		.02 .09		38
Paired Associate - immediate recall	.08 .09		.01 .09		42
Digit Span - Backwards	.10 .11	*	.01 .10	*	40
Serial Learning - speed of learning	.21 .23	**	.15 .23	*	41
Paired Associate - speed of learning	.19 .19	**	.05 .19	**	42

Significance of change in R^2, * $p<0.05$, ** $p<0.01$

Table 6.3 shows the results for the variables measuring retention (these could be more accurately described as measuring recall after a delay as the variables measuring the amount forgotten after a delay were excluded from this section because they did not differ significantly between the groups (Section 4.3.3)). Depression explained a significant amount of the variance in Prose Passage One - delayed recall when entered alone in the equation; the same was not true of anxiety. Neither depression nor anxiety caused a significant increase in the amount of explained variance when added to the equation containing the other variable. This suggests that depression, and not anxiety, was affecting performance on this test. Both depression and anxiety were significantly related to performance on Prose Passage Two - delayed recall when alone in the equation, but neither was when added to the equation containing the other. Something common to both depression and anxiety seems to have been the main determinant of performance on this test. The results for the third variable in this section (Paired Associate - Trial Five) were somewhat different: depression, but not anxiety, was significantly related to performance when entered alone in the equation, and depression caused a significant increase in the amount of explained variance when added to the equation containing anxiety. Thus depression and not anxiety was related to performance on this test.

Scores on the Past Events multi-choice questionnaire were significantly predicted by both anxiety and depression when placed alone in the regression equation (Table 6.3), but neither caused a significant increase in the amount of explained variance when added to the equation containing the other. Thus it must be concluded that something common to both anxiety and depression was the main determinant of performance on this test.

TABLE 6.3 RETENTION AND MEMORY FOR PAST PUBLIC EVENTS: AMOUNT OF VARIANCE EXPLAINED (R^2) WHEN DEPRESSION ENTERS THE REGRESSION EQUATION BEFORE ANXIETY, AND WHEN THE ORDER IS REVERSED.

VARIANCE	R^2 EXPLAINED BY: DEPRESSION + ANXIETY		R^2 EXPLAINED BY: ANXIETY + DEPRESSION		RESIDUAL D.F.
Prose Passage One - delayed recall	.09 .10	*	.06 .10		39
Prose Passage Two - delayed recall	.17 .20	**	.14 .20	**	40
Paired Associate - Trial Five[a]	.16 .16	**	.08 .16	*	41
Past Events multi-choice questionnaire	.11 .16	*	.13 .16	*	40

[a] this variable was squared to overcome heterogenity of variance

Significance of change in r^2, * $p<0.05$ ** $p<0.01$

In summary, depression emerged as the main determinant of performance on Digit Span Backwards, Serial learning - speed of learning, Paired Associate - speed of learning, Prose Passage One - delayed recall and Paired Associate - Trial Five. In addition there was a suggestion that this was also true of performance on Paired Associate - immediate recall and Prose Passage One - immediate recall: although the contribution of depression did not reach statistical significance on these variables, the pattern of results was the same as that of the previous four variables. On four of the remaining variables (Serial Learning - immediate recall, Free Recall - total, Prose Passage Two - delayed recall and Past Events multi-choice questionnaire) both depression and anxiety were significantly related to performance when alone in the equation, although neither significantly increased the amount of variance explained when added to the equation containing the other. It is concluded that something common to both anxiety and depression was related to performance on these variables. The same conclusion was drawn about performance on d' - Trial One, although as these results were not statistically significant they must be regarded with some caution.

6.4 DISCUSSION

Regression analysis was used in this chapter to investigate the relative effects of depression and anxiety on memory. The results showed that it was not possible to distinguish between the effects of depression and anxiety on two of the immediate memory variables (Free Recall - total, Serial Learning - immediate recall) and suggested that this might also be the case on d' - Trial One. It was therefore concluded that something common to both depression and anxiety was the main determinant of performance on these variables. The results of the remaining three immediate memory variables contrasted with this: on Digit Span Backwards depression was a significant predictor of performance both when alone in the regression equation and when added to the equation containing anxiety: the reverse was not true. The results of Paired Associate - immediate recall and Prose Passage One - immediate recall showed the same pattern of results, but the effects of depression were not statistically significant on these variables. It is concluded that depression, rather than anxiety, was related to performance on these variables.

The three variables on which performance was found to be determined by something common to anxiety and depression were the same three variables on which both the retarded and neurotic depressed patients were found to significantly impaired (Section 4.3.1), and which were presumed to be the more difficult immediate memory tasks (Section 4.4.1). In contrast, the three remaining variables were the ones on which only the more severely depressed retarded depressed patients were significantly impaired, and which were presumed to be the easier tasks. This suggests that depression affects performance on easier tasks, whilst some aspect of both anxiety and depression affects performance on more difficult ones.

Depression proved to be the main determinant of performance on both speed of learning variables. Depression was also most closely related to performance on two of the variables measuring recall after a delay: Paired Associate - Trial Five and Prose Passage One - delayed recall. There was evidence on Prose Passage Two - delayed recall that it was not possible to distinguish between the effects of depression and anxiety and that therefore some aspect of both was related to performance on these variables. The same conclusion was drawn from results on the Past Events multi-choice questionnaire.

These results, particularly those for the variables measuring immediate memory, raise the interesting possibility that some aspect common to both depression and anxiety was determining performance on the more difficult tests, while depression was the main determinant of performance on easier ones. This would be consistent with the hypothesis that processing capacity is reduced in depression (probably due to cognitive interference) and that this leads to performance deficits (Hasher and Zacks, 1979). It could also be consistent with Eysenck's (1979, 1982) theory of memory in anxiety, which suggests that processing capacity is also reduced in anxiety (again due to cognitive interference) but that, provided the task does not make too many demands on memory, anxious individuals are able to overcome the effects of reduced capacity by increasing the amount of effort put into the task and consequently the amount of available processing capacity.

If both anxious and depressed individuals have reduced processing capacity due to cognitive interference, then it would be true that something common to both (cognitive interference) was determining performance. In addition, if anxious individuals overcome this deficit by increasing the amount of effort they put into the task then anxiety would not be related to performance on tasks unless the tasks require more processing capacity than they have available, even after increasing effort. There is no reason to hypothesise that depressed people would be motivated to try and overcome the deficit: the evidence rather suggests that depression is related to a reduction in motivation and effort (Section 1.8.1). The depressed patients would therefore be impaired on tasks demanding more processing capacity than they have left after processing task-irrelevant demands (cognitive interference), whilst anxious individuals would be able to increase the available capacity by exerting more effort (Section 4.1.3) and therefore would not show impairment until the task requirements became greater. This would be consistent with these results: on easier tasks depression is related to impairment but anxiety is not, presumably because patients high in anxiety are able to overcome the effects of a reduction in processing capacity by increasing effort. As the tasks become more difficult, they are no longer able to do this and therefore show impairments for the same reason as patients high in depression: a reduction in processing capacity caused by interference from worry and, as Zarantonello et al (1984) suggested, negative evaluations of self and performance. Thus performance on easier tasks is just related to depression but performance on more difficult tasks is impaired in both depression and anxiety due to the cognitive interference common to both.

This hypothesis could explain the results of the effects of depression and anxiety on the immediate memory variables, but it is less certain that it explains the results on the remaining variables. Performance on the speed of learning variables and two of the variables measuring memory after a delay (Prose Passage One - delayed recall andPaired Associate - Trial Five) was determined by depression and therefore, according to the above theory, these should be comparatively easy tasks which make limited demands on memory. It is not apparent that this is the case because the Serial Learning test used unconnected words which on the first trial were considered to make it a demanding task

(Section 4.4.1). In addition some of the remaining tests which were affected by a factor common to both depression and anxiety were prose passage recall tests: these have been considered to be easier tasks because the material is structured and has a degree of redundancy (Section 2.2.1). It is difficult to know how difficult a test is and how much effort and processing capacity it requires: this has not been well-defined by either Eysenck (1979, 1982) or Hasher and Zacks (1979) and it is consequently possible that the delayed recall of the prose passages was more difficult than, for example, the speed of learning tests. There is, however, no good evidence that this is the case.

The hypothesis suggested here is that, although processing capacity is reduced in both depression and anxiety, only individuals high in depression will show deficits on relatively undemanding tasks because anxious individuals are able to overcome the reduction in processing capacity by increasing effort and therefore increasing the available processing capacity. On these types of tasks, therefore, only depression will be significantly related to a reduction in performance. On more demanding tasks, however, the anxious individuals will be unable to increase effort sufficiently to overcome the reduction in processing capacity caused by cognitive interference. Performance will therefore be impaired in both anxiety and depression and by something common to both: cognitive interference leading to a reduction in processing capacity. This hypothesis brings together the work of Hasher and Zacks (1979) on depression and Eysenck (1979; 1982) on anxiety. It remains speculative but seems to have potential in furthering the understanding of memory in depression and anxiety.

One reason why this hypothesis remains speculative (even though the results for the immediate memory variables are supportive) is that caution may be needed in interpreting these results because the effects of psychotropic medication and E.C.T were not taken into consideration before looking at the relative effects of depression and anxiety. It was decided not to do so in this chapter because it was expected that patients high in depression or anxiety would receive different medication, or be more likely to receive medication or E.C.T, than less severely ill patients. If this was so then controlling for the effects of the treatment

variables (Section 5.2.3) would have also controlled for severity and consequently have made it difficult, if not impossible, to explore the relationship between depression, anxiety and performance. In addition the amount of variance explained by depression and anxiety was quite small (less than .23 in all cases). Thus the effect of any common variance being attributed to the treatment variables would again have made it difficult to look at the relative effects of depression and anxiety because neither would be likely to explain a significant amount of the variance. The study reported in this chapter therefore needs to be replicated on a sample of patients not taking any psychotropic medications in order to verify the results. However, the results of Chapter Six suggested only one variable (Prose Passage One - immediate recall) where the results need to be treated with particular caution because it was concluded that the significant differences between the groups observed in Chapter Four may have been due to the treatment the patients were receiving, rather than to their psychiatric condition.

As already noted, the amount of variance explained by the combination of depression and anxiety was quite small on all variables, and on five of the twelve it was ten per cent or less. In contrast the amount of variance explained by group membership on the same variables was never below twenty per cent (Tables 5.2 and 5.4). One reason for the limited relationship between the depression and anxiety scales and performance may have been that the self-rated scales used were not sufficiently sensitive and therefore did not distinguish adequately between different levels of anxiety and depression. The IDA depression scale was found by Kearns et al (1982) to be reasonably good at differentiating between severe, moderate and mild levels of depression in depressed in-patients, but was poor at differentiating between very severe and severe depression. It was also much less good at differentiating between the categories of severity than observer rated scales such as the Hamilton Depression Rating Scale and Montgomery Asberg Depression Rating Scale. Observer rating scales are considered to be more appropriate than self-rating scales in severe depression because psychiatrists have more experience of depression on which to base their decision about severity (Prusoff, Klerman and Paykel, 1972). Self-rating scales have also been noted to be susceptible to response sets such as social desirability (Boyle, 1985), 'faking bad' to attract attention and 'faking good' to

appear mentally healthy (Stravakaki and Vargo, 1986). These points all suggest that self-rating scales may be less sensitive to different levels of depression and, by inference, anxiety than observer rated scales.

This may explain why Van den Hout and Griez (1984) found limited relationships between P.S.E depression syndrome scores derived from the Dutch version of the P.S.E, and Zung depression rating scale scores in neurotic depressed and anxious patients. The correlations between depression scale scores and the special features of depression syndrome (Section 3.2.2) was nonsignificant ($r=.25$), as was that between 'other symptoms of depression' and the depression scale scores ($r=.38$). The 'simple depression' syndrome was however significantly related to depression scale scores ($r=0.59$, $p<0.001$). The relationships between the anxiety syndromes and the Wolpe and Lang Fear Survey Schedule were also assessed. The correlation of the fear schedule and generalised anxiety was .24, as was that with situational anxiety: both were non-significant. In addition to reflecting the general limitations of self-rating scales, the correlations with the Zung Depression Scale may be particularly low because this scale is limited in scope and has been said to be an inadequate measure of depression which should no longer be used (Boyle, 1985). These results, together with the general criticisms of self-rating scales, suggest that better understanding of the relationship between depression, anxiety and memory might come from a study using observer-rated scales as these are likely to be more sensitive to differences in levels of depression and anxiety.

In conclusion, this investigation of the relative contribution of depression and anxiety to memory impairment has to be regarded as preliminary because subjects on psychotropic drugs and with a history of E.C.T were included. This means that the results obtained may have been due to the effects of these treatments rather than to depression and anxiety. In addition self-rated scales of depression and anxiety were used which may have been less sensitive than observer-rated scales. However, the results are largely consistent with the hypothesis that processing capacity is reduced in both depression, as suggested by Hasher and Zacks (1979), and in anxiety, as suggested by Eysenck (1979, 1982). On difficult tasks, subjects high in depression and those high in anxiety will be impaired because of this reduction in processing capacity, which

may be due to cognitive interference. On less difficult tasks, anxious individuals are able to overcome their lack of processing capacity by increasing the effort they put into the task, which consequently increases the available processing capacity and enables them to perform normally on such tasks. Depressed subjects however lack motivation, do not increase effort to overcome the effects of the reduction in processing capacity and thus are more impaired on less demanding tasks than subjects high in anxiety. This is consistent with the fact that performance on what were judged to be the easier tasks was determined solely by depression, while performance on more difficult tasks was determined by something common to anxiety and depression; presumably reduced processing capacity caused by cognitive interference from worry and negative thoughts. This hypothesis is speculative, but it does provide a framework for integrating some of the research on memory in depression with that on memory in anxiety.

The next chapter (Chapter Seven) moves on to the second aspect of memory addressed in this thesis: the reports of depressed people of memory problems in everyday life and the relationship of these reports to performance on laboratory memory tests. The relative contributions of depression and anxiety to the reports of memory problems is also investigated.

COMPLAINTS ABOUT MEMORY PERFORMANCE IN EVERYDAY LIFE

7.1 INTRODUCTION

This chapter is concerned with the second aspect of memory investigated in this thesis; the reports of depressed patients of memory problems in everyday life and the relationship of these reports to performance on the laboratory memory tests. It addresses three questions:

1) Do depressed and anxious patients complain of poor memories?

2) Are the subjects' self-assessments of memory related to depression or to anxiety?

3) Are the subjects' self-assessments of memory in everyday life related to their performance on laboratory memory tests?

Each of these questions will now be considered in turn.

7.1.1 Do Depressed and Anxious Patients Complain of Poor Memories?

There is evidence that depressed subjects frequently complain of poor memories. For instance Kahn et al (1975) found that subjects with normal brain function and high levels of depression, according to the Hamilton Depression Rating Scale (HDRS; Hamilton, 1960) complained significantly more about their memories than subjects with low levels of depression, although the scores of the two groups on memory tests were approximately equal. Popkin et al (1982) compared elderly depressed out-patients with a control group who were not depressed and found that although the groups did equally well on recall and recognition memory tests the depressed patients complained significantly more on three memory complaint measures. Plotkin, Mintz and Jarvik (1985) found that elderly depressed patients had lower scores on a subjective memory complaints scale after treatment with psychotropic medication or psychotherapy than before treatment, presumably due to a reduction in depression. O'Hara et al (1986) compared the memory complaints and memory performance of a group of over 65 year olds fulfilling the Research Diagnositic Criteria (RDC, Spitzer, Endicott and Robins, 1978) for major depression; a group with

similar scores on the CES-D (Radloff, 1977) but not fulfilling these criteria; and non-depressed controls. They found that both depressed groups had significantly higher scores on three memory complaint scales than the non-depressed controls, although the groups did not differ on a free recall task. In addition to these research findings it is also clear that many clinicians have found that depressed patients complain of poor memories (McAllister, 1981).

There is less information on whether anxious people consider themselves to have poor memories. Anxiety has been found to be related to scores on the Cognitive Failures Questionnaire (CFQ), with high levels of anxiety being associated with high scores on the CFQ. For instance Broadbent et al (1982) reported a significant correlation of 0.31 between CFQ scores and State-Trait Anxiety Inventory scores (STAI: Spielberger, Gorsuch and Lushene, 1970) in a sample of student nurses. Hood, MacLachlan and Fisher (1987) found a correlation of 0.35 between the scores of 342 students on the CFQ and their scores on the anxiety scale of the Middlesex Hospital Questionnaire. Martin and Jones (1984) found a correlation of 0.5 between the CFQ scores of twenty students and their scores on the neurotic scale of the Eysenck Personality Questionnaire. Thus, there is evidence that high levels of anxiety in non-patient populations are related to high levels of memory complaints.

However, little is known about whether patients with a diagnosed anxiety state complain of memory impairment and, if so, to what extent. Broadbent et al (1982) report the CFQ scores of a sample of 34 'neurotic' in-patients and found that their mean score of 44.8 was within the limits for other subject groups. In addition there were no significant correlations in this sample between CFQ scores and measures of anxiety. There appear to be no other studies which have looked at the memory complaints of patients with an anxiety state. It is therefore not clear whether or not anxious patients will have high levels of memory complaints.

The first aim of the study described in this chapter was therefore to attempt to replicate the finding of frequent memory complaints by depressed people, and to investigate whether anxious people also have high levels of complaints about their memory performance.

7.1.2 Are the Subjects' Self-assessments of Memory Related to Depression or to Anxiety?

The second question addressed in this chapter is whether the subjects' self-assessments of memory are related to their self-reported depression or anxiety levels.

Several studies have found that memory complaints are related to depression, with high levels of complaints being associated with high levels of depression. For instance Kahn et al (1975) found in a sample of psychiatric out-patients and their relatives that depression, as measured by the HDRS, was significantly related to the degree of memory complaint, although depression was not significantly related to performance on a variety of memory tests. As described above (Section 7.1.2) O'Hara et al (1986) compared memory complaints and memory performance in two groups of depressed elderly people and a group of non-depressed people, all recruited from a community sample. Depression was significantly correlated with scales comparing their current memory performance with their memory performance at age thirty, and with eight items from the Metamemory Questionnaire (Zelinski, Gileviski and Thompson, 1980), but not with a scale comparing memory with that of other people of the same age. Zarit, Gallagher and Kramer (1981) looked at the effects of memory training programmes on memory complaints in elderly people and found that a reduction in depression after training was significantly related to a reduction in memory complaints. Pettinati and Rosenberg (1984) used the Squire Self-rating Scale of Memory Function (Squire, Wetzel and Slater, 1979) which asks depressed patients to compare their current performance with that before the onset of illness; they found that both before and after treatment with E.C.T, memory complaints were significantly correlated with HDRS and Beck Depression Inventory (BDI; Beck et al, 1961) scores. Plotkin, Mintz and Jarvik (1985) found a significant correlation between the reduction in BDI scores and the reduction in memory complaints following treatment in a group of depressed patients. Thus, depression has been shown to be related to the level of memory complaints both in clinically depressed patients and in individuals with symptoms of depression who have not been diagnosed as clinically depressed.

As reported above (Section 7.1.2) anxiety has also been shown to be related to memory complaints; as with depression, high levels of anxiety are related to high levels of memory complaints. Some studies have reported correlations with both anxiety and depression. For instance Hood, MacLachlan and Fisher (1987) found tau coefficients of 0.16 between the depression scale of the MHQ and the CFQ, and of 0.35 between scores on the anxiety scale and the CFQ. Three hundred and forty-two students took part in this study and both correlations were highly significant. These authors combined the tau coefficients for the six groups of subjects used by Broadbent et al (1982) creating an overall group size of 428. They obtained tau coefficients of 0.34 and 0.31 between the depression and anxiety scales of the MHQ and the CFQ; again these were both highly significant. West, Boatwright and Schleser (1984) found significant correlations between BDI scores, STAI scores and scores on metamemory scales in a group of over 65 year olds, while Shelton and Parsons (1987) obtained significant correlations between BDI, STAI and 'Patients' Own Assessment of Functioning Inventory' scores both in a group of chronic alcoholics and in a group of control subjects.

The correlations between anxiety, depression and self-assessments of memory led Chelune, Heaton and Lehman (1986) to conclude that 'patients' subjective perceptions of their cognitive, memory and language functioning are more related to their psychiatric status than to their actual abilities'. The studies reviewed here have predominantly used people who did not have clinically significant levels of anxiety and depression, and therefore indicate that psychiatric patients are not the only people whose perceptions of their abilities are coloured by their affective status.

It might be speculated that the relationships between depression, anxiety and memory complaints are the consequence of the detrimental effects of depression and anxiety on memory (Sections 1.4 and 4.1.3). However, Kahn et al (1975) found that depression was not significantly related to performance although memory complaints were significantly related to depression. Other studies have not found a significant difference in memory performance between groups of subjects with high levels of both depression and memory complaints, and groups with low levels of depression and memory complaints (O'Hara et al, 1986; Popkin et

al, 1982). These results indicate that depressed and anxious people may complain about their memories even when they are not impaired on laboratory memory tests, presumably as part of their general tendency to be negative about their performance (Beck, 1967, 1976). Depression and anxiety may therefore be two of the reasons why self-assessments of memory are not strongly related to performance on laboratory memory tests (Section 7.1.4).

However, it is not clear from these studies whether it is depression, anxiety or something common to both that is most related to self-assessment of memory. Depression and anxiety frequently co-exist and consequently people with high levels of one are also likely to have high levels of the other (Section 6.1). Levels of anxiety need to be taken into consideration when investigating the effects of depression on self-assessments of memory, and vice versa. If this is not done then studies designed to examine the effect of one of these variables may in fact be picking up the effect of the other. None of the studies reported above which have looked at the relationships between both depression and anxiety and self-assessments of memory have taken the effects of depression into consideration when looking at anxiety, or vice versa. It is therefore not clear whether depression, anxiety or something common to both affects peoples' self-assessments of their memory performance. One of the aims of this study was therefore to investigate whether self-assessments of memory are more closely related to depression or to anxiety.

7.1.3 Are The Subjects' Self-assessments of Memory Performance in Everyday Life Related to Their Performance on Laboratory Memory Tests?

There has been a growing awareness in the past fifteen to twenty years that memory processes in the psychology laboratory are rather different than those needed for functioning in everyday life (Baddeley and Wilkins, 1984). As Bennett-Levy and Powell (1980) have observed, laboratory memory tests use paradigms such as paired associate learning which are not encountered outside the laboratory; they tend to assess only 'pure' memory, for instance spatial or verbal memory; they usually measure only relatively short-term memory; the information involved is often trivial and contextual cues are minimal. In contrast in everyday

life aids to memory such as diaries or reminders from other people are often available; the information often cuts across all modalities rather than being restricted just to, for instance, spatial memory; there are many contextual cues and the information is usually pertinent to the individual. In addition in laboratory memory tests the subject is prompted to remember by the investigator, while in everyday life people normally have to remember things without being reminded (Wilkins, 1986): the ability to remember things without being prompted has received little experimental research and is not included in laboratory test batteries. Performance on laboratory memory tests does not, therefore, necessarily reflect memory performance in everyday life.

Most research on memory has consisted of experimental studies in the psychology laboratory. As Neisser (1978) noted, psychologists have not traditionally looked at memory performance in natural settings or at practical memory problems: they have been concerned with working towards a general theory of memory and a scientific understanding of its underlying mechanisms and have therefore concentrated on carefully controlled laboratory experiments. Together with other researchers (Gruneberg, Morris and Sykes, 1978), Neisser has argued that the theories and concepts developed in the laboratory need to be exposed to the more bracing conditions found outside and that, in addition, some effort should be made to investigate aspects of memory which are important to the 'man in the street'.

Interest amongst cognitive psychologists in memory in everyday life has therefore grown. Baddeley and Wilkins (1984) have reviewed the advantages of extending memory research beyond the confines of the laboratory. They argue that to do so will, amongst other things, establish the generality of theories generated in the laboratory, provide a means of checking theories before they are applied to everyday problems and will provide a source of new phenomena to study. This move to extend memory research beyond the laboratory has led to researchers looking at topics previously ignored by psychologists, such as absent-mindedness (Wakeford et al, 1980; Reason and Lucas, 1984) and the ability to remember to do something (Wilkins and Baddeley, 1978; Wilkins, 1986). It has also led them to look at memory in naturalistic settings (Baddeley, 1981a; Chapter Eight). It is clear that there is increased interest in

everyday memory amongst psychologists and that it provides a fruitful area of new ideas and concepts.

The differences between the types of memory measured in the laboratory and those used in everyday life has aroused interest in everyday memory not only amongst cognitive psychologists but also amongst psychologists with clinical interests. The lack of agreement between laboratory tests and memory in everyday life causes problems for those trying to investigate the implications of memory failure for people with, for instance, brain damage caused by head-injury (Sunderland, Harris and Gleave, 1984; Kapur and Pearson, 1983), memory deficits induced by E.C.T (Pettinati and Rosenberg, 1984) or deficits by alcoholism (Shelton and Parsons, 1987). Some way of finding out about memory performance in everyday life is therefore needed in order to identify problem areas and to find out the true capabilities of such subjects. Interest in everyday memory has therefore grown amongst psychologists with clinical interests as well as amongst experimental cognitive psychologists.

One way of investigating everyday memory is to try and replicate real life in the laboratory and to design memory tests which closely resemble the tasks people have to do in everyday life. For instance, Wilkins and Baddeley (1978) designed a task which resembled the everyday activity of remembering to take pills while Becker et al (1983) used a test which resembled the everyday task of associating names and faces. For clinicians, this approach is subject to the limitation that the frequency of memory failures in everyday life will depend on the demands made on memory by the person's environment and life-style. Consequently finding out in the laboratory what type of tasks people have difficulty with will not necessarily give a good idea of the degree of impairment they experience in their daily lives as this will depend on, for instance, how often they have to do such tasks. Another approach is to look at memory performance in an everyday situation: an example of this is presented in Chapter Eight. Finally, the subjects can be asked about their own performance in the hope that this will give a good indication of their memory performance in their daily lives and possibly reveal areas for rehabilitative intervention (Bennett-Levy and Powell, 1980).

The suggestion that an improved understanding of memory performance in everyday life would come from asking people about their own performance has led to the development of a number of what Herrman (1984) has labelled 'metamemory' questionnaires: questionnaires which ask people to indicate how well they recognise and recall knowledge or events, and which assess their beliefs about their memory performance. Herrman (1984) reviewed eighteen different questionnaires which between them cover six different aspects of memory performance: how frequently the respondents forget; how clearly they remember; how memory has changed; how easily they learn; what memory strategies they use; and how they feel about their memory performance. The questionnaires cover a wide range of memory tasks, including memory for common events and tasks, memory for specific episodes, memory for skills and the inclination for perceptual errors. They are, therefore, quite a diverse collection of questionnaires.

These questionnaires have been used with a variety of groups but their validity, as measured by the relationship between them and scores on laboratory memory tests, has often been found to be quite low. For instance Broadbent et al (1982) found no significant correlations between the CFQ scores of twenty subjects and scores on a variety of memory tests, although a study which had a larger sample size and included depressed patients did find a significant correlation between CFQ scores and performance on one of the tests (Weeks, reported by Broadbent et al, 1982).

Shelton and Parsons (1987) used the 'Patients Assessment of Own Functioning Inventory' (PAF) to get the perceptions of alcoholics of their psychological and neuropsychological functioning in everyday life. The PAF scales were not significantly correlated with neuropsychological performance even though the alcoholics had deficits in memory and in overall neuropsychological performance. Bennett-Levy and Powell (1980) found a weak relationship between good memory on a battery of memory tests in a group of alcoholics and controls and reports of good memory on the Subjective Memory Questionnaire. The relationship was strongest on a Face-Name task ($r=0.37$), perhaps because this combined verbal and spatial material and was therefore more like everyday life than the other tasks.

West, Boatwright and Schleser (1984) asked a group of over 65 year olds who had volunteered for a memory training program to complete a metamemory questionnaire and to do digit span and free recall tests. There was no relationship between metamemory and memory performance. Sunderland et al (1986) also investigated the relationship between metamemory and memory performance in elderly subjects. They gave 60 subjects a version of the Everyday Memory Questionnaire (Section 2.2.3), a checklist of memory failures to complete each day and a laboratory test battery. A close relative of the subject, usually the spouse, also completed a questionnaire about the subject's memory. The immediate recall of a story correlated significantly with the patients' scores on the questionnaire and checklist, while the delayed recall version of this test correlated significantly with scores on these and on the relatives' questionnaire. In addition, scores on the checklist were significantly related to the percentage forgotten between the two trials of the story recall test, and to scores on a word recognition test. Out of thirty correlations between the self- and spouse- assessments of memory and scores on the memory tests, only seven were statistically significant and in all cases the correlation was below 0.4.

Sunderland, Harris and Baddeley (1983) carried out a similar investigation using normal subjects and two groups of head-injured patients. Scores on the metamemory questionnaire completed by a relative were significantly correlated with the long-term head-injured patients' scores on six out of fourteen laboratory memory tests. A similar pattern of results was found between scores on the relatives' questionnaire and the test scores of the normal subjects but not between scores on the relatives' questionnaires and the test scores of the recently head-injured group. The strongest correlations were found between the scores of the long-term head-injured patients on immediate and delayed story recall tests and the relatives' questionnaires ($r=0.72$ and 0.62 respectively). The patients' questionnaires were not significantly correlated with any of the laboratory memory tests. The authors concluded that a reasonably valid measure of everyday memory could be obtained by having relatives in daily contact with the patient to complete the metamemory questionnaire rather than the patients themselves.

Kapur and Pearson (1983) found a highly significant correlation in a group of head-injured patients between the degree of memory impairment as perceived by a patient and as observed by a close observer. However, there was a generally low correlation between either subjective or observed memory impairment and scores on clinical memory tests. This was also true when head-injured patients were asked whether various memory functions had changed since the injury and if so, by how much.

A limited or non-existent relationship between scores on metamemory questionnaires and laboratory test performance has also been found in depressed subjects. For example, Kahn et al (1975) found that memory complaints were not related to the degree of impairment on a battery of memory tests, while several studies have shown that depressed subjects had a higher level of memory complaint than non-depressed subjects who performed comparably on memory tests (O'Hara et al, 1986; Popkin et al, 1982).

As already indicated, this lack of correspondence between self-assessments of memory function and scores on laboratory memory tests has often been taken as indicating that the self-assessments lack validity. However, Bennett-Levy and Powell (1980) argue that the lack of correlation does not mean that either the laboratory memory tests or the self-assessments are 'wrong', but that they measure different things. For instance the types of memory measured by the metamemory questionnaires (such as learning and recalling names) differ from those measured by laboratory memory tests such as the Wechsler Memory Scale. West, Boatwright and Schleser (1984) have suggested that the relationship between the memory questionnaires and memory tests would improve if more realistic memory tests were used which bore more resemblance to memory processes in everyday life. Herrman (1984) noted the need for laboratory memory tests to be related to the type of functioning measured by the questionnaire. For instance, Martin and Jones (1984) argued that the cognitive failures reported in the CFQ could result from a failure to distribute attention appropriately; they investigated this and found significant correlations between scores on the CFQ and performance on a test of the ability to distribute attention (Martin and Jones, 1983), which is in contrast to the failure to find correlations between CFQ scores and scores on laboratory memory tests (Broadbent et al, 1982).

This supports the argument that one of the reasons why metamemory questionnaires and laboratory test scores do not correspond is that inappropriate tests are used and consequently they measure different aspects of memory and cognition than the questionnaires.

It has also been suggested (Broadbent, Broadbent and Jones, 1986) that a high incidence of cognitive failures reported on, for instance, the CFQ may represent a preferred method of cognitive organisation which has advantages in some situations and disadvantages in others: if this is the case then high CFQ scores might be associated with good performance on some tasks rather than impairment. Again this suggests that the strength and even direction of the relationship between self-assessments and memory test performance will depend on the nature of the tests involved.

The lack of relationship may, however, also be because people are not a reliable source of information about their own memory performance. As Morris (1984) observed, people differ in their opportunities for memory failure and in what they consider to be normal and therefore worth reporting. They may therefore be quite impaired on a laboratory test but report little impairment in everyday life because they rarely need to use that type of memory, or because such impairment is normal for them. They also need to be able to remember a memory failure long enough to report it, which is likely to be a problem for people with severe memory difficulties. This may be why Sunderland, Harris and Baddeley (1983) found higher correlations between relatives' reports of everyday memory impairment and scores on laboratory memory tests than between patients self-reports and scores on the memory tests. There is some evidence that normal subjects, as well as those with severe memory impairment, may lack insight into their own memory functioning and therefore be unable to rate it accurately. Herrman et al (1983) found either a weak or non-existent relationship between the reported ability to perform ten memory tests and subsequent performance on these tests (<0.42). However when the subjects were asked to rate their ability to perform such tests after completing them eight of the correlations were significant (from 0.30 to 0.87). Thus, their awareness of their memory ability was fostered by recent experience with the memory tasks in question. These results suggest that metamemory questionnaires can elicit accurate self-reports but often fail to do so because people lack insight into their own memory performance.

Sunderland et al (1986) suggested that a strong relationship between reports of memory performance in everyday life and scores on laboratory memory tests would not necessarily be expected, even if the laboratory tests measured appropriate aspects of memory and the subjects had sufficient insight into their memory performance. This is because performance in everyday life is not determined solely by the individual's memory abilities; the demands made by the subjects' lifestyles and the effort they are prepared to go to to avoid memory failures also play a role. A lack of relationship may be due to the importance of these other factors.

Another reason for the lack of a relationship between self-reports of memory and performance on laboratory tests may be that studies have included subjects with a narrow range of scores on laboratory tests and who do not differ greatly in the extent to which they report memory problems. For instance studies using subjects recruited from a subject panel who are not significantly depressed or anxious, and are without organic brain damage, are unlikely to have included subjects with significant memory impairment or high levels of memory complaints (Broadbent et al, 1982). This is also true of studies of memory in elderly subjects and is illustrated by the fact that Sunderland et al (1986) found that the median score was above zero on only eight of the 28 items on their memory questionnaire; the mean score on, for instance, the participants' questionnaire was 35 compared to a maximum score of 224. Similarly, studies which have used chronic alcoholics (Shelton and Parsons, 1987), head-injured patients (Sunderland et al, 1983) or depressed subjects (Kahn et al, 1975) may have had a concentration of subjects with poor memories and high levels of memory complaints. This would have reduced the likelihood of uncovering a relationship between performance and complaint because there would not be a sufficient spread of scores on the memory tests or on the metamemory questionnaires.

This study therefore investigated the relationship between self-assessments of memory and performance on laboratory memory tests in a group of subjects which included depressed and anxious subjects, as well as those who were neither depressed nor anxious. This resulted in a wide range of scores on the memory tests with the depressed subjects doing less well than the controls (Section 4.4.1). In addition, the depressed

and anxious subjects were expected to complain about their memories more than the controls, giving a spread of scores on the memory complaints questionnaires.

The study also investigated whether a stronger relationship would be found between memory complaints and performance when subjects were asked to rate changes in memory, rather than how often various memory slips occurred in a specified period. This was based on the suggestion by Rabbitt (1982) that the difference between elderly people's assessment of their memory performance now compared with their assessment of it at age thirty might be more closely related to performance than the elderly people's assessments of their current memory performance. Sunderland et al (1986) followed up this suggestion and asked elderly subjects to complete an Everyday Memory Questionnaire about their memory now, and an identical one about their memory at age thirty. Contrary to expectations they did not find a stronger relationship between the degree of change between the two questionnaires and memory performance than between scores on the questionnaire about the present and performance. However, there was very little variance in ratings of memory at age thirty and consequently the measure of change between this and ratings of current memory was very similar to the scores for current memory. The methodology in the present study is somewhat different: the depressed and anxious patients were asked to indicate the degree of change in their memory since the the onset of their illness, rather than to complete a questionnaire relating to now and another one relating to before the onset of the illness. It is speculated that a stronger relationship would be found between scores on the Memory Complaints Questionnaire (which measures change) and performance than between scores on the CFQ (which asks how often cognitive slips happen) and performance.

7.1.4 Summary

The self-assessments of depressed and anxious subjects of their memory performance in everyday life, as measured by two self-complete questionnaires, were compared with the self-assessments of subjects who were neither depressed nor anxious. Regression analysis was used to investigate the relationship between the scores on these questionnaires, and self-reported depression and anxiety scores. Finally, scores on the

questionnaires were correlated with scores on the battery of memory tests described in Chapter Four.

7.2 METHOD

This section will give a brief account of the subjects, materials and methods used in this study as they have been described fully elsewhere (materials and procedures, Chapter Two; subjects, Chapter Three).

7.2.1 Materials

Two self-rating memory questionnaires were used in this study: the Cognitive Failures Questionnaire (Broadbent et al, 1982) and the Memory Complaints Questionnaire, a modified version of the Everyday Memory Questionnaire (Harris and Sunderland, 1981; Sunderland, Harris and Gleave, 1984).

The Cognitive Failures Questionnaire (CFQ) asks about cognitive mistakes in daily life; the subject is asked to rate how often various mistakes have happened in the past six months. The Memory Complaints Questionnaire (MCQ) covers memory and concentrational difficulties which may occur in everyday life; the depressed and anxious subjects were asked to compare their memory now with that before the onset of their illness, whilst the controls were asked to compare it with a year ago. Details of both questionnaires are given in Section 2.2.3.

The same battery of memory tests was used in this study as in Chapter Four. Details of the tests are given in Sections 2.2.1 and 2.2.2. Some of the tests resulted in more than one variable as they were designed to measure more than one aspect of memory. Tables 4.1 and 4.2 list the variables included in this study.

The self-report anxiety and depression scales used in the regression analysis are summarised in Section 6.2.2, and described in detail in Section 2.2.4.

7.2.2 Subjects

The first part of the study which compared self-reports of memory in the four subject groups (retarded depressed; neurotic depressed; anxious and controls) was restricted to the depressed and anxious subjects who completed a Present State Examination (Section 3.3.2) and the control subjects. It therefore used the same subjects as the study described in Chapter Four: they are fully described in Section 4.2.2. The same subjects were used in the final part of the study in which the correlations between self-reports and memory test performance were calculated for each of the four subject groups separately. In addition the subjects were also treated as one subject group.

The four subjects excluded from Chapter Four were included in the second part of this study. This used regression analysis to explore the relationship between self-assessments of memory, and depression and anxiety. The subjects were therefore the same as in Chapter Six; they are described in Section 6.2.1.

7.2.3 Analysis

A non-parametric equivalent of the one-way analysis of variance, the Kruskal-Wallis test, was used to test the null hypothesis that there were no significant differences between the scores of the four groups of subjects on the self-report memory questionnaires. This was used because the Bartlett Box test for the equality of variance in the four groups showed that the variances differed significantly between the groups on each test (CFQ, $F=4.46$, $p<0.01$; MCQ, $F=3.1$, $p<0.05$). Multiple comparisons were then carried out to compare the score of each group to that of each other group. Mann-Whitney U tests were used for this with modified significance levels which took into account the fact that several comparisons had been made (Section 4.2.3).

Regression analysis was used to explore the relationship between depression, anxiety and self-assessments of memory performance. As in Chapter Six anxiety was entered into a regression equation without depression, and depression was then entered to the equation. The order was then reversed.

Spearman rank correlation coefficients were calculated to explore the relationship between each memory test variable and scores on the self-assessment questionnaires. They were calculated for each group of subjects separately and for all the subjects combined. One-tailed probability levels were used.

7.3 RESULTS

7.3.1 Do Depressed and Anxious Patients Complain of Poor Memories?

Figure 7.1 shows the scores of the four groups on the CFQ. The differences between the groups were statistically significant ($N=41$, $\chi^2=10.1$, $p<0.05$). Multiple comparisons were carried out using the Mann-Whitney U test with amended significance levels to take account of the fact that several comparisons were being made (Section 4.2.3). The retarded depressed subjects differed significantly from the controls at the five per cent level: there were no other significant differences between the groups.

Figure 7.2 shows the scores of the four groups on the MCQ. Again the differences between the groups were statistically significant ($N=39$, $\chi^2=1.54$, $p<0.001$). Multiple comparisons showed that the anxious subjects, the retarded depressed and the neurotic depressed subjects all differed significantly from the controls ($p<0.05$): in each case they showed significantly higher scores, indicating a greater self-perceived deterioration in memory. There were no other significant differences.

These results show that the depressed and anxious subjects complained of a greater deterioration in their memories, as measured by the MCQ, than the controls. In addition the retarded depressed subjects also showed significantly higher scores than the controls on the CFQ, indicating that they reported more cognitive slips in the past six months.

FIGURE 7.1

SCORES OF THE FOUR SUBJECT GROUPS ON THE COGNITIVE FAILURES QUESTIONNAIRE (MEAN±SD)

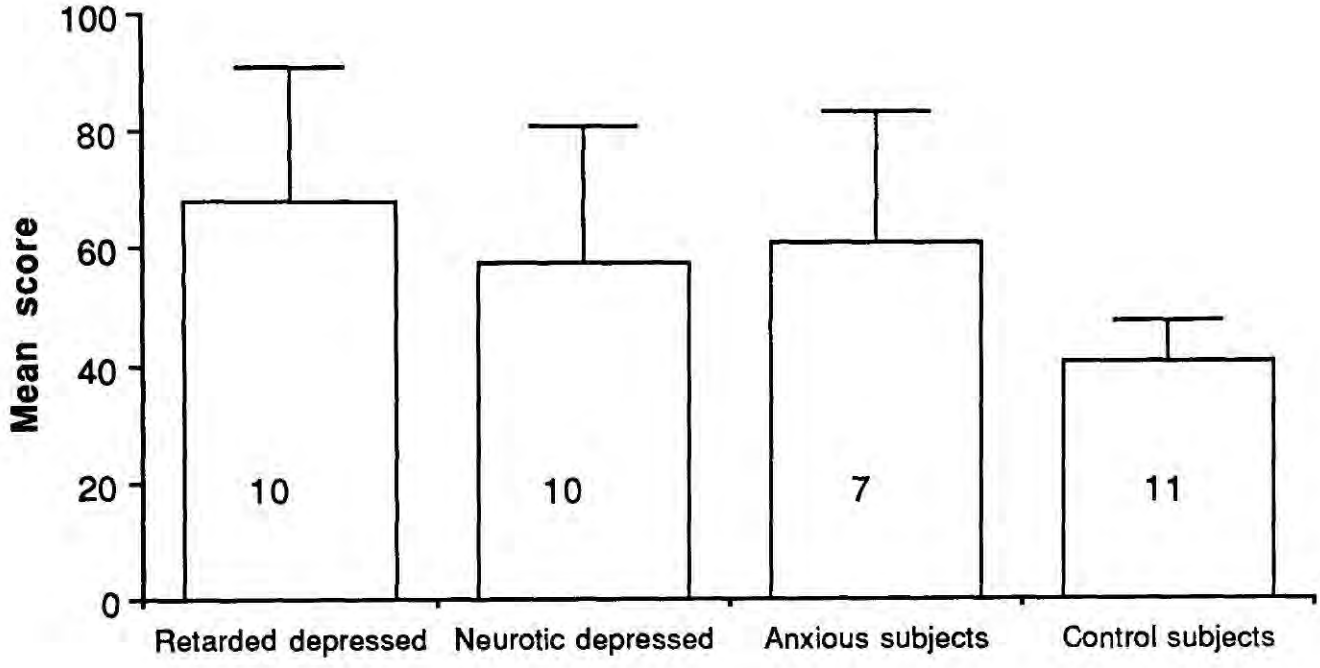


FIGURE 7.2

SCORES OF THE FOUR SUBJECT GROUPS ON THE MEMORY COMPLAINTS QUESTIONNAIRE (MEAN±SD)

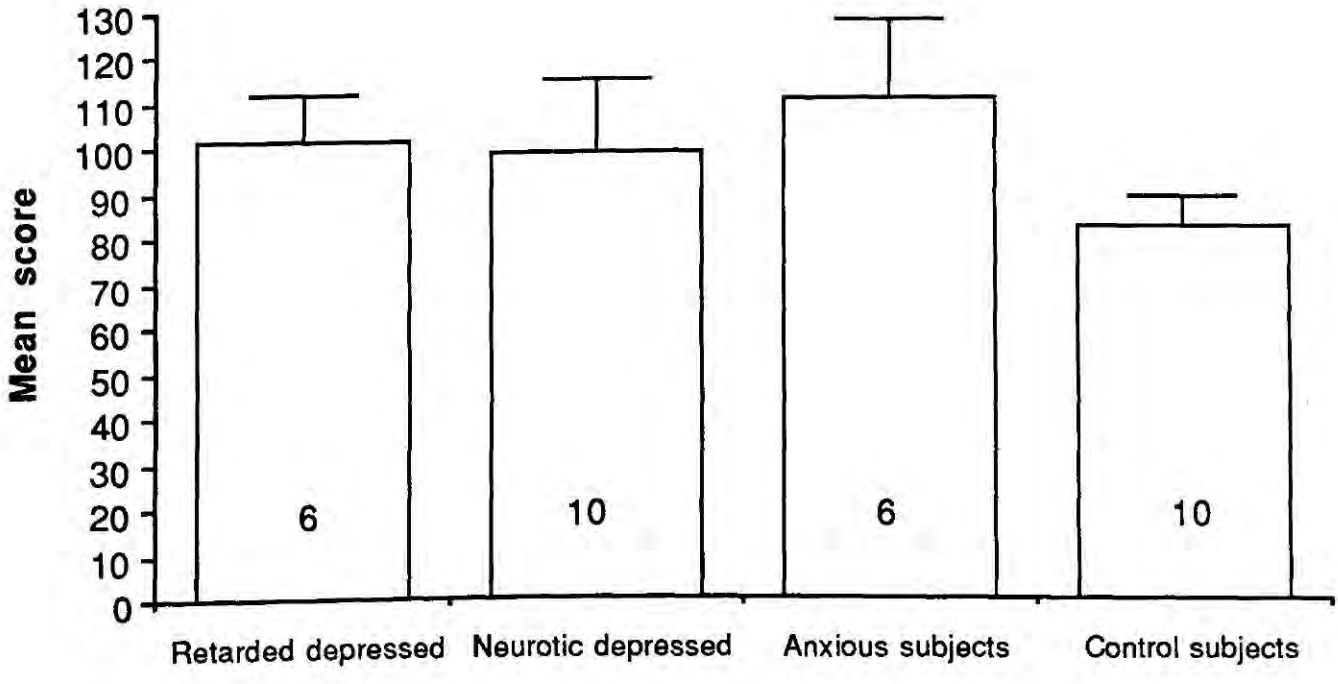


TABLE 7.1 CFQ AND MCQ: THE AMOUNT OF VARIANCE EXPLAINED (R^2) WHEN DEPRESSION ENTERS THE REGRESSION EQUATION BEFORE ANXIETY, AND WHEN THE ORDER IS REVERSED.

VARIABLE	R^2 EXPLAINED BY: DEPRESSION + ANXIETY		R^2 EXPLAINED BY: ANXIETY + DEPRESSION		RESIDUAL D.F.
CFQ	.10	*	.18	**	41
	.18	*	.18		
MCQ	.07		.21	**	40
	.21	**	.21		

Significance of change in r^2, * $p<0.05$ ** $p<0.01$

7.3.2 Are the Subjects' Self-Assessments of Memory Related to Depression or to Anxiety?

Table 7.1 shows the amount of variance explained (R^2) at each stage of the regression analysis. The regression equations for CFQ scores are considered first. It can be seen that anxiety significantly increased the amount of variance explained when it was entered into the regression equation by itself. It also significantly increased it when added into the equation after depression. In contrast, depression significantly increased the amount of variance explained when it was entered into the equation by itself but did not cause a significant increase in the amount of variance explained when added to the equation containing anxiety. It can therefore be concluded that anxiety, and not depression, was significantly related to scores on the CFQ, and that the apparent effect of depression when entered into the regression equation by itself was a consequence of its relationship with anxiety.

The results for the regression equation for MCQ scores are slightly different. In this case depression did not cause a significant increase in the amount of variance explained, either when entered into the regression equation by itself or when added to the equation containing anxiety. Thus depression was not significantly related to MCQ scores. In contrast, anxiety caused a significant increase in the amount of variance explained when entered into the equation by itself, and when added to the equation containing depression. Again it is clearly anxiety, rather than depression, which is significantly related to self-reports of a deterioration in memory.

These results show that it is anxiety, rather than depression, which is related to reports of memory slips or of deterioration in memory. On both the CFQ and MCQ, high anxiety scores were associated with high levels of complaints about memory.

TABLE 7.2 REGISTRATION, IMMEDIATE MEMORY AND SPEED OF LEARNING: SPEARMAN RANK CORRELATIONS BETWEEN THE MEMORY VARIABLES AND COGNITIVE FAILURES QUESTIONNAIRE

	SUBJECT GROUPS				
	ANXIOUS	RETARDED DEPRESSED	NEUROTIC DEPRESSED	CONTROLS	ALL SUBJECTS
Digit Span Forwards	-.84 (6) *	-.41 (10)	.09 (13)	.29 (10)	-.23 (41)
d' - Trial One	-.48 (6)	-.42 (8)	.00 (11)	.09 (11)	-.32 (39) *
Free Recall - total	.39 (7)	.02 (10)	.35 (13)	.05 (11)	-.03 (44)
Serial Learning - immediate recall	.22 (7)	.20 (10)	-.15 (13)	.45 (10)	-.14 (43)
Paired Associate - immediate recall	-.48 (7)	-.47 (10)	-.06 (13)	-.02 (11)	-.32 (44) *
Prose Passage One - immediate recall	.20 (6)	-.24 (13)	.12 (13)	-.03 (9)	-.15 (41)
Digit Span Backwards	-.49 (6)	-.36 (10)	.39 (13)	-.03 (10)	-.22 (42)
Serial Learning - speed of learning	.46 (7)	-.13 (10)	.12 (13)	.31 (10)	-.10 (43)
Paired Associate - speed of learning	-.60 (7)	-.09 (10)	.12 (13)	-.45 (11)	-.36 (44) **

* $p<0.05$ ** $p<0.01$ (N) Number of subjects in group

TABLE 7.3 RETENTION AND PAST PUBLIC EVENTS: SPEARMAN RANK CORRELATIONS BETWEEN THE MEMORY VARIABLES AND THE COGNITIVE FAILURES QUESTIONNAIRE

	SUBJECT GROUPS				
	ANXIOUS	RETARDED DEPRESSED	NEUROTIC DEPRESSED	CONTROLS	ALL SUBJECTS
d' - forgetting	-.31 (6)	.07 (7)	-.53 (10)	.24 (11)	-.50 (36) **
Prose Passage One - forgetting	.17 (6)	.78 (10) **	.20 (13)	.41 (8)	.40 (40) **
Prose Passage Two - delayed recall	-.25 (7)	-.47 (10)	-.13 (13)	-.11 (9)	-.40 (42) **
Prose Passage One - delayed recall	-.02 (7)	-.55 (10) *	.05 (13)	-.14 (8)	-.34 (41) *
Paired Associate - forgetting	.30 (7)	.47 (9)	-.19 (13)	-.04 (11)	.19 (43)
Paired Associate - Trial Five	-.24 (7)	-.46 (9)	.04 (13)	-.23 (11)	-.35 (43) **
Past Events free recall questionnaire	-.32 (7)	-.63 (9) *	-.25 (12)	-.14 (11)	-.58 (42) **
Past Events multi-choice questionnaire	-.47 (7)	.68 (10) *	-.27 (13)	-.35 (11)	-.55 (44) **

* $p<0.05$ ** $p<0.01$ (N) Number of subjects in group

TABLE 7.4 REGISTRATION, IMMEDIATE MEMORY AND SPEED OF LEARNING: SPEARMAN RANK CORRELATIONS BETWEEN THE MEMORY VARIABLES AND THE MEMORY COMPLAINTS QUESTIONNAIRE

	SUBJECT GROUPS: ANXIOUS	RETARDED DEPRESSED	NEUROTIC DEPRESSED	CONTROLS	ALL SUBJECTS
Digit Span Forwards	-.22 (5)	-.07 (10)	.14 (13)	.33 (9)	-.11 (41)
d' - Trial One	-.30 (5)	-.06 (8)	.07 (11)	-.46 (10)	-.35 (38) *
Free Recall - Total	.92 (6) **	-.34 (10)	.06 (13)	.08 (10)	-.08 (43)
Serial Learning - immediate recall	-.37 (6)	-.04 (10)	-.34 (13)	.01 (9)	-.34 (42) *
Paired Associate - immediate recall	-.34 (6)	-.05 (10)	-.20 (13)	.08 (10)	-.37 (43) **
Prose Passage One - immediate recall	-.30 (5)	-.65 (10) *	.01 (13)	-.20 (9)	-.31 (40) *
Digit Span Backwards	-.16 (5)	-.86 (10) **	.27 (13)	.16 (9)	-.27 (41) *
Serial Learning - speed of learning	.94 (6) **	-.60 (10) **	-.00 (13)	.14 (9)	-.15 (42)
Paired Associate - speed of learning	-.40 (6)	-.31 (10)	-.00 (13)	-.19 (10)	-.48 (43) **

* $p<0.05$ ** $p<0.01$ (N) Number of subjects in group

TABLE 7.5 RETENTION AND PAST PUBLIC EVENTS: SPEARMAN RANK CORRELATIONS BETWEEN THE MEMORY VARIABLES AND THE MEMORY COMPLAINTS QUESTIONNAIRE

	SUBJECT GROUPS				
	ANXIOUS	RETARDED DEPRESSED	NEUROTIC DEPRESSED	CONTROLS	ALL SUBJECTS
d' - forgetting	.10 (5)	.36 (7)	-.20 (10)	-.51 (10)	-.51 (35) **
Prose Passage One - forgetting	-.20 (5)	.62 (10) *	.18 (13)	-.26 (8)	.37 (39) **
Prose Passage Two -delayed recall	-.60 (6)	-.59 (10) *	-.11 (13)	.14 (9)	-.39 (39) **
Prose Passage One - delayed recall	-.06 (6)	-.79 (10) **	-.05 (13)	-.24 (8)	-.42 (41) **
Paired Associate - forgetting	.92 (6) **	.52 (9)	.22 (13)	.39 (10)	.44 (42) **
Paired Associate - Trial Five	-.76 (6) *	-.29 (9)	-.19 (13)	-.39 (10)	-.53 (42) **
Past Events free recall questionnaire	-.20 (6)	-.33 (10)	-.12 (13)	.59 (10) *	-.38 (42) **
Past Events multi-choice questionnaire	-.26 (6)	-.46 (9)	-.21 (12)	-.72 (10) **	-.55 (41) **

* $p<0.05$ ** $p<0.01$ (N) Number of subjects in group

7.3.3 Are the Subjects' Self-Assessments of Memory Performance Related to Performance on the Laboratory Memory Tests?

Cognitive Failures Questionnaire (CFQ)

Tables 7.2 and 7.3 give the Spearman rank correlations between scores on the CFQ and the variables derived from the memory test battery (Section 4.2.1). Emphasis is placed on the correlations for the subject group as a whole as this gives the widest range of scores on both the CFQ and the laboratory memory tests and therefore increases the likelihood of uncovering any relationship which does exist between the CFQ and the laboratory tests. The correlations for the individual groups are, however, included for information.

There was a significant relationship between the CFQ and two of the six measures of immediate memory: d' - Trial One and Paired Associate - immediate recall. The relationship was not strong: the correlation coefficents were -0.32 in both cases. The relationship between Paired Associate - speed of learning and scores on the CFQ was also statistically significant but again was not strong (rho=-0.36).

Prose Passage One - forgetting (one of three variables measuring the amount forgotten after a delay) was significantly related to scores on the CFQ with an increase in the amount forgotten being associated with an increase in CFQ scores: again the relationships were quite small in magnitude (rho=.040). d' - forgetting was also significantly related to CFQ scores but in the opposite direction to that expected: high CFQ scores were associated with a small decline in d' scores between the first and fourth trials of the Picture Recognition tests.

All three variables measuring recall after a delay were significantly related to CFQ scores (Prose Passage One - delayed recall, rho=-0.34; Prose Passage Two - delayed recall, rho=-0.40; Paired Associate - Trial Five, rho=-0.35).

The relationships between scores on both the free recall and multi-choice versions of the Past Events questionnaires and the CFQ were much

stronger: in both cases the correlation coefficents were greater than 0.5 (free recall questionnaire, rho=-0.58; multi-choice questionnaire, rho =-0.55).

With the exception of d' - forgetting, high scores on the CFQ were associated either with lower scores on the memory tests or with an increase in the amount forgotten between an initial and a delayed recall trial. Thus subjects who complained most about their memories also performed most badly on the laboratory memory tests.

Memory Complaints Questionnaire

Tables 7.4 and 7.5 give the Spearman rank correlations between scores on the memory variables and scores on the MCQ. Again, emphasis is placed on the correlations for the subject group as a whole.

There were significant correlations between the MCQ and five of the measures of immediate memory: in all cases the correlation coefficient was less than 0.4 (d' - Trial one, rho=-0.35; Serial learning - immediate recall, rho=-0.34; Paired Associate - immediate recall, rho=-0.37; Prose Passage One - immediate recall, rho=-0.31; Digit Span Backwards, rho=-0.27).

There was a highly significant correlation between the MCQ and Paired Associate - speed of learning (rho=-0.48). The correlations between MCQ and the variables measuring the amount forgotten between immediate and delayed recall were also significant (Prose Passage One - delayed recall, rho=0.37; Paired Associate - forgetting, rho=0.44; d' - forgetting, rho=-0.51). As with CFQ scores the relationship between d' - forgetting and MCQ scores was in the opposite direction to that expected, with high MCQ scores being associated with small declines in memory between the first and fourth trials of the Picture Recognition test.

The correlations between the MCQ and the variables measuring recall after a delay were significant (Prose Passage One - delayed recall, rho=-0.42; Prose Passage Two - delayed recall, rho=-0.39; Paired Associate - Trial Five, rho=-0.53).

Highly significant correlations were also found between both versions of the Past Events questionnaires and the MCQ (free recall version, rho= -0.38; multi-choice version, rho=-0.55).

As with the CFQ, in all cases except d' - forgetting a high score on the MCQ was associated either with a poor performance on the memory test or with an increase in the amount forgotten over time. Subjects who complained of the greatest deterioration in memory performed most poorly on the memory tests.

These results show that self-assessments of memory, measured using the CFQ and MCQ, are related to performance on many of the tests in the memory test battery. However, the results presented in Section 7.3.2 have shown that self-assessments of memory are related to anxiety, with high levels of anxiety being associated with negative self-assessments. It has also been shown (Section 6.3) that something common to anxiety and depression is related to performance on many of these memory tests. It is therefore possible that the correlation between self-assessments and performance does not indicate that people can accurately rate their memory performance but, instead, is an artefact due to anxiety being related to both self-assessments and performance. This may be unlikely as the relationship of anxiety to self-assessments showed quite a different pattern to its relationship to memory performance: in the former, anxiety was clearly a predictor but depression was not; whilst in the latter, depression was more closely related to performance than anxiety on some of the tests, and on others something common to both depression and anxiety predicted performance. However, the possibility that anxiety both causes people to be negative about their memory performance and causes memory impairments needs to be considered before it is concluded from the results of this section that people can at least fairly accurately assess their own memory performance.

TABLE 7.6 THE AMOUNT OF VARIANCE EXPLAINED (R^2) WHEN ANXIETY ENTERS THE REGRESSION EQUATION BEFORE CFQ SCORES, AND WHEN THE ORDER IS REVERSED

VARIABLE	R^2 EXPLAINED BY: ANXIETY + CFQ		R^2 EXPLAINED BY: CFQ + ANXIETY		RESIDUAL D.F
d' - Trial One	.06		.15	**	35
	.17		.17		
Paired Associate - immediate learning	.02		.08		40
	.08		.08		
Paired Associate - speed of learning	.06		.09	*	41
	.10		.10		
d' - forgetting	.10		.23	**	33
	.24	*	.24		
Prose Passage One - forgetting	.06		.13	*	36
	.14		.14		
Prose Passage One - delayed recall	.08		.08		37
	.11		.11		
Prose Passage Two - delayed recall	.16	**	.14	*	38
	.19		.19		
Paired Associate - Trial Five	.06		.11	*	39
	.12		.12		
Past Events free recall questionnaire	.07		.28	**	40
	.28	**	.28		
Past Events multi-choice questionnaire	.11	*	.31	**	38
	.31	**	.31		

Significance of change in R^2, * $p<0.05$ ** $p<0.01$

Regression analysis was therefore used to see whether CFQ and MCQ scores continued to be related to performance on the memory tests once anxiety scores were taken into consideration, and vice versa. Table 7.6 gives the results for CFQ scores. On four of the tests (d' - Trial One; Paired Associate - speed of learning; Prose Passage One - forgetting and Paired Associate - Trial Five) anxiety did not cause a significant increase in the amount of variance explained, either when entered into the equation by itself or when added to the equation containing CFQ. CFQ did cause a significant increase in variance when entered into the equation alone, but did not do so when added to the equation containing anxiety. As anxiety was not significantly related to performance on these tests when entered alone in the equation while CFQ was, it can be concluded that the significant correlation between CFQ and performance on these tests was not caused by anxiety. This is also clearly the case on the three variables where CFQ caused a significant increase in variance when entered into the equation by itself and when added to anxiety (d' - forgetting; Past Events free recall and Past Events multi-choice). On Paired Associate - immediate learning neither CFQ or anxiety caused a significant increase in the amount of explained variance at any stage; however, CFQ approached significance when entered alone in the equation ($p=0.056$) while anxiety did not, and so again it is concluded that the significant correlation between CFQ and performance on this test was not caused by the relationship of both of these variables with anxiety.

The results for Prose Passage One - delayed recall and Prose Passage Two - delayed recall were rather different. On the former variable both CFQ and anxiety approached significance when entered alone in the equation, whilst neither caused a significant increase in the amount of variance explained when entered into the equation containing the other. On Prose Passage Two - delayed recall both variables caused a significant increase in variance when alone in the equation but neither did when added to the other variable: it must be concluded that it is not possible to distinguish between the effects of these variables. It is therefore possible that the correlation between CFQ and performance on these tests was caused by anxiety.

TABLE 7.7 IMMEDIATE MEMORY AND SPEED OF LEARNING: THE AMOUNT OF VARIANCE EXPLAINED (R^2) WHEN ANXIETY ENTERS THE REGRESSION EQUATION BEFORE MCQ SCORES, AND WHEN THE ORDER IS REVERSED.

VARIABLE	R^2 EXPLAINED BY: ANXIETY + MCQ		R^2 EXPLAINED BY MCQ + ANXIETY		RESIDUAL D.F.
d' - Trial One	.06		.11	*	35
	.12		.12		
Serial Learning - immediate recall	.18	**	.09	*	39
	.19		.19	*	
Paired Associate - immediate recall	.01		.10	*	40
	.10	*	.10		
Prose Passage One - immediate recall	.01		.07		37
	.07		.07		
Digit Span - Backwards	.00		.04		38
	.04		.04		
Paired Associate - speed of learning	.07		.17	**	40
	.17	*	.17		

Significance of change in R^2, * $p<0.05$, ** $p<0.01$

TABLE 7.8 RETENTION AND PAST PUBLIC EVENTS: THE AMOUNT OF VARIANCE EXPLAINED (R^2) WHEN ANXIETY ENTERS THE REGRESSION EQUATION BEFORE MCQ SCORES, AND WHEN THE ORDER IS REVERSED.

VARIABLE	R^2 EXPLAINED BY: ANXIETY + MCQ		R^2 EXPLAINED BY: MCQ + ANXIETY		RESIDUAL D.F
d' - forgetting	.09 .24	*	.23 .24	**	32
Prose Passage One - forgetting	.06 .13		.12 .13	*	36
Prose Passage One - delayed recall	.04 .13		.13 .13	*	38
Prose Passage Two - delayed recall	.14 .16	**	.08 .16		39
Paired Associate - forgetting	.00 .11		.10 .11	*	39
Paired Associate - Trial Five	.07 .20	*	.20 .20	**	39
Past Events free recall questionnaire	.09 .13		.11 .13	*	39
Past Events multi-choice questionnaire	.16 .23	**	.19 .23	**	39

Significance of change in R^2, * $p<0.05$, ** $p<0.01$

The results for MCQ are given in Table 7.7 and Table 7.8. On six variables (Paired Associate - immediate learning; Paired Associate - speed of learning; Prose Passage One - forgetting; Prose Passage One - delayed recall; Paired Associate - forgetting and Paired Associate - Tria Five) MCQ caused a significant increase in the amount of variance explained when entered alone in the equation and when entered in the equation containing anxiety. It is therefore clear that the significant correlations between these variables and MCQ were not caused by anxiety. On three variables (d' - Trial One; Prose Passage One - forgetting and Past Events free recall) MCQ caused a significant increase in variance when entered into the equation alone, but not when added to anxiety. However, anxiety did not cause a significant increase in variance at any stage so again it is concluded that anxiety was not responsible for the correlation between MCQ scores and these variables. On Prose Passage One - immediate recall and Digit Span Backwards neither MCQ or anxiety caused a significant increase in the amount of variance explained at any stage. However the amount of variance explained by anxiety was minimal i both cases (.01 and .00) and therefore it is unlikely that the significan correlation between MCQ and these variables was an artifact resulting from the relationship of anxiety to MCQ scores and performance.

On two of the remaining variables (Serial Learning - immediate recall; Prose Passage Two - delayed recall) there was evidence that the significant effect of MCQ when entered alone in the equation was caused by its relationship to anxiety. On the third (Past Events multi-choice questionnaire) it was not possible to distinguish between the effects of anxiety and the effects of MCQ.

In summary, on eight of the ten memory variables significantly correlated with CFQ scores, and eleven of the fourteen memory variables significantly correlated with MCQ scores, it was clear that the correlation was not an artifact caused by the relationship of both the self-assessment scores and performance on the memory tests to anxiety.

7.4 DISCUSSION

7.4.1 Do Depressed and Anxious Patients Complain of Poor Memories?

The first aim of the study described in this chapter was to investigate whether depressed and anxious subjects are more likely to complain about their memories than subjects who are neither depressed nc anxious.

There were significant differences between the four subject groups i scores on both the CFQ and MCQ. The retarded depressed group showed significantly higher scores than the controls on the CFQ, indicating tha they reported more cognitive slips in the past six months than the controls. The differences on the CFQ between the neurotic depressed and anxious subjects on the one hand and the controls on the other were in the same direction but were not statistically significant.

The scores of the controls on the MCQ were significantly lower than those of the retarded depressed, neurotic depressed and anxious subjects As explained in Section 2.2.3 the MCQ consisted of twenty-seven questions, each of which was answered on a scale of one to five. Three represented no change in how often the memory failure occurred; one, tha it was occurring much less often, and five that it was occurring much more often. A subject reporting no change in memory at all would therefore score 81. The mean score of the control subjects was 82, indicating that there was little perceived change in memory over a year in this group.

The scores of the other three groups were significantly higher than this, indicating that the anxious and depressed subjects considered that their memories had deteriorated since the onset of their illness, which was on average sixteen and half months before the questionnaires were completed (Section 3.4.2).

The scores on the CFQ can be compared with those found in previous studies (this is not possible with the MCQ scores as this questionnaire was adapted from the Everyday Memory Questionnaire (Sunderland, Harris and Gleave, 1984) for use in this study). The mean score of the control

subjects on the CFQ was 40.8, with a standard deviation of 6.7, whilst those of the depressed and anxious patients were higher (retarded depressed, 68.1 (22.9); neurotic depressed 57.7 (23.5) and anxious 61.4 (22.4); Figure 7.1). These scores compare with a mean of 52.4 (14.5) in a group of student nurses, of 35 (11.5) in car factory production workers and 36.6 (9.4) in car factory skilled men (Broadbent et al, 1982) and a mean of 40.4 (13) in a group of 341 students (Hood, MacLachlan and Fisher, 1987). The scores of the control subjects are therefore comparabl with those found in previous studies, whilst those of the patient groups are higher. This supports the suggestion that depressed and anxious patients have high levels of memory complaints. In contrast Broadbent et al (1982) reported a mean CFQ score of 44.8 in a group of 34 neurotic psychiatric patients. This was comparable with the scores for the non-patient groups and therefore suggested that these patients did not have high levels of memory complaints. No details are given about the neuroti patients and it is therefore not clear whether they were comparable with the patients used in the present study.

These results show that the depressed and anxious subjects complained of a deterioration in their memories since the onset of their illnesses. In addition the most severely depressed subjects, the retarded depressed group (Section 4.4.1), also reported significantly more cognitive failures (such as forgetting appointments) than the controls. The neurotic depressed and anxious patient groups also scored higher tha the controls on the CFQ but the differences were not statistically significant. It is therefore clear that the depressed patients, who have been shown to have impaired memories on laboratory memory tests (Sectic 4.4.1) have negative self-assessments of their memory performance in everyday life. The anxious patients have similarly negative self-assessments although there is less evidence that they were impaired on the laboratory memory tests (Section 4.4.3). This suggests either that they have memory impairments in everyday life which are not well reflected in their performance on the laboratory tests, or that anxious patients complain of memory impairment without actually being impaired.

The results of this section have, therefore, replicated previous findings of high levels of memory complaints in depressed people (Kahn et al, 1975; Plotkin, Mintz and Jarvik, 1985) and of complaints of

deterioration in memory since the onset of depression (Pettinati and Rosenberg, 1984). In addition they also show that anxious patients both report more frequent memory failures and consider their memories to have deteriorated since they became ill. There are apparently no other studies of self-assessment of memory in anxious patients and consequently this is a new finding, although it might have been predicted from the evidence that high levels of anxiety in non-patient populations are related to high levels of complaints about memory (Broadbent et al, 1982).

7.4.2 Are the Subjects' Self-Assessments of Memory Related to Depression or to Anxiety?

The second aim of this study was to investigate whether self-rated depression or anxiety was most closely related to high scores on the CFQ and MCQ.

The results showed that depression, as measured by the self-rating questionnaire, was not a significant predictor of scores on either the CFQ or MCQ once anxiety was included in the regression equation. In contrast self-rated anxiety emerged as a significant predictor of scores on both questionnaires. In both cases high anxiety was associated with a high score and consequently with an increase in self-perceived memory deterioration and the occurrence of cognitive slips.

The results of this study raise the possibility that the significant relationship between depression and memory complaints found in several previous studies (Section 7.1.4) may not have been due to depression at all, but rather to the frequently concomitant anxiety. They highlight the importance of taking both depression and anxiety into consideration when investigating the effects of either depression or anxiety on memory performance or self-assessments of memory.

The results reported in Chapter Six do not suggest that anxiety and not depression was related to performance on the laboratory memory test rather the evidence suggests that depression was related to performance on what were judged to be the easier tasks, whilst something common to both depression and anxiety was related to performance on the more difficult tasks. This raises the question of why anxiety (and not

depression) was related to self-assessments of both the frequency of cognitive slips and the degree of deterioration in memory since the onset of illness, but related to performance on laboratory memory tests.

Martin and Jones (1984) hypothesised that cognitive failures resulted from excessive distributed-attention demands and, in support of this, obtained significant correlations between CFQ scores and performance on a test of the ability to distribute attention (Martin and Jones, 1983). They suggested that this explained why correlations had been found between CFQ scores and anxiety/neuroticism scores (Section 7.1.1): as Eysenck (1982) has observed, anxious subjects are in a dual task (or divided attention) situation because of interference from worry and self-concern, and are therefore likely to do badly on tasks requiring the further distribution of attention. However Martin and Jones (1984) do not take into account the fact that significant correlations have also been found between CFQ and depression scores (Broadbent et al, 1982; Hodd, MacLachlen and Fisher, 1987). If they had done so they might have noted that this explanation could equally be true of depression, especially as depression is also connected with worry and negative self-perceptions and therefore might also be hypothesised to have the effect of placing depressed people in a divided attention situation (Beck, 1967; Hasher and Zacks, 1979). This explanation, like any explanation based on concern about performance and negative self-evaluations, is therefore not unique to anxiety and consequently cannot account for the present findings.

It is possible that anxiety, and not depression, is related to negative self-assessments of memory because anxious individuals are hypothesised to increase the amount of effort they put into a task to overcome the reduction in processing capacity which is presumed to result from worry and self-concern (Eysenck, 1979, 1982). This may make them more aware of memory failures and difficulties with memory than other people who do not have to increase the effort they put into remembering things in order to avoid impairment. Because anxious people exert more effort to overcome these difficulties, they may not be impaired on memory tests unless the test is complex: if it is complex then anxiety will have similar effects on memory to depression, as hypothesised in Section 6.4.

In summary, it is clear that anxiety, and not depression, is related to self-assessments of memory performance in everyday life. These results show that previous studies using depressed subjects were unwise to ignore the fact that depressed individuals tend to be highly anxious as well as depressed (Section 6.1) as the correlations they observed between depression and memory complaints (Section 7.1) may well have been due to the concomitant anxiety and not to depression. It is suggested that anxious individuals may report high levels of memory problems and yet not be impaired on laboratory memory tests because they increase the amount of effort they put into a task. This makes them aware of finding a task difficult but at the same time overcomes the effects of reduced processing capacity and enables them to maintain normal performance.

7.4.3 Are the Subjects' Self-Assessments of Memory Performance in Everyday Life Related to their Performance on the Laboratory Memory Tests?

The final aim of this study was to see whether the subjects' self-assessments of memory performance in everyday life were related to their performance on a variety of laboratory tests.

The results showed that scores on both the CFQ and MCQ were significantly related to scores on many of the variables derived from the memory test battery. In all cases except one, higher scores on the CFQ and MCQ were associated with either lower scores on the memory variables, or with an increase in the amount forgotten between initial and delayed recall (the exception was d' - forgetting, where scores had already been shown not to decline in a linear fashion as expected between the first and fourth trials of the Picture Recognition test (Section 4.3) it therefore produced quite different results to the other 'forgetting' variables). Thus subjects who complained about their memories were likely to do less well than other subjects on the laboratory measures of memory. With a few exceptions (Section 7.3.3) it was clear that these results were not an artefact resulting from anxiety being related to both the self-assessments of memory and memory performance: instead they indicate that people can give a fairly reliable assessment of their memory performance

Two of the variables measuring immediate memory (d' - Trial One and Paired Associate - Trial One) were significantly related to CFQ scores, while five of the variables in this section were associated with MCQ scores (d' - Trial One; Paired Associate - Trial One; Serial Learning - immediate recall; Prose Passage One - immediate recall and Digit Span Backwards). Previous studies have found that the strongest relationships between self-assessment and performance occur with a story recall test, and have suggested that such tests measure the ability to use reconstructive processes which are important in everyday life (Sunderland, Harris and Baddeley, 1983; Sunderland et al, 1986). This study has not replicated this finding as only MCQ scores were significantly associated with Prose Passage Recall - immediate recall. In addition two of the variables showed a stronger relationship with MCQ scores than Prose Passage Recall - immediate recall (Paired Associate - immediate recall ; Serial Learning - immediate recall). The results suggest that relationships can be found with a variety of memory tests ranging from those with a strong hypothesised relationship with memory in everyday life to others which seem less relevant to everyday memory. As expected, more significant correlations were found in this group of variables between MCQ scores (a measure of self-perceived change in memory) and performance than between CFQ scores and performance.

Significant relationships were found between two of the variables measuring the amount forgotten between initial and delayed recall and CFQ scores, whilst all three of these variables were significantly associated with MCQ scores (this group included d'- forgetting, which showed an increase in forgetting with a decrease in CFQ and MCQ scores. However, as already noted, d' scores did not decline in all four groups between the first and fourth trials of the Picture Recognition test (Section 4.3) and so this is not comparable with the other measures of forgetting in which memory did decline between immediate and delayed recall). It is not surprising that correlations are found between the measures of forgetting and the self-assessment questionnaires since twelve of the 27 questions on the MCQ are concerned with forgetting information, whilst the same is true of five of the 25 items on the CFQ. This confirms the assertion of Herrman (1984) that stronger relationships will be found between self-assessments and laboratory memory tests if the tests are chosen to cover aspects of memory measured by the questionnaires.

The variables measuring the amount recalled after a delay were also significantly related to the questionnaire scores, in three out of six instances at or above .4. This was presumably because in everyday life it is often necessary to retain information in memory for quite some time, rather than recalling it immediately. However, the results for this section need to be treated with some caution as it is possible that the correlation between the delayed recall of the two prose passages and the self-assessment questionnaires was caused by the relationship of all these variables to anxiety.

The strongest relationships between self-assessments of memory and memory performance were with the free and multi-choice versions of the Past Events Questionnaires. These measure memory for events which occurred before the onset of depression and, in contrast to the majority of laboratory memory tests which use the intentional learning paradigm, these measure memory for information which was probably incidentally learned. The significant relationship between scores on these and the questionnaires is presumably due to the more realistic nature of these tests: many things in everyday life are learned incidentally and therefore the self-perceived ability to recall them would influence how the memory assessment questionnaires are completed. A previous study has also shown that scores on metamemory questionnaires correlated with recall after incidental learning: Martin, Ward and Clark (1983) found that when people who reported good everyday memory were asked unexpectedly to recall previously presented trait words, they remembered more than people who reported poorer self-assessments of everyday memory. This led Martin (1983) to conclude that significant relationships between metamemory questionnaires and performance might be found if incidental learning tests are used, rather than the more traditional tests measuring intentional learning. The results presented here show that the strongest relationships were found with the incidental task, but that significant correlations between self-assessments and performance were also found with tests of intentional learning.

In summary, the strongest relationships between self-assessments of memory and performance on the laboratory memory tests were found on the tests which were presumed to be most realistic, because they measured ho much information was forgotten over time, recall after a delay or memory

for incidentally learned material. In addition significant relationships were found between scores on the self-assessment questionnaires and variables measuring immediate memory; some of which (Prose Passage Recall and Paired Associate learning) have been judged by previous authors (Sunderland, Harris and Baddeley, 1983; Sunderland et al, 1986) to involve aspects of memory important in everyday life. These results therefore support the assertion that the lack of a relationship between self-assessments of memory and laboratory memory tests is caused by the fact that they tend to measure different aspects of memory and that stronger relationships would be found if the laboratory tests were more realistic and measured aspects of memory frequently utilised in everyday life (Section 7.1.3).

As expected the highest number of significant correlations between self-assessments and performance was found with the MCQ, which measures changes in memory rather than how often memory slips occur. This supports Rabbitt's (1982) suggestion that people are better at assessing changes in memory performance than judging how often memory failures occur. This may be because remembering the latter puts quite a demand on memory and, in addition, is subject both to individual differences in what is considered to be normal memory functioning and in lifestyle, which affect the number and type of failures that occur (Morris, 1984). Procedures which ask about changes in memory are likely to be less affected by such factors.

There are clearly significant relationships between subjects' performances on laboratory memory tests and their self-asessments of memory deterioration and occurrence of cognitive slips when subjects showing wide ranges of scores on both dimensions are used. This suggests that people are at least fairly reliable in assessing their memory performance in everyday life. However, the magnitudes of the relationships were similar to those reported in other studies (eg Sunderland et al, 1986) and in most cases agree with Herrman's (1982) finding that correlations between self-assessments and laboratory memory tests rarely exceed .5: only two of the ten significant correlations with the CFQ did so in this study and only three of fourteen significant correlations with the MCQ. As the correlations are quite low they tend to support the general picture from previous research: that there is a lack of agreement

between self-perceptions of memory performance in everyday life and performance on laboratory memory tests (Section 7.1.3). This is not surprising given the number of factors which affect the accuracy of self-assessments of memory (such as levels of anxiety, the level of awareness of one's own performance and one's expectations of memory) and the fact that laboratory memory tests and metamemory questionnaires often measure different aspects of memory (Section 7.1.3).

If self-perceptions of memory performance are not strongly related to laboratory test performance and it is not clear what relationship either of these has with everyday memory performance (Section 7.1.3), it is not immediately clear what use self-assessments can be to clinicians concerned with patients with memory loss. Sunderland, Harris and Baddeley (1984) suggested that the most fruitful use of self-assessments of memory performance in head-injured patients could be as a source of qualitative data on what sort of memory failures tend to occur in head-injury. Test procedures could then be devised to simulate the everyday tasks that led to these failures: such tests might lead to the best way of assessing the ability of a patient with impaired memory to avoid memory failures in everyday life. This suggestion could also be applied to depression: knowledge of the tasks which depressed people think they find difficult in everyday life could aid the design and selection of appropriate tests for laboratory studies of memory in depression. This could then be theoretically useful to researchers investigating why memory is affected by depression, and clinically useful for those concerned with helping depressed people overcome their impairments.

7.4.4 Summary

The results of this chapter have shown that depressed and anxious patients report more cognitive slips than control subjects and consider their memories to have deteriorated significantly since the onset of their illness. When the relative effects on self-assessments of memory of depression and anxiety were examined it was found that anxiety and not depression was related to negative self-assessments. It was suggested that this is because anxious people increase the effort they put into remembering things and are therefore aware of finding remembering difficult. These results highlight the importance of taking both anxiety

and depression into consideration when looking at memory in depressed and anxious people.

Self-assessments of memory were found to be significantly related to scores on many of the laboratory memory tests used, particularly those considered to be the most realistic and to measure aspects of memory frequently used in everyday life. With a few exceptions it was clear that the correlations between self-assessments and performance were not caused by both variables being associated with a third variable: anxiety. It was therefore concluded that people can give a fairly accurate assessment of their memory performance in everyday life. However, as in previous studies the correlations between self-assessments and performance were quite small and predominantly less than .5. This reflects the fact that laboratory memory tests and self-assessment questionnaires tend to cover different aspects of memory and that the accuracy with which people assess their performance will depend on how anxious they are, what they consider to be normal memory functioning and the opportunities they experience for memory failure. It is suggested that although self-assessment questionnaires may not give an entirely accurate account of memory in everyday life they could provide valuable qualitative information about the types of things depressed people think they have difficulty with. Tests could then be devised to simulate these aspects of memory which could provide useful information both for those concerned with identifying the causes of memory failure in depression, and for those clinicians concerned with helping depressed people overcome their memory deficits.

The next chapter (Chapter Eight) is concerned with another way of investigating memory in everyday life. Depressed and non-depressed patients were compared to see how well they could remember information given to them by their general practitioners. This was in order to see whether depressed people show the same degree of memory impairment in a realistic situation as they do in laboratory memory tests (Chapter Four), and as would be predicted from negative self-assessments of their own memory performance (Chapter Seven).

MEMORY FOR MEDICAL INFORMATION IN DEPRESSED PATIENTS

8.1 INTRODUCTION

This chapter is concerned with the memory of depressed and non-depressed subjects in an important everyday situation: the general practice consultation.

As indicated in Section 7.1.3 interest in everyday memory has grown amongst both experimental cognitive psychologists and amongst psychologists with clinical interests. This has led to attempts to replicate real-life in the laboratory, and to the design of memory tests which more closely resemble tasks which people do in everyday life than traditional laboratory memory tests (Wilkins and Baddeley, 1978; Becker et al, 1983). It has also led to an interest in asking people to assess their own memory performance in everyday life and the development of a large number of metamemory questionnaires (Herrman, 1984; Chapter Seven). In addition, it has led to studies looking at memory in realistic situations.

One such study is that by Baddeley (1981a) who argued that laboratory studies on the effects of alcohol on cognitive functioning were always carried out in an environment very different from the ones in which alcohol is usually drunk. He therefore looked at whether he could find reliable effects of alcohol on performance in a more realistic situation; a residential course for divers who dived during the day and spent the evening in social drinking. He tested the divers on a variety of memory tests and compared their performance on an evening when they agreed to abstain from alcohol with that on a night when they drank as much as they wanted. He found significant effects of alcohol on performance, despite the fact that the amount of alcohol drunk was not controlled and the environment was very different from a laboratory (one test had to be abandoned when tea towels were dried on a vital bit of equipment; such problems do not usually occur in laboratories).

This study illustrates the fact that work outside the laboratory is not easy because it is difficult to achieve the necessary blend of rigour and realism. However, such studies provide a means of testing theories of

memory outside the laboratory and of generating new ideas for research (Baddeley and Wilkins, 1984). In addition they can be used to see if the degree of impairment expected on the basis of the test results of people with memory problems is actually found in everyday life. Such studies could provide important information for clinicians concerned with identifying the extent of impairment and helping patients to overcome it, especially as there is good reason to believe that neither laboratory memory tests nor self-assessments of memory provide an entirely accurate description of memory performance in everyday life (Section 7.1.3). There are, therefore, good reasons for trying to overcome the difficulties experienced when looking at memory performance outside the laboratory.

8.1.1 Everyday Memory in Depressed People

Memory in depression is one area in which it is particularly important to see whether results obtained in the laboratory match those found in more realistic situations. For example, it has been suggested that the memory impairments found in depressed people are caused by a general lack of motivation (Cohen et al, 1982, Section 1.8.1) and that they will be most impaired on tasks requiring sustained effort for successful completion (Section 4.4.1). In addition it has been hypothesised that depression leads to a reduction in processing capacity (Hasher and Zacks, 1978; Ellis, 1985) and that the effects of such reductions can be overcome by increasing the amount of effort put into the task (Eysenck, 1982; Section 6.4). Thus, memory impairment in depression may result from a combination of reduced processing capacity and a lack of motivation: if motivational levels were higher the depressed person may have been able to overcome the effects of reduced processing capacity. If this is so it is likely that the degree of impairment found will vary according to the importance of the situation to the individual, because this will affect their motivation and consequently the effort they make to overcome the reduction in processing capacity. It may be that they are less motivated to remember a list of words presented to them by a researcher than they are to remember things in their everyday life. Alternatively, they may be very anxious to perform well on the laboratory tests and are consequently more motivated than normal; this may enable them to overcome the effects of the reduction in processing capacity. This suggests that researchers need to be cautious

about presuming that the performance of depressed people on laboratory memory tests accurately reflects the degree of memory impairment they experience in their everyday lives.

If laboratory memory tests cannot be relied on to give an accurate impression of the memory problems experienced by depressed people, then an alternative is to ask depressed people about their perceptions of their memory performance in everyday life. As the results of Chapter Seven have shown, there is evidence that people can assess their own memories quite accurately, but that their accuracy is reduced by high levels of anxiety (these increase levels of complaints about memory performance without at the same time decreasing memory performance). The evidence also suggests that this accuracy is affected by individual differences in what are perceived as memory failures and further by differences in what is perceived as normal memory performance (Section 7.4.3). It is not clear, therefore, whether self-assessments of memory performance reflect actual performance in everyday life any better than do laboratory memory tests, especially in depressed subjects since they are likely to have high levels of anxiety (Section 6.1). As there is doubt as to how well either laboratory memory tests or self-assessments of memory reflect the memory of depressed people in everyday life it was decided to look at the memory of depressed people in an everyday situation.

Depressed patients are common in general practice: Sireling et al (1985) estimated that the average general practioner (GP) who holds about 40 surgeries each month initiates four new treatment courses for depression and misses at least five new cases of it per month. As they will have other patients already undergoing treatment for depression these figures underestimate the number of depressed people seen by GPs. Most depressed patients are managed in general practice (Shepherd and Wilkinson, 1988): the 1971 National Morbidity Survey (OPCS, 1974) found a one year consultation rate of 35.5/1000 for depression, whilst in contrast the Chichester/Salisbury Study found an annual referral rate of about three per thousand (Grad de Alarcon, Sainsbury and Costain, 1975). Consultations with a GP are therefore likely to be a common event for depressed people.

This is also one situation in everyday life where a memory impairment could have serious implications: patients are unlikely to be compliant with their doctor's instructions if they have forgotten them by the time they get home. This, therefore, seemed to be a good situation to examine to see if depressed people do have memory impairments outside the laboratory, especially as the factors affecting the patient's memory for medical information have already been investigated in several studies These are reviewed below.

8.1.2 Memory for Medical Information

Ley and Spelman (1965) interviewed 47 new patients at a medical out-patient clinic shortly after they had seen the consultant. They compared the patient's account of the consultation with the doctor's verbatim record and found that on average patients forgot one third of the information they were given, and that they retained proportionally less of it the more they were told. Instructions, prognostic statements and reassurance were forgotten more often than other statements. There was a curvilinear relationship with anxiety, such that patients with both high and low anxiety (as measured by Cattell's 16 Personality Factors questionnaire) remembered less that patients with intermediate scores. In this study they found a negative relationship between age and recall: the younger patients remembered most. This was not however substantiated by the results of two subsequent studies and they conclude that age is not related to performance (Ley and Spelman, 1967).

In order to investigate the factors affecting recall in a more rigorous manner Ley and Spelman (1967) looked at memory for fictitious medical information using healthy normal volunteers as subjects. They completed a multi-choice questionnaire testing knowledge about eleven common illnesses. Recall was best in those with good medical knowledge. The degree of importance that people attach to different types of statements told to them by their doctor was also investigated in this study to see if this helped to explain why the earlier study (Ley and Spelman, 1965) had shown that some types of statements were recalled better than others. The subjects were presented with a list of statements and were asked to indicate how important they would consider each statement to be if they were told it by a doctor and it applied to them.

The list contained diagnostic statements, instructions and other types of statements. The subjects considered diagnostic statements the most important and instructions least important. This was also true when they were given groups of three statements, one from each group, and asked to place the statements in each set in order of importance. In contrast a group of general practitioners who were also asked to rate the statements showed no differences in the importance they attached to each type of statement. The authors conclude that this study shows that laymen and doctors attach different degrees of importance to different types of statements, and that it is likely that there is an association between perceived importance and frequency of recall.

Joyce et al (1969) tape-recorded out-patient consultations and then tested recall after one, two or four weeks, as well as immediately after the consultation. They found that about one half of the items tape-recorded by the doctor were recalled afterwards, regardless of when the recall interview took place. There was a suggestion of a negative relationship between amount told and per cent recalled, but this was not significant. As in the previous studies some types of information were recalled better than others; about 70% of information about further investigations was recalled compared with only 30% of information explaining the disease or the treatment. Less than half of the instructions were recalled. In contrast to earlier studies (Ley and Spelman, 1967) Joyce et al (1969) found a significant negative correlation between age and recall.

Several studies have explored ways of improving the amount of information patients remember. For instance, Ley et al (1973) found that memory for information given during a general practice consultation was improved when the doctors grouped similar items of information together and explicitly labelled each category: for instance they would tell the patient that first they would tell them what was wrong, then how to treat it. Bradshaw et al (1975) found that the use of specific as opposed to general advice to a group of obese patients increased recall by more than 200%, while using sentences of 'high reading ease' (short words in short sentences) increased it by about 40%. Ley (1979) reported that recall can also be improved by repeating information and by presenting important

information early in the consultation or, alternatively, after all other advice has been given.

It is apparent, therefore, that patients with normal memories forget much of the information they are given by their doctor. A number of factors have been shown to be related to the amount recalled. These include the amount of information given during the consultation (Ley and Spelman, 1965; Joyce et al, 1969), number of repeated statements (Ley, 1979), and type of information (Joyce et al, 1969). In addition it has been shown that there are things doctors can do to increase recall, such as using simpler language and giving specific rather than general advice.

Despite the evidence that depression can result in memory impairment, and the fact that many depressed people are likely to be receiving treatment from a doctor, there has been no research into the effects of depression on memory for medical information. It is therefore not known whether depression, like anxiety, affects memory for medical information.

8.1.3 Aims of Study

This chapter describes a study comparing depressed and non-depressed patients' memory for information given to them by their general practitioner. The aim was to see if depressed people had memory impairments in this realistic 'everyday' situation. Similar patients have shown impairments on laboratory memory tests (Chapter Four), have assessed their memory as having deteriorated since the onset of their illness, and have reported a high incidence of cognitive failures (Chapter Seven). Consequently, if laboratory memory tests and self-assessments of memory accurately reflect memory performance in everyday life then these patients would be expected to remember less information than normal patients. If they do not do so, this would suggest that caution is needed when interpreting the results of such tests and self-assessment questionnaires.

Four general practitioners took part in this study. Women consulting them were screened for depression before their consultations; those scoring highly on the screening instruments were asked to return after seeing the doctor, together with some of the low scorers who were about

the same age as the depressed women. Immediately after the consultations (which were taped) the subjects were asked to recall as much as they could remember of what the doctor had said to them. The women who scored highly on the screening instruments were later interviewed at home using the Present State Examination (Wing, Cooper and Sartorius, 1974). The transcripts of the consultations and recall interviews were then subjected to content analysis, and the recall of the depressed and control patients compared.

8.2 METHOD

8.2.1 Materials

Present State Examination (P.S.E)

This is a semi-structured psychiatric interview, designed to assess current mood state, which has been developed for use in research (Wing, Cooper and Sartorius, 1974). The symptom scores produced during each interview are assigned on the PSE Index of Definition (a measure of the certainty that the subject is a psychiatric 'case') ranging from Level One where no symptoms are present, through the threshold level for 'caseness' (Level Five), to the levels of definite caseness (Levels Six to Eight). Subjects at or above the threshold level are allocated by the CATEGO computer program to diagnostic classes. The P.S.E is described in more detail in Section 3.3.3.

Screening Tests for Psychiatric Disorder

a) The '10 Questions' of the P.S.E

These questions make up a screening test for psychiatric disturbance: a score of less than two has been shown to be a very good predictor of a low or negative score on the full P.S.E (Cooper and Mackenzie, 1981). They are intended for use by an interviewer trained to use the P.S.E and consist of key questions from the full interview. They are described fully in Section 3.5.2.

b) Depression Scale

The depression subscale of the Irritability, Depression and Anxiety scale (a self-complete questionnaire; Snaith et al, 1978) was used in this study. It consists of five questions about how the patient has been feeling recently with scores on it ranging from zero to fifteen. The questions were chosen because they correlated well with psychiatrists' ratings of depression. The authors recommend that scores of four to six should be regarded as borderline between clinical and sub-clinical depression. This scale is described in Section 2.2.4.

Recall Questions

a) Free Recall

The first of these questions was intended to elicit as much information as possible from the patient about what the doctor had told them. It was as follows:

'Can you tell me as much as you can remember of what the doctor has just said to you?'.

b) Cued Recall

These questions were intended to prompt the patients' memory for information given to them during a consultation. They were adapted from the categories into which Ley and Spelman (1965) divided all the statements made by the doctor in their study: diagnosis, instructions, further investigations necessary, GP to be informed, further visits necessary, explanation of symptoms, prognostic statements, statements about treatment, reassurance. These were similar to categories used in other studies (Joyce et al, 1969) and it was felt that they would cover most of the information likely to be given during a consultation. They were as follows:

1) Did the doctor tell you what was wrong?

2) Did he give you any instructions?

3) Did he say whether any further visits are necessary?

4) Did he say any tests would be needed?

5) Did he explain your symptoms to you at all?

6) Did he reassure you at all?

7) What did he say the treatment was?

8) Did he say it will get better? If so, how long did he say it will take?

8.2.2 Procedure

All the women aged between 18 and 65 who visited their doctor during a session when the interviewer was at the surgery, and who reported to the receptionist at a time when the interviewer was not busy with another patient, were asked by the receptionist to take part in a study of 'how doctors and patients communicate with one another'. No mention was made of memory.

Preliminary Interview

After she had been introduced to the interviewer the patient was asked if she would mind answering a few questions about how she had been feeling recently. None objected to this. She was first asked the '10 Questions'. If she scored one or zero on this she was asked for some information about herself (described below), thanked for her help and then saw the doctor as normal. The only exception to this was if the woman was close in age to one of the depressed patients, in which case she was asked to come back and see the interviewer after her consultation with the doctor; if she agreed to this she was included in the study as a control subject.

If the woman scored more than one on the '10 Questions' she was asked to complete the depression scale. If she scored less than five on this she was thanked for her help and then proceeded to see the doctor

as normal. If she scored five or more she was asked to come back and talk to the interviewer after she had seen the doctor.

After completing the '10 Questions' and, in some cases the depression scale, each woman was asked her date of birth; occupation (or husband's occupation if not currently working); age at which she had left full-time education; and the number of times she had been to the doctor in the past month and in the past year.

Consultation with the Doctor

The consultations of patients participating in the study needed to be taped so that what the patient remembered about it afterwards could be directly compared to what the doctor had said. It was important that the doctor did not know exactly who was in the study, as if he did there was a possibility that he might alter his style and present information in a more memorable way to these patients. The doctor therefore asked all the women he saw during the sessions when the interviewer was at the surgery if she minded her consultation being taped for 'research purposes'. This meant that he did not need to know who was in the study. If the patient had no objection to the consultation being taped the tape-recorder was switched on and the consultation continued as normal.

Recall Interview

If the patient was taking part in the study she came back to see the interviewer immediately after her consultation with the doctor. She was told that the study was concerned with helping doctors to find better ways of presenting information to patients, and that in order to do this it was necessary to know how much patients could remember at the moment. She was then asked if she minded this section of the interview being taped and the interviewer then went through the recall questions about the consultation.

If the patient was taking part in the study as a control for a depressed patient she was then thanked for her help and given the opportunity to ask any questions she had about the study. If the woman was depressed, and therefore a study patient, she was asked if the

interviewer could visit her at home in order to talk in more detail about how she had been feeling recently. If she agreed, a time was then arranged for the forthcoming week.

Home Interview

At this interview the patients were interviewed using the Present State Examination (Wing, Cooper and Sartorius, 1974). This was concerned with their psychiatric symptoms over the past month.

8.2.3 Subjects

Four general practioners took part in this study. Three had practices in Durham itself while the fourth practiced in a mining village about six miles outside the city. They were selected because they were already known to the Department of Psychology at the University of Durham and because they were in easy travelling distance of the university.

Subjects were recruited for the study at a total of 98 sessions at the four participating practices. Three practices took part in the study for about a year (thirteen months in two cases and eleven months in the third). The fourth practice withdrew after two months as structural alterations to the practice meant that there was no longer a spare room for the interviewer to use.

Only women patients between the ages of 18 and 60 were included in the study. Several studies have shown that the prevalence of psychiatric problems is higher in women in the general practice population than in men; for instance Cooper, Fry and Kalton (1969) found that the annual prevalence for psychiatric morbidity was 60:1000 in men and 172:1000 in women, while Finlay-Jones and Burvill (1978) reported a one-day prevalence of minor psychiatric morbidity in general practice patients of 3.72 per 1000 general population in women and 1.74 per thousand in men. It was therefore decided that it would be cost-effective to concentrate upon women as this would mean that fewer people would need to be screened for depression to obtain the number of subjects required in the study.

Study Group

This study is concerned with the effects of clinical depression upon memory, rather than the effects of less severe, sub-clinical depression. The criteria for inclusion in the study were therefore stringent. A patient was included in the study group if she fulfilled three criteria:

(1) She scored more than one on the '10 Questions'. These questions were used primarily to screen for women who were not depressed and did not have other psychiatric problems and, therefore, were not suitable for the study. With the exception of a small number of these women who form the control group, women who scored one or zero on these questions did not participate in the study beyond this initial screen.

(2) She scored five or more on the depression scale. As described above, this is in the recommended borderline zone between clinical and sub-clinical depression.

(3) She was described by the Catego program of the P.S.E as having neurotic or retarded depression at Index of Definition levels Five or above. As described in Chapter Three of this thesis, these are taken to be clinically significant levels of disorder (Section 3.2.2).

During the course of the study 165 women were interviewed before they saw the doctor and screened for depression. Fifty-nine scored one or below on the '10 Questions' and were therefore not screened further. Fifty-four women scored two or more on the '10 Questions' but less than 5 on the depression scale. Ten of the remaining patients were not willing to come back and talk to the interviewer after their consultation; five said they were too busy while the remainder gave no reason for their refusal.

This leaves 42 patients. Five were eliminated at this stage: three because the general practioner forgot to tape the consultation and two because they did not want their consultation to be taped. The remaining patients were all asked if the interviewer could visit them at home to

talk further about how they had been feeling. Six women refused. Two women made arrangements for a visit but later cancelled them. Twenty-nine patients were therefore interviewed using the P.S.E.

Of these, twelve patients did not reach the predetermined cut-off point of an Index of Definition level of Five or above. Six of the seventeen patients who reached this level of severity were described as having an anxiety state rather than depression. This left eleven patients who form the subject group in this study. Five were from one practice, four from another and one from each of the remaining two.

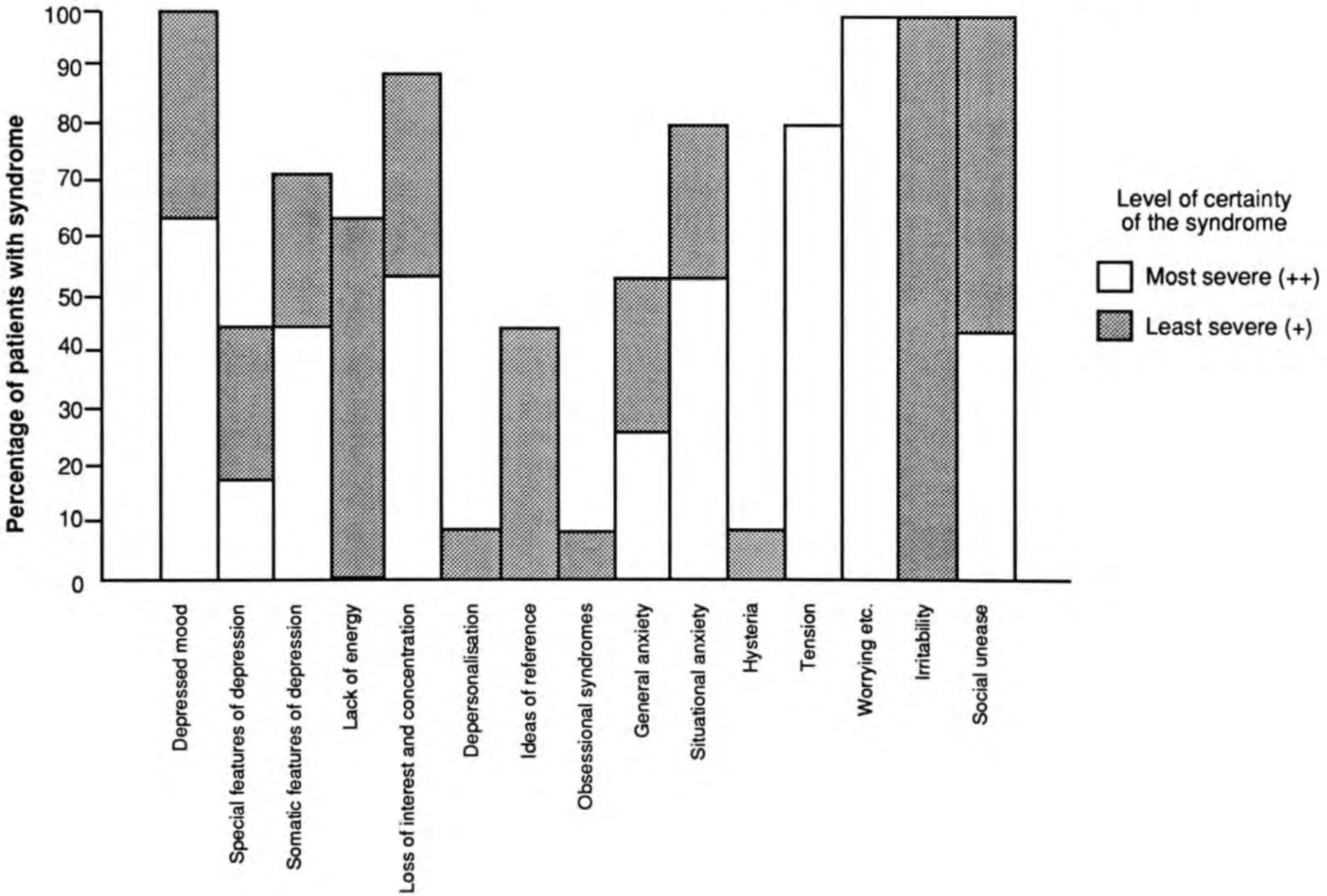
Six of these women reached Index of Definition Level Five: five were assigned to Catego Class 'N' (neurotic depression) and one to Class R (retarded depression). The remaining five women reached Level Six; four were assigned to Class N and one to Class R.

The CATEGO program can be used to produce a 'syndrome profile' of patients. Descriptions of the various syndromes and an explanation of the process by which they are produced can be found in Section 3.3.3. Figure 8.1 gives the syndrome profile of the subjects in this study. It shows the percentage of patients experiencing each syndrome at one of two levels of severity: + or ++ according to the pattern and severity of symptoms within that syndrome. It can be seen that all the women had the syndrome 'depressed mood' which is characterised by hopelessness, inefficient thinking and suicidal ideation in addition to depressed mood. Somatic symptoms of depression (syndrome 'OD') were present in about 70% of the group, while special features of depression (ED), such as self - depreciation and pathological guilt, were present in 45%.

As would be expected in a group of depressed women (Section 6.1), the majority had some anxiety symptoms: 80% reported experiencing situational anxiety (SA) and 54% had autonomic anxiety symptoms and/or experienced panic attacks (GA). Non-specific symptoms were also very common: over 80% had the syndrome TE (tension pains, muscular tension and rest-lessness), while all the women had the syndromes WO (worry, tiredness, nervous tension etc) and SU (social unease). Loss of interest and concentration were reported by 90% of the women (IC).

FIGURE 8.1

CATEGO SYNDROME PROFILE OF THE DEPRESSED PATIENTS, SHOWING THE PERCENTAGE OF PATIENTS WITH THE SYNDROME IN ITS MOST SEVERE FORM (++), AND THE LEAST SEVERE FORM (+)



Control Group

Once a woman had fulfilled the first two criteria for the study and agreed to a home interview for the P.S.E, attempts were made to match her to a suitable control. The next patient who was up to five years younger or older than the putative subject, and who scored less than two on the '10 Questions', was included in the study as a control. Eleven patients screened for psychiatric disorder and within five years of age of a study patient were recruited in this way.

The control and depressed patients were not matched for social class or educational level. This was because there was no evidence from previous studies that these factors were related to recall of medical information, unless it is presumed that high social class or educational level are linked to medical knowledge, which is related to recall (Ley and Spelman, 1967). In addition the practicalities of recruiting control subjects from women visiting their GP made it time-consuming to get enough subjects of the right age: it was not felt feasible to match on other variables as well.

8.2.4 Analysis

Transcripts of Consultations

The recordings of the consultations were transcribed and everything the doctor said was divided into statements on the basis of predetermined rules. A statement was defined as one piece of information. This did not necessarily correspond to a sentence: if it was felt that a patient could remember one part of the sentence and forget another then it contained more than one statement. For instance the sentence 'you have got high blood pressure and I want you to come back in a fortnight to test it again' consists of four statements:

(1) you've got high blood pressure
(2) I want you to come back
(3) in a fortnight
(4) to test it again

During consultations doctors frequently repeated information. A statement was classified as a repeat if it contained the same information as a previous one, not just if exactly the same words was used. Thus 'come back in two weeks' was classified as a repeat of 'I'll see you in fortnight'.

Once the transcripts had been divided into statements, each statement was assigned to a category. These corresponded to the areas covered in the recall questions and were as follows:

(1) Diagnosis
(2) Explanations of diagnosis, symptoms and signs
(3) Treatment; what it is, what side-effects there are, why the treatment is given
(4) Prognosis; whether the condition will get better, how long this will take
(5) Further visits and tests
(6) Instructions and advice
(7) Reassurance
(8) Other statements

Some statements were difficult to assign to any one category. When this was the case the statement was placed in the first category which was appropriate in the above list. For example a statement which might be classed as information about a treatment or as an instruction was placed in the treatment category.

Recall Interview Transcripts

The recall interviews were transcribed and patients' statements relating to what the doctor had said during the consultation were classified in the same way as those derived from the consultation itself. They were then matched against the transcripts of the consultations to ensure that the doctor had actually said what the patient had recalled them as saying. Statements recalled in response to the first general question were analysed separately from those recalled following the other, more specific, recall questions.

Statistical Analysis

T-tests were used to test the significance of differences between the two subject groups, except where the data was not suitable for a parametric test, in which case a Mann-Whitney U test was used instead. Product-Moment correlation co-efficients were calculated to assess the relationship between variables; Spearman's rho was used for data that was not normally distributed. Regression analysis was used to explore the relationship between several variables which had previously been shown to affect memory for medical information (Section 8.1) and recall: a backwards regression program was used to identify the best predictors of recall.

8.3 Results

8.3.1 Content Analyses

As described in Section 8.2.4 content analysis was used to divide the transcripts into statements for analysis. This process demands some judgment and it is possible that the resulting data was biased in some way. The content analysis was therefore done twice: once by the investigator and once by someone blind to the hypothesis under investigation. The relationship between the data from the two content analyses was explored. Product-Moment correlation coefficients were calculated between the two sets of data for the number of statements given, number recalled and percentage recalled. In all cases the correlation was positive and high (number of statements given, $r=.85$, $df=20$, $p<0.001$; number recalled, $r=.76$, $df=20$, $p<0.001$; percentage recall, $r=.72$, $df=20$, $p<0.001$). Whilst there was not total agreement between the two sets of data the strength of the relationship was felt to be satisfactory.

The relationship between the number of statements allocated to the different categories in the two content analyses was also determined, this time using Spearman's rho as the data tended to be very skewed. The median number of statements given in each category and the appropriate rho value are given in Table 8.1. In all cases the relationship between

the two sets of data is significant at the 1% level, and in all but three cases rho was larger than .7. In the remaining three cases it ranges from .67 for statements in the 'visit' category to only .56 to those assigned to the 'reassurance' category. These figures cast doubt on the reliability of these categories and suggest that the judges had particular difficulty in deciding whether or not to allocate a statement to the reassurance category. This means that the results obtained for this category need to be treated with some caution.

In addition to determining the relationship between the data derived from the two content analysis, both sets of data were analysed in the same way to see if the same pattern of results were obtained. Overall this was the case; in particular it should be noted that the pattern of results regarding differences in recall between the groups, and the relationship of this to other factors which might affect recall, did not differ between the sets of data. There were some minor discrepancies, particularly with the individual categories; this again suggests that the results for some of these should be regarded with some caution.

Because there are high positive correlations between the scores obtained from the two analyses for the main variables (amount of information given, number of statements recalled and percentage recall), and the overall pattern of results did not differ in any important respects between the two analyses, it is concluded that the results obtained from these analyses are reliable, except, perhaps for those from some of the categories.

Unless otherwise specified the results presented here are from the content analysis carried out blind (Content Analysis 2 in Table 8.1).

TABLE 8.1 REPLICATION OF CONTENT ANALYSIS: RELATIONSHIP BETWEEN THE TWO SETS OF DATA

	CONTENT ANALYSIS ONE (by investigator)		CONTENT ANALYSIS TWO (blind)			
	MEAN	(S.D)	MEAN	(S.D)	r	p
Number of statments given	22.5	(7.7)	19.6	(7.9)	.85	<0.001
Number of statements recalled	10.5	(4.3)	7.3	(2.7)	.76	<0.001
Percentage recall	48.3	(14.3)	39.8	(16.3)	.72	<0.001
CATEGORY	MEDIAN	(MIN, MAX)	MEDIAN	(MIN, MAX)	rho	p
Questions	11.5	(1, 63)	10	(1, 63)	0.99	<0.01
Diagnosis	0	(0, 9)	0	(0, 5)	0.83	<0.01
Explanation	4	(0, 21)	2	(0, 14)	0.83	<0.01
Treatment	6	(0, 18)	2	(0, 12)	0.72	<0.01
Prognosis	1	(0, 7)	2	(0, 5)	0.78	<0.01
Further visits	1.5	(0, 8)	1	(0, 5)	0.67	<0.01
Instructions	1	(0, 12)	6	(0, 15)	0.61	<0.01
Reassurance	1	(0, 4)	3.5	(0, 7)	0.56	<0.01
Other	6.5	(0, 25)	9	(0, 25)	0.75	<0.01

8.3.2 Patient Characteristics

Although all depressed patients were matched for age (+/- five years) with non-depressed patients, they were not matched for social class or educational level. It was therefore necessary to check that the distribution of social class and educational level did not differ between the two groups.

The mean number of years spent in education are given in Table 8.2; the mean for the depressed and non-depressed patients did not differ on this variable. Mean age in the two groups is also shown. As the groups were matched for age it was expected that this would not differ significantly between the two groups; this was the case (a paired t-test was used because of the matching, t=-.64, df=10, p>0.05). The numbers in each social class also did not differ between the groups (U=67.5, U'=53.5, critical value for U when Na=10 is 23, p>0.05: data was missing for two patients).

The relationship of these variables to the number of items correctly recalled was also determined. Years spent in full time education was not significantly related to recall (r=.07, N=22, p>0.05), whilst social class was; the higher the social class, the more the subject remembered (rho=.04, p<0.05). Age was not significantly related to recall (r=-.07, n=22, p>0.05), but, as already described, the subjects had been matched on this variable. This raised questions about the most appropriate statistical tests to use in the remainder of the analysis: if the samples were matched in such a way that their scores were correlated then a paired t-Test would be appropriate as this presumes that much of the variation in scores between individuals has been removed. The subjects in this study had been matched, but on a variable which proved to be unrelated to recall; matching has not, therefore, reduced the variability in scores as it had been expected to do. This suggests that a t-Test for independent samples should be used. In order to check this, recall scores for the matched pairs were correlated to see if they were related. The relationship was low and insignificant (r=.18, df=9, p>0.05). There is, therefore no evidence that the two samples are correlated. Consequently it was decided not to use paired t-Tests in this analysis.

TABLE 8.2 PERSONAL CHARACTERISTICS OF SUBJECTS

	DEPRESSED PATIENTS MEAN	S.D.	NON-DEPRESSED PATIENTS MEAN	S.D.	U	NA	NB	t	(df)	p
Age (years)	36.7	(11.2)	37.5	(13.1)				-.64	(10)	NS
Years of education	11.9	(2.7)	11.9	(2.1)	54	11	11			NS

Social Class	DEPRESSED PATIENTS N	(%)	NON-DEPRESSED PATIENTS N	(%)
1	1	(9.1)	0	(0)
2	2	(18.2)	4	(36.4)
3N	2	(18.2)	3	(27.3)
3M	4	(36.4)	1	(9.1)
4	0	(0.0)	1	(9.1)
5	1	(9.1)	1	(9.1)

FIGURE 8.2

TOTAL AMOUNT SAID TO DEPRESSED AND NON-DEPRESSED SUBJECTS DURING THE GENERAL PRACTICE CONSULTATION (MEAN±SD)

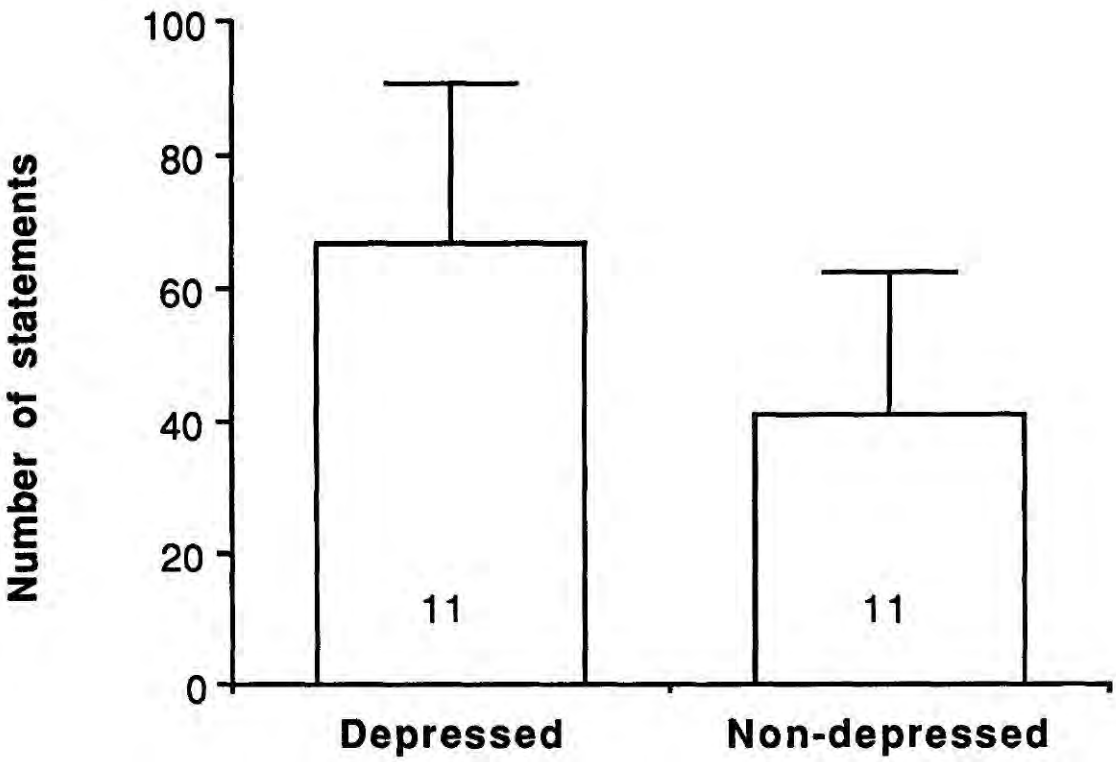


FIGURE 8.3

NUMBER OF ITEMS OF INFORMATION GIVEN TO DEPRESSED AND NON-DEPRESSED SUBJECTS (MEAN±SD)

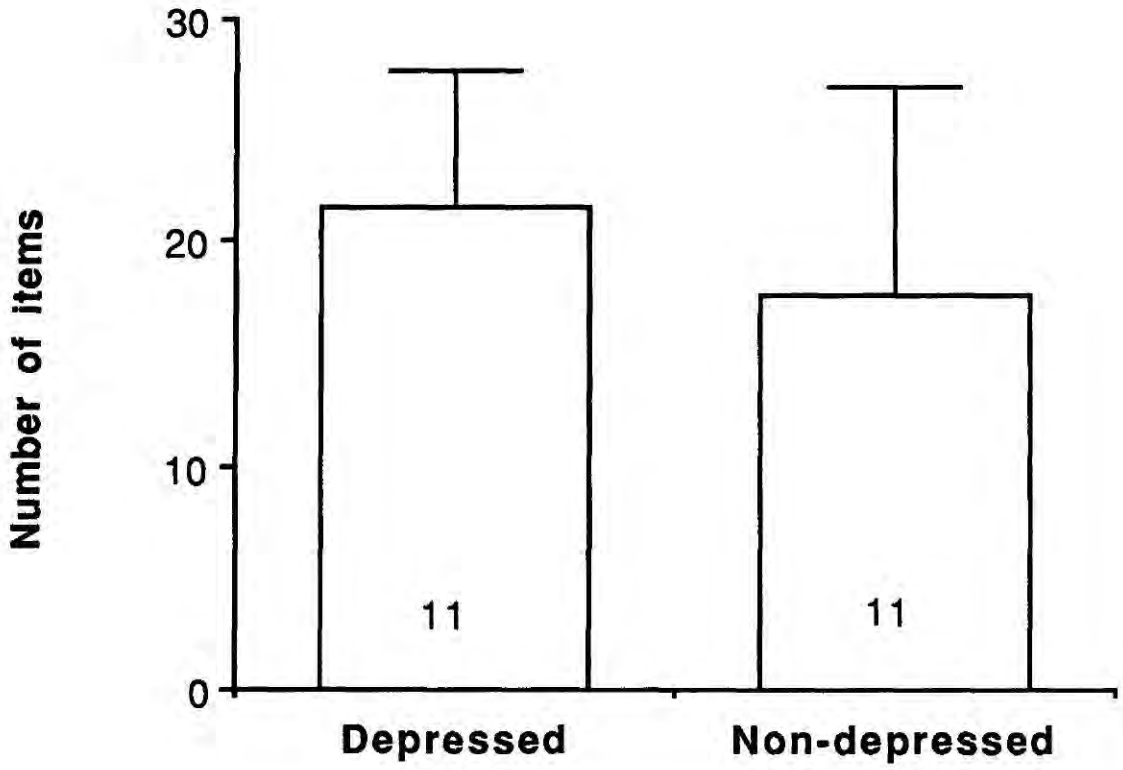
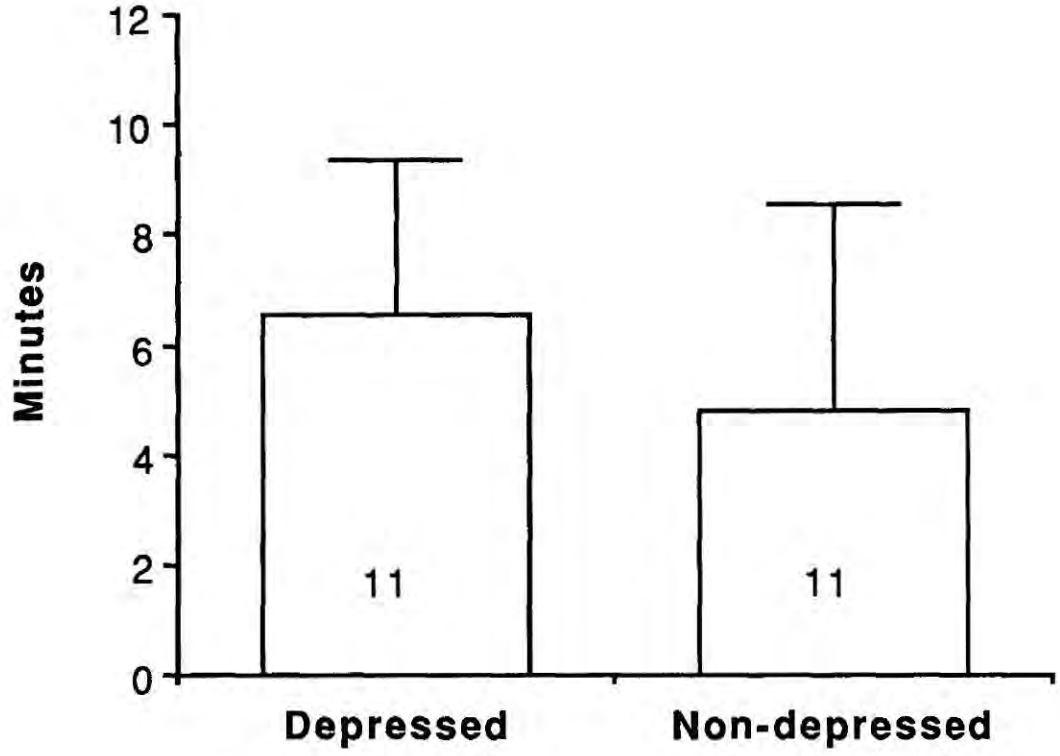


FIGURE 8.4

LENGTH OF CONSULTATION OF DEPRESSED AND NON-DEPRESSED SUBJECTS (MEAN±SD)



8.3.3 Characteristics of the Consultations

There was considerable variation in the total amount the doctors said during the consultation. When questions, social conversation and repeated information are included the total amount said ranged from 16 to 112 statements, with a mean of 53.9. The doctors said significantly more to the depressed than the non-depressed patients, as illustrated in Figure 8.2 (t=2.68, p=0.01).

As the aim of this study is to look at how well patients remember the information they are given by the doctor, statements which did not contain information and therefore did not fit into one of the first seven recall categories (Section 8.2.4) were excluded from the analysis. Such statements included questions and those classified into 'other statements', typically conversation about holidays or the children. In addition statements which are repeats of previous statements were also excluded as these did not increase the amount of information the patient had to remember. The remainder of the analysis is therefore concerned with the information the doctor gave the patient, and the patient's memory for it.

The doctors gave the patients a mean of 19.6 statements containing information, with a range of 7 to 35. There was no significant difference between the depressed and other patients in the amount of information they were given. (t=1.17, p>0.05, Figure 8.3). The mean length of the consultation also did not differ significantly between the groups (the distribution of this variable was skewed, so the data was squared to normalise it, t=.68, p>0.05, Figure 8.4).

FIGURE 8.5

NUMBER OF STATEMENTS CORRECTLY RECALLED BY DEPRESSED AND NON-DEPRESSED SUBJECTS (MEAN±SD)

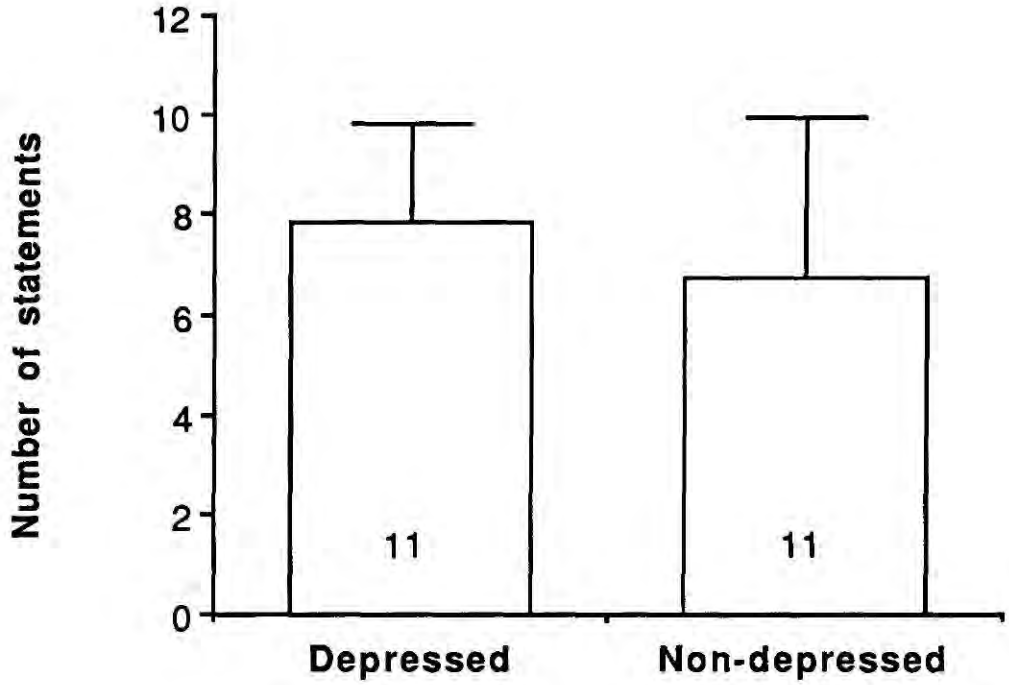
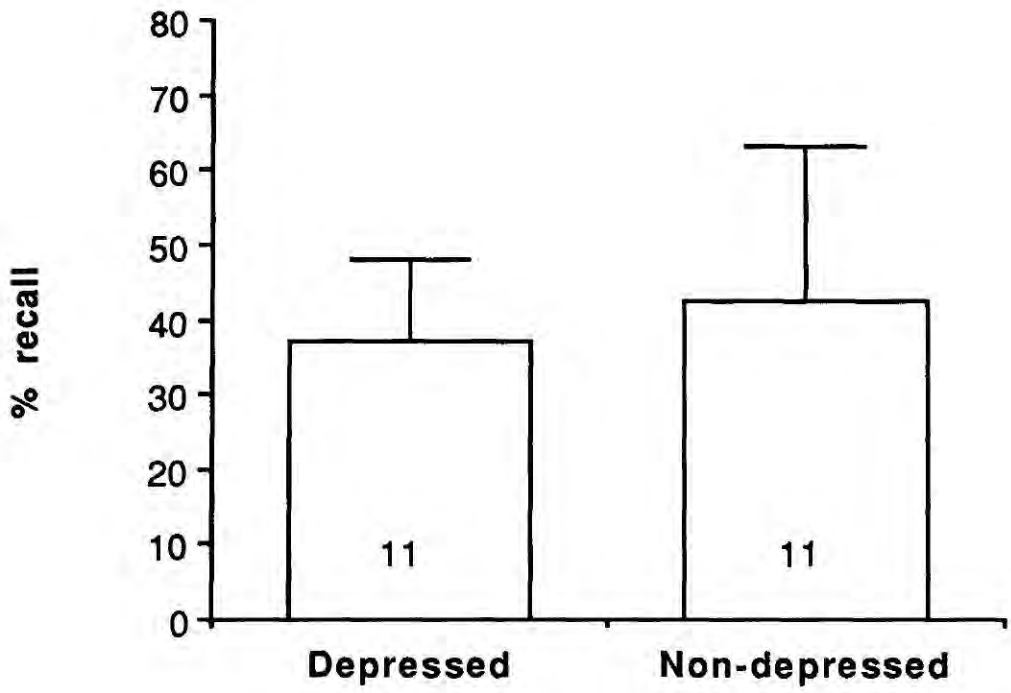


FIGURE 8.6

PERCENTAGE RECALL OF INFORMATION BY DEPRESSED AND NON-DEPRESSED SUBJECTS (MEAN±SD)



8.3.4 Amount Recalled

The mean number of statements recalled by the patients was 7.2. This ranged from 3 to 12 and, as shown in Figure 8.5, did not differ significantly between the two groups ($t=.94$, $p>0.05$). Percentage recall scores were also calculated as these take into account the amount of information that the patients were given. On average the patients recalled 39.8% of the information presented by the doctor. The F-test for uniform variances approached significance in this case ($f(10,10)=.27$, $p=.053$), and therefore a log transformation was used to equalise the variances between the groups. The difference between the two groups was not statistically significant ($t=-.48$, $p>0.05$, Figure 8.6).

8.3.5 Factors Affecting Recall

It would be premature to conclude that depressed patients do not have impaired memory for medical information before looking at several aspects of the consultation which have been shown in previous studies to affect memory for this type of material (Section 8.1). The consultations of the depressed and non-depressed subjects may have differed in such a way that those of the depressed patients were easier to recall than those of the non-depressed. If this was the case the depressed patients should have recalled more information than the other patients: the fact that they did not would therefore indicate that their memories were impaired despite the apparent evidence to the contrary.

Regression analysis was therefore used to investigate the difference between the two groups of patients whilst statistically controlling for the effect of variables which affect recall. The number of items recalled was the dependent variable in each case. Each variable which might affect recall was considered in turn. First, this variable was entered to the regression equation by itself, and then Group (depressed/nondepressed) was added to this. The order was then reversed. If depression did affect recall then Group would explain a significant amount of the variance when added to the equation containing the other variable. The results are given in Table 8.3.

TABLE 8.3 MEMORY FOR MEDICAL INFORMATION: THE AMOUNT OF VARIANCE (R^2) EXPLAINED IN THE NUMBER OF STATEMENTS RECALLED WHEN THE PREDICTOR VARIABLE ENTERS THE REGRESSION EQUATION BEFORE GROUP, AND WHEN THE ORDER IS REVERSED.

PREDICTOR VARIABLE	R^2 EXPLAINED BY: PREDICTOR VARIABLE + GROUP		R^2 EXPLAINED BY: GROUP + PREDICTOR VARIABLE		RESIDUAL D.F
Total amount said	.01 .04		.04 .00		19
Number of items of information	.46 .46	**	.04 .46	**	19
Length of consultation	.01 .07		.04 .07		19
Number of repeated statements	.25 .25	*	.04 .25	*	19
Number of times repeated statements were given	.05 .06		.04 .06		19
Visits to doctor in past year	.12 .23		.08 .23		16
Visits to doctor in past month	.00 .04		.04 .00		18

Significance of increase in R^2, * $p<0.05$ ** $p<0.01$

Total Amount Said

Figure 8.2 shows the total amount said to the depressed and non-depressed patients. As indicated above, the difference between the two groups in the total amount said to them during the consultation was significant: the doctors said more to their depressed patients. Regression analysis was therefore used to explore the relationship between this variable, Group (depressed/non-depressed) and recall.

First, Group was entered into the equation by itself (this is included for illustration only: as there were no significant differences between the depressed and non-depressed patients the amount of variance explained by group could not be significant). Group explained four per cent of the variance in the dependent variable; as expected this was not significant.

The total amount said was then entered into an equation by itself. As the results in Table 8.3 show, it did not explain a significant amount of variance when entered by itself. Group was then added to this equation. It did not cause a significant increase in the amount of explained variance. Thus there was no significant relationship between group membership and recall, even when the effects of the difference in the total amount said between the groups was held constant.

Items of Information Given

The number of items of information given to the patients explained a significant amount of variance when entered in the regression equation by itself: as would be expected, the more information the patient was given the more she recalled (a patient could only recall ten items if she had been given that many in the first place). This variable also significantly increased the amount of explained variance when added to the regression equation containing Group. The reverse was not true: Group did not explain a significant amount of variance when alone in the regression equation or when added to the equation containing the number of items of information given. There is, therefore, no evidence that the depressed patients have a memory impairment which is hidden by the effects of this variable.

FIGURE 8.7

NUMBER OF STATEMENTS REPEATED TO DEPRESSED AND NON-DEPRESSED SUBJECTS (MEAN±SD)

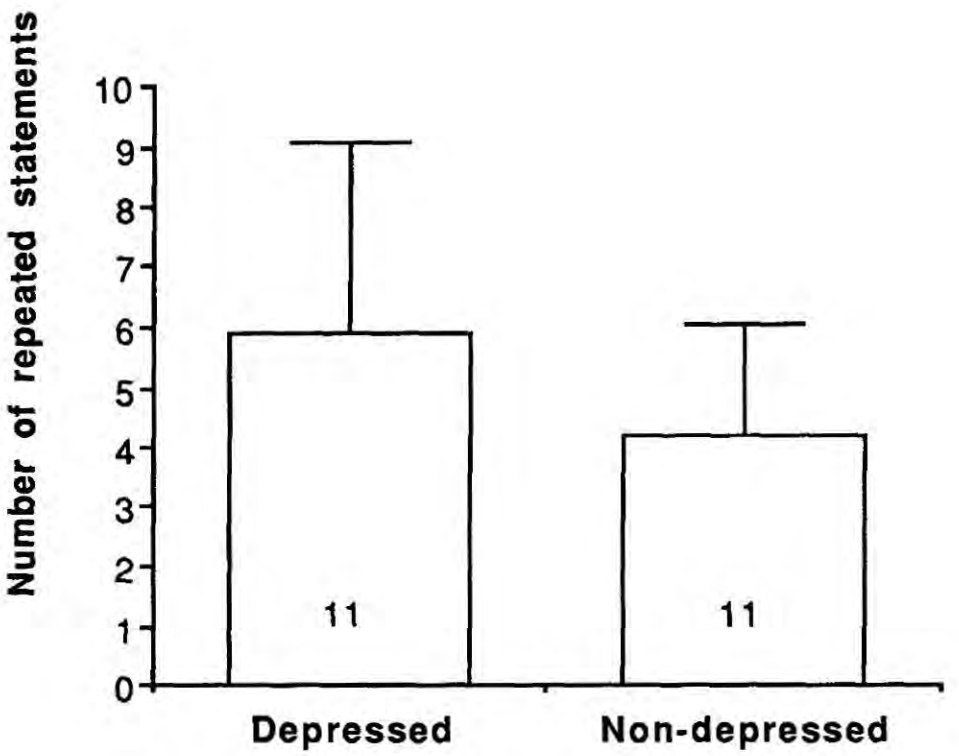
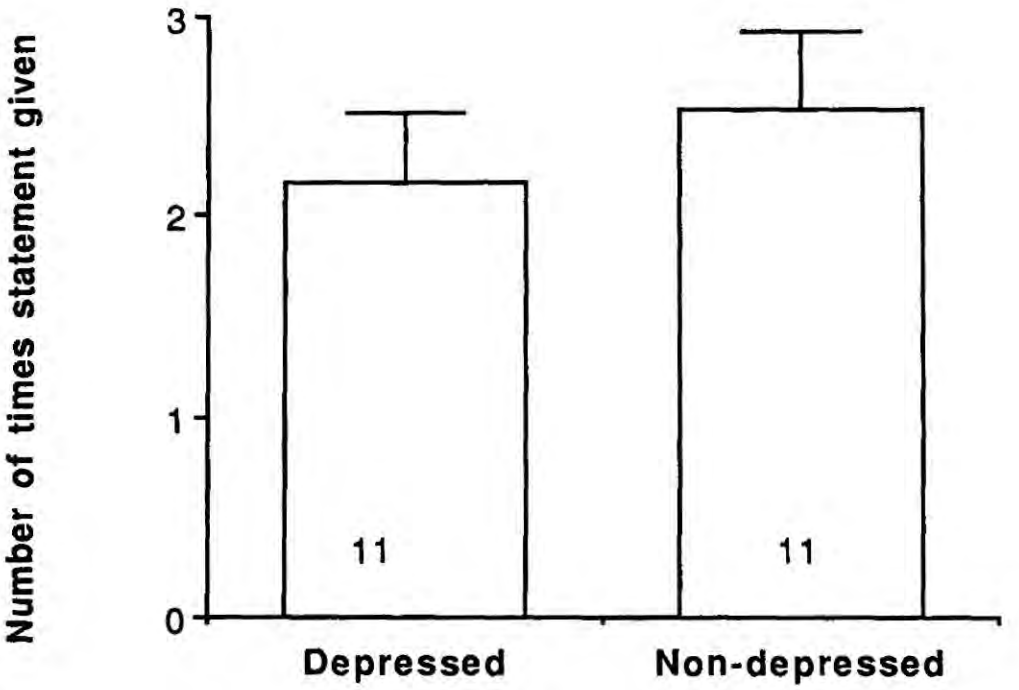


FIGURE 8.8

NUMBER OF TIMES THAT REPEATED STATEMENTS WERE GIVEN TO DEPRESSED AND NON-DEPRESSED SUBJECTS (MEAN±SD)



Length of Consultation

The length of the consultation did not explain a significant amount of variance when entered into the regression equation by itself, or when added to Group. Group also did not cause a significant increase in the amount of explained variance at any stage of the proceeding. Again this substantiates the original finding; that the recall of the depressed and non-depressed patients does not differ significantly.

Repeats

Ley (1979) reported that recall can be improved by repeating information. Consequently if the consultations of the depressed patients contain more repeated statements than those of the non-depressed patients, then they would be easier to recall: the depressed patients would be expected to recall more information than the non-depressed and the fact that they did not would indicate that they did have a memory impairment.

Doctors repeated a mean of 5.9 statements to the depressed patients and a mean of 4.2 to the other patients, with an overall mean of 5.1 (Figure 8.7). Although the difference was in the predicted direction it did not approach significance ($t=1.55$, $p>0.05$). The number of repeated statements caused a significant increase in the amount of variance explained when entered into the regression equation. The regression coefficient was positive (0.5) indicating that, as predicted, an increase in the number of repeated statements was related to an increase in the amount of information recalled. In contrast, Group did not explain a significant amount of variance when entered alone in the equation or when added to the number of repeated statements. This indicates that the depressed patients do not have a memory impairment which is covered up by the effects of the number of repeated statements.

The number of times that repeated statements were given was also explored. This differed significantly between the two groups: statements repeated to the depressed subjects were given on average 2.5 times, whilst those repeated to non-depressed subjects were given 2.2 times

(t=-2.22, p<0.05; Figure 8.8). In contrast to the number of repeated statements this did not cause a significant increase in the amount of explained variance when entered alone in the regression equation and was not, therefore, significantly related to recall. Again, Group did not explain a significant amount of variance when alone in the regression equation or when added to the equation containing the other predictor variable. Although this variable did differ between the groups, there is no evidence that it made the consultations of the depressed group easier to recall than those of the non-depressed subjects: if it had done then Group would have been significantly related to recall on this variable and caused a significant increase in the amount of explained variance when added to the equation containing the number of times that repeated statements were given.

Previous Visits to the Doctor

The number of visits the patient has made to the general practitioner in the recent past may affect recall. For instance the information may be familiar to the patient and therefore easier to recall, or information given at previous consultations may interfere with memory for new facts the patient is given. The patients were therefore asked how many times they had visited the doctor in the previous month, and in the previous year (Figure 8.9).

Mann-Whitney U tests were used to analyse the difference between the depressed and non-depressed subjects in the number of recent visits because the distribution of the data deviated considerably from normal. The two groups did not differ in the number of consultations in the past month (U=54, U'=56, NA=11, ns at 5% level) or in the past year (U=26.5, U'=65.5, NA=9, ns at 5% level; Table 8.3). Neither the number of visits in the previous year nor in the previous month caused a significant increase in the amount of explained variance when entered alone in the regression equation (Table 8.3). In both cases Group also did not cause a significant increase in variance when added to the regression equations. There is no evidence, therefore, that the recall of the depressed and non-depressed patients differ significantly once the effects of the number of visits in the past year and past month are held constant.

FIGURE 8.9

NUMBER OF VISITS TO THE DOCTOR BY DEPRESSED AND NON-DEPRESSED SUBJECTS IN THE PAST MONTH, AND THE PAST YEAR

TABLE 8.4 CORRELATION MATRIX OF VARIABLES WHICH MAY HAVE BEEN RELATED TO RECALL OF MEDICAL INFORMATION

	GROUP	ITEMS OF INFORM-ATION	TOTAL AMOUNT SAID	LENGTH OF CONSULT-ATION	NUMBER OF REPEATED STATE-MENTS	NUMBER OF TIMES REPEATED S'MENTS GIVEN	VISITS IN MONTH	VISITS IN YEAR
Group	1.00 (22)							
Number of items of information	.25 (22)	1.00 (22)						
Total amount said	.51 * (22)	.66 ** (22)	1.00 (22)					
Length of consultation	.27 (22)	.50 * (22)	.85 ** (22)	1.00 (22)				
Number of repeated statements	.44 * (22)	-.02 (22)	.17 (22)	.01 (22)	1.00 (22)			
Number of times repeated statements given	.36 (22)	.68 ** (22)	.47 * (22)	.36 (22)	.11 (22)	1.00 (22)		
Visits in past month	.04 (21)	-.01 (22)	-.16 (21)	-.07 (21)	-.10 (21)	.13 (21)	1.00 (21)	
Visits in past year	.12 (19)	.02 (19)	.25 (19)	.19 (19)	.04 (19)	.17 (19)	.22 (19)	1.00 (19)

Pearson correlation coefficients, * $p<0.05$ ** $p<0.01$, two tailed significance. (N) Number of subjects

To summarise, there is no evidence that the consultations of the depressed patients were easier to recall than those of the non-depressed patients because of differences between the groups in aspects of the consultations that were related to recall. Group did not explain a significant amount of the variance in recall when placed in a regression equation by itself and, more importantly, did not cause a significant increase in the amount of explained variance when added to the regression equations containing any of the other predictor variables. There is therefore no evidence that the recall of the two groups differed once the effects of other variables such as the length of the consultation and the number of repeats were taken into consideration.

The regression analyses also showed that only some of the variables hypothesised to affect recall in fact did so. Only the amount of information given by the doctor and the number of repeated statements were related to recall. However several of the independent variables used were clearly inter-related, as shown by the correlation matrix given in Table 8.4. For instance the total amount the doctor said was related to the amount of information he gave, and to the length of the consultation. In addition the number of repeated statements was also clearly related to the amount of information given. This means that the relationship of one variable with recall may be obscured by the effects of a related variable. Regression analysis was therefore used to investigate the relationship of these variables with recall while holding the effect of other related variables constant. Group was included in the equation to check that it was not related to recall when not only the factors which may affect recall but also the relationships between them were taken into consideration.

TABLE 8.5 PARTIAL REGRESSION COEFFICIENTS OF THE PREDICTOR VARIABLES IN A REGRESSION EQUATION USED TO INVESTIGATE FACTORS AFFECTING THE NUMBER OF ITEMS OF INFORMATION RECALLED BY PATIENTS AFTER A G.P CONSULTATION

PREDICTOR VARIABLES	REGRESSION COEFFICIENTS	t	p
Group	0.58	0.67	NS
Number of items of information given	0.33	5.65	<0.001
Total amount said	-0.03	-0.88	NS
Length of consultation	0.29	-1.49	NS
Number of items repeated	0.01	0.05	NS
Number of times repeated statements given	1.84	2.56	<0.05
Visits in past month	-0.10	-0.25	NS
Visits in past year	-0.23	-2.05	NS
(Constant)	(-2.50)		

Relationship between Different Aspects of the Consultation and their Relationship to Recall

All eight predictor variables which were hypothesised to affect recall were entered in the same regression equation: as before, the dependent variable was the number of items correctly recalled. The variables were: Group; the amount said to the patients; the amount of information they were given; the length of the consultation; the number of repeated statements; the number of times statements were given; the number of visits in the previous year and the number of visits in the previous month. Together they explained 90% of the variance in the number of statements recalled: this was highly significant ($F(8,10)=14.1$, $p<0.01$). Table 8.5 gives the partial regression coefficient for each variable, together with the associated t value and significance level.

The total amount of information given by the doctor was still a highly significant predictor of recall when the effects of other predictor variables were controlled for. However the number of repeated statements, which significantly predicted recall when alone in the equation, was no longer significant when the effects of other independent variables were taken into consideration. The number of repeated statements must depend on the total number of statements given: the more statements that are given, the more that can be repeated. It is therefore hypothesised that the significant effect of this when alone in the equation was a consequence of its relationship to the total number of statements given.

In contrast, the number of times repeated statements were given explained a significant amount of the variance when combined with the other variables: the more often repeated statements were given, the more the patient recalled. None of the other variables reached significance and, most importantly, Group did not have a significant effect on recall when the effects of all the other predictor variables were held constant.

A backwards regression program was then used to remove from the equation the variable which produced the smallest change in R^2 when the effects of all the other predictor variables were controlled for. The equation was then re-computed and the procedure repeated until only variables which explained a significant amount of the variance remained

in the equation. The number of statements which were repeated was removed first, followed by the number of visits in the past month. Group was removed in the third step, and like the above variables, caused a reduction in the amount of variance explained of less than one per cent (R^2 change=-.005, f(1)=.55, p>0.05). It is clear that this was not a significant predictor of performance. This therefore supports the conclusion drawn above; there was no evidence that depressed patients had a memory impairment which was hidden by differences between the consultations of the two groups of patients in the prevalence of factors which were related to recall.

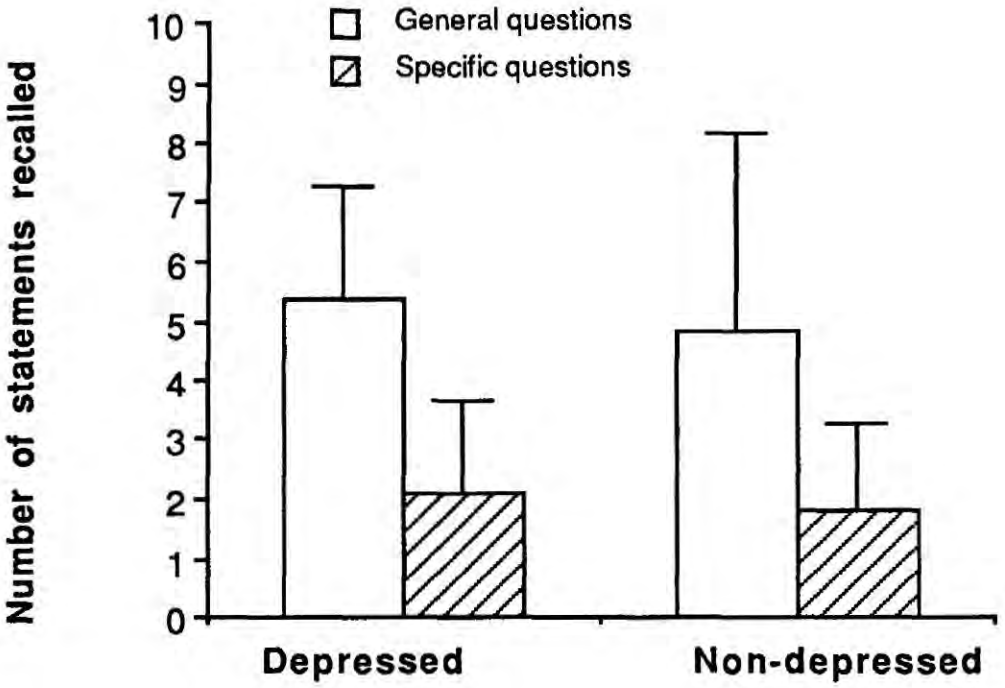
Although Group had been removed from the equation the backwards regression program was continued to see which factors emerged as the best predictors of performance. The total amount said during the consultation was removed on the next step (R^2 change=-.004, f=.57, p>0.05), and the final regression equation therefore contained the following independent variables: the amount of information given; the length of the consultation; the number of times repeated statements were repeated and the number of consultations in the previous year. It accounted for 89% of the variance in the amount recalled by the patients: this was highly significant (f(4,14)=28.5, p<0.0001). The regression equation was as follows:

Amount recalled=-.25 + 0.31 (amount of information) -.43 (length of consultation) +1.85 (times repeated statements repeated) -.26 (visits in past year).

Patients recalled more information the more they were given and the more repeated statements were repeated. Patients with long consultations recalled less than those with shorter ones, and those who had visited the doctor often in the past year recalled less than those with few previous visits.

FIGURE 8.10

NUMBER OF STATEMENTS RECALLED BY THE DEPRESSED AND NON-DEPRESSED SUBJECTS IN RESPONSE TO THE GENERAL AND SPECIFIC QUESTIONS (MEAN±SD)



Other Factors which may Affect Recall

There are two other factors which may affect recall which have not been investigated so far. These are the effect of prompting the patient's memory and the different types of information given in the consultation. These need to be considered before final conclusions are drawn about the memory for medical information of the depressed patients in this study.

The Effect of Cueing Recall

The interviewer cued the patients' memory by asking about specific areas which the doctor may have covered (see Section 8.2.1). Figure 8.10 shows the number of unique statements that were recalled in response to the general question (free recall) and to the more specific ones (cued recall). The free recall variable was transformed using a log transformation because the distribution was skewed, causing the normal plot of the residuals to deviate considerably from the linear. The number of statements recalled in response to the free recall question did not differ significantly between the two groups ($t=.38$, $p>0.05$) and neither did the number recalled in response to the questions intended to prompt memory ($t=.42$, $p>0.05$). There is, therefore, no evidence that cueing helped the depressed patients overcome a memory problem which otherwise would have been evident in this situation.

Type of Information Given to the Patients

As summarised above (Section 8.1) previous studies have found that some types of information are remembered better than others; for instance Ley and Spelman (1965) found that instructions, prognostic statements and reassurance were forgotten more often than other types of information.

TABLE 8.6 NUMBER OF STATEMENTS GIVEN BY GPs IN EACH CATEGORY OF INFORMATION

TYPE OF STATEMENT	DEPRESSED PATIENTS MEDIAN	(MIN, MAX)	NON-DEPRESSED PATIENTS MEDIAN	(MIN, MAX)	U	U'	P
Reassurance	4.0	(2, 7)	2.0	(0, 6)	21.5	99.5	<0.05
Questions	11.5	(7, 63)	6.5	(1, 37)	31.5	89.5	>0.05
Other statements	12.0	(0, 25)	6.0	(3, 20)	38	83	>0.05
Diagnosis	0.0	(0, 5)	0.0	(0, 4)	58	129	NS
Explanation	3.0	(0, 14)	1.0	(0, 11)	41.5	79.5	NS
Treatment	3.0	(0, 10)	2.0	(0, 12)	47.5	73.5	NS
Prognosis	2.0	(0, 5)	1.0	(0, 5)	46	71	NS
Further visits	1.0	(0, 2)	1.0	(0, 5)	77.5	43.5	NS
Instructions	6.0	(0, 15)	5.0	(2, 12)	59	62	NS

FIGURE 8.11

NUMBER OF STATEMENTS IN EACH CATEGORY GIVEN TO THE DEPRESSED AND NON-DEPRESSED SUBJECTS (MEDIAN)

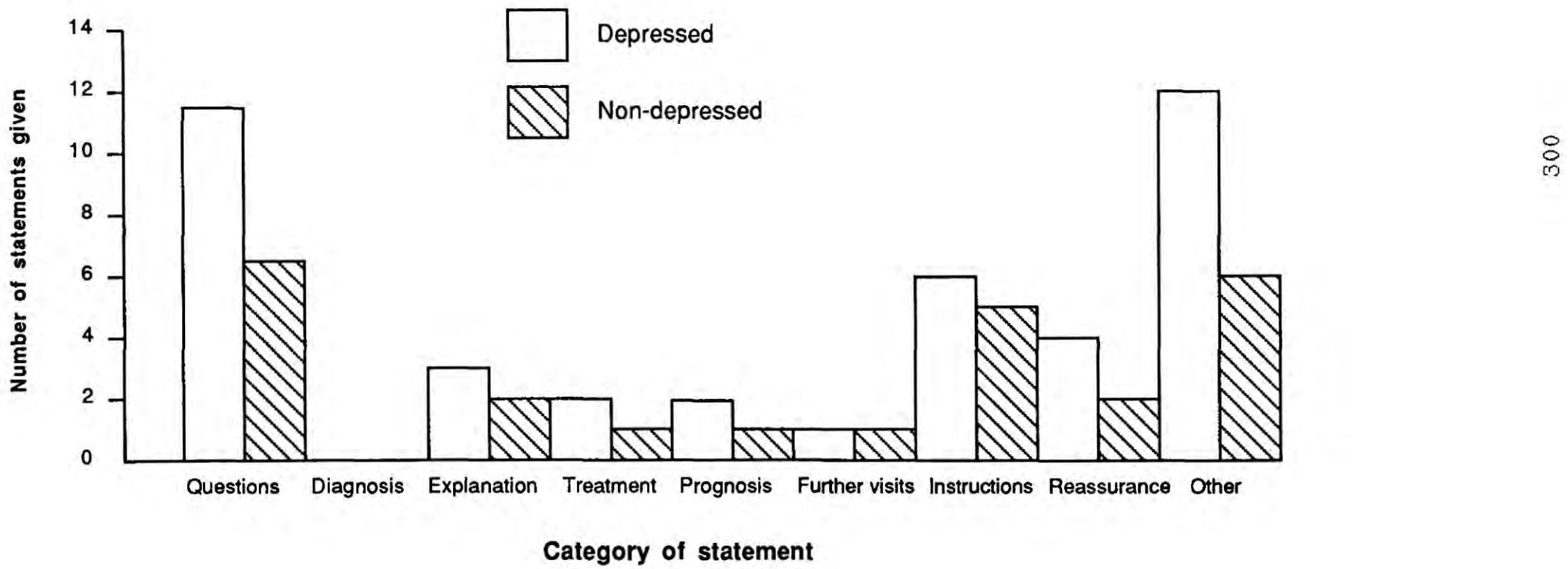


FIGURE 8.12

PERCENTAGE RECALL OF INFORMATION IN EACH CATEGORY BY THE DEPRESSED AND NON-DEPRESSED SUBJECTS (MEDIAN)

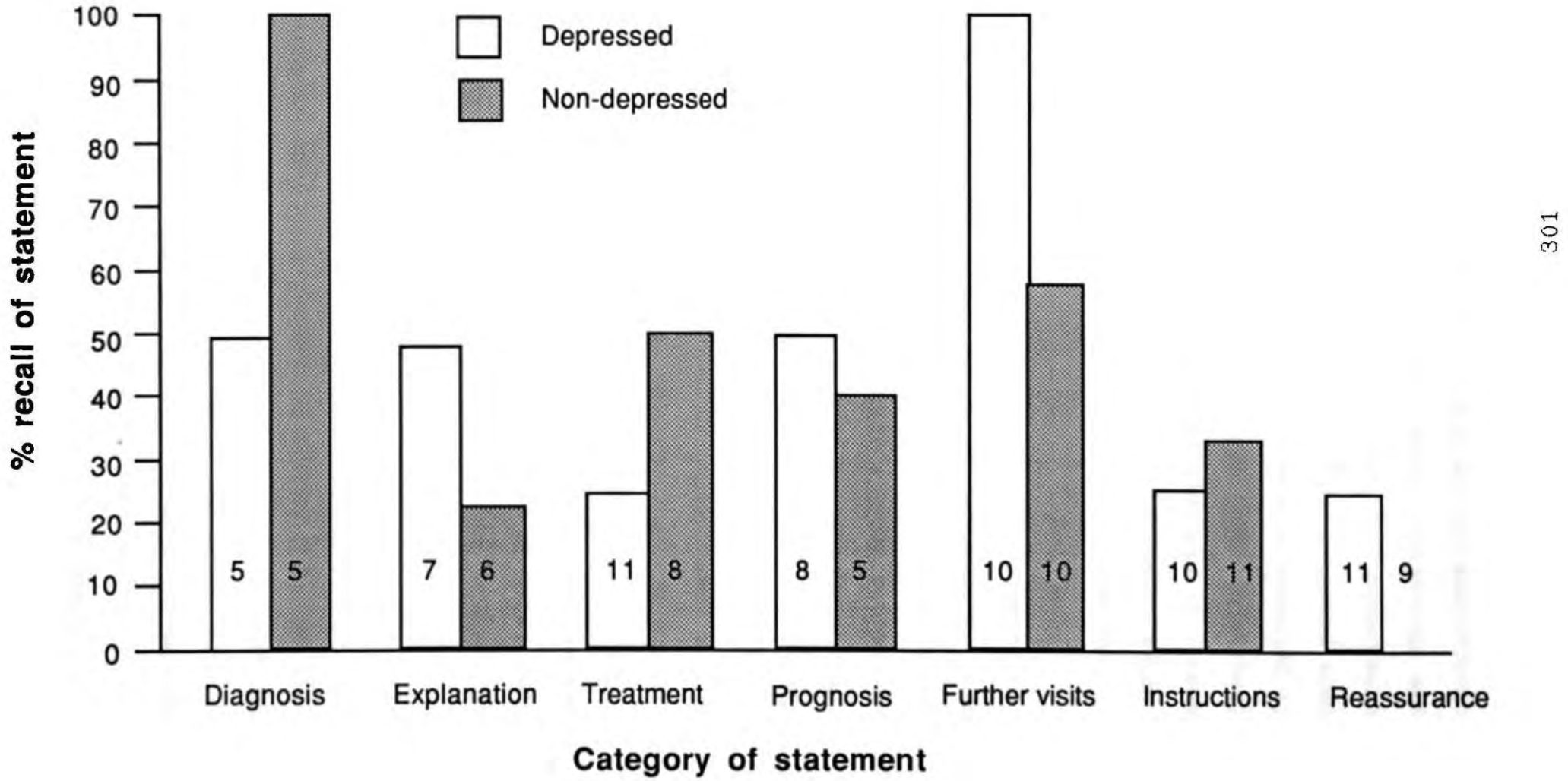


Table 8.6 shows the median number of unique statements given in each category to the two groups of patients (repeats of statements are excluded); the data is illustrated in Figure 8.11. Non-parametric statistics were used to test for differences between the groups because in most cases the distribution was very skewed. The differences between the depressed and non-depressed patients in the number of questions asked and the amount of general conversation ('other' category) both approached significance (questions, U=31.5; general conversation, U=38; critical value for U at 5% level is 30). There was a significant difference in the number of reassuring statements (U=21.5, $p<0.05$) although this should be treated with some caution because the data for this category was not reliable (Section 8.3.1).

The median percentage recall of the patients for each type of information are illustrated in Figure 8.12. There is limited evidence that some types of information are recalled better than others: the median percentage recalls of statements about diagnosis and further visits were 75% and 79% respectively; for information about prognosis, statements explaining the diagnosis, symptoms or signs, describing the treatment or giving instructions it was less than 50% (45%, 34%, 37%, 27% respectively); whilst for reassurance it was only 12%. In addition there is some suggestion from the data that the two groups differed in their recall of some categories of information: non-depressed patients recalled more statements about diagnosis, instructions and treatments than depressed patients. In contrast the depressed patients recalled more explanations, more information on future visits and more statements containing reassurance.

As each type of information did not occur in every consultation the number of subjects given information in some of the categories was very small. In addition the number of statements given in each category was frequently small and so percentage recall was not normally distributed: if patients were given only one piece of information in a category the only possible rates of recall were 0% (item forgotten) or 100% (item remembered). It was therefore decided that it would be inappropriate to use statistical tests with these data and for this reason no firm conclusions can be drawn from these data.

The important thing to note, however, is that the consultations of the depressed patients did not contain more statements than those of the non-depressed patients from the categories which seemed to be well recalled. If they had done this might suggest that the consultations with depressed patients were easier to recall, and that the depressed patients should have recalled more information than they did. Since this is not the case there is therefore no evidence that the depressed patients' memory for medical information was impaired.

8.4 Discussion

The aim of this study was to investigate whether depressed patients had a memory impairment in an important everyday situation: the general practice consultation. The memory of clinically depressed patients for information given by the general practioner was compared to that of non-depressed patients. The two groups did not differ in the percentage of the information they recalled, nor in the absolute number of statements recalled. These results suggest that the depressed patients did not have a memory impairment in this realistic situation.

However, it is possible that the consultations of the depressed and non-depressed patients differed in such a way that the consultations of the depressed patients were more memorable than those of the other patients. If this was the case then they should have recalled more information than the non-depressed patients, and the fact that they did not do so would suggest that their memories were impaired. Several aspects of the consultation were considered which have been shown in previous studies to affect the recall of medical information. These included the amount of information given (Ley and Spelman, 1867; Joyce et al, 1969), the number of statements repeated (Ley, 1979) and the type of information (Ley and Spelman, 1965, 1967).

It was found that the amount of information given and the number of statements repeated did not differ between the two groups and that Group (depressed/nondepressed) was not related to recall when the effects of these variables was controlled for statistically. There was some suggestion that there were significant differences between the groups in the number of statements given in different categories: for instance the

depressed patients were given more reassuring statements. However, there was no evidence that they were given more information in the categories (such as statements about visits or prognosis) which were particularly well recalled, or less information in the categories which seemed to be more difficult to remember (such as explanations). These results suggest (as do those for the amount of information given and the number of repeated statements) that there is no reason to suppose that the consultations with the depressed patients were particularly easy and that therefore they should have recalled more information than they did.

However, it should be noted that depressed patients may differ from other patients in the type of information they find easy to recall. For instance they may remember bad news and worrying information particularly well as a consequence of their tendency to selectively recall negative material (Section 1.7.4). There was a suggestion that depressed patients recalled more reassuring statements and information about future visits than non-depressed patients. Depressed patients did not, therefore, show a tendency to recall negative material; instead they seemed to recall more positive reassuring information than the non-depressed patients. As the two groups did not seem to differ in their recall of information from any category there is no reason to suppose that their consultations differed in such a way as to make them more memorable to one group than to another.

Other variables which might affect recall were also considered to see if there were differences between the two groups on these variables. The total amount said during the consultation differed significantly between the two groups although, as already noted, the amount of information given to the two groups did not. This suggests that the doctors chatted more to the depressed patients and asked them more questions. This variable was not significantly related to recall and therefore, despite the fact that the two groups differed on it, there is no suggestion that it caused the consultations of the depressed patients to be easier to remember. In contrast the number of times repeated statements were given did differ significantly between the two groups, and was related to recall once the effects of the other predictor variables (such as the amount of information given and the number of repeated statements) werecontrolled for statistically. However, there was no evidence that Group was related to recall once the effects of this variable were removed statistically.

Other variables which might have been related to recall did not differ significantly between the two groups. These included the length of the consultation; the longer the consultation the less the patients recalled (this was not related to recall when entered alone in the regression equation but emerged as one of four variables related to recall when the backwards regression procedure was used; the other variables were the number of items of information given; the number of times repeated statements were repated and the number of visits in the past year.). There was no evidence that Group was related to recall once the effects of this variable were controlled for.

The number of visits to the doctor in the previous month and the previous year were also considered. Ley and Spelman (1965) used new patients at an out-patient clinic in their study of memory for medical information, as did Joyce et al (1969). This makes it less likely that memory for information given in the present consultation will be affected by memory for information given previously. If the information given on the two occasions is similar it may make recall on the latter occasion easier because of increased familiarity with the material. If it differs, confusion may occur between information given on the two occasions and this may make recall more difficult. Ideally, therefore, all the patients in this study would have been consulting the doctor with a new illness. This was not possible as it would have made recruiting subjects extremely difficult. The patients were therefore asked how many times they had visited the doctor in the previous month and in the previous year in order to look at the effect of previous consultations on memory.

The number of visits to the doctor in the previous year was not significantly related to recall when placed alone in the regression equation. However it emerged as one of the variables significantly related to recall when a backwards regression procedure was used to examine the relationship between eight predictor variables and recall: the more visits the patient had made, the less they recalled (the relationship between the number of visits in the previous month and recall was not significant). This may be for a variety of reasons: the patients may concentrate less on information they have been given before; the content of the consultations may vary so that those of patients with a large number of

previous consultations contain more statements from categories which are poorly recalled such as instructions and explanations; patients may get confused between information given on different occasions. The important thing to note, however, is that if the depressed patients had fewer previous consultations than the non-depressed patients they would be expected to recall more information, and the fact that they did not would suggest that they did have a memory impairment after all. In fact, the numbers of previous consultations in the previous month and the previous year did not differ significantly between the two groups and when the effects of these variables were controlled for statistically, Group still did not emerge as a significant predictor of recall. There is, therefore, no reason to suppose that the depressed patients' consultations were particularly easy to recall because of the pattern of previous consultations.

The final factor to be considered was the effect of cueing recall. The interviewer asked each patient about various areas the doctor may have covered during the consultation, for instance 'did the doctor tell you what was wrong?'. This may have helped depressed patients to overcome their putative memory impairment by reducing the amount of effort needed to retrieve information from memory (Section 1.8.1). If this were so, then depressed patients would be expected to recall less than non-depressed patients in response to the first general question, and more in response to the cue questions. However, the two groups did not differ in the number of statements they recalled in response to the first question, or in the number they recalled in response to the other questions. There is, therefore, no evidence that the depressed patients were helped to overcome their memory impairment in this way.

There is, therefore, no evidence that the consultations with the depressed patients were easier to recall than those with the non-depressed patients. Although recall was positively related to the total number of items given and the number of times statements were repeated, and negatively related to the length of the consultation and the number of visits in the previous year, there was no evidence that Group was related to recall once the effects of these variables was held constant. Since this is the case there is no reason to suppose either that the depressed patients should have recalled more information than the others

or that the fact that they did not shows that they had a memory impairment despite the apparent evidence to the contrary. The results of this study therefore show that the depressed patients did not have a memory impairment in this realistic everyday situation.

These results contrast with the finding of memory impairment on laboratory memory tests reported in Chapter Four. There are two possible explanations of this; these will now be considered in turn.

Although the non-depressed patients were screened for depression they were not also screened for anxiety. In the light of both Ley and Spelman's (1965) finding that the recall of medical information was poor in highly anxious patients, and other evidence of memory impairments caused by anxiety (Section 6.1) it might be argued that the non-depressed patients were anxious about visiting the doctor and therefore did not recall as much information as they might otherwise have done. If this were so, then this study would show that the recall of depressed patients did not differ significantly from that of another group of patients with impaired memories. Although this possibility cannot be excluded in the absence of a measure of anxiety there is good reason to believe that the non-depressed patients had low levels of anxiety. There is considerable evidence that measures of anxiety and depression tend to be highly correlated (reviewed in Section 6.1): for example scores on the Irritabilty-Depression-Anxiety depression subscale were found to be highly correlated ($r=.62$) with scores on the anxiety subscale in the group of subjects used in Chapter Six of this thesis, which included controls who were neither depressed or anxious as well as clinically depressed patients and those with an anxiety state. As the non-depressed patients in this study were selected because they had low depression scores they were also likely to have had low anxiety scores. It is therefore unlikely that the depressed patients did not appear to have a memory impairment for medical information because they were compared to a group who also had memory impairments, in this case due to anxiety.

The second possible explanation for the discrepancy between the results of this study and that reported in Chapter Four is that the depressed general practice patients used in this study were less depressed than the depressed psychiatric patients used in the previous

study. Nine out of eleven of the general practice patients were assigned to Catego Class N (neurotic depression) with eight of these reaching Index of Definition Level Five (the borderline level) while one reached Level Six. The remaining two patients were assigned to Class R (retarded depression), one at Level Five and the other at Level Six. In contrast, nine out of ten retarded depressed patients and nine out of thirteen neurotic depressed patients in the earlier study reached at least Level Six (Section 3.2.3). The general practice patients were therefore less severely ill than the psychiatric patients.

Hasher et al (1985) hypothesised that whether there are memory deficits in depression will depend on the severity of the depression, while Ellis (1985) suggested that impairment would only be found on more difficult tasks. A similar conclusion was reached in Chapter Four of this thesis, where it was found that the less severely depressed neurotic patients were impaired only on what were judged to be the more difficult tasks, whilst the more severely ill retarded depressed patients were also impaired on less difficult tasks. It was hypothesised that both groups had reduced processing capacity due to interference from worry and depressive thoughts (Section 1.8.4) and that this was particularly true of the retarded depressed group who had insufficient capacity remaining even for the less difficult tasks.

In the present study, the depressed general practice patients may not have shown a memory impairment in this situation because they were not sufficiently ill and therefore showed only a slight reduction in processing capacity. In addition, the material was probably not as difficult to remember because it was meaningful to the patients and did not therefore make as many demands on memory. These patients might have been impaired on a task which made more demands on memory. It might also be that it was important to the patients that they recalled this information and that therefore they were more motivated than in other, less important, situations. If this was the case, they may have increased the amount of effort they put into the task and consequently overcome the effects of any reduction in processing capacity resulting from cognitive interference (Section 1.8.4). This was particularly likely to be the case as the depressed patients also had significant symptoms of anxiety (Section 8.2) and it is hypothesised in Chapter Six that subjects with

high levels of anxiety are able to overcome the effects of reduced motivation by increasing effort (Eysenck, 1979, 1982).

It can therefore be concluded that the depressed general practice patients were not impaired in this realistic everyday situation because the task was not difficult and therefore did not require much processing capacity and effort; because the depressed patients were not severely depressed and consequently did not have much reduction in processing capacity resulting from cognitive interference; and because they were motivated to do well and therefore were able to overcome the effects of reduced processing capacity by increasing the amount of effort they put into the task. However, these conclusions can only be preliminary because neither effort nor processing capacity were directly measured in this study.

This study illustrates the difficulties inherent in examining memory in realistic everyday situations. Baddeley (1980) observed that such studies tend to be very expensive, time-consuming (this was the case in this study: 98 recruitment sessions were needed to obtain eleven suitable depressed patients) and are also liable to provide data with more than one possible interpretation. He argued that this showed that both observational studies of everyday memory and experimental approaches had their limitations and that both are needed in order to adequately understand everyday memory. Wilkins (1986) has also pointed out that everyday memory tasks and laboratory tests have different strengths and weaknesses: for instance laboratory tests lack relevance to everyday memory and consequently have low 'ecological validity', whilst studies describing memory in everyday life have high 'ecological validity' but may be less reliable because of the highly variable and idiosyncratic cues available in everyday life. It is therefore clear that studies of memory undertaken in everyday situations and in the laboratory have different strengths and that both are needed for an adequate understanding of memory in depression.

It has already been suggested that the best use of self-assessments of memory may be as a source of qualitative data which can be used to generate memory tests which are relevant to the problems depressed people experience in everyday life (Section 7.4.3). The same may be true

of studies of memory in depression in realistic everyday situations. For instance it might be appropriate to follow up the results of the present study with more controlled laboratory tests looking at, for instance, the recall of depressed patients for material which is meaningful to them and for material which has no personal relevance for them; the effects of increasing the difficulty of the task; the effects of manipulating the motivational levels of the subjects; and the effects of depression on processing capacity. In this way the hypothesis that the memory of depressed people depends on the difficulty of the task (presumably because of the demands it makes on processing capacity) and the severity of the depression (presumably due to reduced processing capacity and reduced motivation) which seems to explain the results obtained in this everyday situation could be systematically tested.

In summary, the results of this study have shown that depressed general practice patients are not impaired in an important everyday situation: the general practice consultation. It is hypothesised that this was because the demands of the task did not exceed the processing capacity of these depressed patients: if they had been more severely depressed then the combination of the reduction in processing capacity (which is hypothesised to be caused by cognitive interference) and reduced motivation may have resulted in impairment in this situation. It is suggested that such studies of memory in depression in everyday situations might be used as a source of hypotheses which could then be tested systematically using appropriate laboratory memory tests.

In the next chapter (Chapter Nine) the results of this study are discussed in the light of those reported in earlier chapters of this thesis.

GENERAL DISCUSSION

This thesis has investigated three aspects of memory in depression: the performance of clinically depressed people on laboratory memory tests; their reports of memory problems in everyday life and the relationship of these reports to performance on the laboratory tests; and the memory of depressed people in an important everyday situation, the general practice consultation. The findings are discussed in this final chapter.

9.1 THE RELATIONSHIP OF THE SEVERITY OF DEPRESSION TO MEMORY IMPAIRMENT.

There is some evidence to suggest that memory impairment and the severity of depression are related. For example most studies reviewed in Section 1.4, which found that depressed people performed more poorly than people who were not depressed on memory tests, used depressed psychiatric in-patients as subjects; studies which used depressed medical in-patients (Cole and Zarit, 1984), psychiatric out-patients (Davis and Unruh, 1980; Popkin et al, 1982) or elderly depressed people recruited from an epidemiological survey (O'Hara et al, 1986) have not found evidence of significant memory impairment. It was therefore concluded (Section 1.4) that a memory impairment may only be found in people with symptoms severe enough to warrant psychiatric in-patient treatment. Further evidence for a relationship between the severity of depression and memory impairment came from studies showing that memory impairments observed in depression disappeared on the remission of the depression (Section 1.6).

The relationship between the severity of depression and memory impairment was explored in Chapter Four of this thesis. This describes a study in which the memories of two groups of depressed in- and day-patients (retarded depressed and neurotic depressed) were compared to those of anxious patients and controls, who were neither depressed or anxious. The retarded depressed patients had higher Index of Definition levels (Section 3.2.3, Wing et al, 1978) than the neurotic depressed group, imdicating that they had both more and more severe symptoms, and were consequently more severely ill. They were significantly impaired compared

to the control subjects on variables measuring immediate learning, the speed of learning, recall after a delay and memory for past public events; but were not impaired on variables measuring the retention of information once learned, or on β, a measure of response bias. The neurotic depressed subjects showed the same pattern of results. They scored lower than the control subjects on all of these variables and were significantly impaired on all but five of those variables on which the retarded depressed subjects were significantly impaired; the exceptions were the least difficult three immediate learning variables, and two variables measuring recall after a delay which were judged to be undemanding (Section 4.4.1). The performance of the two depressed groups differed significantly on only two variables (d' - Trial One and Paired Associate-speed of learning). In both cases the less severely ill neurotic depressed group showed significantly higher scores and were less impaired. These results therefore show that the more severely ill retarded depressed group scored significantly lower than the neurotic depressed group on two memory variables, and were significantly impaired compared to control subjects on more variables. However, the two groups of depressed subjects showed the same pattern of results.

Patients who were taking psychotropic medication or who had been given E.C.T in the previous year were included in this study (Section 3.2). Regression analysis was therefore used to see if the same pattern of results was obtained when the effects of medication and E.C.T were controlled for statistically (Chapter Five). The results of this analysis suggested that differences between the groups on the variables measuring prose passage recall could have been due to medication and E.C.T rather than to the psychiatric condition of the subject. However, apart from this the results of the previous analysis were substantiated - the two groups of depressed patients showed the same pattern of impairment but the less severely ill neurotic depressed group were significantly impaired on fewer variables. These results therefore support the hypothesis that the degree of memory impairment in depression is related to the severity of the depression: patients in the more severely depressed retarded group were significantly impaired on more tests of memory than those in the less severely depressed neurotic depressed group.

In the study reported in Chapter Eight depressed general practice patients were found not to have impaired memories for medical information. It was argued that one reason for this was that these patients were less severely depressed than the psychiatric in- and day-patients included in the study reported in Chapter Four, which showed that depressed patients had significantly impaired performance on laboratory memory tests. Nine out of eleven patients in the general practice study reached Index of Definition Level Five, the borderline level, whilst eighteen out of the 23 depressed psychiatric patients reached at least Level Six. The results of this study therefore support the conclusion reached in Chapter Four that the degree of memory impairment found in depressed people will depend upon the severity of the depression. This also agrees with the conclusion reached in Section 1.4 that most of the studies reporting memory impairment in depression had used psychiatric in-patients and that impairments may therefore only be found in people sufficiently depressed to need in-patient care. It also agrees with the results of studies showing that as clinical depression is alleviated by treatment, the memory impairments abate (Section 1.6).

Several studies have looked at the correlation between measures of the severity of depression and measures of the severity of memory impairment; these are reviewed in Section 1.5. Of eighteen such studies only seven found a significant relationship between the two; in each case memory declined as the severity of depression increased. This suggests that memory impairment in depression is not strongly related to the severity of depression. However, many of these studies may have not found a significant relationship for methodological reasons such as using an inadequate measure of depression, having a narrow range of depression scores or using too few subjects (Section 1.6). They do not, therefore, provide good evidence of a minimal relationship between the severity of depression and the degree of memory impairment.

The difference between these results and those from studies comparing depressed and control subjects which suggest that the severity of depression may be related to the severity of memory impairment (Section 1.4), may also result from confusion as to what the term 'depression' signifies. It can refer to the psychiatric condition of 'clinical depression' (Section 1.2), in which symptoms of depression

predominate but in which other symptoms (such as anxiety and tension) frequently occur (Section 2.2.4). It can, however, also refer to the symptoms of depression themselves, which are most common in clinical depression but which also occur in other psychiatric conditions such as anxiety (Section 6.1). The severity of memory impairment in clinically depressed people may be related to the overall severity of clinical depression (which will depend only in part on the severity of symptoms of depression), but not specifically to the severity of the symptoms of depression. For instance, people who are severely clinically depressed will obviously have a number of symptoms specific to depression but are also likely to show significant symptoms of anxiety (Section 6.1). If memory impairment is related to anxiety, then severely clinically depressed people with many anxiety symptoms would have more impaired memories than less depressed people who show fewer anxiety symptoms; memory impairment would therefore be related to the overall severity of clinical depression but not necessarily to the severity of symptoms of depression alone. It is therefore possible that the overall severity of clinical depression, but not the severity of depression symptoms, will be strongly related to the severity of memory impairment. This means that a weak relationship between depression and memory performance might be found, even if clinically depressed people with impaired memories were used as subjects; this would be particularly likely if the measure of depression used was a good measure of the symptoms of depression, rather than of the overall severity of clinical depression.

Chapter Six describes a study which looked specifically at the relationship between memory and symptoms of depression in a group of subjects which included clinically depressed, clinically anxious and control subjects; the clinically depressed subjects had already been shown to be impaired on the memory tests used (Chapters Four and Five). In contrast to the studies reviewed in Section 1.6, which just considered the effects of depression, the relationship between the syndrome of anxiety and memory was also examined in this study; depression and anxiety are highly correlated (Section 6.1) and therefore the effects of one need to be taken into consideration when assessing the effects of the other. Depression, rather than anxiety, was related to performance on three of the immediate memory variables, as well as one speed of learning variable, and on two variables measuring recall after a delay. It was not

possible to distinguish between the effects of depression and anxiety on the remaining variables and it was therefore concluded that something common to both was related to performance.

The proportion of variance explained by depression and/or anxiety was quite small; on five of the twelve variables it was ten per cent or less and on all twelve it was less than 23%. In three, the amount of variance explained by depression or anxiety was not statistically significant, even though the clinically depressed patients were impaired on these tests. One reason for this may have been that the self-rating questionnaires used to measure depression and anxiety are not sufficiently sensitive and therefore did not distinguish adequately between different levels of depression and anxiety; similar methodological problems may explain why some other studies have also failed to find a significant correlation between depression and memory (Section 1.6).

The results of Chapter Six are therefore similar to those of studies reviewed in Section 1.6: on some variables there was a significant relationship between depression (or something common to both anxiety and depression) and memory performance, whilst on others the amount of variance explained was not statistically significant. In all cases high levels of depression and/or anxiety were associated with poor performance. These results highlight the importance of taking both depression and anxiety into consideration when assessing the role of either in causing memory impairment in depression: if only depression is measured, the results could be misleading because an apparent relationship between depression and memory could in fact be an artefact of the relationship between anxiety and both depression and memory.

The importance of considering both depression and anxiety is further supported by the finding reported in Chapter Seven. Both the number of memory failures and the degree of self-perceived memory change reported by the same group of subjects as used in Chapters Four to Six were related to anxiety, rather than to depression. Previous investigations of memory complaints in depression (Section 7.1.3) have not considered both and have therefore concluded that a high number of memory complaints was caused by depression: these results suggest that that is not the case.

In this study, the more severely ill retarded depressed patients reported both significantly more cognitive failures and greater memory deterioration than control subjects. The neurotic depressed patients were not significantly different from the controls on the reports of cognitive failures, but did report a significantly greater deterioration in memory. Thus the highest number of memory complaints is found in the most severely ill depressed patients, even though depression per se is not significantly related to memory complaint. This supports the hypothesis that significant correlations between the symptoms of depression and memory may not be found even when clinically depressed subjects have significantly impaired memories because the impairment may be related to other symptoms which are common in clinical depression, such as anxiety.

In summary, the results of Chapter Four and Chapter Eight have both indicated that the memory impairment found in clinically depressed people will vary according to the severity of clinical depression. People with clinical depression have high levels of depression but they would also have high levels of anxiety. The fact that severely clinically depressed people have memory impairments does not, therefore, necessarily indicate that the impairment is caused by depression; it might, for instance, be caused by anxiety. The results reported in Chapter Six showed that on some tests performance was related to the severity of depression, while on others it was related to something common to both depression and anxiety. In contrast memory complaints were related to anxiety: the more anxious the subjects, the more they complained about their memory (Chapter Seven). It can therefore be concluded that, whilst the extent of memory impairment and the level of memory complaints vary with the severity of the clinical depression, there is less evidence that they are specifically related to the symptoms of depression; memory complaints are clearly related to anxiety rather than depression, while performance on some memory tests seemed to be related to factors common to both depression and anxiety. This indicates the importance of assessing anxiety before concluding that performance is affected by depression, and vice versa.

9.2 THE RELATIONSHIP OF MEMORY IMPAIRMENTS TO THE OVERALL LEVEL OF PSYCHOPATHOLOGY.

This thesis has examined the possibility that memory impairments observed in people with clinical depression are a consequence of anxiety, rather than of depression itself. Another possibility is that impairments are not related specifically to either the symptoms of depression or those of anxiety, but instead are more closely related to the overall impairment in functioning which results from psychiatric illness; that is, that the impairment is related to the overall level of psychopathology rather than to any particular symptoms or groups of symptoms. If this were so, then some correlations would be expected between measures of depression and memory because the overall level of psychopathology will be determined partly by the severity of depression. Similarly scores on anxiety questionnaires would also be expected to be correlated with performance. However, the overall level of psychopathology depends on the severity of both depression and anxiety, as well as on the severity of other symptoms not included in these questionnaires, and therefore would be more closely related to performance than either depression or anxiety separately.

The possibility that performance is determined by the level of overall psychopathology rather than by specific symptoms, such as those related to depression, has been investigated in relation to thought disorder in depression and schizophrenia. Harrow and Quinlan (1977) proposed that disordered thinking was not, as commonly presumed, unique to schizophrenia but that instead there was a continuum of increasing thought disorder with increasing general psychopathological deficit. This has been labelled the continuum model of thought disorder (Braff, Glick and Griffin, 1983) and was based upon the finding of Harrow and Quinlan (1977) that acutely ill depressed patients and patients with personality disorders, as well as acutely ill schizophrenic patients, showed evidence of disordered thinking. The possibility that thought disorder was related to the severity of psychopathology was further investigated by Braff, Glick and Griffin (1983). They looked at the performance of depressed, schizophrenic and normal subjects on the Shipley Hartford Scale and Gorham Proverbs Test, and correlated performance on these with scores on two measures of general psychopathology (the Brief Psychiatric Rating

Scale and the Global Assessment Scale), and with scores on the Beck Depression Inventory. As expected the normal subjects showed the best functioning, as assessed by the two psychopathology scales, followed by the depressed patients, with the schizophrenics having the worst functioning. Both the schizophrenics and depressed patients had significantly decreased abstraction ability compared with normal subjects In addition, the schizophrenics showed large increases in idiosyncratic abstraction. When the three subject groups were combined, both general psychopathology scales were significantly correlated with the tests of thought disorder, but the depression scale was not. These results show that the relationship between increased psychopathology and thought disorder exists across all subjects, from normals to the severely impaired psychiatric patients, and supports the hypothesis by Harrow and Quinlan (1977) of a continuum of thought disorder.

Braff et al (1988) carried out a similar study using the same assessment instruments and again found that across all three groups of subjects thought disorder (abstraction difficulties) correlated with general psychopathological impairment, rather than with the level of depression. In addition they followed the subjects through from their admission to psychiatric hospital to their discharge. They found that changes in the abstraction measures over this period were correlated significantly with the improvement in the general clinical state of the patient, but not with the improvement in depression. They used stepwise regression analysis to investigate further the relationship between psychopathology, depression and thought disorder and found that depression did not account for a significant amount of the variance in scores on the measures of thought disorder once the variance associated with the measures of general psychopathology was partialled out. They do not present the figures for the proportion of variance explained by general psychopathology once the effects of depression were partialled out. Nevertheless, the authors concluded that their results support the hypothesis that general psychopathology, rather than depression, can produce thought disorder. The results of previous studies linking depression to thought disorder (eg Sprock et al, 1983) may stem primarily from the correlation between depression symptoms and general psychopathology rather than reflecting the effects of depression per se. Braff et al (1988) argue that there is little evidence that abstraction

dysfunction is specifically induced by any specific psychiatric condition, although they stress that this does not mean that there are no forms of thought disorder specific to a particular psychiatric condition; for instance there is some evidence (Braff, Glick and Griffin, 1983) that idiosyncratic and autistic thinking may be unique to schizophrenia.

It is possible that, as Johnson and Magaro (1987) have recently suggested, memory impairments (like thought disorder) are due to the general level of psychopathology, rather than specifically related to any psychiatric diagnosis. Some evidence for this comes from the failure to find memory impairments which are specific to depression and not also found in other psychiatric conditions. In his extensive review of cognition in depression Miller (1975) concluded that there was little evidence for impairments on cognitive, motor or perceptual tasks which were unique to depression. More recent studies comparing the memory of depressed people with that of manic or schizophrenic patients (Section 1.9.2) have failed to find evidence of memory impairments in depression which are not also found in the other psychiatric conditions. Several studies have shown that schizophrenic subjects are more impaired; for instance Taylor and Abrams (1983) found more dominant hemisphere and global impairment in schizophrenic subjects than in depressed subjects. This would be expected if the schizophrenics had higher overall levels of psychopathology, as both Braff, Glick and Griffin (1983) and Braff et al (1988) found that they did.

Some preliminary evidence that memory impairments are not specific to any psychiatric diagnosis but are related to overall psychopathology can be found in Chapter Four of this thesis. The memory performance of a small group of anxious patients was compared to that of the depressed patients. The anxious patients were less severely ill than the depressed patients, particularly the retarded depressed group: 70% of the retarded depressed patients reached Index of Definition Levels Seven and Eight compared with none of the anxious patients. The scores of the anxious subjects on the laboratory memory tests fell between those of the depressed and control subjects but, in most cases, were significantly different from neither, presumably due to the small number of anxious subjects. If the degree of impairment was related to the overall level of psychopathology the anxious patients would be expected to be less

impaired than the depressed patients because they had both fewer and less serious symptoms and therefore lower levels of overall psychopathology.

However, the anxious patients also had lower depression scores and lower anxiety scores than the depressed patients (Section 6.2.2) and therefore may have been less impaired because of this; depression scores were found to be significant predictors of performance on some of the memory tests, whilst some aspect of both anxiety and depression was related to performance on others (Section 6.3). If this was the case it would support the argument that memory impairments are not specific to a particular psychiatric condition, because the symptoms of depression and anxiety can occur to differing extents in a variety of psychiatric conditions. However, it would conflict with the hypothesis that the impairment is due to the overall level of psychopathology.

The hypothesis that memory impairment in depression (and other psychiatric conditions) is caused by the overall level of psychopathology rather than by condition-specific factors, or by symptoms of depression and anxiety which can occur in a variety of psychiatric conditions, has yet to be adequately tested. In order to do so a sample of schizophrenic, depressed, anxious and normal subjects would be needed to obtain a sufficiently range of psychopathology. The level of overall psychopathology would need to be assessed, as would levels of anxiety and depression. In addition a range of memory tests would be needed to see if the various subject groups showed the same patterns of memory impairment. If they did, regression analysis could then be used to investigate whether overall psychopathology, depression or anxiety was most closely related to memory impairment.

In the absence of such a study it is not clear if memory impairment in depression is caused by the same factors affecting memory in other psychiatric conditions, such as anxiety, mania and schizophrenia. The failure to find memory impairments specific to clinical depression suggests that the impairments are indeed caused by a factor common to all these conditions. Whether it is the overall severity of psychopathology (as suggested in relation to abstractive ability; Braff et al, 1988) or specific symptoms such as those of depression and anxiety which

occur in a variety of psychiatric conditions such as depression or anxiety as suggested above, has yet to be determined.

9.3 THE RELATIONSHIP OF TASK DIFFICULTY TO MEMORY IMPAIRMENT IN DEPRESSION.

As reviewed in Section 1.8.1, several authors have concluded that depressed people are most impaired on tasks which are complex and which require sustained effort or motivation for completion; for instance Glass et al (1981) concluded that depressed people would be impaired on any sufficiently complex tasks.

The results presented in Chapter Four of this thesis largely support this and, in particular, show an interaction between the level of difficulty of the task and the severity of depression; the most severe impairments are shown by the most depressed subjects on the most difficult tasks, as suggested by Ellis (1985). While the retarded depressed subjects were significantly impaired on all of the immediate learning variables, the less severely depressed group of neurotic depressed patients were not impaired on a paired associate test and a prose passage recall test. These were judged to be less demanding than the tests of memory for unconnected words on which the neurotic depressed patients were impaired; the former because it involved cued recall and relatively over-learnt pairs of words, and the latter because it had a definite structure with inbuilt redundancy. Thus the more severely depressed group of retarded depressed patients were impaired on all the immediate learning tests, whilst the less depressed neurotic depressed group were only impaired on the more difficult tests.

Contrary to expectations, the neurotic depressed patients also had significantly lower d' scores (calculated from scores on the Picture Recognition test) than the control subjects. This was surprising, because it was presumed that the recognition test required less effort for successful completion than either free or cued recall tests, and was consequently one of the easier tasks. However, when scores on the recognition test, a cued recall test and free recall test were standardised there was no interaction between mode of recall and the psychiatric group of the subjects, indicating that the depressed patients

did not do relatively better on this task than on the others and that it did not, as expected, demand less effort than the other tasks. It was therefore concluded that other aspects of the task were determining the amount of effort required; for example the Snoopy cartoons used in the Picture Recognition test may have been insufficiently distinct from one another. Watts and Sharrock (1987) similarly found that free and cued recall were equally affected by depression despite the fact that the latter was presumed to require less effort; they also concluded that there were other types of effort involved in these tasks which made them less difficult than anticipated.

However, it is obviously unsatisfactory to conclude, after depressed subjects have failed to show a deficit on a task considered to be undemanding, that it must have been demanding after all. There does not seem to be any consensus on how to decide how difficult a task is and how much effort it therefore requires. Eysenck (1983) presumed that difficult tasks were those that made greater demands on working memory capacity (Baddeley and Hitch, 1974), while Weingartner et al (1981) worked on the basis that 'deeper' processing strategies such as semantic encoding were more demanding than more shallow processes such as structural encoding (this is compatible with Eysenck's view because more elaborative processing will presumably make more demands on working memory). Other studies have talked in terms of 'complex' tasks without defining what makes a task complex (Cohen et al, 1982). Thus although it can be concluded that depressed people are impaired on tasks requiring effort for completion (Section 1.8.1), it is not always clear what tasks fit into this category as the results for the Picture Recognition Test included in Chapter Four illustrate.

The results for the questionnaires measuring memory for past public events generally support the hypothesis that depressed people will be impaired on any task that requires sufficient effort for successful completion, regardless of the stage of memory involved. Both groups of depressed patients were significantly impaired on the multi-choice version of these questionnaires and showed a trend towards impairment on the free recall version (this was true even when the time taken to complete the questionnaire was controlled for statistically, indicating that the depressed subjects did not show low scores solely because they

were slow at completing these timed tests). These tests differed from the rest of the test battery in that they did not measure new learning but, instead, memory for events which happened before the testing session and, indeed, presumably before the onset of depression. As the depressed subjects did not differ from the controls in their ability to retain information in memory (at least over a short period of time), these results suggest that depressed people have difficulty retrieving information from memory. It may, however, be that memory for this material was unaffected by depression and that instead the depressed subjects were not motivated to complete these questionnaires, did not exert sufficient effort and were therefore impaired. This is supported by the finding that depressed subjects tended to omit more questions than controls on the free recall version. However, the fact that the depressed subjects did less well on the multi-choice version of the questionnaire than on the free recall version (even though the latter was presumed to be most demanding) may seem to contradict this. This again illustrates the difficulty in assessing how difficult memory tasks are; the multi-choice questionnaire may have needed considerable effort and concentration for completion because subjects had to choose between the responses rather than, as expected, being less demanding because the responses were provided.

Further support for the hypothesis that the extent of memory impairment shown by depressed people will vary according to how difficult the task is comes from the finding that depressed general practice patients did not have impaired memories for medical information (Section 8.3). As already noted (Section 9.1), these patients were less depressed than the psychiatric patients who showed impairments on the laboratory memory tests (Chapters Four and Five); this presumably was one reason why they were not impaired. In addition, it is likely that the information given to the general practice patients was easier to recall than the material used in the laboratory memory tests because it was relevant to the individual, meaningful and structured. The general practice patients may not, therefore, have been impaired both because they were not severely depressed and because the information was not difficult to recall.

In summary, the results presented in this thesis are generally consistent with those of previous studies (Section 1.8.1) which have shown that memory impairment in depression is related to the complexity of the task and the amount of effort it requires. They support the suggestion that impairments in depression are not restricted to any particular stage or process, but will be exhibited on any sufficiently complex task (Cohen et al, 1982). Further research is required, especially research in which the operational definition of 'task difficulty' is clearly specified and in which levels of difficulty can be experimentally manipulated; for instance by using a letter transformation task (Hamilton, Hockey and Rejiman, 1977) such as that used by Eysenck (1983) to investigate whether 'difficult' tasks make greater demands on working memory than less difficult tasks. Although there is evidence that more difficult tasks are more impaired in depression, few studies have clearly specified why a task is considered to be difficult or effortful. There are a few exceptions: for instance Weingartner et al (1981) presumed that 'deeper' memory processes were more effortful and compared the performance of depressed people on a variety of tasks requiring different processing strategies, while several studies have compared the performance of depressed and control subjects on tasks based on the distinction made by Hasher and Zacks (1979) between effortful and automatic processing (Roy-Byrne et al, 1986; Hart et al, 1987c). Overall, there is evidence that depressed people are impaired on difficult or demanding tasks, but further research based on specified theories of memory is needed to investigate why some tasks are demanding and why depressed people are impaired on them.

As reviewed in Section 4.1.3 it has also been suggested that the memory impairments of anxious people are most evident on difficult tasks; on easier tasks they may actually perform better than less anxious subjects. Eysenck (1979, 1982) has put forward a model to account for this which will be summarised here (it is reviewed in more detail in Section 4.1.3). This has been labelled the 'working memory capacity theory' (Leon and Revelle, 1985). It is based upon the assumption that anxious people divide their attention between task requirements and task-irrelevant cognitive activities such as worry and self-criticism, and that therefore less working memory processing capacity is available for the task than in non-anxious subjects. As the working memory is thought to

be involved in a wide range of cognitive tasks (Eysenck, 1979) this might be expected to produce a poorer performance in most situations. However, Eysenck (1979, 1982) suggests that this is not always so, because anxious subjects attempt to compensate for the reduction in available processing capacity by increasing the effort they put into the task. According to Kahneman (1973) one of the main determinants of the amount of effort expended by an individual is their evaluation of task demands; consequently if anxious people have reduced processing capacity because of task-irrelevant processing they find the task more demanding and therefore increase the amount of effort they exert. The increase in effort in turn increases the amount of processing capacity (Kahneman, 1973; Dornic, 1977; Eysenck, 1982; Section 1.8.1).

Anxious subjects may therefore show enhanced performance on easy tasks because the task requirements are greater for them, and therefore they exert more effort than non-anxious subjects. However, as task-difficulty increases the amount of required processing capacity increases. In addition, there is evidence that worry about failure also increases as task difficulty increases and that this in turn increases the level of anxiety (Tennyson and Woolley, 1971; Weiner and Schneider, 1971). This will further reduce the amount of processing capacity allocated to the task and the anxious subject eventually has insufficent processing capacity available to maintain performance.

This model has been investigated (Leon and Revelle, 1985) to see if it explains performance on tests of analogical reasoning any better than the cue utilisation theory (Easterbrook, 1959) or attentional theory (Mandler and Sarason, 1952). There was little evidence to support either the cue utilisation theory or Leon and Revelle's conceptualisation of working memory capacity theory (Eysenck, 1979, 1982). However, their conceptualisation of this model does not mention the fact that anxious subjects are thought to compensate for the reduction in working memory capacity by increasing effort expenditure; it therefore cannot be regarded as an adequate test of this theory. Although there is evidence for the key concepts of the theory (such as increased impairment on difficult tasks, an association between worry and performance, and increased effort expenditure by anxious subjects (see Section 4.1.3)) the model has not yet been verified.

Can this model be applied to memory in depression and, indeed, to memory in other psychiatric conditions? There is some evidence that depressed people, like anxious people, experience cognitive interference from intrusive thoughts and worry (Section 1.8.4) and that this is related to poor performance on memory tests (Watts and Sharrock, 1985). Cognitive interference in depression has received little attention and there have apparently been no investigations of whether depressed people have reduced processing capacity due to cognitive interference. It can however be presumed that worry and intrusive thoughts have the same effect on depressed people as on the anxious, and that therefore anxious people are not alone in having reduced processing capacity due to cognitive interference. This implies that the first part of Eysenck's theory, stating that processing capacity is reduced due to interference from worry and intrusive thoughts, can be applied to depression as well as anxiety.

The second part of Eysenck's theory states that anxious subjects compensate for their reduced processing capacity by increasing the effort they exert on a task. This is a motivational explanation of memory performance; anxious subjects perceive the task demands as being high, they are motivated to avoid impairment and therefore increase the effort they put into the task. Eysenck (1979, 1982) has noted that anxiety will not lead to an increase in effort in all situations; for instance it is unlikely to do so if subjects believe the chance of success on the task is low (Revelle and Micheals, 1976), or believe they are unlikely to alleviate their anxiety by doing well on the task. Whether or not anxiety leads to impairment will therefore depend at least in part on how motivated the anxious person is to successfully complete the task. Similarly, as already noted, memory impairment in depression has been attributed to a lack of motivation and effort (Section 1.8.1). It can therefore be hypothesised that memory in both depression and anxiety is affected by cognitive interference and changes in levels of motivation. Eysenck's theory of working memory capacity can therefore be applied to depression as well as to anxiety: processing capacity would be reduced by task-irrelevant worry and intrusive thoughts, as in anxiety; however unlike anxious subjects, depressed subjects would lack motivation and therefore would not be expected to increase effort expenditure to

compensate for the effects of reduced processing capacity. They would therefore be expected to show impairment on a wider range of tasks.

It is possible that Eysenck's theory can be applied not just to anxiety and depression but to psychopathology in general. As noted in Section 9.2, memory impairments in psychiatric conditions may be related to the overall level of psychopathology rather than being specific to any particular psychiatric condition. Johnson and Magaro (1987) suggested two ways in which the overall severity of illness could affect cognitive performance. The first was that the presence of a psychiatric illness may produce low levels of effort, which would be more severe in more severe illnesses. The second was that global pathology may disrupt storage and recall because of intrusive or illogical thoughts 'crowding' short-term memory. These suggestions are clearly very similar to Eysenck's model which is also based upon the effects of effort and of cognitive interference. In contrast to Johnson and Magaro (1987), however, Eysenck has drawn up a model of how these two factors operate together to produce, or protect against, memory impairment. Eysenck has not extended his model beyond anxiety but it seems appropriate to do so. According to this extension memory performance will depend upon two things. The first is the extent of cognitive interference from worry, intrusive thoughts or, in psychotic illness, from illogical thoughts which will have the effect of reducing the amount of working memory capacity available to process the task. The second is on the level of motivation, which will determine whether the amount of effort exerted on a task is increased to overcome reduced processing capacity.

The results of the studies reported in this thesis are generally supportive of this extension of Eysenck's theory. For instance, there is some evidence that anxious patients were less impaired than depressed patients on the laboratory memory tests (Section 4.4.3), as would be expected if they were able to overcome the effects of reduced processing capacity by increasing the effort exerted on the tasks. In addition, the depressed general practice patients did not have impaired memories for medical information (Chapter Eight). They were likely to have been motivated to remember this information because it was relevant to them and may therefore have increased the amount of effort exerted in order to overcome the effects of cognitive inteference. Perhaps more interesting

are the results presented in Chapter Six. These showed that symptoms of depression in a group of clinically depressed, anxious and control subjects were related to performance on the easier tests in the memory test battery, whilst it was not possible to distinguish between the effects of the symptoms of depression and the symptoms of anxiety on the more difficult tests. It was therefore concluded that something common to both anxiety and depression was related to performance on these tests, and argued that this was the reduction in processing capacity common to subjects with high levels of anxiety and those with high levels of depression. On the easier tasks, only depression was related to performance because highly anxious subjects were more motivated and therefore able to increase the amount of effort they expended in order to avoid impairment. On the more difficult tasks, however, anxious subjects were not able to increase effort sufficiently and so both high levels of anxiety and high levels of depression were related to poor performance on these tests.

These results are therefore generally supportive of the theory that the degree of memory impairment found in people suffering from psychiatric disorders will vary according to their level of motivation and the degree of cognitive interference. The level of motivation will influence the amount of effort exerted on the task, and the degree of cognitive interference will affect the amount of capacity available to process material to be remembered, and therefore the amount of effort needed to avoid impairment. This theory remains speculative although, as indicated above, the available evidence generally supports it. It would warrant further investigation, although this is not easy because it is based upon two concepts which are difficult to measure: the amount of effort exerted and the amount of processing capacity available. Some ways of investigating this theory are now discussed.

The amount of effort exerted on a task could be measured in two ways. Firstly, subjects could be asked how difficult they found a memory test, and how much effort they put into it. Secondly, the test could be carried out in the presence and absence of incentives (perhaps monetary rewards) on the basis that the effect of the incentives would be greatest in those subjects who were exerting less effort initially; highly motivated subjects would be unable to further increase the effort they

put into the test and therefore would not benefit from the incentives (Eysenck, 1985). Care would be needed to ensure that the incentives were motivating for all subjects, as depressed subjects seem to be relatively indifferent to most incentives (Layne et al, 1982). If the incentives did not increase motivation in the less motivated subjects (hypothesised to be the more depressed subjects) they would not increase their performance in the presence of incentives. This could lead to the conclusion that they were exerting considerable effort to begin with; this would not necessarily be so. This indicates the difficulty of establishing how much effort subjects, and particularly depressed subjects, are exerting.

The second part of the hypothesis suggested above is that processing capacity is reduced in both depression and anxiety by cognitive interference. One way of investigating this would be to see whether such subjects report intrusive thoughts, and whether this correlates with poor performance. Subjects could be asked to indicate each time their mind wanders when completing a memory test (Watts and Sharrock, 1985), or how much time they estimate they spent attending to the test and how much time they spent thinking about other things (Deffenbacher, 1986). These measures could then be correlated with performance, which would indicate if performance is impaired by cognitive interference.

This would not, however, indicate directly that processing capacity is reduced by cognitive interference. One way of doing this would be to investigate the performance of depressed and anxious subjects on a test in which they had to divide their attention between a main task, varying in complexity, and a concurrent attention-demanding subsidiary task (Eysenck, 1982). It would be expected that the greatest deficits would be shown on the complex main tasks, presumably because these demand more processing capacity and insufficient capacity remains after processing task-irrelevant cognitions and the concurrent subsidiary task.

These suggestions for further research would address the separate components of the extension of Eysenck's theory; that motivation is related to performance on memory tests, and that processing capacity is reduced by psychiatric conditions such as depression and anxiety. Both components need to be combined, however, in order to throw light on this theory. One way of doing this might be to use a test in which subjects

had to divide their attention between a main task (varying in complexity) and a subsidiary task, both in the presence and absence of incentives. It would be hypothesised that less motivated subjects would be impaired on less complex tasks (which require less processing capacity) than more motivated subjects, who are thought to increase processing capacity and therefore avoid impairment until the tasks become more complex. If this was the case, incentives should improve the performance of subjects who were originally impaired on the less complex tasks. If subjects were asked how hard they tried on the original tasks (without incentives) it would be anticipated that subjects with lower scores on such a measure would show more improvement in the presence of incentives than subjects who reported exerting more effort. In addition, it would be expected that the severity of anxiety would be related to improvement, with the most improvement occurring in the less anxious subjects; more anxious subjects would be hypothesised to be more aroused or motivated and therefore to benefit less from incentives.

In summary, this theory has value in that it has the potential to integrate research on memory in anxiety with that on memory in depression and, in addition, it extends Johnson and Magaro's (1977) hypothesis that impairment in all psychiatric disorders is due to the effects of effort and cognitive interference by stating the relationship between the two. It would therefore seem worthy of further investigation.

However, this theory does not provide the only possible interpretation of the data on memory in depression. For instance, a model put forward by Williams and Teasdale (1982) to account for findings in the learned helplessness literature may be applicable to memory in depression. The model was developed to explain the apparently discrepant finding that experimentally induced failure sometimes produced helplessness and sometimes facilitation. It is based on the hypothesis that the expectancy of success on a task is linked to the amount of effort the subject intends to exert: as perceived task difficulty increases the subject believes s/he will have to expend more effort for the same expectancy of success (Kukla, 1972b). However, the subject will not always increase the amount of effort expended, even if s/he perceives the need to do so in order to be successful on the task. According to Williams and Teasdale (1982) this is because exerting effort has a cost and therefore

acts as a negative incentive: the greater the effort needed to achieve success the greater the negative incentive. If the positive incentives to succeed on the task (such as importance of success and expectancy of success) are insufficient they will be exceeded by the negative incentive and the subject will cease to exert effort and give up. It is therefore hypothesised that subjects chose the effort level which maximises the difference between the positive incentives and the negative incentives (level of estimated effort and cost of that effort). The model suggests that as perceived difficulty of important tasks increases progressively more effort will be exerted and performance will be facilitated until a transition point is reached at which the cost of the effort necessary exceeds the importance and expectancy of success. At this point the subject abruptly ceases to expend effort and debilitated performance will result.

Although the model was tested in non-depressed subjects the authors suggest that it could account for impaired performance in severely depressed patients. They argue that depressed patients complain of fatigue which may lead to increased cost of exerting effort. In addition they show a pervasive loss of interest which may reduce the importance of success on a task. It can also be hypothesised that, as part of their negative view of themselves (Section 1.7.4) they are likely to have a reduced expectancy of success. This would lead them to view tasks as requiring more effort for successful completion than would non-depressed subjects, and therefore to be more difficult. It is therefore likely that in many circumstances the negative incentives (the cost of effort and the amount of effort perceived to be necessary) would outweigh the positive incentives (importance and expectancy of success) and that this would lead to performance deficit. This model has not been investigated in depression to date, although it has recently been suggested (Williams et al, 1988; p48) that it might account for performance deficits in depression and therefore may repay further investigation.

Some aspects of this model were based on work by Kukla (1972b) who investigated the effects of a number of inter-related factors on 'the vigour with which a task is undertaken' or the amount of effort expended. The factors included a task's perceived difficulty, the subject's perception of his/her own ability and experience of success and failure.

Kukla (1972b) argues that as the level of perceived difficulty is increased the amount of effort expended will also increase until a point is reached at which the level of difficulty is such that even the maximum possible level of effort would not produce success. In such circumstances the subject will not exert any effort. This relationship between effort and perceived difficulty is supported by the results of a study in which subjects were asked to indicate how much effort they would expend if they were to undertake tasks of varying difficulty (Weiner et al, 1972). The results showed that the relationship between task difficulty and intended effort expenditure is an inverted-U shaped function.

In addition to arguing that perceived difficulty determines effort expenditure, Kukla (1972b) also argued a role for perceived ability. According to this, subjects who believe that they have the ability to succeed on a task will expend less effort than those who believe their ability to complete the task is low. There is evidence that individuals who do not differ in their ability on a task do differ in their estimation of ability (Kukla, 1972a). High estimation of ability is related to high resultant achievement motivation (Kukla, 1972b) which in turn is related to anxiety: those high in resultant achievement motivation tend to be low in anxiety. It is hypothesised that, on a task which two subjects perceive to be equally difficult, the subject who believes his/her ability to be higher will expect that a smaller amount of effort is necessary for success. If this is the case then low perceived ability would lead to increased performance on easy tasks because subjects low in perceived ability judge the task to be more difficult than it is and to require more effort for completion. However, it would lead to decreased performance on a more difficult task because the subjects would reach the point at which the perceived effort requirement exceeds the available effort on tasks which are lower in objective difficulty than tasks on which subjects high in perceived ability reach this point. This pattern of results was found by Weiner (1966) who showed that subjects high in anxiety (and therefore low in perceived ability) were superior to those low in anxiety on subjectively easy tasks while the reverse was true for subjectively difficult tasks. These results therefore support the hypothesis that perceived ability can reduce performance on a task, presumably by increasing the amount of effort believed to be necessary

for successful completion and consequently the perceived level of difficulty of the task.

Finally, Kukla (1972b) argued that the experience of success and failure can influence effort expenditure. If a subject is successful on a task the perceived difficulty and effort expenditure will decrease and the reverse is true if failure is experienced. The effect of success on performance is hypothesised to vary according to the perceived difficulty of the task and the perceived ability of the subject. For instance success will improve the performance of subjects low in perceived ability on tasks initially perceived to be of intermediate difficulty (one which subjects with high opinion of their ability believe to be within their capacity while those with low opinions do not). This is presumably because such subjects perceive the task as requiring less effort after experience of success, see it as within their capacity and therefore begin to exert effort on the task. In contrast success will lead to reduced performance in subjects high in perceived ability, presumably because they also perceive it as requiring less effort and therefore reduce their effort expenditure. Failure is hypothesised to lead to decreased performance in those low in perceived ability (because the perceived amount of effort required exceeds that available) while leading to increased performance in those high in perceived ability. This pattern of results is supported by Weiner (1966).

Thus, according to Kukla (1972b) the amount of effort expended on a task will depend upon three inter-related factors: perceived task difficulty, perceived ability and experience of success or failure. It can be hypothesised that depressed people are likely to perceive tasks as being more difficult than others because, as argued above, they under-estimate the likelihood of success due to their negative opinion of themselves. It might be expected that this would lead to increased expenditure of effort and therefore improve performance. However, according to Kukla's model, this will not always be the case. Subjects are hypothesised to exert the minimum level of effort necessary to achieve success and consequently if the perceived effort requirement exceeds the available effort and consequently success cannot be achieved no effort will be expended. Depressed subjects are likely to reach this point and become impaired on tasks which are objectively easier than those on

which other subjects begin to experience impairment because of this tendency to over-estimate the difficulty of the task.

As outlined above, the model developed by Williams and Teasdale (1982) enhances this explanation of impairment in depression by suggesting that the likelihood of effort being expended and performance being improved will depend on whether the positive incentives (importance and expectation of success) outweigh the negative incentives (cost of effort and amount of effort required), not just on whether perceived effort requirement exceeds available effort. The strength of positive incentives is likely to be reduced in depression (by, for instance, the loss of interest common in depression leading to reduced importance of success) and the strength of negative incentives (for instance, the amount of effort believed to be necessary for success) is likely to be increased. This will lead to impairment on tasks requiring more effort (negative incentive) than the level of postive incentives justify.

The hypothesis that impairment in depression is related to a tendency to over-estimate the difficulty of the task (and therefore the amount of effort involved) and to the reduced importance of success seems to have the potential to explain memory deficit in depression. However, it is argued above (Section 9.3) that this is also true of Eysenck's working memory capacity model of impairment. How do these models differ?

First, Eysenck's model (Eysenck, 1982) has two components. These are cognitive interference from intrusive thoughts and worry which reduces the available processing capacity, and effort which can be increased to overcome the effects of reduced capacity. In contrast the Williams and Teasdale model does not specify a role for cognitive interference. Instead it attributes performance decrement to the balance between the amount of effort judged to be required for success on the task (which is determined by factors such as perceived task difficulty) and the strength of postive incentives (such as the importance of success). The evidence that memory deficit in depression is related to cognitive interference is, as outlined in Section 1.8.4, limited. However, even if it were to be found to be stongly related to performance this would not necessarily indicate that Eysenck's model is a better explanation of memory impairment than the alternative model. This is because it could be hypothesised that cognitive

interference is another factor which determines how difficult the subject judges a task to be: if some processing capacity is unavailable due to cognitive interference the subject may judge that the task is more difficult than would otherwise be the case and that it requires more effort for successful completion. This would be consistent with the suggestion by Kahneman (1973) that one of the determinants of the amount of effort expended by subjects is task demands: if depressed subjects have fewer processing capacities the task demands are likely to be greater and therefore perceived task difficulty will also be greater. Thus, although the two models differ in that only that developed by Eysenck postulates a role for cognitive impairment, Williams and Teasdale's model could be extended to include such a role. It is therefore not necessarily the case that evidence showing that memory impairment is related to cognitive interference would indicate that Eysenck's model should be accepted in preference to the model developed by Williams and Teasdale.

The two models also differ in that Eysenck hypothesises that the level of motivation will determine both how much effort is put into a task and whether the amount of effort will be increased to overcome the effects of reduced processing capacity. The model developed by Williams and Teasdale asserts that the determinants of the amount of effort expended are more complex than simply 'motivation' and include such inter-related factors as perceived task difficulty, perceived ability and the importance of success. Further research is needed to determine the relative importance of these factors but available research in non-depressed subjects does suggest that the amount of effort expended is determined by factors such as these (see above). This model may therefore be able to specify the amount of effort put into a task and the resultant performance rather better than Eysenck's theory which uses the more global and ill-defined concept of motivation.

The model developed by Williams and Teasdale (1982) may explain memory deficit in depression as well as the model developed by Eysenck (1982): further research is needed to determine whether this is the case. Eysenck's model, however, was developed to account for cognitive performance in anxiety (Section 4.1.3) and part of its attraction as an explanation of memory deficit in depression was its apparent ability to

account for impairment in a range of psychiatric disorders (Section 9.3). Can Williams and Teasdale's model also explain performance in anxiety and other psychiatric disorders as well as in depression?

As outlined in Section 4.1.3, the relationship between anxiety and performance can be described by an inverted U shape with optimal levels of anxiety decreasing as task difficulty increases. According to William and Teasdale's model anxious subjects show increased performance on easy tasks because they have low self-perceived ability and therefore believe that the task is more difficult and requires more effort for successful completion than a non-anxious subject would (Kukla, 1972b). As the objective task difficulty increases the amount of effort required also increases. As anxious subjects exerted more effort than non-anxious subjects on easier tasks they reach the point at which the negative incentive to exert effort outweighs the positive incentive sooner than non-anxious subjects. Thus this model can account for the pattern of performance found in anxious subjects.

It may also be able to account for impairment in other psychiatric conditions. For instance Johnson and Magaro (1987) suggest that the overall level of psychiatric disorder in subjects with a range of diagnoses may be related to memory impairment firstly because psychopathology leads to cognitive interference and secondly because it reduces the amount of effort exerted on the task. This model has the potential to explain why the amount of effort is reduced and to hypothesise in which situations this will lead to impairment. In the absence of evidence that impairment is related to cognitive impairment rather than reduced effort the model put forward by Williams and Teasdale can be seen to be able to account for impairment in conditions other than depression and anxiety. In addition, as argued above, the model could be modified to include a role for cognitive interference. There is, therefore, no reason to chose Eysenck's model in preference to that of Williams and Teasdale on the basis that the former may explain impairment in a range of psychiatric conditions.

It has been argued above (p327) that the results obtained in the studies reported in this thesis were supportive of Eysenck's model of memory impairment. However, it can be argued that this is also true of

Williams and Teasdale's model. For instance anxious patients were found to be less impaired than depressed patients on a battery of laboratory memory tests (Section 4.4.3). This could be explained by the anxious subjects having a negative view of their ability (Kukla, 1972b) and therefore increasing their estimation of task difficulty and the amount of effort required for successful performance. This would also be true of the depressed subjects. However, in the depressed subjects the increased perceived task difficulty would be combined with loss of interest leading them to view success as unimportant. This could lead to them reaching the point at which the amount of effort required (the negative incentives) exceeds the positive incentives for increasing effort on tasks which are easier than those on which anxious subjects reach this point. However, as the anxious subjects view the task as difficult and consequently as requiring more effort for successful completion, they would reach reach this point before normal subjects. The model could account in a similar way for the finding that anxiety is only related to performance on difficult tasks while depression is related to performance on both easy and difficult tasks (Section 6.4.

The model could also explain why depressed patients did not show impaired memory for information given to them by their general practitioners. This may be because the information is seen as important by the subjects and therefore the positive incentive of the importance of success is high and outweighs the negative incentives. Alternatively (or in addition) it may be that as the depressed general practice patients were less depressed than the psychiatric patients used in the other studies (Section 8.4) their negative self-image and expectancy of failure may have been less developed. In addition the cost of exerting effort may have been less in these subjects. The positive incentives may have outweighed the negative for these reasons.

This model can also account for the conclusion reached in the literature review (Sections 1.10 and 9.5) that memory deficit in depression is related to both task difficulty and the severity of depression. Increasing objective task difficulty would presumably lead to increasing perceived task difficulty and the subjects would therefore increase their estimations of the effort required for successful completion of the task. This would increase the negative incentive to

exert more effort. Increasing severity of depression may increase the expectation of failure and therefore the perceived difficulty of the task, while at the same time causing the depressed subject to lose interest in the task thus reducing the positive incentive of importance of success. Thus both increased task difficulty and increased depression would increase perceived task difficulty and the strength of the negative incentive to exert effort, while increased depression would also decrease the value of the positive incentives. The negative incentives would therefore be likely to outweigh the positive incentives, resulting in impairment. Thus the Williams and Teasdale model, like that of Eysenck, has the potential to explain previous research findings.

It has already been argued that research is needed to investigate both components of the model devised by Eysenck (1982), and to test the combination of these components to see if they operate to produce impairment in the way suggested by the model. In addition, it seems that the alternative model devised by Williams and Teasdale (1982) is also worthy of further research. Such research might, for instance, include experimental designs such as that used by Weiner (1966) which separated objective and perceived task difficulty by giving false norms for performance on tasks. Such a study would investigate the hypothesis that perceived task difficulty rather than objective difficulty is important in determining effort expenditure and consequent impairment. This could also be investigated by the direct approach of asking depressed and non-depressed subjects how difficult they expect a task to be and how much effort they expect to need to exert to succeed on the task. Other studies could involve the manipulation of the positive incentive of the importance of success by the use of rewards although, as argued above, this may prove ineffective with depressed subjects who have been shown to be impervious to most rewards (Layne et al, 1982).

In conclusion, two models applicable to memory in depression have been discussed: the working memory capacity model devised by Eysenck (1982) and the model devised by Williams and Teasdale (1982), which is based on the hypothesis that perceived task difficulty combined with positive incentives such as the importance of success will determine the amount of effort exerted on a task and the consequent likelihood of impairment. Both models have been shown to provide a reasonable

explanation of previous research findings and to have at least some evidence to support them. Further research is therefore needed to test specific hypotheses derived from these models and to test which of the models provides the best understanding of the causes of memory impairment in depression.

9.4 THREE APPROACHES TO MEMORY IN DEPRESSION

This thesis has taken three approaches to memory in depression: it has investigated the performance of depressed people on laboratory memory tests; their perceptions of their memory in everyday life; and their actual memory in an important everyday situation, the general practice consultation. Each of these approaches has different strengths and weaknesses, and different contributions to make to an understanding of memory in depression.

As regards the first, laboratory memory tests have an important role to play in that they can be used to test specific theories of memory in depression: for example, that the extent of memory impairment shown by depressed people (and indeed by those suffering from other psychiatric disorders) depends on how motivated the person is and the extent of interference from worry and intrusive thoughts. There is clearly a need for experimental investigations which are based on models and theories of memory in depression; most studies to date, like those described in this thesis, have not been designed to test a particular theory but to describe the performance of depressed people on a range of tasks, and on this basis make inferences about the causes and specific nature of impairment. Such studies have an important role to play in identifying the pattern of impairment and its relationship to impairment in other disorders, but the next stage must be to investigate models of memory which can account for this descriptive data. Experimental studies of memory in depression which can be carefully controlled, and in which the parameters of concern can be isolated and identified, are obviously needed to do this.

Laboratory memory tests are therefore needed to test models of memory in depression. However, they may be of less use to clinicians, who are

interested in developing some understanding of the problems which depressed people experience with memory in their everyday lives. Performance on laboratory memory tests does not necessarily give a good impression of the memory performance of depressed people in their everyday lives. They may be less motivated than usual when asked to complete laboratory memory tests, perhaps because they are pessimistic about their ability to do them (Revelle and Micheals, 1976; Strack et al 1985). If so, the tests will give a very negative picture of the amount of impairment they normally experience. Or they may make a special effort; if so the results on such tests will underestimate the problems they experience in their normal lives. In the absence of knowledge about the motivational levels of the depressed person, the amount of effort they are putting into the task, and how this relates to the maximum amount of effort they are able to sustain it is difficult to predict from laboratory memory tests how much of a memory problem the depressed people are experiencing. One way of using laboratory memory tests to at least give an idea of what depressed people are capable of might be to try and increase their motivational levels to a maximum, perhaps by the use of monetary rewards. This is, however, likely to be difficult as there is evidence that they are quite indifferent to most rewards and therefore difficult to motivate (Layne et al, 1982). It is not therefore certain whether the results of laboratory memory tests represent the best depressed people can do, or whether they would be capable of better performance in situations in which they are more motivated. They therefore give only a limited impression of the memory problems which depressed people experience in their everyday lives.

The second approach to memory in depression used in this thesis was the use of self-rating metamemory questionnnaires which ask people about their perceptions of their memory performance in everyday life. As reviewed in Chapter Seven these questionnaires, like laboratory memory tests, have their limitations. For instance correlations between scores on these questionnaires and scores on memory tests are usually minimal or even absent (Section 6.1.3). Although strong correlations might not be expected, because the questionnaires and memory tests tend to measure different aspects of memory, in the absence of stronger relationships psychologists have been reluctant to conclude that metamemory questionnaires give an accurate picture of memory. The results presented

in Chapter Seven suggest that people are at least fairly accurate in rating the incidence of cognitive failures in the previous six months, and in assessing the degree of change in their memories (particularly the latter). However, both the ratings of the frequency of cognitive failures and the assessments of change in memory were affected by anxiety; the more anxious the subject was, the more negative his/her assessment of his/her memory. Such questionnaires do not, therefore, necessarily give clinicians a good picture of the problems which depressed people experience with remembering things in their everyday lives.

Nevertheless, metamemory questionnaires have some value to psychologists interested in understanding more about memory in depression because they indicate what areas of memory depressed people think they find difficult. Such areas could then be explored systematically using laboratory memory tests, leading to a greater understanding of memory in depression. The most appropriate way to find the views of depressed people about their memory problems would be to ask a group of patients about them, and then to draw up a questionnaire based on their reports of memory problems; this is similar to the procedure used by Sunderland, Harris and Baddeley (1983), who were concerned with memory problems experienced by head-injured patients. This would give a better impression of the type of problems depressed people think they have than using questionnaires designed for other subject groups (such as the two questionnaires used in the study described in Chapter Seven; the MCQ was designed for use with head-injured patients (Sunderland, Harris and Baddeley, 1983) although it has also been used with the elderly (Sunderland et al, 1986), while the CFQ was intended for use in a general population (Broadbent et al, 1982)). One questionnaire has been designed specifically for use with depressed people (Squire, Wetzel and Slater, 1979) but this was concerned primarily with the effects of E.C.T rather than the effects of depression. A metamemory questionnaire specifically concerned with the memory problems experienced by depressed people, and based upon their accounts of memory failures, may therefore be useful in indicating areas in which depressed people think they have problems. These can then be explored experimentally; such a questionnaire might also be of use to clinicians concerned with finding out how much of a problem with memory patients are experiencing, provided that their anxiety level is taken into consideration when interpreting answers on the questionnaire.

The third approach to memory in depression taken in this thesis was to investigate performance in a realistic everyday situation; the general practice consultation. Like the two approaches discussed above, this too has its limitations. It is difficult to generalise from one such situation to another because there are different contextual cues and task demands in each situation and, because such studies are observational rather than experimental, such factors cannot be precisely measured and compared. In addition such studies can, as Baddeley (1981a) observed, be expensive and time-consuming; this was certainly the case with the study described in Chapter Eight of this thesis. Such studies may be useful in seeing whether or not depressed people experience the type of problems in everyday life that would be expected on the basis of their performance on laboratory memory tests, and on the basis of models of memory in depression. For instance it would be expected that, as depressed people are hypothesised to be less impaired when they are motivated and therefore increase the effort they exert to overcome the effects of cognitive interference, that they would not show much evidence of memory impairment when the information is personally relevant and important; the finding that depressed people did not have impaired memories for medical information supports this hypothesis.

In summary, it is clear that all approaches to memory in depression have limitations and that all three approaches are needed to obtain a full picture of the type of memory problems depressed people experience in their everyday lives and the reasons for impairment: metamemory questionnaires give an idea of the type of problems depressed people think they have and these self-reports can be used to generate hypotheses which can be investigated experimentally using laboratory memory tests; experimental investigations are essential to test and generate models of memory in depression and are necessary if understanding of the reasons for memory impairment in depression is to grow; investigations of memory in everyday situations can be used to check out these models and to ensure that theories generated in the laboratory are applicable outside. The fullest understanding of memory in depression will therefore be obtained by using all three approaches to the problem.

TABLE 9.1 SUMMARY OF EXPERIMENTAL STUDIES OF MEMORY IN DEPRESSION: I. SHORT-TERM MEMORY SCANNING (ADDITIVE FACTOR METHODOLOGY)

TYPE OF MATERIAL	TYPE OF DEPRESSED SUBJECT			
	DEPRESSED PSYCHIATRIC IN-PATIENTS	DEPRESSED PSYCHIATRIC OUT-PATIENT OR MIXTURE OF IN- AND OUT-PATIENTS	DEPRESSED SUBJECTS DEFINED ON PSYCHOMETRIC GROUNDS	INDUCED DEPRESSED MOOD
i) Verbal stimuli	Depressed patients had slower response latencies than non-depressed controls on four tasks and showed slower memory scanning on two of the four tasks (Brand and Jolles, 1987c)	Elderly depressed patients had the same slope function (and therefore same memory scanning speed) as non-depressed controls but had slower response latencies (Hart et al 1987c) Depressed patients had slower response latencies than non-depressed subjects but showed no difference in memory scanning process (Glass et al, 1981)	Elderly people with high levels of depression showed slower response latencies but similar memory scanning to those with low levels of depression (Hilbert, Niederehe and Kahn, 1976)	
ii) Picture stimuli		No evidence of slower memory scanning but some evidence of slower response latencies (Hart and Kwentus, 1987)		

TABLE 9.2 SUMMARY OF EXPERIMENTAL STUDIES OF MEMORY IN DEPRESSION: II. INTENTIONAL LEARNING - FREE RECALL

TYPE OF MATERIAL	TYPE OF DEPRESSED SUBJECT			
	DEPRESSED PSYCHIATRIC IN-PATIENTS	DEPRESSED PSYCHIATRIC OUT-PATIENT OR MIXTURE OF IN- AND OUT-PATIENTS	DEPRESSED SUBJECTS DEFINED ON PSYCHOMETRIC GROUNDS	INDUCED DEPRESSED MOOD
i) Words - unrelated, no processing instructions	Learning impaired compared non-depressed controls; no difference in retention, and no correlation between depression and immediate recall scores (Wolfe et al, 1987) Impaired compared to non-depressed controls in recall of words varying in emotional connotations (Frith et al, 1983) Impaired compared to own performance when less depressed (Henry, Weingartner and Murphy, 1973) Impaired on this aspect of a neuro-psychological test battery (Caine et al, 1973) Impaired compared to own performance when depression in remission (Whitehead, 1973) No significant difference between depressed and non-depressed controls (Silberman et al, 1983)	Significant difference from non-depressed controls in proportion with scores more than two standard deviations below mean for controls (Coughlan and Hollows 1984) No significant difference between elderly depressed and non-depressed controls on immediate or delayed recall (Popkin et al, 1982) No significant difference between recall of depressed and non-depressed subjects; no difference in amount of subjective organisation imposed on material (Davis and Unruh, 1980)	No significant relationship between severity of depression and performance in elderly people (West, Boatwright and Schleser, 1984) No significant difference between elderly subjects with high levels of depression and those low in depression (O'Hara et al, 1986)	
ii) Words differing in category membership or level of imagery.	a) Impaired compared to depressed controls only when list structure is not immediately apparent b) Impaired compared to non-depressed controls on recall of categorised words (Weingartner et al, 1981)	Impaired compared to non-depressed controls on recall of high imagery words, but not on recall of low imagery words (Hart et al, 1987c)		

TABLE 9.2 (cont) SUMMARY OF EXPERIMENTAL STUDIES OF MEMORY IN DEPRESSION: II. INTENTIONAL LEARNING - FREE RECALL

TYPE OF MATERIAL	TYPE OF DEPRESSED SUBJECT			
	DEPRESSED PSYCHIATRIC IN-PATIENTS	DEPRESSED PSYCHIATRIC OUT-PATIENT OR MIXTURE OF IN- AND OUT-PATIENTS	DEPRESSED SUBJECTS DEFINED ON PSYCHOMETRIC GROUNDS	INDUCED DEPRESSED MOOD
ii) Words differing in category membership or level of imagery (cont)	Recall impaired compared to non-depressed controls, and less clustering of categories shown in recall (Calev and Erwin, 1985) No significant difference between recall of depressed subjects and that of non-depressed controls (Silberman et al, 1985)			
iii) Trigrams of letters or digit-span tasks	Impaired on digit span backwards task compared to non-depressed controls, but not on digit span forwards task (Breslow, Kocsis and Belkin, 1980) Significant impairment on digit span task (Rapaport, 1945) No significant difference on digit span forwards task between depressed subjects and non-depressed controls (Kopelman, 1986)	Significant relationship between levels of depression and number of trigrams recalled after delay intervals of 3 to 18 seconds (Cohen et al, 1982)	No significant difference between subjects with high depression scores and those with low scores, and no relationship with severity of depression (Gass and Russell, 1986)	
iv) Prose	Significant impairment compared to non-depressed controls (Breslow, Kocsis and Belkin, 1981) Significant impairment only on positive themes, no difference between depressed subjects and controls in recall of neutral or negative themes (Breslow, Kocsis and Belkin, 1981)	No significant difference between depressed subjects and controls in proportion with scores two deviations below mean for controls on immediate or delayed recall (Coughlan and Hollows, 1984)	No significant difference between subjects with high depression scores and those with low scores (Gass and Russell, 1986)	

TABLE 9.2 (cont) SUMMARY OF EXPERIMENTAL STUDIES OF MEMORY IN DEPRESSION: II. INTENTIONAL LEARNING - FREE RECALL

TYPE OF MATERIAL	TYPE OF DEPRESSED SUBJECT			
	DEPRESSED PSYCHIATRIC IN-PATIENTS	DEPRESSED PSYCHIATRIC OUT-PATIENT OR MIXTURE OF IN- AND OUT-PATIENTS	DEPRESSED SUBJECTS DEFINED ON PSYCHOMETRIC GROUNDS	INDUCED DEPRESSEI MOOD
iv) Prose (cont)	Significant impairment compared with controls on immediate and delayed recall, but not on retention; no correlation with with severity of depression (Kopelman, 1986) Significant improvement in recall scores following treatment with ECT (McAllister et al, 1987) Significant relationship with severity of depression (Stromgren, 1977) Significant impairment (Rapaport, 1945) Significant impairment compared to non-depressed controls and significant relationship with severity of depression (Watts and Sharrock, 1987) Significant relationship between recall and severity of depression (Watts and Sharrock, 1985) No significant difference between performance of subject when depressed and performance following remission of depression (Whitehead, 1973)	No significant difference between elderly depressed patients and controls (Popkin et al, 1982)	No difference between students with high depression scores and non-depressed students in overall recall, or on recall of themes with varying affective tones (Hasher et al, 1985) No significant difference between subjects over 50 years of age who are high in depression, and those low in depression (Kahn et al, 1975)	

TABLE 9.2 (cont) SUMMARY OF EXPERIMENTAL STUDIES OF MEMORY IN DEPRESSION: II. INTENTIONAL LEARNING - FREE RECALL

TYPE OF MATERIAL	TYPE OF DEPRESSED SUBJECT		
	DEPRESSED PSYCHIATRIC IN-PATIENTS	DEPRESSED PSYCHIATRIC OUT-PATIENT OR MIXTURE OF IN- AND OUT-PATIENTS	DEPRESSED SUBJECTS DEFINED ON PSYCHOMETRIC GROUNDS	INDUCED DEPRESSE MOOD
v) Non-verbal material	Significantly impaired compared to non-depressed controls (Breslow, Kocsis and Belkin, 1981) Significant relationship between performance on immediate and delayed reproduction and severity of depression (Stromgren, 1977) Significant improvement following treatment with ECT (McAllister et al, 1987) Significant impairment compared to non-depressed controls, relationship between severity of depression and performance on immediate and delayed recall tasks, and improvement on remission of depression (Fisher, Sweet and Pfaetzer-Smith, 1986)	Depressed prisoners significantly impaired compared to non-depressed prisoners (Robertson and Taylor, 1985) Significantly more depressed subjects than non-depressed controls had scores more than two standard deviations below mean score for control group (Coughlan and Hollows, 1984)		

TABLE 9.3 SUMMARY OF EXPERIMENTAL STUDIES OF MEMORY IN DEPRESSION: III. INTENTIONAL LEARNING - CUED RECALL

TYPE OF MATERIAL	TYPE OF DEPRESSED SUBJECT			
	DEPRESSED PSYCHIATRIC IN-PATIENTS	DEPRESSED PSYCHIATRIC OUT-PATIENT OR MIXTURE OF IN- AND OUT-PATIENTS	DEPRESSED SUBJECTS DEFINED ON PSYCHOMETRIC GROUNDS	INDUCED DEPRESSE MOOD
i) Words	Significant impairment compared with non-depressed controls (Cutting, 1979) Significant relationship between severity of depression and performance (Stromgren, 1977) Significant impairment compared to non-depressed controls (Cronholm and Ottosson, 1961; Kopleman, 1986; Steif et al, 1986) Significant impairment compared to non-depressed controls, and improvement on remission of depression (Sternberg and Jarvik, 1976) No significant difference between depressed subjects and non-depressed controls (Breslow, Kocsis and Belkin, 1981) No significant difference between performance when depressed and performance when in remission (Whitehead, 1973)		No significant difference between scores of elderly subjects with high levels of depression and those with low levels (Kahn et al, 1975)	
ii) Prose	Significant impairment compared to non-depressed controls and significant relationship with severity of depression (Watts and Sharrock, 1987) No significant relationship with severity of depression (Watts and Sharrock, 1985)			
iii) Non-verbal material	Significant difference between performance of depressed and non-depressed controls when photographs were used to cue facts learnt earlier (Cronholm and Ottosson, 1961; Frith et al, 1983; Sternberg and Jarvik, 1976; Steif et al, 1986)			

TABLE 9.4 SUMMARY OF EXPERIMENTAL STUDIES OF MEMORY IN DEPRESSION: IV. INTENTIONAL LEARNING - RECOGNITION

TYPE OF MATERIAL	TYPE OF DEPRESSED SUBJECT			
	DEPRESSED PSYCHIATRIC IN-PATIENTS	DEPRESSED PSYCHIATRIC OUT-PATIENT OR MIXTURE OF IN- AND OUT-PATIENTS	DEPRESSED SUBJECTS DEFINED ON PSYCHOMETRIC GROUNDS	INDUCED DEPRESSED MOOD
i) Words	Significant impairment compared to non-depressed controls (Calev et al, 1986; Frith et al, 1986; Roy-Byrne et al, 1986; Wolfe et al, 1987) Significant impairment compared to non-depressed controls and, unlike controls, better performance on recognition test than on free recall test (Calev and Erwin, 1985) No overall difference in recognition rates, but depressed patients had better detection rates (d') for negative words and worse recognition rates for positive words than controls (Dunbar and Lishman, 1984) Depressed patients had lower d' rates than non-depressed controls but did not differ from controls in response bias, except in vocalisation condition (Watts, Morris and MacLeod, 1987) No difference between performance when depressed and performance when depression was alleviated (Whitehead, 1973)	No significant difference between depressed subjects and non-depressed controls (Davis and Unruh, 1980) No significant difference between depressed subjects and non-depressed controls in proportion with scores more than two standard deviations below the mean score for the controls (Coughlan and Hollows, 1984)		
ii) Prose	No significant difference between depressed subjects and non-depressed controls ((Watts and Sharrock, 1987)			

TABLE 9.4 (cont) SUMMARY OF EXPERIMENTAL STUDIES OF MEMORY IN DEPRESSION: II. INTENTIONAL LEARNING - RECOGNITION

TYPE OF MATERIAL	TYPE OF DEPRESSED SUBJECT		
	DEPRESSED PSYCHIATRIC IN-PATIENTS	DEPRESSED PSYCHIATRIC OUT-PATIENT OR MIXTURE OF IN- AND OUT-PATIENTS	DEPRESSED SUBJECTS DEFINED ON PSYCHOMETRIC GROUNDS / INDUCED DEPRESSED MOOD
iii) Non-Verbal Material	Significantly impaired compared to non-depressed controls on immediate and delayed recognition; no difference in forgetting (Cronholm and Ottosson, 1961; Sternberg and Jarvik, 1976; Cutting, 1979)	No significant difference between depressed and non-depressed elderly subjects in d' but depressed subjects showed greater response bias (Miller and Lewis, 1977)	
	Significantly impaired compared to non-depressed controls (Calev (Calev et al, 1986)	Depressed prisoners significantly impaired compared to non-depressed prisoners (Robertson and Taylor, 1985)	
	Elderly depressed subjects took longer to reach threshold on on a recognition test but showed similar retention (Hart et al, 1987a)	No significant difference between depressed and non-depressed control subjects in proportion getting scores more than two standard deviations below the mean score for the controls (Coughlan and Hollows, 1984)	

TABLE 9.5 SUMMARY OF EXPERIMENTAL STUDIES OF MEMORY IN DEPRESSION: V. INCIDENTAL LEARNING

TYPE OF MATERIAL	TYPE OF DEPRESSED SUBJECT		
	DEPRESSED PSYCHIATRIC IN-PATIENTS	DEPRESSED PSYCHIATRIC OUT-PATIENTS OR MIXTURE OF IN- AND OUT PATIENTS	DEPRESSED SUBJECTS DEFINED ON PSYCHOMETRIC GROUNDS	INDUCED DEPRESSED MOOD
i) Free Recall	Significant impairment compared to non-depressed controls following task involving comparative judgements about pairs of words (Roy-Byrne et al, 1986) Significant impairment in recall of semantically processed words but not in recall of acoustically processed words (Weingartner et al, 1981) Significant impairment in number of random words recalled after sorting task, but no impairment in recall of related words (Weingartner et al, 1981) Significant impairment following task involving rating words for emotionality (Silberman et al, 1985)		No difference between depressed students and non-depressed students on incidental learning task (Hasher et al, 1985)	Induced mood led to: a) recall of fewer words and failure to show expected benefit from level of elaborative detail in sentences b) fewer words recalled overall, but subjects benefitted from semantic orientation c) subjects failed to show expected benefit from use of difficult words in sentence-completion tasks (Ellis et al, 1984)
ii) Cued recall				Depressed mood induced after rating sentences for complexity led to an overall reduction in recall, but elaborati led to superior recal of target words (Ellis et al, 1985)
iii) Recognition	Significant impairment following incidental learning task (Sliberman et al, 1985)			

9.5 THE NATURE OF THE MEMORY DEFICIT IN DEPRESSION

Further investigations of memory in depression need to be directed towards elucidating the nature of the memory impairment for, as indicated in the previous section, most previous studies have been descriptive rather than being intended to test particular models or theories of memory impairment in depression. There have been some exceptions which are reviewed in Section 1.7. However, these studies were discussed only in relationship to the particular theory which originally inspired the investigation. In this section previous work on memory in depression will be examined in the light of current theoretical understanding of memory in order to investigate more fully the nature of the deficit in depression and to give some indication of areas and issues which warrant further research.

Tables 9.1 to 9.5 gives a summary of research on memory in depression, sub-divided by the type of task used and by the type of depressed subject. The tasks used and the results will be discussed below but first the reason for sub-dividing depressed subjects into the groups used will be discussed.

Depressed subjects were divided into psychiatric in-patients, psychiatric out-patients or mixtures of in- and out- patients, subjects defined as depressed on psychometric grounds (that is scores on depression inventories) who were not receiving psychiatric help, and subjects in whom depressed mood was induced. These divisions were used partly because they reflect the putative relationship between severity of depression (or general psychopathology) and extent of impairment which has been discussed earlier in this thesis (Hasher et al, 1985; Sections 9.1 and 9.2). However they were also chosen because of the difficulty in comparing the diagnostic group of depressed subjects used in different studies which in turn reflects the variety of terms used to describe sub-groups of depression (Section 1.3.2). Given that there is evidence to support a distinction between the syndrome of severe or endogenous depression and other forms of depression (Section 1.3.2) it may have been fruitful to separate off those studies which labelled their subjects as suffering from endogenous depression or used one of the synonymous labels. This would not necessarily have resulted in a homogenous group of

subjects, however, as various definitions of endogenous depression do not always produce comparable groups, as indicated by differences in response to anti-depressant therapy between subjects classified as endogenous by the Newcastle Scale and DSM-III melancholics (Carney, Reynolds and Sheffield, 1986).

The difficulty in differentiating studies which have included endogenous depressed subjects from those which have not may be limiting understanding of memory in depression as there is some evidence that endogenous depressed patients differ on some factors which may be related to memory impairment. For instance it has been shown that endogenous depressed patients (classified according to the Research Diagnostic Criteria (Spitzer, Endicott and Robins, 1978)) have a cognitive component to psychomotor retardation in addition to the motor component common to endogenous and non-endogenous depression (Cornell, Suarez and Berent, 1984). Psychomotor retardation has been suggested as a cause of impairment in depression (Section 1.8.3) and it may be that the additional cognitive component in endogenous depression makes endogenous depressed people particularly likely to experience memory impairment. This may help to explain why only one out of four studies on short-term memory scanning in depression has found any evidence of reduced information-processing speed ((Section 1.7.2; Table 9.1). Although the information given in the paper is too limited to draw firm conclusions, it is possible that this study (Brand and Jolles, 1987) included more subjects with endogenous depression. This example indicates how the endogenous versus non-endogenous differentiation may be an important one to consider in future studies and, indeed, some researchers have already begun to take it into consideration (for example, Silberman et al, 1985; Watts, Morris and MacLeod, 1987).

Other characteristics of depressed subjects, besides the potentially important distinction between endogenous and non-endogenous depression, may need to be taken into account as well. As outlined in Section 9.3, it is possible that memory deficit in depression (and possibly in other psychiatric conditions) is a product of reduced processing capacity due to intrusive thoughts and a lack of effort. Progress in the field may be enhanced if ways of assessing these factors, such as those discussed in Section 9.3, are developed and included in future studies to make

comparison of subjects on these factors possible, and to examine the relationship between these and performance on memory tasks. The development of such measures is problematic but may be important in reaching a better understanding of memory in depression.

In the absence of robust ways of dividing studies into those with endogenous subjects and those without, and of assessing how motivated were subjects in different studies and how much processing capacity they had available, it was decided the most useful division of subjects in previous studies was that outlined above which uses psychiatric patient status as a proxy for severity of depression.

It can be seen in Tables 9.1 to 9.5 that the majority of investigations of memory in depression have used psychiatric in-patients: only in Table 9.2 (intentional learning - free recall) are there substantial number of studies using either psychiatric out-patients or subjects defined on psychometric grounds. On 22 of the 26 free recall tasks given to depressed in-patients there was evidence of significant impairment in relation to non-depressed controls, a significant relationship with the severity of depression or significant improvement on the remission of depression (on some tasks, for instance that used by Breslow, Kocsis and Belkin (1981), the depressed subjects were impaired on one aspect of the task (recall of positive themes) and not on others (recall of negative or neutral themes). Such tasks are classified as indicating impairment). Depressed out-patients showed impairment on six out of nine tasks, while those who were classified as depressed on psychometric grounds were impaired on none of the five tasks they were given. The highest incidence of impairment was therefore found in the depressed in-patients. This is consistent with the argument that the presence of memory impairment in depression is dependent on the severity of depression, and that memory impairment may only be found in people with symptoms severe enough to warrant psychiatric in-patient treatment (Sections 1.4 and 9.1).

However, results for the incidental learning paradigm (Table 9.5) show that depressed mood induced in non-depressed subjects can cause impairment on such tasks. This is surprising given the failure to find impairment in subjects defined as depressed on psychometric grounds and may, as argued in Section 1.10, be due to tighter experimental control in

the induced mood studies. However, the studies using induced mood and those using psychometrically defined subjects are not comparable as the former involved incidental learning and the latter intentional learning. There are no investigations involving incidental learning which have used either out-patients or psychometrically defined subjects, and no studies using subjects in whom depressed mood is induced which have used intentional learning free recall tests. It is therefore not possible to draw firm conclusions from these studies: it may be that induced mood produces impairment on tasks which mild clinical depression and high levels of depressive symptomatology do not affect, or that the latter conditions would cause impairment on incidental learning tasks if tested on appropriate subjects. The finding of memory impairment in subjects in whom depressed mood has been induced does not, therefore, necessarily provide a challenge to the hypothesis that memory impairment in depression is related to the severity of depression. It does, however, indicate the need for investigations of the performance of depressed subjects differing in severity on the same battery of tests.

The study reported in Chapter Four of this thesis used two groups of depressed subjects who differed in the severity of depression, as assessed by the Present State Examination (Wing, Cooper and Sartorius, 1974). The most severe memory impairment was found in the more severely depressed group of subjects (Retarded Depressed) and these subjects were impaired on the largest number of tests: the less depressed Neurotic depressed group were impaired only on what were judged to be the more difficult tests. The finding that depressed general practice patients did not show impaired recall for information given to them by their general practitioner (Chapter Eight) was also considered to support the suggestion that impairment in depression is related to the severity of depression. These results are therefore consistent with impressions gained from the review of previous studies.

However, it is not clear why severity of depression is related to memory impairment in depression. As indicated above, it may be that what is important is whether the subjects have endogenous or non-endogenous depression: this may be difficult to disentangle from the effects of severity of depression because groups such as those used in this thesis differ both in severity and symptom pattern. Alternatively, it may be

that what is important is the overall level of psychopathology rather than depression per se at all (Section 9.2). The question of why the severity of depression is related to memory impairment in depression is therefore worthy of further research.

Such studies would throw light on the incidence of memory impairment in depression and may help to establish why impairment occurs. They would do little however to elucidate the nature of memory impairment in depression. As already noted (Section 9.4) much of the literature on memory in depression is descriptive and concerned with identifying what tests depressed people are impaired on: only a minority of studies have been concerned with testing specific hypotheses based on current theoretical understanding of memory in order to investigate precisely where the deficits lie (Section 1.7). The remainder of this section is concerned with bringing together the results of previous studies to establish what is currently known about the nature of the deficit in depression, and to highlight directions for future research.

The majority of studies of memory in depression have been concerned with verbal rather than non-verbal memory: eleven of the 50 tasks given to depressed in-patients were non-verbal tasks, and three of the fourteen given to out-patient groups. There is some suggestion that performance on tasks using non-verbal material may be more consistently impaired than that on verbal tasks: depressed in-patients were impaired on all eleven non-verbal tasks compared with 28 of the 39 verbal tasks while out-patients were impaired on three of the four non-verbal tasks and four of the nine verbal tasks. However, it is difficult to draw conclusions unless the same group of subjects have completed both verbal and non-verbal memory tasks since there are likely to be individual differences in memory for words and pictures. For instance Woodhead and Baddeley (1981) selected subjects according to performance on a facial recognition task and found that although good recognizers on this test performed better than poor recognizers on recognition tests using faces and paintings, the groups did not differ on a word recognition test.

Cutting (1979) gave a verbal and a non-verbal test to the same group of depressed patients in order to investigate whether depression is a right-. left-, or bilateral hemisphere problem (Section 1.8.5). The

depressed patients were significantly impaired compared to non-depressed controls on both tests which Cutting argues indicates that there is a bilateral problem. However, the two test scores were not directly compared and therefore the possibility that the depressed subjects were more impaired on one type of test than the other cannot be ruled out. Direct comparison of the tests used in this study would be difficult to interpret as the verbal test used cued recall while the non-verbal test involved recognition memory. This means that the tests differed on dimensions other than type of material involved. Steif et al (1986) overcame this problem by using two recognition tests and found that depressed subjects performed more poorly on the word recognition test than on that using faces. This does not necessarily, however, provide strong evidence for a differential deficit in depression as the face recognition test was found to be less discriminating than the word test and may therefore have been less sensitive to differences between the patients and controls.

These results highlight the difficulty of comparing results for tests differing in psychometric properties where differential deficits may reflect differences in sensitivity of the tests to impairment or other differences between the tests such as the level of difficulty, rather than different effects of depression on the dimension under investigation. As has recently been noted (Williams et al, 1988) interaction effects, whereby depression affects performance more on one test than on another, are of particular interest as they shed light on the nature of deficit in depression. However they are beset by methodological problems such as these. One solution is to use matched task methodology whereby two tests are developed which give equivalent scores when used on a normal population. Calev et al (1986) developed matched tasks to investigate the effects of depression on verbal and non-verbal memory and found no differential deficit in the two types of memory. There is, therefore, no evidence at present that the effects of depression on verbal or non-verbal memory differ. However, the available research is limited and, as indicated, beset by methodological problems. More research is therefore needed in this area especially as such a differential deficit may support the argument that changes in cerebal function underlie at least some of the memory deficits observed in depression (Section 1.8.5).

These studies on the differential effects of depression on verbal and non-verbal memory are some of the few studies which have been concerned with testing a particular hypothesis about memory performance in depression, rather than describing performance on a variety of tests and then trying to draw conclusions from the pattern of results. Another study concerned specifically with elucidating the nature of the memory impairment in depression is that of Weingartner et al (1981) which was based on the 'levels of processing' model of memory (Craik and Lockhart, 1972; Section 1.7.3). The results of this study showed that depressed in-patients did not benefit from semantic processing instructions which would be expected to improve memory in normal subjects but that they did not differ from controls on an acoustic processing task. This was taken to indicate that the depressed subjects did not process material as 'deeply' as controls, thus reducing the likelihood of it being recalled later. The results also showed that the depressed subjects did not make use of encoding strategies which would be useful in organising input and facilitating later recall.

It is likely that 'depth' of processing, as indicated by semantic rather than acoustic or structural processing, is not the only determinant of how well something is processed and later recalled. For example there is evidence that elaboration is also important: Craik and Tulving (1975) found that an unexpected memory test after sentence elaboration tasks showed that, although 'deep' processing was involved throughout, more words were recalled from elaborative sentence frames. The effect of depression on the recall of elaborative material has been investigated by Ellis et al (1984) in a study which used subjects in whom depressed mood was induced. The results showed that these subjects did not show the expected benefit from the level of elaborative detail, which was taken to indicate that depressed mood led to ineffective processing. Subjects did benefit from semantic orientating instructions suggesting,as Weingartner et al (1981) argued for depressed patients, that such subjects can make use of 'deeper' (or more elaborative) encoding strategies when their attention is drawn to it but do not engage in such processing spontanteously. Induced depressed mood also led to subjects failing to benefit from another manipulation designed to produced 'deeper' more detailed processing. Normal subjects recall difficult words in sentence completion tasks better than easy words, presumably because more

resources are put into processing them (Tyler et al, 1979): depressed subjects did not show this pattern of results.

Other studies have shown that clinically depressed subjects fail to benefit from characteristics of material and/or processing instructions which usually lead to enhanced recall in non-depressed people. For instance, Silberman et al (1985) found that the emotionality of words did not enhance later recall and recognition as it would be expected to do (and did) in non-depressed controls. Hart et al (1987c) found that depressed patients were significantly impaired compared to controls in the recall of high imagery words, but not in the recall of low imagery words. Again this indicates that they did not benefit from a characteristic of the material which would normally lead to better recall, presumably because it leads to more elaborative rehearsal. There is also evidence that depressed subjects are less able to structure material at encoding and do not show the normal tendency of non-depressed subjects to order randomly presented words into semantic categories (Calev and Erwin, 1985; Weingartner et al, 1981). Related to this is some evidence that depressed subjects benefit less than non-depressed controls from increasing structure in word lists (Levy and Maxwell, 1967).

In summary, a number of studies of memory in depression have manipulated factors designed to lead to 'deeper' or more elaborative processing which are known to result in enhanced recall in normal subjects. It is presumed that they have this effect because they maintain the representation of the input in active memory for longer, thus making it more likely that associations would be formed between the representation and semantic nodes in memory (Glass and Holyoak, 1986) and, as Craik and Lockhart (1972) have argued, ensuring that more aspects of the representation are attended to, thus producing a more discriminable memory trace with more associations to other nodes in memory. Studies which have manipulated these factors in depression have shown that depressed patients (and those in whom depressed mood has been induced) do not benefit from factors such as semantic processing instructions or high imagery words which should lead to better recall, although there is some evidence that they can benefit from these factors when their attention is drawn to it (Weingartner et al, 1981; Ellis et al, 1984). There is, therefore, evidence that both depressed patients and

those with induced depressed mood do not engage spontaneously in active processing at the encoding stage which will enhance later recall.

If this is the case, it might be expected that depressed patients would show more impairment in the recall of unassociated words (which lack structure and may require considerable effort to process in a way that is likely to lead to complex and elaborative memory traces which will aid recall) than in the recall of stories or prose passages which have an obvious and well-established hierarchical structure (which aids processing and can be used to help construct the story at recall). Such a pattern of results would be consistent with the finding of Weingartner et al (1981) that depressed patients only benefit from structure which is obvious at presentation, as it is in a story or prose passage.

There is little difference between the number of studies with depressed in-patients as subjects which have found impairment on a prose passage recall task (eight out of nine) and the number which have found impairment on a word recall task (eight out of ten). Neither of the studies using depressed out-patients found impairment on a prose passage task, while three of the four studies using word recall tasks reported impairment. Studies using psychometrically defined subjects found no impairment either on prose passage tasks (three studies) or word recall tasks (two studies). The number of studies using out-patients limit the conclusions that can be drawn. However, if it is presumed that prose passage recall does demand less effort than word recall the pattern of results seems consistent with the conclusion drawn earlier in this thesis (Sections 4.4.1 and 9.3) that there is 'an interaction between the severity of depression and the difficulty of the task such that impairments will be most consistently be found on difficult tasks in severely depressed patients' (pg 169).

However, it is difficult to draw firm conclusions from this summary of previous studies as it is not apparent that the subjects used in different studies were comparable: the two studies using prose passage recall with out-patients may have had less severely depressed subjects that those using word recall tasks and this may account for the higher incidence of impairment on the latter tasks. One of these studies measured both word and prose recall (Coughlan and Hollows, 1984) and

reported impairment on the word recall task but not on the prose recall task. However a stringent definition of impairment was used (scores more than two standard deviations below the mean score of the control group) and impairments may have been found on both tasks if the means of the two groups had been compared directly. Whitehead (1973) also used both word and prose recall tasks, this time with depressed in-patients. She found impairment compared to the performance of the same subject when depression had alleviated only on the word recall task. Again, however, the study was flawed as there is some doubt as to how it was decided that the depression was in remission. There may, therefore, have been residual depression which may have accounted for this finding (Section 1.6). The study reported in Chapter Four of this thesis found that severely depressed subjects ('Retarded Depressed') were impaired on both prose passage recall and word recall tests, while the less severely depressed neurotic depressed subjects were only impaired on the word recall test. These results suggest that less severe depression is needed to produce impairment on the word recall test than on a prose recall test which provides some evidence that depressed subjects are less likely to be impaired on a task where the structure is evident than on one where there is no (or little) apparent structure.

The question of whether clinically depressed subjects show less impairment on structured material such as prose passages than on less structured material remains unsettled and would warrant further research. It is clear, however, that the finding reported by Ellis and Ashbrook (1987) that induced depressed mood does not affect prose recall does not apply to clinical depression: nine studies (including that reported in this thesis) have found significant impairment on prose recall tests. Further research could usefully be directed to establishing whether depressed subjects show the same benefit from structure in material as non-depressed controls or whether, as suggested by the findings reported by Levy and Maxwell (1968) and Weingartner et al (1981), non-depressed subjects benefit more from structure and depressed subjects can only utilise it when it is obvious. Such a finding would not necessarily conflict with the suggestion that structured material is less affected by depression than unstructured material: depressed subjects may benefit sufficiently from structure to remember structured passages more easily

than unconnected words while still benefitting less from the structure than non-depressed controls.

In summary, there is evidence that encoding is affected by depression with depressed subjects failing to engage in 'deep' or elaborative encoding or to benefit from characteristics of the material such as imagery or structure which would result in increased recall for non-depressed people. This is presumably because insufficient effort is available or utilised to engage in such processing (Section 4.4.1).

Until recently most studies of memory in depression which have moved beyond the descriptive and attempted to test particular hypotheses have concentrated on the encoding stage of information processing. This has reflected a lack of interest in retrieval generally amongst psychologists: indeed the 'levels of processing' approach to memory dismissed retrieval as 'probably automatic' (Craik and Lockhart, 1972). However, there is increasing interest in retrieval amongst psychologists. Some of the current ideas about retrieval will be discussed next, followed by a review of what is known about the effects of depression on retrieval.

The traditional view of retrieval, as outlined in Tulving's Encoding Specificity Principle (Tulving and Thomson, 1973), is that whether or not a cue succeeds in retrieving a memory will depend entirely on whether the information related to the cue was encoded on the memory trace at encoding: the memory trace is an elaborative structure and retrieval is simply a matter of comparing cues with traces until a match is found. This is known as direct access retrieval. There is some evidence to support this hypothesis; for instance Tulving and Watkins (1975) presented evidence that different contexts at encoding favoured the production of different memory traces with, for instance, a rhyme context favouring the creation of memory traces with rhyme cues encoded on them. One effect of 'deep' or elaborative encoding may, therefore, be to increase the number of potentially successful retrieval cues by increasing the amount of information encoded on the memory trace. In contrast to the direct access approach to retrieval, however, it is now recognised that in at least some situations retrieval may be an active process in which associated concepts may be activated in memory at retrieval, thus

allowing associations to the target which were not encoded on the memory trace at encoding to be effective as cues.

This has led to the development of a number of generate-recognise models of retrieval, which argue that there are two stages to retrieval: the first stage involves the generation of potential targets while the second involves the identification of the correct target. According to this view the success of retrieval is not dependent upon what is encoded on the memory trace as associations can be generated between memory nodes to aid recall at the retrieval stage. Several generate-recognise models have been developed and these will not be reviewed in detail here (see Le Voi, 1986 for a review). The debate about which version of the model best explains the available data will continue, but it is now recognised that both direct-access retrieval and generate-recognise retrieval may be available for use in different situations (Le Voi, 1986). For instance Jones (1978) has argued that direct access retrieval may operate when the cue is intrinsic (or contained within the original stimulus) while generate-recognise retrieval may be necessary when the cue is extrinsic (not contained within the original stimulus). From the perspective of understanding the nature of the memory deficit in depression the important aspects of these models is the recognition that retrieval may not be a matter of directly accessing material from memory with all active processing and organising of material taking place at encoding, but that it may involve an active and elaborative retrieval process.

It is difficult to differentiate between processing at the encoding stage of information processing and processing at the retrieval stage because the two are clearly intertwined. For instance, one explanation of the results of 'distractor paradigm' experiments (Brown, 1958) (in which a distractor is placed between presentation and recall, leading to a dramatic deterioration in performance over a series of trials) is that the distractor prevents subjects from forming associations between the targets and other concepts in memory that could serve as retrieval cues (Glass and Holyoak, 1986). Thus inefficient or inadequate processing can affect the success of retrieval and it would be difficult to determine whether the results reflected inadequate encoding, inefficient retrieval or both. Despite the difficulty in differentiating between the two, the

new emphasis on active retrieval processes has raised the possibility that encoding operations may not be the sole, or the most important, determinant of successful retrieval.

To date, there has been little systematic exploration of the effect of depression on retrieval: as the above review has indicated the emphasis, as in research on memory in non-depressed subjects, has been on encoding. Indeed, the studies which have systematically investigated encoding in depression (for instance Weingartner et al, 1981; Ellis et al, 1985) have almost exclusively used free recall tests and have therefore shown little interest in retrieval. There has, however, been some suggestion that recognition memory is less affected by depression than free recall (Watts and Sharrock, 1987; Williams et al, 1988). This suggestion was originally based upon an study by Miller and Lewis (1977) which concluded that elderly depressed patients did not have impaired memories when recognition tests were used. However, there is some doubt as to whether the conclusion reached in this study that recognition memory is less affected by depression than free recall is supported by the results of other studies. For instance depressed in-patients were found to be significantly impaired on 22 out of 26 free recall tests (85 per cent), nine out of twelve cued recall tests (75 per cent) and ten out of twelve recognition tests (83 per cent) (Table 9.1). Depressed out-patients showed impairment on six of nine recall tests (66 per cent) and one of five recognition tests (20 per cent). Thus there is little evidence that depressed in-patients are more likely to be impaired on free recall tests than on either cued recall or recognition memory tests although there is some suggestion that the out-patient group (who are presumed to be less depressed (Section 4.4.1)) may be less impaired on recognition tests than on free recall tests. The initial view that recognition memory is less affected by depression may have reflected the fact that Miller and Lewis (1977) used elderly depressed patients who were in the care of the psycho-geriatric service and who may have been mildly depressed. The authors give no information on the type or severity of depression so it is difficult to establish whether this was the case. These results might therefore be consistent with the results for the less depressed out-patients, but not applicable to the more severely depressed psychiatric in-patients.

It appears, therefore, that the view that recognition memory is less likely to be affected by depression than free recall is not supported in depressed in-patients. It is possible that such an effect is shown by the depressed out-patients which may reflect an interaction between the severity of depression and the difficulty of the task (Section 4.4.1). However, a review of previous studies cannot provide a conclusive answer to the question of whether particular aspects of memory are more affected by depression than others as the characteristics of both subjects and tests used differ between studies. This may be particularly important here because, as indicated above, there are individual differences between subjects in whether they find verbal and non-verbal tasks easier and this makes comparisons between different studies using different materials difficult. Direct comparisons of free recall and recognition memory tasks given to the same group of subjects are needed. This is problematic as the tests may differ in ways other than that under investigation, the recognition test may be easier for instance. Matched task methodology is therefore needed in order to detect a differential effect of depression on free recall tests as compared to recognition tests, or vice versa. Calev and Erwin (1985) used such methodology to ensure the free recall and recognition tests were comparable and found that depressed subjects, unlike non-depressed controls, performed better on the recognition test than on the recall test although they were significantly impaired on both.

Watts and Sharrock (1987) have also examined whether depressed subjects show more impairment on free recall tests than on recognition tests. They used the 'Circle Islands' prose passage, in comparison to Calev and Erwin (1985) who used a word recognition test, and found that depressed subjects had significantly lower scores than non-depressed controls on the free and cued recall tests but not on the recognition test. They note, however, that the evidence that recognition memory is less affected by depression is not compelling as there was no interaction between subject group and type of recall on a two-way ANOVA using standardised scores. This was calculated to see if the depressed and non-depressed groups differ less on the recognition test than on the other two tests. It was also possible that the recognition test was less discriminating and therefore less sensitive to a depression-related deficit. The results were also somewhat unexpected in that the depressed

subjects did not show benefit from cued recall. This may indicate that depression does not affect the ability to generate appropriate cues but such a conclusion remains tentative. The question of whether recognition memory and cued recall are less affected by depression than free recall is not, therefore, conclusively answered by this research.

Three types of recall (free recall, cued recall and recognition) were also tested in the study reported in Chapter Four of this thesis. In common with the study by Watts and Sharrock (1987) no interaction was found between type of recall and subject group, suggesting that the depressed and non-depressed subjects did not differ more on one type of test than on another. However, this must be regarded as a poor test of the hypothesis that recognition memory is less affected by depression because the recognition test differed from the others in testing non-verbal memory rather than memory for words. There was also some indication that the recognition test differed from the free recall and cued recall tests on other dimensions such as level of difficulty. It might be expected that a recognition memory task would need least effort for successful completion as the subject does not need to generate potential targets but instead has just to recognise them, and that cued recall would be intermediate between recognition and free recall as cues are provided to aid the search through memory. Indeed, it is because such tests are presumed to differ on this dimension that using matched task methodology is acknowledged to be important. However, there is some suggestion that the recognition test used in this study may have been particularly demanding and that the cued recall test actually demanded less effort than the recognition test, the reverse of what was expected. For instance the retarded depressed patients were impaired on all three tests but the less depressed neurotic depressed patients were not significantly impaired on the cued recall test. In addition the two groups of depressed patients differed significantly on the d' measure calculated from the recognition memory test and the more depressed retarded depressed group performed worst. This was the only measure on which the two groups differed significantly. The general pattern of results indicate that there was an interaction between the severity of depression and the level of difficulty of the task (Section 4.4.1) so these results suggest that the Picture Recognition test was more effort-demanding than expected. This means it is inappropriate to draw conclusions from these

results about the relative effects of depression on recognition, cued and free recall.

These results indicate the importance of matching tasks for difficulty because recognition and free recall tasks are likely to differ on this dimension and not always, as this study shows, in the expected direction. Recognition memory should demand less effort because the target is given and only has to be compared to concepts in memory until a match is found. However, a recognition memory test may require the same two-stage retrieval process as a free recall test if the distractor items are too similar to the target and therefore seem equally familiar to the subject (Bower and Glass, 1976; Glass and Holyoak, 1986). This would mean that the subject could not identify the target as 'seen before' on the basis of familiarity and would therefore need to generate possible candidate concepts which could then be recognised, just as in a free recall task. This means that it is possible to fail to recognise something which it is later possible to recall (see Ogilvie et al, 1980). It is not, therefore, always the case that recognition involves less effort than free recall and will always succeed if recall succeeds.

It cannot, therefore, be presumed that recognition demands less effort than recall and is therefore less affected by depression: it may actually demand a lot of effort for successful completion depending on the choice of, for instance, the distractor targets. In the Picture Recognition test used in this thesis the distractor targets were all black and white cartoons featuring the same cartoon characters as the target pictures (Section 2.2.1). It is possible that it was difficult to discriminate between the distractors and targets and that this was therefore a difficult effort-demanding test to complete. A similar criticism can be applied to the Past Public Events Multi-Choice questionnaires used in this thesis (Section 2.2.2). The distractor items were chosen because they were plausible answers to the questions and it may, therefore, have been difficult to decide between the distractors and targets on the basis of familiarity alone which would have necessitated the generation of possible answers to the questions rather than the simple recognition of the correct answer. This may help to explain why the depressed patients were significantly impaired on the multi-choice versions of the Past Public Events questionnaires and not on the free recall versions, despite

the prior presumption that the former would be easier and help the depressed subjects to overcome any effects of depression on retrieval (Section 4.3.4).

The difficulty in establishing the amount of effort required by recognition tests therefore makes it difficult to compare the results of different studies and to draw conclusions from the results of recognition and recall tests given to the same subjects. It highlights the importance of using matched tasks when investigating differential effects of depression on recognition and free recall as this ensures that the results are due to the dimension under investigation (the effects of depression on retrieval) rather than to the amount of effort required by the tests. As has recently been pointed out (Williams et al, 1988) this may mean that the tests differ on other dimensions, for instance length of test. However, it would make it easier to draw conclusions about the effects of depression on retrieval than the present situation where matched tasks have rarely been used and comparisons both between and within studies are extremely difficult.

The effects of depression on retrieval clearly warrant further research as do the reasons why retrieval may be affected in depression. For instance Ellis et al (1984) argued that the ease of retrieval would depend upon the effort exerted at encoding. According to this view retrieval failures would indicate an encoding rather than a retrieval problem. One way of differentiating the effects of depression on encoding and retrieval is to induce depressed mood after encoding. Ellis et al (1985) induced depressed mood in subjects after an incidental learning task and found that it led to a reduction in recall, although subjects showed the expected benefit from elaboration. This may, of course, indicate that the failure to show benefit from elaboration in other studies (eg Ellis et al, 1984) is caused by an encoding deficit. It is not, however, clear what the relationship between depressed mood and clinical depression is (Section 1.2.2) and it is not therefore apparent that results from studies using induced depressed mood are applicable to the depressed psychiatric in-patients who feature in most studies of memory in depression. Nevertheless, induced mood studies may provide a useful way forward as they make it possible to differentiate between effects of mood on encoding and retrieval.

Another way of investigating retrieval deficits in depression would be to use Past Public Events questionnaires to look at memory for events presumed to have been encoded before the onset of depression. However, as indicated in Section 2.2.2, these have limitations, including the difficulty of being certain that the event was originally learnt. It would also be difficult to equate the level of difficulty of these questionnaires with tests of new learning which would be important in establishing the existence of a retrieval deficit rather than an impairment due to the level of difficulty of the test. Results on such questionnaires would indicate whether retrieval is affected by depression but studies with tighter experimental control and more carefully defined hypotheses would be necessary to be sure that there was a genuine effect of depression on retrieval mechanisms rather than a general inability to sustain effort, resulting in impairment on any sufficiently complex task, regardless of the type of memory involved (Section 1.8.1). For instance it has been found that patients suffering from Korsakoff's syndrome do not show the usual release from proactive inhibtion when new categories are presented. This is taken as evidence that they are unable to generate their own cues to aid retrieval (Winocur, Kinsbourne and Moscovitch, 1981). Similar experiments designed to investigate specific aspects of retrieval would help increase understanding of the effects of depression on retrieval.

It would not be surprising if both the encoding and retrieval stages of information processing were affected in depression. According to the working memory model of memory (Baddeley and Hitch, 1974) both encoding and retrieval processes are likely to involve the operation of the central executive of working memory. The central executive is a somewhat vague and ill-determined concept (Baddeley, 1981) which is presumed to have limited capacity and to be involved in allocating attention to competing tasks and in directing the operation of other components of working memory. Eysenck (1979, 1982) has proposed that the available capacity of working memory is reduced in anxiety due to task-irrelevant cognitive activities such as worry and self-criticism. It is argued in Section 9.3 of this thesis that this is also likely to apply to depression and that the effects in depression are likely to be more pronounced as depressed subjects are unlikely to increase the amount of effort they exert on a task in order to overcome the effect of reduced processing capacity. If

this is so then depressed subjects with reduced processing capacity may be as likely to fail to use effective active retrieval stategies as they are to fail to process material sufficiently 'deeply' or elaboratively. It is therefore quite feasible that there is a generalised deficit in depression which affects each stage of memory involving the active processing of information. However, there have been so few investigations of memory in depression designed either to find differential effects of depression on different stages of processing or to test a particular hypothesis about memory in depression that it is difficult to draw such a conclusion: as deficits have been found on almost every aspect of memory tested in depression (Table 9.1) the evidence may point in the direction of a generalised deficit but at present it is too limited.

In conclusion, it is clear that many questions about memory in depression remain unanswered. For instance does depression result in a generalised deficit affecting any aspect of memory requiring sufficient effort for completion, or does it affect some aspects of information-processing more than others? Now that it has been established that there are memory deficits in depression, there is a need to turn away from studies such as those reported in this thesis which are predominately descriptive. The need is now for more studies of memory in depression which take insights from the current theoretical understanding of memory in non-depressed subjects, and which attempt to answer clearly defined questions about how information is processed in depression. The review of the existing literature has shown that most studies have not done this. For instance little use has been made of procedures such as matched task methodology which would enable conclusions to be reached about the differential effects of depression on one test compared to another. Such studies would shed light on the nature of the memory deficit in depression. Previous studies have, for example, concentrated on verbal memory and consequently little is known about whether there are differential effects of depression on verbal and non-verbal memory.

Several illuminating studies have used subjects in whom depressed mood has been induced. Such studies have advantages because they permit the separation of the effects of depression on, for instance, encoding and retrieval. However, the relationship between induced depressed mood and clinical depression is uncertain and therefore conclusions reached from

studies using induced mood methodology need to be tested on clinically depressed subjects before they can be assessed properly. As well as designing studies specifically to reduce the uncertainty about the nature of the memory defict in depression, future studies could usefully consider the distinction between endogenous and non-endogenous depression when selecting subjects, as it is possible that different or less extensive deficits may be found in the two groups. Finally, studies designed to investigate the nature of memory impairment in depression would do well to consider both the similarities between memory performance and the hypothesised reasons for impairment in depression, anxiety and schizophrenia. Comparisons between these groups could do much to further understanding of memory deficits in depression (Sections 9.1 and 9.2).

9.6 GENERAL CONCLUSIONS

This thesis has been concerned with three aspects of memory in depression: the performance of clinically depressed people on laboratory memory tests; their reports of memory failures in everyday life and the relationship of these reports to performance on laboratory memory tests; and their memory in an important everyday situation, the general practice consultation. It is concluded that experimental studies of memory, metamemory questionnaires and investigations of memory in everyday situations all have a contribution to make to the study of memory in depression and that the fullest understanding will come from using all three approaches.

The memory on laboratory memory tests of two groups of clinically depressed psychiatric patients (retarded depressed and neurotic depressed) was compared to that of anxious patients and controls, who were neither anxious or depressed (Chapter Four). The depressed patients had impaired ability to learn new material, to recall material after a delay and to remember past public events; they did not forget more information between immediate and delayed recall than the controls, and did not have a more conservative response bias. These impairments were not due to the psychotropic medication the patients were receiving nor to the after-effects of E.C.T. (Chapter Five). The more severely retarded depressed patients were more impaired than the less depressed neurotic depressed group, who were impaired only on what were presumed to be the

more difficult tests. The scores of the clinically anxious patients were intermediate between those of the depressed and control subjects, but not significantly different from either; this was probably due to the small number of subjects in the anxious group. The four groups of subjects were combined in order to investigate the relative effects of depression and anxiety on memory. Self-rated depression was related to performance on the easier tests, whilst self-rated anxiety had no effect once the effect of depression was partialled out. In contrast, it was not possible to distinguish between the effects of depression and anxiety on the more difficult tests and it was therefore concluded that something common to both was affecting performance on these tests. The retarded depressed subjects reported more cognitive failures in the previous six months than the other subjects; both groups of depressed subjects and the anxious subjects complained of more deterioration in memory than the controls. There were moderate correlations between the estimates of cognitve failures and changes in memory on the one hand, and performance on the laboratory memory tests on the other. Anxiety was significantly related to the self-assessments of memory performance whilst depression was not.

The memory of clinically depressed general practice patients for information given to them by their general practitioners was compared to that of patients who were not depressed. The depressed patients remembered as much information as the non-depressed patients. The consultations of the two groups did not differ in such a way as to make those of the depressed patients easier to recall. It was therefore concluded that depressed patients did not have impaired memories in this important everyday situation.

These results are consistent with the hypothesis that the degree of memory impairment shown in depression will depend both on the severity of depression and the level of difficulty of the task (eg Ellis, 1985; Section 9.5). They have been discussed in the light of the suggestion by, amongst others, Johnson and Magaro (1987) that memory impairments may not be specific to depression but instead may be related to the overall level of psychopathology. Johnson and Magaro (1987) suggest that memory impairment in psychiatric disorders is related to a lack of effort and to the disruptive effect of interference from worry, intrusive and illogical thoughts; the reduction in effort and cognitive interference both increase

as the overall severity of the psychiatric illness increases. Eysenck (1979, 1982) proposed a model of cognitive function in anxiety in which he stated that anxious subjects have reduced working memory capacity due to capacity being taken up by worry and self-preoccupation. He argued that this did not always lead to cognitive impairment because anxious subjects will increase the effort they put into the task; this will have the effect of increasing processing capacity (Dornic, 1977; Eysenck, 1979, 1982). This is obviously similar to the suggestion by Johnson and Magaro (1987) with the advantage that it presents a model of the way in which effort and cognitive interference are supposed to interact to produce performance decrements. This model is therefore extended to memory in depression and other psychiatric disorders. It is hypothesised that worry, intrusive and illogical thoughts pre-empt some of the capacity of working memory and therefore, as Eysenck suggests, always reduce the effectiveness of performance. However, whether or not the quality or efficiency of performance is affected will depend both on the level of motivation of the subjects (which determines whether they increase the effort they exert on the task) and on the level of difficulty of the task (which affects the processing capacity required).

This model has the advantage of bringing together research on the effects of anxiety on memory with that on the effects of depression, and it may also explain some of the effects of other psychiatric conditions, such as mania and schizophrenia, on memory. It is consistent with the evidence that depressed people are most impaired on tasks requiring effort for successful completion (Section 1.8.1), and on the more sparse evidence that cognitive interference plays a role in producing memory impairments (Section 1.8.4). However, it is not the only feasible interpretation of the available information on memory in depression.

An alternative model was devised by Williams and Teasdale (1982) to account for results in the learned helplessness literature. This presumes that the amount of effort exerted on a task (and the resulting performance) depends upon the perceived difficulty of the task which varies according to the difficulty of the task, the subjects perception of their ability and their experience of success and failure (Kukla, 1972). Subjects will, however, only increase the amount of effort exerted in response to increased perceived task-difficulty if the positive incentives

for exerting effort (importance and expectancy of success) outweigh the negative incentives of the cost of effort and the amount of effort required. Like the model proposed by Eysenck (1982), this model can account for many of the previous findings in the literature on memory in depression. It is argued that it can also be applied to memory in anxiety and possibly to memory in other psychiatric disorders. Further investigation of this model may, therefore, be fruitful.

In conclusion, further investigation of memory in depression is needed which, as argued in Section 9.5, moves beyond the descriptive and seeks to investigate specific hypotheses about the nature and causes of memory impairment in depression. Both the model proposed by Eysenck (1982) and that proposed by Williams and Teasdale (1982) seem to have the potential to help elucidate the causes of impairment in depression and therefore seem worthy of further investigation.

PAST EVENTS QUESTIONNAIRE

FORM A

1. What was the name of the Welsh village where a slag tip slipped and engulfed a school killing 146 children and 23 adults in October 1966? .....

2. Which Eastern European country was invaded by the Russians in 1968 after the appointment of Dubcek as Party Secretary had led to a series of reforms? .....

3. Who followed Harold Wilson as Prime Minister after the General Election in 1970? .....

4. 11 athletes were killed by terrorists during the Olympic Games in 1972. Which national team did they belong to? .....

5. Which American politician resigned in 1974 following threats of impeachment? .....

6. Who became leader of the Labour party and Prime Minister when Harold Wilson resigned in 1976? .....

7. Which Member of Parliament was assassinated by a bomb which exploded as he drove out of the House of Commons in 1979? .....

8. Which famous popstar was shot dead in New York in 1980? .....

9. Which British M.P. was acquitted at the Old Bailey in 1979 of plotting to Kill Mr. Norman Scott? .....

10. In 1976 Israeli commandos carried out a raid to release Jewish hostages from a hijacked plane. Where was the plane at the time? .....

11. What was the name of the U.S. newspaper heiress who was kidnapped by the Symbionese Liberation Army in 1974? .....

12. Which union went on strike in 1972 causing the proclamation of a State of Emergency and nation-wide power cuts? .....

13. In which Central American country was the World Cup held in 1970? .....

14. Who did Mehmet Ali Agca try to assassinate in St. Peter's Square, Rome in 1981? .....

15. Which American civil-rights campaigner was assassinated by James Earl Ray in 1968? .....

16. Who was convicted with Ian Brady of committing the Moors murders in 1966? .....

17. What was introduced in Britain on February 15th 1971? .....

18. Who did Margeret Thatcher succeed as leader of the Conservative Party in 1975? .....

19. Who was stripped of his knighthood in 1979 following his admission that he had been spying for Russia? .....

20. Who succeeded Lyndon Johnson as President of the U.S. in 1968? .....

21. Which country were US troops finally withdrawn from in 1973? .....

22. Which American President visited Britain in May 1977? .....

23. What was the name of the President of an Arab country who was assassinated by his own soldiers in 1981? .....

24. What was the name of the Liberian tanker that went aground on the Seven Stones rock near Lands End in 1967 causing an oilslick of nearly 100 square miles? .....

25. What did Mr. Faulkner authorise in Northern Ireland under the Special Powers Act in 1971? .....

26. A passanger train was hijacked by South Moluccan extremists in 1975. Which country did this happen in? .....

27. Which country elected its first majority-rule Parliament in 1979 and got its first black Prime Minister? .....

28. Which famous race horse created a new record by winning the Grand National for the 3rd time in 1977? .....

29. What was the name of the new political party formed in Britain in 1981? .....

30. Which British golfer won the Golf Open Championship in 1969 after 17 years of foreign domination? .....

31. Which ethnic group was forced to leave Uganda in 1972? .....

32. Where was Prince Charles married on 29th July 1981? .....

33. Which country prevented Britain joining the Common Market in 1967? .....

34. Which Asian country was India at war with in December 1971? .....

35. Whose death led to the investiture of Prince Juan Carlos as King of Spain in 1975? .....

36. President Sadat signed the peace treaty between Israel and Egypt for the Egyptians in Washington in 1979. Who signed it for the Israelis? .....

37. Name the American Senator who was driving the car in which Mary-Jo Kopechne drowned after a party at Chappaquiddick Island in 1969? .....

38. Which country did Egyptian and Syrian forces attack in October 1973? .....

39. Who did England beat in the final of the World Cup in 1966? .....

40. Which British tennis player won the ladies singles at Wimbledon in 1977? .....

PAST EVENTS QUESTIONNAIRE

FORM B

1. What did Britain join on the 1st January 1973? .....

2. Which famous pop group disbanded in 1970? .....

3. In 1976 a Member of Parliament was imprisoned after having been found guilty of charges of false pretences arising from his faked disappearance from Florida in 1974? What was his name? .....

4. Who was blown up by an IRA bomb on board his boat in Mullaghmore Harbour in the Irish Republic in 1979? .....

5. Which Eastern European country was invaded by the Russians in 1968 after the appointment of Dubcek as Party Secretary had led to a series of reforms? .....

6. Which member of the Royal Family died in France in 1972 and was buried at Frogmore in Windsor Park? .....

7. Who resigned as leader of the Liberals in 1976? .....

8. What is the name of Poland's Independent Trade Union which was recognised by the Polish authorities in 1980? .....

9. A member of a well-known American political family was assassinated by Sirhan Bishara Sirhan in 1968. What was his name? .....

10. Where were Princess Anne and Captain Mark Phillips married in November 1973? .....

11. Which member of the Royal Family was separated from her husband in 1976? .....

12. In which industry were more than 50,000 redundancies announced in 1980 which left whole towns almost jobless? .....

13. Where was the World Cup competition held in 1966? .....

14. Which British boxer lost the British, European and Commonwealth heavy-weight titles to Joe Bugner in 1971? .....

15. Which embassy in Iran was overrun in 1979 by students demanding that the Shah should be returned for punishment? .....

16. Whose death led to the investiture of Prince Juan Carlos as King of Spain in 1975? .....

17. Which American politician survived an assassination attempt in Washington during March 1981? .....

18. What stretch of water was closed as a result of the 'Six Day' Arab-Israeli war in 1967? .....

19. Which young Australian player won the ladies final at Wimbledon in 1971? .....

20. What country had Haile Selassie ruled for 44 years before he was deposed in 1974? .....

21. Who returned to Iran in 1979 after being in exile for 14 years? .....

22. Which British iceskater won the European, Olympic and World figureskating titles in 1976? .....

23. Who was the first man to walk on the moon? .....

24. Which American politician visited China in 1972 bringing to an end 20 years of hostility between the U.S.A. and China? .....

25. Who did Mr. Foot succeed as leader of the Labour Party in 1980? .....

26. What epidemic led to over 300,000 animals being slaughtered in 1967? .....

27. Which famous British company went bankrupt in 1971 and was later nationalised? .....

28. Which member of the Royal Family was attacked in her car on the Mall by an armed man in 1974? .....

29. Which woman tennis player beat the previous record of 19 Wimbledon titles in 1979 when she won the ladies doubles with Martina Navratilova? .....

30. Which country was King Constantine forced to leave in 1967 after an unsuccessful attempt to overthrow the military junta? .....

31. Where did the investiture of Prince Charles as Prince of Wales take place in 1969? .....

32. Which famous woman horserider won the individual prize in the 3-day event at Burghley Horse Trials and then became the 1971 'Sportswoman of the Year'? .....

33. Who was the Prime Minister responsible for introducing the three-day week in 1973? .....

34. What action was taken in 1975 to see if Britain would stay in the European Community? .....

35. Which famous rock and roll star died in 1977? .....

36. Whose 80th birthday was marked by a service in St. Pauls Cathedral in 1980? .....

37. Which aeroplane made its maiden flight in 1969? .....

38. Who set out to sail round the world in 1966 and was knighted at Greenwich on his return in 1967? .....

39. Why were a string of bonfires lit all over Britain on the 6th June 1977? .....

40. Which European country experienced a severe earthquake in 1980 which resulted in more than 3,000 deaths and left 200,000 people homeless? .....

PAST EVENTS QUESTIONNAIRE

FORM A (Multiple choice)

1. What was the name of the Welsh village where a slag tip slipped and engulfed a school killing 146 children and 23 adults in October 1966?

   a) Aberfan
   b) Tregaron
   c) Caerphilly
   d) Cwmbran

2. Which Eastern European country was invaded by the Russians in 1968 after the appointment of Dubcek as Party Secretary had led to a series of reforms?

   a) Rumania
   b) Bulgaria
   c) Czechoslavakia
   d) Yugoslavia

3. Who followed Harold Wilson as Prime Minister after the General Election in 1970?

   a) Edward Heath
   b) Michael Foot
   c) Margeret Thatcher
   d) Alec Douglas-Howe

4. 11 athletes were killed by terrorists during the Olympic Games in 1972. Which national team did they belong to?

   a) Rhodesian
   b) Dutch
   c) Hungarian
   d) Israeli

5. Which American politician resigned in 1974 following threats of impeachment?

   a) Henry Kissinger
   b) Edmund Muskie
   c) President Nixon
   d) President Ford

6. Who became leader of the Labour party and Prime Minister when Harold Wilson resigned in 1976?

   a) James Callaghan
   b) Margeret Thatcher
   c) Michael Foot
   d) Dennis Healey

7. Which Member of Parliament was assassinated by a bomb which exploded as he drove out of the House of Commons in 1979?

   a) Brian Faulkner
   b) Airey Neave
   c) Ian Macleod
   d) Antony Crossland

8. Which famous popstar was shot dead in New York in 1980?

   a) Marc Bolan
   b) Sid Vicious
   c) John Lennon
   d) Keith Moon

9. Which British M.P. was acquitted at the Old Bailey in 1979 of plotting to kill Mr. Norman Scott?

a) Patrick Jenkin
b) Tom Litterick
c) Reg Prentice
d) Jeremy Thorpe

10. In 1976 Israeli commandos carried out a raid to release Jewish hostages from a hijacked plane. Where was the plane at the time?

a) New York
b) Entebbe
c) London
d) Dar-es-Salaam

11. What was the name of the U.S. newspaper heiress who was kidnapped by the Symbionese Liberation Army in 1974?

a) Patty Hearst
b) Jacqueline Hill
c) Jeanette Tate
d) Lesley Whittle

12. Which union went on stike in 1972 causing the proclamation of a State of Emergency and nation-wide power cuts?

a) The Transport Union
b) National Union of Mineworkers
c) National Union of Railwaymen
d) National Union of Teachers

13. In which Central American country was the World Cup held in 1970?

a) Mexico
b) Panama
c) Nicaragua
d) Honduras

14. Who did Mehmet Ali Agca try to assassinate in St. Peter's Square, Rome in 1981?

a) Aldo Moro
b) Signor Bertini
c) The Pope
d) Cardinal Hulme

15. Which American civil-rights campaigner was assassinated by James Earl Ray in 1968?

a) Andrew Young
b) Martin Luther-King
c) Ralph Nader
d) Steve Biko

16. Who was convicted with Ian Brady of committing the Moors murders in 1966?

a) Dr. Rose Dugdale
b) Ruth Ellis
c) Judith Ward
d) Myra Hindley

17. What was introduced in Britain on February 15th 1971?

a) Decimal currency
b) Comprehensive schools
c) V.A.T
d) Credit cards

18. Who did Margeret Thatcher succeed as leader of the Conservative Party in 1975?

a) William Whitelaw
b) Alec Douglas-Home
c) Harold Macmillan
d) Edward Heath

19. Who was stripped of his knighthood in 1979 following his admission that he had been spying for Russia?

a) Sir Colin Buchanan
b) Sir Peter Medawar
c) Sir Anthony Blunt
d) Sir Richard Marsh

20. Who succeeded Lyndon Johnson as President of the U.S. in 1968?

a) Richard Nixon
b) John Kennedy
c) Jimmy Carter
d) Ronald Reagan

21. Which country were US troops finally withdrawn from in 1973?

a) West Germany
b) Thailand
c) Cambodia
d) South Vietnam

22. Which American President visited Britain in May 1977?

a) President Nixon
b) President Ford
c) President Carter
d) President Reagan

23. What was the name of the President of an Arab country who was assassinated by his own soldiers in 1981?

a) President Bani-Sadr
b) General Sadat
c) Idi Amin
d) Bishop Muzorewa

24. What was the name of the Liberian tanker that went aground on the Seven Stones rock near Lands End in 1967 causing an oilslick of nearly 100 square miles?

a) Andros Patria
b) Tattershall Castle
c) Torrey Canyon
d) Ben Asdale

25. What did Mr. Faulkner authorise in Northern Ireland under the Special Powers Act in 1971?

a) Use of rubber bullets
b) Carrying firearms
c) Internment of suspects
d) Army's continued presence in the region

26. A passanger train was hijacked by South Moluccan extremists in 1975. Which country did this happen in?

a) Italy
b) Portugal
c) West Germany
d) Holland

27. Which country elected its first majority-rule Parliament in 1979 and got its first black Prime Minister?

a) Jamaica
b) South Africa
c) Rhodesia
d) Mozambique

28. Which famous race horse created a new record by winning the Grand National for the 3rd time in 1977?

a) Red Rum
b) Nijinsky
c) Brigadier General
d) Mill House

29. What was the name of the new political party formed in Britain in 1981?

a) Ecology Party
b) Social Democratic Party
c) National Front
d) Marxist-Leninist Party

30. Which British golfer won the Golf Open Championship in 1969 after 17 years of foreign domination?

a) Tony Jacklin
b) Peter Oosterhuis
c) Lee Trevino
d) Jack Nicklaus

31. Which ethnic group was forced to leave Uganda in 1972?

a) Europeans
b) Japanese
c) Chinese
d) Asians

32. Where was Prince Charles married on 29th July 1981?

a) Westminster Abbey
b) St. Paul's Cathedral
c) St. George's Chapel, Windsor
d) Westminster Cathedral

33. Which country prevented Britain joining the Common Market in 1967?

a) Italy
b) Belgium
c) West Germany
d) France

34. Which Asian country was India at war with in December 1971?

a) Afghanistan
b) Iran
c) Pakistan
d) Sri Lanka

35. Whose death led to the investiture of Prince Juan Carlos as King of Spain in 1975?

a) President Tiko
b) General Franco
c) President Makarios
d) General de Gaulle

36. President Sadat signed the peace treaty between Israel and Egypt for the Egyptians in Washington in 1979. Who signed it for the Israelis?

a) General Rabin
b) Mr. Sharon
c) General Dayan
d) Mr. Begin

37. Name the American Senator who was driving the car in which Mary-Jo Kopechne drowned after a party at Chappaquiddick Island in 1969?

a) Edward Kennedy
b) Vance Hartke
c) Eugene McCarthy
d) Edmund Muskie

38. Which country did Egyptian and Syrian forces attack in October 1973?

a) Iran
b) Iraq
c) Lebanon
d) Israel

39. Who did England beat in the final of the World Cup in 1966?

a) Italy
b) Brazil
c) Argentina
d) West Germany

40. Which British tennis player won the ladies singles at Wimbledon in 1977?

a) Betty Stove
b) Virginia Wade
c) Sue Barker
d) Rosemary Casals

PAST EVENTS QUESTIONNAIRE

FORM B (Multiple Choice)

1. What did Britain join on the 1st January 1973?

   a) European Community
   b) E.F.T.A.
   c) The Commonwealth
   d) N.A.T.O.

2. Which famous pop group disbanded in 1970?

   a) The Monkeys
   b) The Who
   c) The Beatles
   d) The Rolling Stones

3. In 1976 a Member of Parliament was imprisoned after having been found guilty of charges of false pretences arising from his faked disappearance from Florida in 1974? What was his name?

   a) Nicholas Fairbairn
   b) John Stonehouse
   c) Jack Ashley
   d) John Hannam

4. Who was blown up by an IRA bomb on board his boat in Mullaghmore Harbour in the Irish Republic in 1979?

   a) Earl Mountbatten
   b) Viscount Montgomery
   c) Rev. Robert Bradford
   d) Mr Christopher Ewart-Biggs

5. Which Eastern European country was invaded by the Russians in 1968 after the appointment of Dubcek as Party Secretary had led to a series of reforms?

   a) Rumania
   b) Bulgaria
   c) Yugoslavia
   d) Czechoslavakia

6. Which member of the Royal Family died in France in 1972 and was buried at Frogmore in Windsor Park?

   a) Duke of Gloucester
   b) Duke of Windsor
   c) Princess Alice of Athlone
   d) Princess Maria, Duchess of Kent

7. Who resigned as leader of the Liberals in 1976?

   a) Cyril Smith
   b) David Steel
   c) Jeremy Thorpe
   d) Clement Freud

8. What is the name of Poland's Independent Trade Union which was recognised by the Polish authorities in 1980?

   a) The United Workers Trade Union
   b) Solidarity
   c) Unity
   d) Polish Workers' Union

9. A member of a well-known American political family was assassinated by Sirhan Bishara Sirhan in 1968. What was his name?

   a) J. Edgar Hoover
   b) Aristotle Onassis
   c) Robert Kennedy
   d) Nelson Rockefeller

10. Where were Princess Anne and Captain Mark Phillips married in November 1973?

a) Westminster Abbey
b) St Paul's Cathedral
c) Sandringham
d) Chapel of St. George, Windsor

11. Which member of the Royal Family was separated from her husband in 1976?

a) Princess Alexandra
b) Duchess of Kent
c) Princess Anne
d) Princess Margaret

12. In which industry were more than 50,000 redundancies announced in 1980 which left whole towns almost jobless?

a) Coalmining
b) The Car Industry
c) Ship-building
d) Steel

13. Where was the World Cup competition held in 1966?

a) Britain
b) Uruguay
c) Spain
d) Argentina

14. Which British boxer lost the British, European and Commonwealth heavy-weight titles to Joe Bugner in 1971?

a) George Foreman
b) Cassius Clay
c) Henry Cooper
d) Joe Frazier

15. Which embassy in Iran was overrun in 1979 by students demanding that the Shah should be returned for punishment?

a) Canadian
b) Egyptian
c) French
d) American

16. Whose death led to the investiture of Prince Juan Carlos as King of Spain in 1975?

a) President Tito
b) General Franco
c) General de Gauille
d) President Makarios

17. Which American politician survived an assassination attempt in Washington during March 1981?

a) President Reagan
b) General Haig
c) Mr Harry Mondale
d) Henry Kissinger

18. What stretch of water was closed as a result of the 'Six Day' Arab-Israeli war in 1967?

a) Gulf of Aqaba
b) Red Sea
c) Panama Canal
d) Suez Canal

19. Which young Australian player won the ladies final at Wimbledon in 1971?

a) Evonne Goolagong
b) Judy Daiton
c) Margaret Court
d) Betty Stove

20. What country had Haile Selassie ruled for 44 years before he was deposed in 1974?

a) Thailand
b) Ethiopia
c) Tanzania
d) Iran

21. Who returned to Iran in 1979 after being in exile for 14 years?

a) Ayatollah Huhammad Beheshti
b) The Shah
c) President A. Bani-Sadr
d) Ayatollah Khomeni

22. Which British iceskater won the European, Olympic and World figure-skating titles in 1976?

a) John Curry
b) Warren Maxwell
c) Glyn Watts
d) Jan Hoffman

23. Who was the first man to walk on the moon?

a) Colonel Komarov
b) John Swigart
c) Neil Armstrong
d) James Lovell

24. Which American politician visited China in 1972 bringing to an end 20 years of hostility between the U.S.A. and China?

a) Richard Nixon
b) Jimmy Carter
c) George Wallace
d) Henry Kissinger

25. Who did Mr. Foot succeed as leader of the Labour Party in 1980?

a) Hugh Gaitskell
b) Dennis Healey
c) James Callaghan
d) Harold Wilson

26. What epidemic led to over 300,000 animals being slaughtered in 1967?

a) Swine venicular disease
b) Foot and mouth disease
c) Rabies
d) Tuberculosis

27. Which famous British company went bankrupt in 1971 and was later nationalised?

a) British Steel
b) I.C.I
c) Rolls Royce
d) British Leyland

28. Which member of the Royal Family was attacked in her car on the Mall by an armed man in 1974?

a) Princess Anne
b) Duchess of Kent
c) Princess Margaret
d) Queen Mother

29. Which woman tennis player beat the previous record of 19 Wimbledon titles in 1979 when she won the ladies doubles with Martina Navratilova?

a) Virginia Wade
b) Billy-Jean King
c) Margaret Court
d) Chris Evert

30. Which country was King Constantine forced to leave in 1967 after an unsuccessful attempt to overthrow the military junta?

a) Greece
b) Spain
c) Portugal
d) Egypt

31. Where did the investiture of Prince Charles as Prince of Wales take place in 1969?

a) St David's Cathedral
b) Cardiff Castle
c) Caernarvon Castle
d) Harlich Castle

32. Which famous woman horserider won the individual prize in the 3-day event at Burghley Horse Trials and then became the 1971 'Sportswoman of the Year'?

a) Caroline Bradley
b) Princess Anne
c) Lucinda Prior-Palmer
d) Mary Gordon-Watson

33. Who was the Prime Minister responsible for introducing the three-day week in 1973?

a) Edward Heath
b) Sir Alec Douglas-Home
c) Harold Wilson
d) James Callaghan

34. What action was taken in 1975 to see if Britain would stay in the European Community?

a) Debate in parliament
b) National referendum
c) General Election
d) Conference of European Heads of State

35. Which famous rock and roll star died in 1977?

a) Bill Haley
b) Carl Perkins
c) Eddie Cochrane
d) Elvis Presley

36. Whose 80th birthday was marked by a service in St. Pauls Cathedral in 1980?

a) The Queen Mother
b) Harold Macmillan
c) Princess Alice of Athlone
d) Duchess of Windsor

37. Which aeroplane made its maiden flight in 1969?

a) Boeing 747
b) B.E.A Vanguard
c) DC10
d) Concorde

38. Who set out to sail round the world in 1966 and was knighted at Greenwich on his return in 1967?

a) Robin Knox-Johnston
b) Francis Chichester
c) Clay Blyth
d) John Fairfax

39. Why were a string of bonfires lit all over Britain on the 6th June 1977?

a) Queen Mother's 80th birthday
b) Queen's Silver Jubilee
c) Anniversary of the sailing of the Armada
d) Queen's first grandchild

40. Which European country experienced a severe earth-quake in 1980 which resulted in more than 3,000 deaths and left 200,000 people homeless?

a) France
b) Italy
c) Spain
d) Greece

COGNITIVE FAILURE QUESTIONNAIRE

NAME..... AGE ..... SEX.....

The following questions are about minor mistakes which everyone makes from time to time, but some of which happen more often than others. We want to know how often these things have happened to you in the past six months. Please circle the appropriate number.

		Very often	Quite often	Occasionally	Very rarely	Never
1.	Do you read something and find you haven't been thinking about it and must read it again?	4	3	2	1	0
2.	Do you find you forget why you went from one part of the house to the other?	4	3	2	1	0
3.	Do you fail to notice signposts on the road?	4	3	2	1	0
4.	Do you find you confuse right and left when giving directions?	4	3	2	1	0
5.	Do you bump into people?	4	3	2	1	0
6.	Do you find you forget whether you've turned off a light or fire or locked a door?	4	3	2	1	0
7.	Do you fail to listen to people's names when you are meeting them?	4	3	2	1	0
8.	Do you say something and realise afterwards that it might be taken as insulting?	4	3	2	1	0
9.	Do you fail to hear people speaking to you when you are doing something else?	4	3	2	1	0
10.	Do you lose your temper and regret it?	4	3	2	1	0
11.	Do you leave important letters unanswered for days?	4	3	2	1	0
12.	Do you find you forget which way to turn on a road you know well but rarely use?	4	3	2	1	0

13. Do you fail to see what you want in a supermarket (although it's there)?	4	3	2	1	0
14. Do you find yourself suddenly wondering whether you've used a word correctly?	4	3	2	1	0
15. Do you have trouble making up your mind?	4	3	2	1	0
16. Do you find you forget appointments?	4	3	2	1	0
17. Do you forget where you put something like a newspaper or a book?	4	3	2	1	0
18. Do you find you accidently throw away the thing you want and keep what you meant to throw away - as in the example of throwing away the matchbox and putting the used match in your pocket?	4	3	2	1	0
19. Do you daydream when you ought to be listening to something?	4	3	2	1	0
20. Do you find you forget people's names?	4	3	2	1	0
21. Do you start doing one thing at home and get distracted into doing something else (unintentionally)	4	3	2	1	0
22. Do you find you can't quite remember something although it's 'on the tip of your tongue?	4	3	2	1	0
23. Do you find you forget what you came into the shops to buy?	4	3	2	1	0
24. Do you drop things?	4	3	2	1	0
25. Do you find you can't think of anything to say?	4	3	2	1	0

MEMORY QUESTIONNAIRE

This questionniare contains a list of questions about minor slips of memory which happen to us all at some time in our everyday lives.

We would like you to think about each question, and consider whether that particular sort of slip happens to you more or less often now than it did before you became ill.

Please choose the number from the following scale which best indicates your choice.

1	2	3	4	5
Much less often	Less often	The same	More often	Much more often

Put the number you have chosen on the line provided opposite each question.

NAME..... AGE.....

Use the 1-5 scale. Put the appropriate number on the line by each question.

1. Forgetting where you have put something. Losing things around the house. -----

2. Failing to recognise places that you are told you have often been to before. -----

3. Finding a television story difficult to follow. -----

4. Unable to cope with a change in your daily routine, such as a change in the time something happens. Following your old routine by mistake. -----

5. Having to go back to check whether you have done something that you meant to do. -----

6. When thinking of the past, forgetting when something happened. For example, forgetting whether something happened yesterday or last week. -----

7. Completely forgetting to take things with you, or leaving things behind and having to go back and fetch them. -----

8. Forgetting that you were told something yesterday or a few days ago, and maybe having to be reminded about it. -----

9. Starting to read something (a book or an article in a newspaper or a magazine) without realising you have already read it before. -----

10. Letting yourself ramble on to speak about unimportant or irrelevant things. -----

11. Failing to recognise, by sight, close relatives or friends that you meet frequently. -----

12. Unable to pick up a new skill. For example, having difficulty in learning a new game or in working some new gadget that you have practised once or twice. -----

13. Finding that a word is 'on the tip of your tongue'. You know what it is but cannot quite find it. -----

14. Completely forgetting to do things you said you would do, and things you planned to do. -----

15. Forgetting important details of what you did or what happened the day before. -----

16. When talking to someone, forgetting what you have just said. Maybe saying, 'What was I talking about?'. -----

17. When reading a newspaper or magazine, being unable to follow the thread of a story; losing track of what it is about. -----

18. Forgetting to tell somebody something important. Perhaps forgetting to pass on a message or remind someone of something. -----

19. Forgetting important details about yourself, e.g. your birthdate or where you live. -----

20. Getting the details of what someone has told you mixed up and confused. -----

21. Telling someone a story or joke that you have told them once already. -----

22. Forgetting details of things you do regularly, whether at home or at work. For example, forgetting details of what to do, or forgetting at what time to do it. -----

23. Finding that the faces of famous people seen on television or in photographs look unfamiliar. -----

24. Forgetting where things are normally kept or looking for them in the wrong place. -----

25. (a) Getting lost or turning in the wrong direction on a journey or a walk that you have OFTEN been on before. -----

    (b) Getting lost or turning in the wrong direction on a journey or a walk that you have ONLY BEEN ON ONCE OR TWICE before. -----

26. Doing some routine thing twice by mistake. For example, putting two lots of tea in the teapot, or going to brush/comb your hair when you have just done so. -----

27. Repeating to someone what you have just told them or asking them the same question twice. ----

I.D.A SCALE

NAME: **DATE:**

INSTRUCTIONS FOR USE

First write your name and the date in the space above. This form has been designed so that you can show how you have been feeling in the past few days.

Read each item in turn, and then UNDERLINE the response which best shows how you are feeling or have been feeling in the past few days.

I feel cheerful:

Yes, definitely
Yes, sometimes
No, not much
No, not at all

I can sit down and relax quite easily:

Yes, definitely
Yes, sometimes
No, not much
No, not at all

My appetite is:

Very poor
Fairly poor
Quite good
Very good

I lose my temper and shout or snap at others:

Yes, definitely
Yes, sometimes
No, not much
No, not at all

I can laugh and feel amused:

Yes, definitely
Yes, sometimes
No, not much
No, not at all

I feel I might lose control and hit or hurt someone:

Sometimes
Occasionally
Rarely
Never

I have an uncomfortable feeling like butterflies in the stomach:

Yes, definitely
Yes, sometimes
Not very often
Not at all

The thought of hurting myself occurs to me:

Sometimes
Not very often
Hardly ever
Not at all

I'm awake before I need to get up:

For 2 hours or more
For about 1 hour
For less than an hour
Not at all, I sleep until it is time to get up

I feel tense or 'wound up':

Yes, definitely
Yes, sometimes
No, not much
No, not at all

I feel like harming myself:

Yes, definitely
Yes, sometimes
No, not much
No, not at all

I have kept up my old intersts:

Yes, most of them
Yes, some of them
No, not many of them
No, none of them

I am patient with other people:

All the time
Most of the time
Some of the time
Hardly ever

I get scared or panicky for no very good reason:

Yes, definitely
Yes, sometimes
No, not much
No, not at all

I get angry with myself or call myself names:

Yes, definitely
Yes, sometimes
No, not much
No, not at all

People upset me so that I feel like slamming doors or banging about:

Yes, often
Yes, sometimes
Only occasionally
Not at all

I can go out on my own without feeling anxious:

Yes, always
Yes, sometimes
No, not often
No, I never can

Lately I have been getting annoyed with myself:

Very much so
Rather a lot
Not much
Not at all

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