The Role of the Frontal Lobes in Human Memory

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Thesis submitted for the degree of Doctor of Philosophy
in Neuropsychology at the Institute of Neurology,
This thesis is an examination of the memory problems that are considered pathological of frontal lobe dysfunction in humans. It is divided into three main sections. The first section concentrates on the findings from the experimental literature, and includes the development of two measures sensitive to frontal lobe involvement. These tests were validated on a group of 152 patients with localised cerebral lesions, and were used in order to discover the role of executive functions in patients' performance on traditional neuropsychological memory tests.

The second section puts forward the notion that executive functions and prospective memory are intimately linked, and describes the investigation of three single case studies who showed organisational problems in everyday life. It is proposed that one of the core problems shown by such patients is a deficit in prospective memory functions.

The last section presents the development of a model of the role of the frontal lobes in human memory with particular reference to confabulation. The model is based upon empirical evidence from a study of how normal subjects recall autobiographical events.
ACKNOWLEDGEMENTS

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This thesis is an investigation of the cluster of memory symptoms that are presently held to follow damage to the frontal lobes in humans. Recently the cluster of memory characteristics shown by these patients has been referred to as "frontal amnesia" (eg. Shallice, 1988). The thesis attempts to answer the two questions of whether frontal amnesia exists, and if so, is the area of the brain which subserves the processes damaged in such a syndrome indeed located in the frontal lobes. As we shall see, this requires consideration of the inter-relationship between executive and memory functions.

Research into the role of the frontal lobes in memory has been approached in two ways. Firstly there are group and single case studies of patients with known frontal lobe pathology, with empirical investigation of these patients' neuropsychological characteristics. Secondly there have been group and single case studies of the memory phenomena that are often considered to be secondary to frontal lobe pathology (eg. confabulation). There need not necessarily be overlap between these two approaches of course, but in practice this is rarely the case. This thesis will use both approaches in considering frontal memory phenomena. This chapter will therefore consider the relative merits of both the approaches and the data gleaned from them, in an attempt to justify such a broad methodological approach. This will be performed by first considering how the cluster of memory phenomena that have been considered to follow frontal lobe damage differ from those considered central to "classical amnesia". The strength of the evidence for each of these phenomena will then be considered in turn. This examination raises a number of methodological issues which are dealt with in the next section. The last section traces the use of this empirical evidence in current theories of frontal lobe amnesia. This is designed to reflect broadly the organisation of the thesis as a whole.
1.1 Introducing frontal amnesia

"Frontal amnesia" is a term which has only recently begun to appear in the literature (eg. Baddeley and Wilson, 1986; Shallice, 1988), although discussion of the memory problems that can follow anterior cortical damage has been appearing in the literature for many years (eg. Bolton, 1903). In part, the reluctance of researchers to use the term "frontal amnesia" to describe these memory problems reflects the present uncertainty about the syndrome: there is a fine but important difference between saying that patients may suffer from memory problems secondary to frontal dysfunction, and saying that frontal lobe lesions may cause "frontal amnesia". The root of this cautiousness seems to be whether the problems that frontal patients show can be properly described as "amnesia" and for this reason the concept of frontal amnesia is inextricably linked with that of "classical amnesia".

"Classical amnesia", as a term, has also enjoyed a recent rise in popularity (see Baddeley and Wilson, 1986; McCarthy and Warrington, 1990; Shallice, 1988). However, it is difficult to determine whether the use of this term "classical amnesia" has increased as a response to a greater understanding of this particular syndrome, or whether it has increased only out of a need to differentiate it from frontal amnesia. The result of the use of both terms is that one is almost forced by terminology alone into one or other of two main camps of opinion regarding frontal amnesia. Thus if one accepts the term "frontal amnesia" one is implicitly accepting that there are two types of amnesia, and if one will only stretch as far as describing "memory problems secondary to frontal lobe dysfunction" then one is implicitly suggesting that these problems do not warrant the term "amnesia". Either way, it is difficult to understand what is meant by frontal amnesia without first understanding "classical amnesia" since the debate as to whether frontal amnesia is a form separate from classical amnesia
is not yet decided, and one view (represented most clearly by Baddeley and Wilson, 1988) is that frontal amnesia is a classical amnesia plus other characteristics.

Fortunately, while frontal amnesia is as yet a poorly defined syndrome, the characteristics of classical amnesia have been the focus of exhaustive studies (see Mayes [1988] for a review) and there are now some features which are generally accepted.

1.1(a) Classical Amnesia

Classical amnesia (in pure form) refers to a state in which a patient has an anterograde memory loss and a retrograde loss the degree and nature of which is still debated. This occurs in the context of spared intellectual functioning (as for instance, measured by the WAIS) and short-term memory (Mayes, 1988). As pointed out by Shallice (1988), criteria for defining the point at which a patient is categorised as "amnesic", vary. However, most studies use patients scoring between two and three standard deviations below the means of age-matched controls on tests such as the Wechsler Memory Scale (WMS) or Warrington’s Recognition Memory Test (RMT). Other agreed characteristics of the classical amnesic have been elicited by dissociation methodology and thus much is made of their performance on tasks where one might intuitively have expected them to be impaired, but in fact they are normal or near-normal. Examples are:

1. Preserved motor-skill acquisition: Much of the research in this area owes a debt to Brenda Milner, who was the first to convincingly demonstrate that an amnesic (in this case, H.M.) was able to learn a motor-skill, albeit at a slower rate than normals. Thus he was taught to trace an outline drawing whilst looking in a mirror rather than at his hand, and showed the benefits of this practice three days later (Milner, 1962, 1965). Other examples of this type of learning
being preserved in amnesia include learning to use novel tools (Talland, 1965), pursuit rotor learning (Brooks and Baddeley, 1976; Corkin, 1968), and piano playing (Starr and Phillips, 1970). Indeed in the case of the Brooks and Baddeley study, the amnesics learnt the pursuit rotor task at normal rates.

2. Preserved perceptual learning: Also known as repetition priming (Ellis and Young, 1988 p. 298), the finding that amnesics can be near normal on tasks involving perceptual learning stems from the early study of Warrington and Weiskrantz (1970: see also 1968). Using repeated presentations of variously degraded words or pictures, they were able to show that, under certain conditions, amnesics may show the same priming benefits from repeated presentations as controls. Warrington and Weiskrantz (1968) demonstrated that some of these gains were still evident in one amnesic three months later. This finding has since been replicated with other forms of perceptual learning tasks (eg. Moscovitch, 1982).

3. Preserved completion recall: Many experiments have shown this to be robust characteristic of classical amnesia (eg. Jacoby and Witherspoon, 1982; Schacter, 1985; Shimamura, 1986; Warrington and Weiskrantz, 1970). For instance, Graf, Squire and Mandler (1984) tested amnesics' and normals' memory for previously studied words under four conditions: free recall, cued recall (using the first three letters of the words as cues), recognition memory, and fragment completion recall. The amnesic patients were impaired under all conditions except for completion recall.

4. A number of priming effects have been shown to be relatively preserved in amnesia. Johnson, Kim and Risse (1985) showed that amnesic patients preferred previously heard melodies, in favour of new ones, despite not being able to tell which they had heard before. Patients may also show improved speed of reading of primed material (Schacter, 1987), or be
faster in a task where the patient is required to generate cues to make difficult-to-comprehend sentences easier to comprehend (McAndrews, Glisky and Schacter [see Schacter, 1987]). In addition reaction times to primed serially presented material may be quicker (Nissen and Bullemer, 1987) and patients may show a variety of what McCarthy and Warrington (1990) describe as "verbal facilitation effects" (p. 303), in such tasks as naming, category identification, and the generation of opposites (Warrington and Weiskrantz, 1982).

That there are preserved priming effects in classical amnesia is now, of course, generally accepted, and this information has been used to argue for separate 'types' of memory, memory trace, or memory processing. The most commonly known terminology for this distinction is perhaps the episodic vs. semantic explanation of Tulving (1972, 1983). However, according to Shallice (1988), this explanation has receded in favour due to its inability to deal comprehensively with all the different types of learning. Instead, broader taxonomies have become more popular - the most obvious example being the procedural vs. declarative explanation (Cohen and Squire, 1980; Squire, 1986) which seems to have become so much a part of neuropsychological terminology that in some texts the term "procedural memory" is used without even reference to the artificial intelligence background from whence it came (Winograd, 1975) (see for instance Baddeley, 1990). The explanatory power of this taxonomy is demonstrated by Mayes (1988), who argues that both semantic and episodic memory can be thought of as forms of "declarative memory", leaving the "procedural memory" component free to explain the various priming, conditioning and skill acquisition phenomena that can still be preserved in a dense amnesic. It is becoming clear, however, that even the procedural/declarative taxonomy, broad though it is, cannot encompass all forms of human memory.

There is increasing evidence for a type of cognitive process which is involved in "remembering to remember"
ie. recalling a prior intention at a time remote from the setting-up of that intention (Harris, 1983; Loftus, 1971; Meacham and Leiman, 1982). It is most commonly termed "prospective memory". Additionally, it has been suggested that prospective memory functioning may work independently from all forms of retrospective memory (Kvavilashvili, 1987; Wilkins and Baddeley, 1978). Chapter six will propose that it is possible to suffer from a severe impairment in this type of memory functioning, and yet not be classically amnesic. However, firstly the pattern of performance of frontal amnesics on retrospective memory tasks will be contrasted with that of the classical amnesic pattern already described.

1.1(b) Frontal Amnesia

Frontal amnesia should of course refer to an amnesic state secondary to frontal lobe involvement. This would seem straightforward enough, but in fact even at this stage there are problems. The most immediate is that the term itself mixes descriptive levels, being a combination of syndrome and localisation. It also begs two important questions. Firstly, what has been termed here "classical amnesia" is what is generally considered to be "amnesia". So if frontal amnesia has different characteristics, then we need to be clear about they ways in which it qualifies as an amnesic syndrome. Secondly, there is considerable doubt about whether the lesion which produces the characteristics of "frontal amnesia" is indeed in the frontal lobes. These two questions will be examined in more detail, starting with the topic of whether the disruption in memory performance that frontal lobe dysfunction causes can indeed be considered an amnesia.
1.2 In what way do the memory problems experienced by patients with frontal lobe dysfunction differ from classical amnesia?

Empirical evidence supporting a distinction between classical and frontal memory problems has only recently started to emerge, and much of that evidence is equivocal (Baddeley and Wilson, 1988). In addition, many of the characteristics of classical amnesia are shared by amnesic patients with frontal lobe damage. However, suggestions have been made that the following characteristics are shown by patients with frontal lobe dysfunction:


2. Failure to release from proactive interference (Moscovitch, 1982; Squire, 1982)

3. Dissociations of performance on free recall vs recognition memory tasks, with either free recall normal and recognition impaired, or the converse pattern (Delbecq-Derouesne et al, in press; Hirst, 1985; Jetter et al, 1986; Moscovitch, 1989; Kapur and Coughlan, 1980)

4. Frontal lobe patients are often not normal on procedural learning tasks such as jigsaw learning or reading transformed script (Baddeley and Wilson, 1988; Mayes, 1988 p.118).

5. It has been argued that frontal lobe patients perform tasks involving cued recall performance poorer than classical amnesics matched for the severity of their amnesia by, for instance, free recall performance (Parkin, et al, 1988; although see also Damasio et al, 1985 and Vilkki, 1985).

6. Increased susceptibility to interference (Parkin et al, 1988; Volpe and Hirst, 1983).
7. Poor temporal discrimination abilities (Fuster, 1980; Milner et al, 1985; Parkin et al, 1988)

Of all these characteristics, perhaps the recall/recognition dissociations are the most puzzling, since this is the only indication that frontal amnesics can be better than classical amnesics at some tasks - otherwise they just seem to be more universally impaired (ignoring for the moment the complicated issue of what confabulation is). In addition, some authors (eg. Stuss and Benson, 1986) have contended that frontal lobe patients may show memory-related problems without being "amnesic". So it would seem at first sight that at least some of the memory problems experienced by frontal lobe patients do differ from those experienced by classical amnesics. However, as will be shown later, the situation is far from being straightforward.

1.3 The pathology of patients with "frontal lobe" memory problems

There are a number of groups of patients who are most commonly reported to show the characteristic signs of frontal memory impairment as described above. However their presentation does vary with pathology, as has the way in which the data from these patients has been interpreted. These will be discussed in turn.

1.3(a) Korsakoff's Psychosis

Probably the earliest reported examples of the amnesic syndrome were of Korsakoff's patients (Korsakoff, 1889, 1890, although some cases showing a Wernicke-Korsakoff pattern were reported even before these [see Talland, 1965 for a review]) and much of the data which has been used to define "classical amnesia" has come from studies of Korsakoff patients (see for instance Mair et al, 1979).

Recent evidence has emerged that the lesioning in Korsakoff patients may be more widespread than that
considered necessary and sufficient for a classical amnesia. Thus in nonalcoholic Korsakoff disease lesions are found in the regions of the 3rd and 4th ventricles, and of the aqueduct. The mamillary bodies are, of course, most consistently involved and often show macroscopic petechial haemorrhages. But macroscopic haemorrhages are also occasionally seen in the grey matter around the 3rd ventricle and aqueduct, the inferior colliculi, parts of the medial thalamic nuclei and the region of the vestibular and dorsal vagal nuclei (Victor, Adams and Collins, 1971). However, alcoholic patients may have even more widespread lesioning. These patients, in addition to the lesioning above, may also have additional, widespread cortical atrophy. Confusing the issue still further, there is growing evidence for widespread neurotransmitter system disorder in Wernicke’s encephalopathy (Joyce, 1987), which makes interpretation of cognitive impairments in terms of a local structural disorder very difficult indeed.

It is possible that different patients with Korsakoff disease may therefore be able to provide evidence for both a mamillary-thalamic type of amnesia (ie. classical) and for a less pure amnesia which includes frontal cortical involvement. However, unless the patient under study has been investigated at post-mortem (eg. Mair et al, 1979) - and perhaps even then, given the neurotransmitter system evidence - it may not be possible to know exactly where the dysfunction is in any particular Korsakoff patient.

A number of recent studies contrast the performance of Korsakoff’s patients with patients that supposedly have lesioning confined to areas outside the frontal lobes, in a "subtraction" methodological design. The idea is that the cognitive phenomena shown by the Korsakoff’s patients that are not shared by the classical amnesics is therefore due to frontal lobe dysfunction (eg. Janowsky et al, 1989a, 1989b; Parkin et al, 1988). This raises a number of issues which will be dealt with separately in the later discussion on methodological issues, but
needless to say the fact that we cannot be sure of the extent of the lesioning in Korsakoff's psychosis means that we have to be very careful in interpreting data from these patients, if we are using that data to make inferences about the localisation of memory functions. In any event, Korsakoff patients cannot be thought of as providing unequivocal evidence for a frontally located amnesia as distinct from classical amnesia since their lesions are not confined to the frontal lobes (Butters, 1984).

1.3(b) **Anterior Communicating Artery Aneurysms (ACoAA)**

Patients who have suffered an ACoAA may show confabulation (Delbecq-Derouesne et al, 1990;) and recall/recognition dissociations (Volpe and Hirst, 1983) together with other frontal amnesic signs (Alexander and Freedman, 1984; Shallice et al, 1989). In addition, the pathology of the damage leads to quite circumscribed lesions which are often located anteriorly (see for instance Shallice et al's case LE). So if it could be shown conclusively that ACoAA lesions are confined to the frontal lobes, then the concept of frontal amnesia is secure. However, the situation is far from straightforward. Volpe and Hirst's (1983) two ACoAA patients showed no evidence of frontal lobe damage on CT, and Volpe and Hirst contend that a variety of mechanisms at work following aneurysm rupture can cause damage remote from the original site. Most notably, they suggested that this may include transtentorial herniation of the mesial temporal lobes, an assertion supported by Volpe et al's (1984) PET study a year later. Alexander and Freedman (1984) went even further, stating, on the basis of their study of 11 ACoAA cases, that "gross infarction of the frontal lobes is not a requirement for the syndrome [of amnesia following ACoAA rupture]" (p. 752). Instead, they contend that the critical sites are the medial septal nuclei, the paraventricular nucleus of the anterior hypothalamus, and the medial forebrain bundle. Other supporting evidence comes from Gade's (1982) study, which discusses the wide variety of
candidate lesion sites following damage to the AComA perforators, concluding that "the most likely candidate as critical area in the cases of ACoA amnesia may be the columns of the fornix". Crowell and Morawitz (1977) make the point that the distribution of the ACoA includes the basal forebrain, the anterior cingulate, the anterior hypothalamus, the anterior columns of the fornix, the septal nuclei, the anterior commissure and the corpus callosum.

Given that there appears to be such individual variation in the distribution of the AComA (Vincentelli et al, 1991) it is hardly surprising that great variation also exists in the nature of the amnesic impairment in such patients. While there is little doubt that some patients can become densely amnesic following ACoAA (see example OE below), this is by no means always the case. It is clear, for instance, that Delbecq-Derouesne et al’s (1990) patient, RW was not densely amnesic in that he could remember day-to-day events (albeit the details and contexts were confused) in a way that a classical amnesic would not. In addition, the group study which is most often quoted as evidence for memory impairment following ACoAA is not without its problems. Alexander and Freedman (1984) document 11 cases of patients with memory problems following ACoAA which were investigated in a single case fashion. Unfortunately the only neuropsychological memory data available for these cases is overall Wechsler Memory Scale [WMS] Memory Quotient [MQ] and Wechsler Logical Recall scores (immediate recall and either 20 min. or 5 min. delay, depending on which page you are reading) and qualitative information about the Rey Auditory-Verbal Learning Test (RAVLT) of 6/11 cases.

However, if one calculates their patients’ Full-Scale IQs from the Verbal and Performance scores given, and compare those with the WMS MQ given, Alexander and Freedman’s assertion that these patients are amnesic becomes a little tenuous. Thus one patient’s WMS MQ is actually 12 points above his IQ, and of the 10 patients whose MQ is lower than their IQ, the mean discrepancy is just 14.6
point (SD 11.1), which is less than one standard
deviation lower than it should be. Using Shallice's
(1988) criteria for defining patients as amnesic
("severely amnesic": >30 points MQ-IQ discrepancy;
"mild/moderate amnesics": 20-30 points MQ-IQ
discrepancy), only one of Alexander and Freedman's eleven
patients would be considered severely amnesic (Case 4,
with MQ-IQ discrepancy of 43 points), with 2 further
patients classified as mildly amnesic (Cases 7 and 9 with
discrepancies of 21 and 23 points respectively). But 8 of
the 11 patients would be classified as having no
significant memory disorder at all. There is even some
doubt about the true nature of the amnesia in the patient
with a 43 point discrepancy: his MQ was still 97, which
is average for the population as a whole.

In conclusion, patients who have suffered an ACoAA may or
may not be amnesic, and some cases who perform at a level
commensurate with a dense amnesia (in a classical
amnesic) on some memory tests, may not be clinically
amnesic (eg. Delbecq-Derouesne et al, 1990). Some studies
give too little information for the reader to assess the
extent of the memory problem (eg. Lhermitte and Signoret,
1976). In addition, there is good evidence that
anatomically the distribution of damage following ACoAA
can be highly variable, and there is currently no need to
assume that the memory impairment experienced by patients
who have suffered an ACoAA is informative about the role
of the frontal cortex in memory (see Kapur, 1988 for a
full discussion).

1.3(c) Frontal Lobe Space Occupying Lesions (SOLs) and
Localised Traumatic Damage

There is considerable disagreement as to whether a
prefrontal cortical lesion, of whatever size, is a
sufficient condition to produce an amnesic syndrome. Thus
some maintain firmly that amnesic disorders do occur in
patients with frontal lesions (ie. Hecaen, 1964;
Shallice, 1988) whilst others do not (eg. Shimamura,
Janowsky and Squire, in press). After exhaustive review of the literature, Stuss and Benson (1986) conclude that "No consistent demonstration of frontal structural abnormality associated with memory disturbance in the classic amnesia pattern has been presented in the human" (p. 185). However, later they go on to explain "Most current research demonstrated that damage to the frontal lobes does not produce classic amnesia, but, rather, results in frontal cognitive impairments that influence the successful functioning of memory". However, there is considerable empirical evidence for frontal lobe SOLs causing similar deficits to other lesions on memory tests. Thus the largest and most closely controlled study comparing different lesion site patients on a memory test is that of Warrington (1984). Warrington tested 152 SOL patients on a forced choice memory paradigm and found no significant differences in patient’s performances according to lesion site within the same hemisphere - ie. left frontals were similarly impaired to left temporals, for instance, on her FCW test. Mayes has argued with this data, contending that "Although in a large-scale normative study, Warrington (1984) has reported recognition impairments in patients with frontal lobe lesions, one cannot exclude the possibility that lesions in some of her patients may have extended into other regions where damage can cause mild amnesia" (p. 110).

However, if Warrington’s data is examined closely it appears that this is an unlikely explanation. Consider Table 1, where the distribution of each lesions group’s score on Warrington’s tests are presented. It is apparent that the distributions of scores within the lesion groups are very similar indeed. If Mayes’s argument is that a few frontal lesion cases who were amnesic skewed the group data, then the distributions would not be as similar. Mayes’s argument is interesting from another point of view. Mayes is of course founding his argument on the basis that it is damage to the posterior regions that 'causes' amnesia. If however one changes the assumption, his argument might equally (on the basis of Warrington’s data) hold for the posteriors. In other
words, supposing for the moment that it is anterior rather than posterior damage that leads to amnesia, then one could argue that when posteriors fail the RMT it is because they have lesions which extend to frontal regions. Warrington’s data equally well fits either notion.

Table 1
Distribution of forced-choice recognition memory scores achieved by patients with cortical lesions according to lesion site from Warrington (1984)

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<td>37</td>
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<td>25</td>
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<tr>
<td>50</td>
<td>44</td>
<td>46</td>
<td>44</td>
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</tbody>
</table>

1 Chance in this context refers to a score of 25/50 or less. Clearly however some scores higher than this may still represent chance levels.

Key: R = right  L = left  F = frontal  T = Temporal  P = parietal

Newcombe’s (1969) study of 83 war veterans with localised penetrating head wounds, studied approximately 20 years post-trauma, is also instructive. Thus she found that on immediate and delayed recall of a story (Talland, 1965
Text D p. 236) frontally lesioned patients were not significantly better as a group than either temporals, parietals or occipitals (although the usual hemispheric differences emerged). Indeed, there were no significant differences between any of the lesion sites, echoing Warrington's data. However on another measure - Verbal Associate Learning [Inglis, 1959], the left parietal lesioned patients were significantly worse than any other, with temporal patients nonsignificantly but nevertheless worse than the unimpaired frontals.

1.3(d) Surgically Induced Lesions

There have been two main reasons for inducing frontal lobe lesions surgically (surgery for removal of space occupying lesions is considered in the previous section). The first is for remediation of certain psychiatric conditions (in the 1940s and 50s this was schizophrenia, more recently it is more usually chronic intractible depression) and the second is for intractible epileptic disorders. These surgical procedures have yielded considerable data of varying significance.

As pointed out by Hecaen and Albert (1978), psychosurgery cases are contaminated by their pre-surgical pathology. Traditionally, however, this has only been treated as a considerable problem for leucotomy cases. This is because cases which undergo surgery for epilepsy may have excisions which spare the frontal lobes whereas leucotomy patients necessarily have no such non-frontal controls. That much of the leucotomy data should be treated with caution is underlined by recent findings of the wide variety of cognitive impairments found in schizophrenia and depression (see Shallice, Burgess and Frith, 1991; Coughlan and Hollows, 1984). It is possible to have a control group of patients who have not had the operation, of course, but this raises the question of why it was that they had not also undergone surgery, given that they were matched for factors such as severity and form of disorder (see for instance Stuss et al's [1981] study). Kartsounis, Poynton, Bartlett and Bridges (in press)
sidestepped this methodological problem by performing assessments on patients pre-operatively, so that the patients acted as their own control. This study found that while the patients showed significant decreases in memory performance on some memory tests two weeks post-operatively, these differences had disappeared at testing six months post-operatively (see Table 5).

Since the lesion made by the operation remains at six months, but the considerable oedema which may extend in some patients beyond the frontal lobes does not, it seems that the memory impairments shown by these patients is most likely due to the oedema. However, the situation is complicated by the fact that postoperatively there is ventricular enlargement in most patients. In addition, the lesion itself is not cortical. The lesion is always in the white matter of the orbital gyri, posterior in the frontal lobe and beneath the head of the caudate nucleus, and is quite large, covering an area of between 16 to 22mm, lying between 2 and 6mm lateral to the medial border of the lobe. Bartlett (personal communication) contends that in some cases the lesion may affect areas distant to the actual site of the lesion, particularly within various limbic system structures and, critically, the dorsomedial nucleus of the thalamus. While the various methodological issues surrounding this procedure are as yet unresolved (Kartsounis et al, in press) it is clear that the nature of both the neuropsychological and neurosurgical data mean that data from these patients cannot provide unequivocal evidence for the role of the frontal cortex in memory (see also Stuss et al 1981, 1982 for a full criticism of older methods).

Since the early 1960s, Brenda Milner and her colleagues at Montreal have studied extensively patients undergoing unilateral cortical excisions for the relief of focal epilepsy (eg. Milner, 1962, 1964, 1968, 1975, 1980, Milner and Teuber, 1968; Petrides, 1985; Petrides and Milner, 1982). These studies have yielded considerable useful data. However many of the findings of frontal-lobe memory impairment are equivocal. Those studies where
patients with frontal lobe excisions are compared
directly with patients with posterior excisions on memory
tasks have shown little impairment in the frontal groups
relative to patients with extra-frontal lesions (eg.
Milner, 1985; Smith and Milner, 1984).

In other studies, the ways in which the results were
analysed makes interpretation according to lesion site
problematic. For instance, Milner (1968) studied five
subject groups - normal subjects plus groups of patients
with either right and left temporal, frontal or parietal
lesions, on measures of visual recall and recognition
using faces as stimuli. Throughout the study, Milner did
not distinguish between left and right hemisphere for the
frontal- or parietal-lesioned patients, but grouped them
together for comparison with the right or left temporal
groups. Thus, for instance, when she looked at the
performance of right temporal lesions on a measure of
visual retention after an interpolated task, the
statistical comparison was made between this group, the
left temporals and a group of right and left frontals
considered together, and a group consisting of right and
left parietals. So we cannot tell how right temporals
compare with right frontals or right parietals. Since the
left temporals were generally better than the right
temporals, the apparent differences between the frontals,
parietals and the right temporals may be due to a
between-hemisphere effect only. Clearly this study
enables us to say little about frontal vs. temporal
lesion effects on memory.

Similarly, Petrides and Milner (1982) studied groups of
patients with unilateral frontal or temporal lobe lesions
on a task requiring the organisation of a sequence of
pointing responses. Since the task requires the subject
to remember those items previously pointed to, the task
clearly has a memory component. As such, the task may be
relevant to theories of cortical involvement in temporal
sequencing. However Petrides and Milner do not perform
direct comparisons between the temporal groups and the
frontal groups, instead comparisons are made between the
lesion groups and the normals. While the left frontals are consistently the most impaired on these tasks, regardless of the type of stimuli used, the mean errors of the frontal groups are often only slightly greater than the temporals. Presumably the comparisons of groups within hemisphere were not significant, but this is not mentioned. The same points hold true for Petrides's study of spatial conditional associative learning reported in Milner (1982).

In summary, as pointed out by Stuss and Benson (1981, 1986), the information gained so far from studies of surgically-induced lesions has yielded little that is not equivocal regarding the role of the frontal lobes in memory.

1.4 Evidence for "Frontal Amnesia"

Studies concentrating on localisation issues relevant to frontal lobe amnesia have largely proved of limited value. In the following sections, the evidence for the neuropsychological aspects of frontal lobe amnesia will be discussed. For convenience, the individual phenomena which have been considered characteristic of frontal amnesia will be explored in turn.

1.4(a) Free Recall vs. Recognition Memory Dissociations

The evidence for recall-recognition discrepancies in patients who display other characteristics of frontal amnesia is growing quite rapidly (see Moscovitch, 1989). But what is the quality of this evidence? The view that such discrepancies do occur, and that they are significant for our understanding of amnesia is pursued most vigorously by Volpe and Hirst. Their 1983 paper reported 2 cases of memory problems following ACoAA, one of whom would be classified by Shallice's (1988) criteria as having a mild to moderate amnesia (26 point discrepancy) whilst the other would be considered severely amnesic (33 point discrepancy). The study is
most remarkable for its finding that in these two patients, their memory performance when measured by a recognition paradigm, was significantly superior (p<0.05) to their performance measured under free recall conditions. The data, in adapted form, where patients’ scores are quoted as percentiles of the control data are quoted in Table 2.

One can make both specific and general points regarding Volpe and Hirst’s interpretations of their data. A specific criticism is that there are some statements which are difficult to understand. For instance, they remark that "Visual recognition was...comparable to that of the controls" (p. 707). However when the data is considered in terms of percentiles of the controls performance, the two patients performed at the 1%ile and 10%ile levels respectively. It is doubtful whether this can be considered a "comparable" performance. Another is that they maintain that "cued recall [as opposed to free recall] improved the patients’ performance by almost

**TABLE 2**

Memory Test Performances of AcoAA Patients Reported in Volpe and Hirst (1983)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Patient 1</th>
<th>Patient 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>WAIS FSIQ</td>
<td>113</td>
<td>116</td>
</tr>
<tr>
<td>WMS MQ</td>
<td>87</td>
<td>83</td>
</tr>
<tr>
<td>MQ-IQ Discerepancy</td>
<td>26</td>
<td>33</td>
</tr>
<tr>
<td>Free Recall (%iles of control)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Verbal Immediate</td>
<td>34</td>
<td>4</td>
</tr>
<tr>
<td>30s Delay</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>5 min Delay</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>Visual 5 min Delay</td>
<td>0.5</td>
<td>0.6</td>
</tr>
<tr>
<td>Recognition</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Verbal Immediate</td>
<td>82</td>
<td>46</td>
</tr>
<tr>
<td>30s Delay</td>
<td>14</td>
<td>21</td>
</tr>
<tr>
<td>5 min Delay</td>
<td>50</td>
<td>31</td>
</tr>
<tr>
<td>Visual 5 min Delay</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Free vs. Cued Recall (words)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Cued</td>
<td>38</td>
<td>3</td>
</tr>
</tbody>
</table>
three times the free recall performance rate" (p. 701). This may be true as far as raw scores are concerned, but when one considers the improvement in terms of relative positions within the distribution of scores, this only appears to be true for one patient (Case 1), who improved from the 4th percentile (of the controls) under free recall, to the 38%ile level under cued conditions. Case 2 is however less convincing. This patient improved from the 1st percentile level to the 3rd percentile level - hardly a remarkable difference.

A general methodological criticism is that we cannot know the significance of the recall-recognition discrepancies indicated here until we know the pattern of performance by patients with other cortical lesions, or indeed patients with extracerebral lesions. If the free recall task was merely more "resource demanding" (Shallice 1988; Schacter, 1987) than the recognition task, one might get this pattern with any form of illness which affects cognitive resources. For instance McNeil (1991) found recall-recognition discrepancies in a group of schizophrenic patients. However she also found recall-recognition discrepancies in some of a group of age- and NART-matched normal controls. When the groups were compared directly, the schizophrenics were significantly poorer at free recall (both story recall and list learning) than the controls, but there was no significant difference between the groups' abilities on a a recognition memory task (Warrington’s RMT for words). However an analysis of covariance using current level of intellectual functioning (Ravens Matrices) as a covariate, removed the differences between the groups completely. Analysis of factors predictive of performance on recognition or free recall tasks found considerable differences in the demands the differing tasks make upon general cognitive resources. Many factors (such as age, current and pre-morbid IQ, and a number of measures of language abilities and problem-solving) were influential upon the free recall tasks, whereas this was much less the case for the recognition memory task.
In any case, then, it is apparent that Volpe and Hirst's recognition-recall pattern cannot be considered peculiar to ACoAA cases. Moreover there are a number of potentially influential factors that must be carefully controlled for in such studies.

These criticisms however probably only relate to the significance of Volpe and Hirst's patients' pattern of performance, rather than to whether they had an amnesia per se. Shallice (1988) makes the following criticism of the WMS: "It is now widely agreed that the WMS does not provide a very adequate measure of amnesia. A number of its subtests (e.g. Digit Span) can be performed relatively well by amnesics because they tap processes other than the critical memory one - for example short-term memory skills that are not impaired in the amnesic syndrome.." (p. 354). The corollary of this is that if some measures are not sensitive to amnesia, in the patient who is otherwise intact (as measured by WAIS scores etc.), a poor score on the WMS is particularly indicative of a memory problem. Thus those patients' performance on the Logical Memory, Paired Associates and Visual reproduction parts of the WMS were probably very poor indeed.

Three years later Hirst et al (1986) tackled the issue of recall-recognition discrepancies1. In an attempt to get round the criticism that amnesics' discrepancies are due to the fact that recognition tasks are generally easier, they raised the amnesic's recognition performance until it equalled normal recognition and then compared free recall of amnesics and controls. They managed this by presenting the stimuli (words printed on cards) to the amnesics for 8 seconds each, but only 0.5 secs each for the normals. Subjects were then asked, after 5 minutes of arithmetic problem-solving, to free recall the word they had seen, followed by 2-choice recognition of the targets, with confidence ratings. Interestingly, the

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1 This study does not concern itself directly with recall-recognition discrepancies as a characteristic of frontal lobe involvement. It is included because of its value in highlighting the methodological issues outlined.
increased study time was not successful in equating the recognition of their Korsakoff group. Their other group of patients, consisted of 3 patients amnesic following ACoAA, 3 patients who had suffered diffuse hypoxic ischemia and one closed head injury case. This group did show the expected benefit from increased stimulus exposure. We are not given equivalent background neuropsychological data on the two patient groups, and so are unable to be sure, therefore, whether the groups were matched for severity of amnesia. As Hirst et al admit, their findings therefore might only be applicable to those who have a mild amnesia. If recall-recognition discrepancies are indeed characteristic of amnesia (as opposed to anything else), then surely those who are more amnesic should be more rather than less likely to show the discrepancy. But this was not the case - those that were most amnesic merely failed all measures. Conversely, if recall-recognition discrepancies in normals arise because of the different "resource demands" of the tasks, then those amnesics who are most like the normals (ie. least amnesic) would be most likely to show discrepancies. And this was the case.

Shallice (1988) presents a more sophisticated criticism of Hirst et al's (1986) findings. He argues that "the very rapid one stimulus per 0.5 sec presentation rate used with the normal group would be likely to lead to a large variance in trace strength across stimuli due to variability in attention and encoding. If this is the case, matching the groups on a low criterion (for recognition) would not ensure that the groups were matched on a higher one (for recall). The group with the larger variance would exceed a higher criterion more frequently. Thus a higher recall level would be expected in the control group than in the amnesics because recall would in general require higher "trace strength". In fact, the control group did show a higher difference in confidence between the correctly recognised items and the incorrectly recognised one than did the Korsakoff patients, indicating a greater variance in the
distribution of trace strengths across subjects in that group" (pp. 355).

Two years later, Shallice and his colleagues (Delbecq-Derouesne et al. 1990) reported one of the most striking cases of a recall-recognition discrepancy. Their patient RW, who had suffered an ACoAA "produced a normal number of correct responses in tasks of recall, whereas his performance was comparable to that of amnesics in recognition tasks" (p.1046). Their case is of interest, they contend, because there are "no neuropsychological arguments in favour of a dissociation between the two processes of retrieval, one involved in recall, the other in recognition" (p. 1046). This is perhaps a little unfair to Hirst et al (1986), whose evidence they cite in the next sentence. However, Delbecq-Derouesne et al’s point that in amnesia, it is often claimed that recognition is superior to recall because recognition is a less "resource demanding" (Shallice, 1988) is valid argument. To find, therefore, a patient who shows a pattern of impairment the opposite of what this would predict is potentially very interesting. There is one possible explanation for these findings which Delbecq-Derouesne et al do not consider which will be dealt with later. For the moment, however, we will consider their case in more detail.

RW was 54 at the start of Delbecq-Derouesne et al’s investigation of his problem, which was approximately eight years after his operation to clip an ACoAA. He achieved a Progressive Matrices score at the 90th percentile, and had a WAIS VIQ of 102 and PIQ of 87. His language, perceptual and praxic skills were intact, and he failed only one of thirteen tests considered sensitive to frontal lobe dysfunction. On the WMS, he achieved a MQ of 94, which "is in the dull normal range and not significantly different from his IQ on the WAIS" (p. 1052).

The study concentrates on two aspects of RW’s presentation: his recall-recognition discrepancy and his
confabulation. His confabulation will be dealt with later, so for the moment the nature of his memory test performances will be considered.

Essentially, RW showed normal or near-normal recall of word lists or short stories, but Delbecq-Derouesne et al contend that his performance on recognition tests lay significantly below the expected value given his recall scores.

One obvious problem with their data as presented, is that they for the most part are comparing free recall of verbal material, with forced-choice recognition of visual material. There is of course no reason why these should be correlated in a brain-damaged patient. However Delbecq-Derouesne et al do present tasks on which verbal recall and recognition can be compared. The first were the standard neuropsychological tests of story recall from the WMS and Warrington’s (1984) RMT for words. We are told that he scored 34/50 on the RMT which is a performance at the 2%ile for his age (however the task was not identical to that of Warrington (1984) since the task is translated). This compares with his performance on the WMS logical memory which was approximately at the 15%ile for his age. Whereas for the visual forced-choice tasks, Delbecq-Derouesne et al give RW’s performance compared with 75 control subjects, for the forced-choice words, we are only told that his performance was worse than 15 age-matched controls. Since we are not given the distribution of these scores, it remains possible that RW’s performance was at or even above the 15%ile on this task. In any case, there must be some doubt as to the significance of a 15%ile-2%ile discrepancy, since Warren and Groome (1984) found a similar pattern in some of their depressed patients. Interestingly, the recall-recognition discrepancy in these patients was even greater when verbal free recall (story recall) is compared with a visual recognition task (Warrington’s [1984] RMT for faces) - which is what Delbecq-Derouesne et al concentrate on for the majority of the paper. In addition, the direction of the discrepancy (the mean
score for the depressed group who received high-energy sine wave ECT before treatment on story recall was at the 16%ile of normals, the mean score on forced-choice faces was below the 1%ile) was the same as that shown by RW. It is doubtful, therefore, that RW’s performance on the standard neuropsychological tests represents unequivocal evidence for a significant recall-recognition discrepancy. So what of the experimental tests?

Delbecq-Derouesne et al gave RW a test based on Craik and Tulving’s (1975) depth of processing paradigm. It consisted of a series of cards, each containing a sentence with a word missing and a target word. For each sentence he had to say whether the target would fit well into the sentence. There were 30 target words that occurred once and 30 that occurred twice, on each occasion with a different sentence frame. After a one minute delay RW was given five minutes to remember, by free recall, as many words as he could. He was then presented with all the 60 targets together with an equal number of distractors and had to select the words that he had seen before. He was not told how many targets he had to recognise.

RW’s performance on the recognition measure of the Semantic Orienting Task was essentially at chance. This compares with his free recall performance which was at the low end of the normal range. In the free recall measure he produced many more confabulatory responses than any normal. RW’s story recall ability was also at the lower end of normal. On other "free recall" tasks (immediate recall of 10 or 15 word lists and delayed recall of one of the lists) his score was within the normal range - but not high within it.

Delbecq-Derouesne et al present one statistical analysis of the significance of such a discrepancy. Using a group of just 10 control subjects (about whom we have no details), a regression line was obtained for the recognition score (corrected for guessing) that would be expected for a given level on the recall test (they state
the correlation between the two values as being 0.48). The recognition score obtained by R.W. lay significantly below the expected value given his recall score (z = 2.6; p < 0.01).

Given that many factors can influence a subject’s performance (differentially) on free recall and recognition paradigms, it is very important to know the exact details of the Delbecq-Derouesne’s control group. The danger is that a selection artefact may occur in such a small control group which would lead to artificially high correlations between the free recall and the recognition measures. This might give the impression that R.W.’s recognition-recall discrepancy was significant when a broader sample would reveal that such findings are not altogether uncommon in the normal population.

McNeil’s (1991) finding that differing abilities traditionally considered unrelated to memory (such as reading ability, problem-solving, IQ and so forth) may differentially affect recall or recognition paradigms underlines the crucial importance of using a very carefully matched control group. In some senses of course it may never be possible to find an adequate normal control group for neurological patients since it would be impossible to achieve a perfect match for such factors as pre-morbid IQ as well as current IQ. These sorts of factors are clearly important however, as McNeil’s study found that the best predictor (from a range of factors) of recognition performance was NART score, whereas this was not predictive at all of performance on free recall tasks. Similarly, Matrices IQ was a highly significant predictor of free recall performance, but was not an influential factor in recognition memory score. Thus discrepancies on different test may be related to many possible task/ability interactions2.

The low correlations between differing forms of the same memory test, between different tests and generally low

2 These influences upon memory test performance are investigated further in chapters 4 and 5 of this thesis.
test-retest reliability (see Coughlan and Hollows, 1985; Wilson, Cockburn and Baddeley, 1985 [Cockburn, personal communication] underline the fact that performance on memory tests is quite fragile, and drawing conclusions from correlations between scores is fraught with difficulty.

The traditionally used method of calculating individual difference scores involves testing a group of carefully matched controls on both tests, calculating test reliability using split-half correlation, and then applying an equation calculating the standard error of the difference between the two scores based upon their standard errors of measurement (see Anastasi, 1988). This conventional approach (used in chapter 5 in considering individual difference scores) is however not followed by Delbecq-Derouesne et al.

These objections also hold for other studies, such as those carried out by Moscovitch (1989) or Kapur and Coughlan's (1980) case, whose recall-recognition discrepancy pattern was the converse of RW. Complicating the interpretation of the significance of these discrepancies are of course the original objections raised by Shallice (1988) and Delbecq-Derouesne et al (1990).

It should also be remembered that not all patients who show other signs of frontal amnesia also show recall-recognition discrepancies (see case LE reported by Shallice et al (1989).

In summary, then, the issue of whether recall-recognition discrepancies are a feature of frontal amnesia is a complicated one. Since there are at present so few studies and too many possible methodological questions surrounding those studies, it is doubtful whether this can yet be considered a significant phenomenon in its own right.
1.4(b) Cued recall vs. Free recall

That cued vs free recall may not follow the classical amnesic pattern in patients with "frontal memory impairment" has been noted by Parkin, Leng, Stanhope and Smith (1988). They presented the case of a 42 year-old man who had suffered a ACoAA, and a CT scan nine months post-operatively demonstrated left frontal low density with an enlarged left frontal horn. At this time he scored zero on the RBMT, which by any standards indicates a considerable memory impairment. He confabulated freely, in a fantastic manner (Berlyne, 1972). However his condition improved steadily over the next 21 months until the time of the investigation reported.

JB had a WAIS FSIQ of 115 with an estimated premorbid IQ at the bottom of the superior range (NART = 120). His WMS MQ was 89, which indicates a moderate amnesia by Shallice's criteria. The paper focusses on JB's pattern of performance on memory tests, and his performance

Table 3
Memory Test Performance of Case JB from Parkin, Leng and Stanhope (1988)

<table>
<thead>
<tr>
<th>Paradigm</th>
<th>JB</th>
<th>Korsakoff</th>
<th>Bitemps</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brown-Peterson</td>
<td>2</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Massed/Practice</td>
<td>2</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Release from PI</td>
<td>1</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td>Forgetting Rate</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Free Recall</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Cued Recall</td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Related/Unrelated Word Lists</td>
<td>1</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td>Paired Associate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rote Learn</td>
<td>1</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Self Images</td>
<td>1</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Images Given</td>
<td>1</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Verbal Med.</td>
<td>1</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Temporal Discrimination</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recognition</td>
<td>0</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td>Discrimination</td>
<td>1</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td>Source Forgetting</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Facts</td>
<td>0</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td>Source</td>
<td>1</td>
<td>No Data</td>
<td>No Data</td>
</tr>
<tr>
<td>Concentration</td>
<td>0</td>
<td>No Data</td>
<td>No Data</td>
</tr>
</tbody>
</table>
compared with a group of 7 Korsakoffs and a group of 6 bitemporals. The essence of the patterns is presented in Table 3, in which the relative impairments of the groups is represented by ranks (where 3 is poorest) and zero indicates no impairment relative to controls. Where two groups scored similarly, they are given the same rank.

From this table it is clear that JB was not the most impaired of the groups on any task (for which we have data). Additionally, on those tasks which contrasted mode of recall/presentation (eg. Temporal discrimination/Source forgetting) there is no data for the other groups, so it is difficult to interpret the meaning of the relative performances. It is not enough to state that performance on one of the stages of the experiment was impaired (relative to controls) more than another since this might be a resource threshold artefact of the type described by Shallice (1988, p. 233). Interpretation is not made easier by the fact that no standard deviations are quoted for the raw scores presented, and no comparative statistical analyses are presented.

Despite these factors, some of the patterns of performance look interesting. On a test where JB was given 10 words to recall spontaneously after a 30 minute delay, he was within the normal range. However when presented with a cue of the first three letters of the word and asked to recall the target, he did not perform within normal limits. This is an interesting finding, which would be more so if sufficient data were presented to show that no individual patient or control behaved in a fashion similar to JB: it should be explained that the scores quoted for the groups are means, whereas JB’s score is that of one trial only, and we have no information regarding the standard deviation of the controls over trials. It should also be borne in mind that if the percentage increase of the cued recall score over the free recall condition is considered (ie. the percentage difference to your score that the benefit of
cues makes over free recall) is considered, JB (100%)
performed more like the controls (254%) than did either
the Korsakoffs (1283%) or the Bitemporal (2400%).
Perhaps, then, at least as interesting a question
regarding this data is why the Bitemporal and Korsakoffs
benefit so much from cueing, rather than why JB’s cued
recall performance fell short of the normal range (see
Figure 1).

If one accepts this later argument, then combined with
the fact that the data (see Table 3), shows JB to be
overall less amnesic than either of the groups as a
whole, means that all that can be concluded from this
data is that JB performs worse on tests of memory than do
controls, but better than Bitemporal or Korsakoffs - in
other words, that his memory disorder is less severe. In
the absence of anything else, the finding that a patient
with a (at least according to CT) unilateral lesion
confined to one lobe is less amnesic than a group of
patients who will almost certainly have bifrontal and
bitemporal damage (Korskoffs) or a group with bilateral
lesions (Bitemporals), hardly seems remarkable.

Figure 1
Data on Free Recall and Cued Recall Performances of an
ACoAA patient (JB), a Bitemporal Group (B) and a group of
Korakoffs (K), From Parkin et al, 1988

Free Recall  Cued Recall
1.4(c) **Increased Susceptibility to Interference**

There are a number of views regarding what constitutes an investigation of interference effects, and to some extent the matter is bound up, understandably, with issues of the release from proactive interference. This will be dealt as a separate topic in the next section.

The study that is most often quoted in reference to interference effects following frontal lobe involvement is that of Stuss et al (1982). However this paper is actually very careful about its interpretations. Stuss et al acknowledge that, in their leucotomized schizophrenic sample many factors such as "patient state (eg. chronic vs acute), lesion location, and operational characteristics of the interference" (p. 923) may affect the results of interference paradigms. Their caution is well founded: the study used small groups of cases (five groups consisting of five cases each) and many of the criticisms of studies of surgically-induced lesions outlined above apply to this paper. In their favour, Stuss et al do not avoid pointing out the flaws in the study, and this leads them to their guarded conclusions. Not so guarded, however, are Parkin, Leng and Stanhope (1988), who make one of the most unequivocal suggestions that increased susceptibility to interference might be characteristic of "frontal impairment". This observation was made in the context of being one characteristic of a pattern of performance that they felt represented a "memory impairment..qualitatively different from that encountered in patients with the [Korsakoff] amnesic syndrome" (p. 231). The actual data they present for this assertion was based on their study of patient JB, particularly his performance on the Brown-Peterson paradigm. This data, together with the clinical details of JB have already been dealt with, and so will not be repeated. It may be that JB's performance on the Brown-Peterson task was significant as regards understanding the characteristics of anterior amnesia, but since no statistical comparisons are performed which would
facilitate an understanding of the significance of his level of performance the reader cannot know. Until either the data is presented in such a way as to enable the reader to understand how JB’s performance compares with the other groups, or the authors present conventional statistical comparisons, the suggestion that this poor Brown-Peterson performance is characteristic of those with frontal impairment remains only a suggestion. An added possibility is that these interference effects are just associated deficits.

Evidence from other sources that increased susceptibility to interference may be part of an amnesic disorder different from the classical syndrome is rare. The supporting evidence used by Parkin et al for their contention above was the study of Volpe and Hirst (1983). They, however, used a rather different measure from that used by Parkin et al (1988). Volpe and Hirst gave their two ACoAA cases ten free recall lists with ten words per list. Their measure of interference was the degree of proactive interference between lists. They calculated this by dividing the average of prior list intrusions (the number recalled that were from previously recalled lists) by the average number of responses. Their controls were six normal subjects matched for age and educational level. The proportion of prior list intrusions shown by the controls was approximately 1%, and for cases 1 and 2 were 14.4% and 38.5% respectively. However the actual number of prior list intrusions occurring is so small in all groups (controls average = 0.07; case 1 = 0.66; case 2 = 1.5) that statistical comparisons were presumably not possible (since they were not done). In addition Volpe and Hirst present no data for amnesics with other pathologies so we cannot know whether these levels are indeed (as they suggest) unusual amongst amnesics. Evidence from Warrington and Weiskrantz (1968a and 1968b) suggests that they are not. An additional minor methodological point is that it would be informative if the controls had been matched on something other than educational level - current IQ, for instance.
1.4(d) Release from Proactive Interference (PI)

The most cited study claiming that failure to show release form PI is characteristic of frontal involvement is Milner and Moscovitch’s experiment, reported in Moscovitch (1982). They tested patients who had undergone either temporal- or frontal-lobectomies on a release from PI task, adapted from Craik and Birdwhistle (1971). Giving five lists of 12 words, where the first four lists all came from the same semantic category, with a fifth from a new category, they only used patients who demonstrated buildup of PI between lists 1 and 4. Subjects were considered to have shown release from PI when the number of items recalled from list 5 was more than the number they had recalled on list 4. Patients who had undergone a left frontal lobectomy, especially those who had performed poorly on the WCST (<4 categories achieved) demonstrated the least amount of release from PI. Patients with a left temporal lobectomy who had had more than 1 cm of their hippocampus removed, showed a normal release from PI despite having a verbal memory that (on average) was lower than any other lesion group.

Leaving aside for the moment the complex questions that are raised by Moscovitch’s decision to study only a hand-picked sub-group of the cases, there are a few other points that need to be made about their results. These can be summarised as follows:

1. The proportion of patients with left temporal (LT) lesions without hippocampal removal that showed release from PI was smaller than that in the group of patients with LT plus hippocampal removal.

2. In the right temporal (RT) group, a greater proportion showed release from PI than in the control group.

3. The left frontal group who performed poorly on the WCST actually recalled more words across each of trials 1-4 than the left frontal group who did well on the WCST (although the left frontals with good WCST recalled more
The first two points seem to suggest that having a brain lesion makes you more likely to show release from PI, and that the bigger the lesion, the more likely this is. This is curious indeed, if we are to assume that to show release from PI is indeed a characteristic of normals. The third point is contradictory, however. It suggests that "good" frontals (i.e. good WCST performance) tend to show release from PI whereas "bad" frontals (poor WCST performance) do not (agreeing with Moscovitch's overall hypothesis). However, the bad frontals showed less buildup of interference in the initial trials, with subsequent poor release from PI. This raises the question of dependency. Clearly whether one shows release from PI will be dependent upon the degree to which one has encoded the previous trials - if for instance you have paid little attention to trials 1-4, then you will benefit less from the change condition, in proportional terms. However this is not explored.

An alternative explanation of the data would be that there is so much "noise" that individual cases may seem to show a variety of effects on this paradigm.

Other evidence for frontals' lack of release from PI is not strong. For instance, Parkin et al's (1988) patient JB showed less release from PI than their controls (although he did show some), but his performance is not compared with patients with other brain lesions, so we cannot be sure that it is not just a non-specific effect of any form of brain damage. Butters and Cermaks' (1980) influential study reported the performance of alcoholic Korsakoff patients, and thus is difficult to interpret in terms of specific frontal effects (as noted above, but this issue is also dealt with in more detail in the methodology section below). Other evidence suggests that release from PI may not be specific to patients with frontal lesions. Thus 2 of Graff-Radford et al's (1990)
four cases who had bilateral thalamic infarctions showed
defective release from PI. In addition "in all patients
'executive control functions' (as measured by the
Controlled Oral Word Association Test and the Wisconsin
Card Sorting Test) was mildly to moderately impaired "
(p. 11). Additionally, their conclusion is instructive in
relation to localisation studies in general. They contend
that "all our patients had lesions which could compromise
dorsomedial nucleus connections with he frontal lobe..".
Clearly if 'frontal' effects (not restricted to memory
tasks) can be caused by lesions remote from the frontal
lobes, much of the localisation methodology must be
reconsidered.

1.4(e) Poor Temporal Discrimination Abilities

Fuster (1980) advanced the view that a primary function
of the prefrontal cortex is "temporal integration" (p. 135). More specifically, the frontal cortex is involved
in what Fuster calls "provisional memory", which is "a
form of memory that permits referring any event in a
behavioural sequence to preceding events or to the
original schema" (p. 135). Certainly that one aspect of
confabulation can be the inability to recall events in
their correct sequence is well documented (see cases by
Experimentally, the ability to make temporal order
judgements have been shown to be affected in a wide range
of amnesics (Huppert and Piercy, 1978; Hirst and Volpe,
1982; Winocur and Kinsbourne, 1978), and so it seems
unlikely that this is a characteristic peculiar to memory
disorders following anterior dysfunction. However the
phenomenological evidence from confabulators is quite
compelling. Moscovitch (1989) goes as far as to suggest
that temporal sequencing problems are primary to
confabulation, and gives a good example of the type of
confused recall that can occur in confabulators:

E: "What date is (it)?
HW: The 30th of September
E: What year?
HW: 1987
E: It’s now may of 1987. You jumped a little bit ahead. can you just tell me a little bit about yourself? How old are you?
HW: I’m 40, 42, pardon me, 62
E: Are you married or single?
HW: Married.
E: How long have you been married?
HW: About four months.
E: What’s you wife’s name?
HW: Martha.
E: How many children do you have?
HW: Four. (he laughs) Not bad for 4 months.

Clearly there is something wrong with this patient’s ability to recall the correct sequence of events. He goes on to state that his oldest child is 32, the youngest being 22, and gives a variety of other information, most of which was no doubt correct at some point in the past, but which makes little sense now in the context of other information he can recall. There does seem, therefore to be something to the contention that one characteristic of frontal amnesia is disordered temporal recall, but the area requires further investigation of both a phenomenological and an experimental nature.

However, not all confabulation can be accounted for by the temporal disintegration account, as pointed out by Kapur (1988). Confabulation can be fantastic (Berlyne, 1972) in nature, bearing little relation to facts that have actually happened. What then do we know about confabulation, and is the lesion necessary for its occurrence frontally located?

1.4(f) Confabulation and Paramnesic Phenomena

The phenomenon of confabulation has been well known for some time (eg. Bonhoeffer, 1901). It is therefore perhaps surprising that it has received such scant attention,
certainly from the point of view of empirical investigation. Originally confabulation was considered a characteristic of Korsakoff's psychosis, but now it is generally accepted that it can occur following a number of different neurological events (Stuss and Benson, 1986), the most common of which is probably ruptured aneurysm of the anterior communicating artery (Shallice, 1988). The assertion that confabulation occurs as a consequence of frontal lobe damage has more empirical support than for some of the frontal amnesic phenomena already described. Thus if one considers those paramnesic cases where pathology has been detected by radiological evidence (some confabulators have clear scans eg. Dalla Barba, Cipolotti and Denes, 1989) the case that frontal lobe damage is a pre-requisite for paramnesia looks firm (see Table 4). However the data warrants closer examination. Of the 41 cases in the literature that have such radiological evidence, 13 of these cases have evidence of frontal without additional posterior involvement, and of those cases 9 are unilaterals. This of course would be more than enough evidence were the cases convincing. However of the 13 cases, 6 had lesions following traumatic brain damage, 3 had suffered ACoAAs, 1 had a subarachnoid haemorrhage and two (from Levine and Grek, 1984) had suffered intraventricular haematomas. Given that traumatic injury generally produces diffuse damage, and that in ACoAA cases, as already noted, subcortical and posterior involvement cannot be ruled out, there are only 3 cases in which involvement outside the frontal lobes seems unlikely. One of these cases was 75 years old (case 38 in Table 4) and Levine and Grek observe that their patients invariably had some degree of cerebral atrophy in addition to the vascular lesion. If one accepts these criticisms, then this leaves only 1/41 case in which frontal-only involvement seems likely.

3 The complex question of the interrelationships between the differing forms of paramnesia - of which confabulation is but one - is beyond the scope of this chapter. For the moment the term "paramnesia" will be used to refer to those family of phenomena which include reduplications, the Capgras syndrome and confabulation, following McCarthy and Warrington (1990).

11 Given that the memory for the word was stronger,
(Kapur and Coughlan’s 1980 case). Given that in this case we do not know the actual site of his aneurysm, and that the only radiological measure reported was a CT scan one week post-operatively (CT scans are hardly considered definitive in accuracy or detection rates these days) this case cannot be regarded as conclusive evidence on its own. So at the moment the issue of whether a frontal lesion alone is sufficient for the development of a paramnesic disorder is undecided. For the moment, however, it seems reasonable to assume from the data presented in Table 4, that whilst a frontal lesion alone may not be sufficient to produce a paramnesic state, those case which do show such symptoms usually do have some degree of frontal (usually nondominant hemisphere) involvement.
## TABLE 4

**SUMMARY OF CASE REPORTS OF CONFABULATION, PARAMNESIAS, REDUPLICATIVE PHENOMENA, CAPGRAS SYNDROME, FREGOLI SYNDROME AND DELUSIONS WITH KNOWN NEUROLOGICAL PATHOLOGY**

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KEY

5. Stuss et al (1978)
13. Alexander, Stuss and Benson (1979)
15. Lewis (1987)
17. Delbecq-Derouesne et al (1990)

S= sex
A= age
O= occipital; P= parietal; T= temporal; F= frontal; D= diffuse
Th= Specifically Thalamic
DB= Diffuse Bilateral
L = Left
R = Right
ER= Environmental Reduplication
C= Confabulation
RP= Reduplicative Paramnesia
Del= Complex delusions including capgras etc
CAP= Capgras
FRE= Fregoli
Haem = Haematoma
Aneur = Aneurysm
IVH = Intraventricular Haematoma
Traum = Trauma
AcoAA = Anterior Communicating Artery Aneurysm
SOL = Space Occupying Lesion
SDH = Subdural Haematoma
SAH = Subarachnoid Haemorrhage
Confabulation (and other paramnesic disorders) are of considerable significance as a symptom of "frontal amnesia" because while other aspects of the frontal amnesic syndrome, as we have already seen, are both disputed and disputable, the fact that confabulation exists as a phenomena is hardly in doubt. What is in doubt is the nature of the impairment which leads to confabulation and on this matter there are a number of different views, although there is greater agreement amongst more recent investigators. Bonhoeffer (1901, as quoted by Berlyne, 1972) described two forms of confabulation, the first of which was motivated by the embarrassment felt by amnesics when they were asked questions which revealed their memory "gaps". The second form was a much more spontaneous confabulation with fantastic content, and were related to delirium-like states. In both cases the patient believed what s/he was saying.

Some older investigations into confabulation emphasised the suggestibility and/or the personality of the patient (Williams and Rupp, 1938), or psychopathological reactions to the patients' illness (Zangwill, 1953, 1966). However, more recently investigators have produced accounts of confabulation which have concentrated upon the neuropsychological and/or neurological features of confabulators (e.g. Berglund, Gustafson, and Hagberg, 1979; Kapur and Coughlan, 1980; Kopelman, 1987; Shallice, 1988; Shapiro, Alexander, Gardner and Mercer, 1981; Stuss, Alexander, Lieberman and Levine, 1978; Mercer, Wapner, Gardner and Benson, 1977; McCarthy and Warrington, 1990). The definition of what constitutes "a confabulation" also seems to have broadened, now often including certain characteristics of performance on traditional neuropsychological tests of memory (e.g. Kopelman, 1987; Delbecq-Derouesne et al, 1990) as well as autobiographical recall disturbances. These empirical investigations have allowed the emergence of some explanations of confabulation at the theoretical level,
which have clear implications for theories of normal memory as well as amnesia.

1.4(f)ii Paramnesic Disorders

McCarthy and Warrington (1990) are not alone in suggesting that confabulation is "can be considered a paramnesic disorder" (p. 313). Other investigators (eg Mayes, 1988; Benson, Gardner and Meadows, 1976) have suggested that confabulation should be seen as one form of paramnesia, alongside phenomena such as the Capgras syndrome, environmental or other reduplicative paramnesias and other rare exotica such as the Fregoli Syndrome. However the link between these disorders has been made primarily either on the basis of localisation issues (eg. Mayes, 1988) or on the fact that the patients who show these symptoms do not generally conform to the classical amnesic pattern (eg. McCarthy and Warrington, 1990). Categorising according either to association or (even worse) exclusion criteria is of course unsatisfactory and would hardly be acceptable if applied to phenomena from other areas of neuropsychological study. However the distinctions have been made for purely pragmatic reasons since, as Mayes (1988) puts it "the precise cause of these unusual reduplicative disorders is uncertain (p. 112). However, an understanding of the different mechanisms variously involved in these disorders is crucial since not all these disorders need exist in the same patient, and that most of the paramnesic/confabulating patients reported have also been those who have shown other characteristics of the "frontal amnesic syndrome" (see for instance Delbecq-Derouesne et al, 1990). The precise contribution of frontal lobe damage to the formation of these phenomena (particularly confabulation) will be the topic of Chapter 7 of this thesis.
Studies of memory problems secondary to frontal lobe involvement utilise three main methods of investigation. As we have seen, there are those studies which study a group of patients selected on the criterion of having a certain form of pathology which is thought to involve frontal structures. These patients are then tested on a particular paradigm, and then these results are compared with the performance of patients considered to have involvement of structures outside the frontal lobes.

A second general approach to studying frontal amnesia has been to concentrate on the phenomena which are held to be part of the frontal anmesic syndrome. As we have seen, the pathology of these patients is considered less important. However there is a third general approach to the issue of frontal lobe memory deficits which relies upon a number of logical deductions. This approach either takes patients with anterior as well as posterior damage and compares them with patients held to have only posterior damage (the subtraction methodology), or considers associations between memory test performance and tests which have been shown to be sensitive to frontal lobe dysfunction (the association methodology).

The value of these third forms of study rest critically upon the legitimacy of the reasoning behind the methodology. Whilst quite powerful there are a number of potential pitfalls to such approaches. Obviously the credibility of the conclusions reached by such studies rests largely upon successful negotiation of these pitfalls, and since this thesis will be using modified versions of both the subtraction and the association methods in later chapters, these pitfalls need to be examined closely.

1.5(a) Arguments against the Subtraction Methodology

It has become increasingly common for investigators to test two or more groups of patients, none of whom have
exclusively anterior cortical lesions, and use a subtraction-type methodology in an attempt to elucidate the nature of frontal impairments of memory. The argument is broadly that if there are a group of posterior patients (Group A) - usually bitemporals - and a group of patients with a more widespred disease (group B), but one which has been shown by pathology studies to generally affect the frontal lobes as well as other structures, then the effects that group B show that group A do not must be due to frontal involvement (see for instance studies by Janowsky et al, 1989; Leng and Parkin, 1988; Leng and Parkin, 1989; Parkin et al, 1988; Shoqeirat et al, 1990; Squire, 1982). There are a number of difficulties with this rationale, however.

1) Korsakoff patients are generally accepted to have bitemporal damage in addition to varying degress of frontal atrophy. However the nature of the bitemporal damage is unlikely to be equivalent to those whose bitemporal damage is due to vascular events of (rarely) surgery.

2) Korsäkoff patients have more widespread posterior damage than bitemporal vascular cases. The main lesions in non-alcoholic Korsakoff’s disease are found in the regions of the 3rd and 4th ventricle, and of the aqueduct. The mamillary bodies are most consistently involved and often show macroscopic petechial haemorrhages. Macroscopic haemorrhages are also occasionally seen in the grey matter around the 3rd ventricle and aqueduct, the inferior colliculi, parts of the medial thalamic nuclei and the region of the vestibular and dorsal vagal nuclei (Victor, Adams and Collins, 1971). In addition to varying degrees of lesioning in these areas, alcoholic Korsakoff patients may have additional, more widespread cortical atrophy. To make the issue yet more difficult, there is growing evidence for widespread neurotransmitter system disorder in Wernicke’s encephalopathy (Joyce, 1987), which makes interpretation of cognitive impairments in terms of local structural disorder very difficult indeed.
It is perfectly plausible that Korsakoff patients will have more widespread, diffuse damage than bitemporals. Thus interpreting characteristics shown by the Korsakoff patients but not shown by the bitemporal patients, (or vice-versa), as purely "frontal" symptoms may be misleading. It may merely be that they are more impaired due to more widespread lesioning. Even if one was to use a WAIS quotient in an attempt to co-vary for degree of general impairment this still does not solve the problem. The critical lesion sites in Korsakoffs that are causing the difference in memory performance still need not be anterior at all.

3) If the type of impairment in Korsakoff's syndrome that bitemporals do not show is due to frontal involvement, why cannot these effects be demonstrated in many studies of frontal lesion cases?

4) Some authors have used a "bitemporal group" which consists wholly or partially of patients whose amnesia is secondary to a herpes simplex encephalopathy (eg. Leng and Parkin, 1989). Other patients included in the "bitemporal group" are commonly PCAA cases. While PCAs may lead to reasonably localised posterior lesions (although not necessarily confined to the temporal lobes), acute necrotizing encephalitis leads, in varying degrees, to widespread and asymmetrical necrosis. Whilst this is noticeable (at post-mortem) particularly in the temporal lobes, other areas of the neocortex and the digitate white matter may also be affected. More specifically, areas of lesioning characteristically include the anterior parts of the parahippocampal, the fusiform and the inferior and middle temporal gyri and the posterior orbital cortex. It may also extend through the superior temporal gyrus to become continuous with necrosis in the insula. Additional areas include the hippocampus, the amygdaloid nucleus and inferior pole of the putamen, and the medial and lateral surfaces of the frontal lobe in the most severely affected hemisphere (Adams, 1969; Blackwood and Corsellis, 1976). Particularly important here is of course the frontal lobe.
involvement, which has been consistently demonstrated at post-mortem (McMenemy, 1966). As if this didn’t complicate matters enough, it is widely accepted amongst neuroradiologists that brain scanning is largely ineffective in the detection of neuropathology following encephalitis (Britton, 1983), which gives even less credence to the idea of including post-encephalitic cases in a "bitemporal" group, even if scan data were available (and in some studies it is not: eg. Leng and Parkin, 1989).

1.5(b) Arguments Against the Association Methodology

1) The difficulty with taking a group of Korsakoff patients and correlating memory performance and frontal lobe tasks, in an attempt to determine whether those with more evidence of 'frontal dysfunction' perform the tests more poorly is simply that this can be explained by the fact that their lesions are bigger. Other data suggests that some of the Korsakoffs used in these studies may be atypically good at some cognitive tasks. For instance, in the study by Leng and Parkin (1989), their Korsakoff group obtained a mean number of categories on the MWCST of 2.6, which accords well with Nelson’s figure (mean 2.92 sd 1.9) for patients with frontal lesions. However their bitemporal group managed a mean of 5.3 categories, which is considerably higher than Nelson’s unilateral posterior lesion group (mean cats 3.75 sd 1.9) and higher even than Nelson’s control group (mean cats 5.0 sd 1.64)! More convincing would be a dissociation methodology where some patients with evidence of frontal impairment as measured by tests considered sensitive to frontal lobe dysfunction show a particular pattern of performance which would include better as well as worse scores on certain measures. Clearly this is not possible, however, if we are to assume that which we are hypothesising to be "posterior" functions are equivalent in the two groups. If they are not, it is doubtful that they should be compared in the first place.
Most tests considered sensitive to frontal lobe dysfunction are very poor at detecting patients with frontal lesions (Shallice, 1982; see also Burgess and Alderman, 1990). This stems from the fact that most of the frontal lobe tests were developed experimentally, and therefore not concerned with accuracy of rates of detection - even of problem-solving deficits, let alone localisation. As one example, consider for instance Nelson's (1976) study using her modified version of the WCST. The difference between her posterior and anterior lesions groups in the number of categories they achieved failed to reach significance at the .05 level. She therefore recommends, for clinical purposes, calculating the number of perseverative errors as a percentage of the total errors. The cut-off she recommends is 50%. However, clearly if a patient only made 2 errors in the whole test (which would be a remarkably good performance) but one of them was perseverative, then their performance would be considered abnormal by one measure but not the other (i.e., categories achieved).

Attempting to use the number of categories achieved as predictive of a frontal lesion is equally hopeless. Thus if (according to Nelson's data) one were to use a 5%ile cut-off for the test, one would correctly classify 48% of frontally lesioned cases as having an anterior rather than posterior lesion, which seems satisfactory. However one would also classify 32% of cases with a posterior lesion as being frontal, which is hardly acceptable. Using a more stringent criteria of a 1%ile cut-off does not improves matters. Using this method, one would detect 36% of frontal cases as being frontal, and misclassify 18% of posterior lesioned cases as having a frontal lesion, which is marginally better than before. But what of a false negative rate of 64%? Thus the test does not really seem sensitive enough for most clinical applications.

Clinicians are fond of considering Milner's 128-card version of the WCST as more sensitive, but in fact Milner's data does not support this as regards the
frontal lobes as a whole. As she remarks in her 1964 paper, "the patients with lesions of the dorsolateral frontal cortex alone showed consistent impairment on the card sorting test" (p. 315). Her patients with lesions in other areas of the frontal cortex were not impaired on the test at all, and in fact were used for comparison purposes with the dorsolaterally-lesioned frontal patients in her analysis of the results. Additionally, her subjects were hardly representative of neurological patients as a whole, not only because of their pathology, but primarily because their average age was in the late twenties. Parkin (personal communication) has found that performance on the WCST is correlated with age, and so we cannot be sure what the results might be with a "typically" aged neurological group with a mean approximately twenty years older than Milner’s. Possibly, then, the best that can be said about the WCST is that it is sensitive to an unknown (and probably not very high) degree to lesions in one specific part of the frontal cortex. However which part is still not certain. This is supported by Robinson et al’s (1980) failure to replicate certain aspects of Milner’s findings with their frontal SOL case. As they put it "Milner (1963) examined the number of categories achieved on the WCST and found that none of her frontal cases were able to achieve more than three categories, whereas 74% of her nonfrontal cases achieved 4 or more categories. A chi-square performed on her data was highly significant. In contrast, when we attempted to replicate this part of Milner’s study, 43% of our frontal subjects achieved four or more categories and 39% of our nonfrontal subjects achieved fewer than four. The resulting chi-square was not significant" (p. 610). To confuse matters further, there is the question of laterality. Thus in Milner’s study the patients that performed most poorly were those left dorsolateral lesions, whereas in Nelson’s study the right frontals were worst. If this were not enough to suggest that the Wisconsin is unreliable, Drewe (1974) found significant differences between lesions groups and methods of scoring the test, with patient groups performing differently
according to whether one considered total test errors, perseverative errors or number of categories achieved!

These sorts of problems are not, however, restricted to the WCST. Other tests considered sensitive to frontal lobe dysfunction fare little better. Shallice (1982) reports an unpublished study of his, which he conducted in collaboration with Warrington and Oldfield. He reports that when he tested a group of patients with localised lesions on a battery of 10 tests which "were simplified versions of ones where from the literature one might expect a frontal deficit" (p. 202), including three tests developed by Luria, as well as Milner’s (1964) word fluency, the Stroop (Perret, 1974) and Shallice’s own Cognitive Estimates test (Shallice and Evans, 1978) only one of the tests (Weigl’s sorting task) "produced a significant effect of anterior-posterior location in the basic analysis" (p. 202).

This finding is particularly important, since the most commonly used "frontal test" other than the WCST is probably Verbal Fluency. There are a number of different versions of this test, but they all require the subject to generate as many words as they can, within a set period, which either belong to a particular semantic category, or else start with a specified letter.

The study probably used more than other to support the contention that this test is sensitive to frontal lobe lesions is Benton’s (1968) study. However he examined only patients with frontal lobe lesions and so this study does not help us understand the differential effects of lesion site on verbal fluency. Crockett et al (1986) did test posterior as well as frontal cases on the Verbal Fluency Test. They found that the procedure had greater predictive validity than did the WCST as regards the detection of frontal rather than non-frontal organic involvement. However the mean of their frontal group was at approximately the 30th %ile of their posterior group. This presents difficulties enough when deciding what cut-off criterion to use, but added to this is the fact that
whilst they found that verbal fluency performance was significantly correlated with WAIS-R FSIQ, (and had tested all their patients on this measure) they did not use this as a covariate in their analyses. Thus we cannot know that this small difference is not due to general intellectual decline in their mixed pathology frontal group.

Miller (1984) did however take this variable into account. He found that left and right frontals produced significantly fewer words than right or left posteriors. But since he also found that performance on this test was significantly correlated with IQ (but not age), he presented a regression equation for predicting verbal fluency from WAIS verbal subtest scores \( W = 1.46(T) - 11.73 \) \( SE = 7.18 \), where \( W \) = total of letter FAS with 60 secs each, and \( T \) = total of scaled scores for subtests Similarites, Vocabulary and Comprehension of the WAIS). This eminently sensible idea does however serve to show up the weaknesses inherent in this task. Thus, using Miller’s equation, a normal subject whose current IQ is at the bottom of the Dull Normal range, and is the mean age of Miller’s controls, would be expected to produce a total of approximately 16 words total (ie. an average of 5 or so words for each letter in 60 seconds). The standard error for Millers equation is however 7.18, which means that a subject must perform extremely badly indeed on this task before one can be sure to any degree that the performance is in any way abnormal, let alone that it is a "frontal" as opposed to "posterior" performance. At lower IQs the test becomes completely meaningless (a Borderline IQ, 45 year-old normal subject would be expected to achieve an average of less than three words per minute!). Other peculiarities that need explaining exist across studies with this test, such as the fact that whilst Crockett et al and Miller both found right frontals impaired on verbal fluency, Milner (1964) did not. Another is that the right frontals in Benton’s (1968) study actually performed as well as Miller’s controls - the procedures were identical - which suggests that, given the considerable spread of scores obtained by
normals on this task, these studies would benefit from larger subject groups.

The data for other procedures supposedly sensitive to frontal lobe dysfunction is even less convincing, in terms of their predictive utility (eg. Benton, 1968; Chorover and Cole, 1966; Cicerone, Lazar and Shapiro, 1983; Halstead, 1940; Perret, 1974; Petrides, 1985; Petrides and Milner, 1982; Semmes et al, 1963; Luria, 1966). The point that often seems to be missed is that the criteria used in determining localisation of function when a task is developed experimentally are not necessarily the same when the test is used clinically or as part of the association methodology. The latter two applications require considerably greater discriminative power.

So, then, while the association methodology may in fact be useful, its utility hinges critically upon the accuracy of the tests used, especially since they are only being used in terms of localising significance rather than in terms of the processes that they measure (eg. rule detection etc.). Since existing "frontal tests" are generally inaccurate, and performance has to be considered in the light of a number of other variables such as age, current (and probably) pre-morbid level of general intellectual functioning, it is of doubtful value merely to find a correlation between one test and another and therefore assume that inferences can be made without testing the underlying dynamics of your measures. Nevertheless, if these factors were to be addressed, then the association methodology clearly may have something to say. It is with this in mind that the tests described in Chapters 2 and 3 were developed, and this issue will be addressed further in those parts of this thesis.

1.6 Theories of frontal lobe amnesia

So far, this treatise has concentrated on the nature of the empirical evidence for or against the existence of a
frontal amnesia syndrome. As we have seen, the quality of the evidence is not good in a number of areas. Others look potentially interesting, but need further investigation, and it is in these areas that it is hoped that the present thesis will make a contribution. The next section aims to explore not the quality of the empirical evidence per se, but the way in which that evidence has been used in support of the different theories regarding the role of the frontal lobes in memory, and how well those theories account for these data. Many of these theories provide starting points for the model to be presented in chapter 7.

Fully developed theories of frontal lobe amnesia are rare, and those that do exist are necessarily bound up with theories of amnesia generally. Nevertheless there are a few which are detailed enough to warrant close examination, and these generally fall into one of four categories, although often the theories are not mutually exclusive:

1) The spatiotemporal context theory  
2) The strategic recall theories.  
3) The "partial amnesia" explanation  
4) The "generally frontal " explanation

Since there are a number of exponents of each, each with slight variations over another, no attempt will be made to cover every writer’s theory. Instead the leading exponent of each type of theory will be examined in detail as an example of that "type" of explanation. The choice does not necessarily reflect the originality of that writer’s opinion, but merely the fullness of their argument.
1.6(a) The Strategic Recall Hypothesis

Moscovitch (1989)

Moscovitch believes that, in patients with frontal lobe lesions (FLL), while retention may be also impaired, the main focus of his theory is with retrieval. He distinguishes between 2 types or components of retrieval - strategic/organisational and associative. The strategic retrieval process is largely self-initiated and goal oriented (Craik, 1983) and is/are concerned with "reinstating the temporal and spatial context in which the target is embedded and, having reinstated it, they co-ordinate the various other retrieval processes that utilise general knowledge as well as episodic cues to home in on the target".

Moscovitch alikens this process to problem-solving, where relevant knowledge is recruited to analyse the nature of a problem, and once the way in which the problem is to be solved is found, then lower-level routines are employed to actually perform the calculations or "local routines". Moscovitch argues that the associative retrieval process is analogous to these lower-level routines.

Associative recall does not always follow strategic recall - the two processes are interactive, but independent. In Moscovitch’s view, patients who confabulate and show relatively preserved recognition have damage to the strategic retrieval process which is frontally located. In practice this means that the confabulating patient will not be able to retrieve episodic memories if given the task of finding one (as for instance, by using the Crovitz technique), but if given a clue which happens to have a strong association with an event memory, some details may be retrieved. Moscovitch contends that this sort of deficit probably holds for this semantic memory as well. From the point of
view of the present study, Moscovitch makes a number of important points:

1) Crucial to correct retrieval of an episodic event is a system which can make appropriate encoding and retrieval information available to the hippocampus and can organise and evaluate the ecphoric information that is its output. Moscovitch contends that these control processes are mediated by the frontal lobes and its related structures.

2) Moscovitch makes the point that memory is often fragmented, and that one aspect of an event may be remembered separately from another. Different retrieval information may be necessary to retrieve one aspect of the trace than another, suggesting that the information may also have been encoded differently. If competing information arises, then various deductive or inferential processes must be used in order to decide between them.

3) In the confabulating patient, the system which combines and organises the available information into a "memory" (and which is held anteriorly in the cortex), is damaged. Thus the confabulating patient "...haphazardly combines information from disparate events, jumbles their sequence, and essentially accepts as veridical whatever the ecphoric process delivers to consciousness" (p. 155).

4) In the case of fantastic confabulation (Berlyne, 1972), the output reflects "...recent thoughts, perceptions or fantasises rather than relevant past experiences" (p. 155). In this there is an echo of Delbecq-Derouesne et al’s contention that confabulation arises as a result of an inability to distinguish between associations arising from a stimulus and actual past memories.

Thus Moscovitch contends that the frontal lobes are crucial to correct episodic recall, although their role
is essentially that or organising the output of more posterior structures. He explains that patients who confabulate and whose recall is poor but recognition is unimpaired have a deficit in the strategic recall system, whereas the associative recall system presumably mediates recognition memory. One problem for this view is Delbecq-Derouesne et al’s (1990) patient who confabulated, but showed preserved recall and impaired recognition. If Moscovitch’s hypothesis is correct, this patient’s strategic recall system must have been intact for him to be able to be near normal on free recall tests, and it was his associative recall system which must have been damaged. This is of course the opposite of what Moscovitch contends.

There are however many attractive aspects to Moscovitch’s theory, which stem largely from his examination of the phenomenon of confabulation. His theory has been developed with this as the main focus of attention, rather than empirical data culled from the procedures of experimental psychology. This is an approach which will be examined in the latter half of this thesis.

1.6(b) The Spatio-temporal Context Hypothesis

Schacter (1987)

Schacter’s theory regarding the role of the frontal lobes is broadly similar to many others (eg. Fuster, 1980; Pribram and Tubbs, 1967; Tubbs, 1969) and is the clearest example of the view that the frontal lobes are not involved in event memory, but are involved in what might be termed "event context". Schacter’s view is that frontal lobe lesions lead to deficits on those tasks which require "spatiotemporal" abilities. The term spatiotemporal seems to be an amalgam term which refers to both memory for spatial relations or locations and memory for the temporal order of events. Thus for Schacter, frontal lobe dysfunction does not lead to a pure amnesia syndrome, but in amnesic patients who also
have frontal lobe dysfunction, one can also find deficits in spatiotemporal relations. He sums up his review of the empirical literature thus:

1. Patients with restricted frontal lobe lesions exhibit deficits in remembering certain kinds of contextual or spatiotemporal information

2. Similar deficits have been observed in studies of nonhuman primates with frontal lesions, which may have shown that memory for spatiotemporal information is handled by specific and restricted areas of the prefrontal cortex.

3. Encoding of spatiotemporal information in humans can be accomplished relatively automatically (p.31)

From this, Schacter concludes that:

A) Amnesic patients with additional frontal-related memory problems represent a type of amnesic syndrome that differs often quantitatively from that established by patients who are entirely free of frontal signs but is equally central to our understanding of memory disorders.

B) Patients showing this form of amnesia may provide insights into the nature of normal memory function that are different from those provided by the study of so-called "pure" amnesic patients.

Up to this point there is little new in Schacter’s views, but where he departs from some other investigators is that he theorises about the causes of these spatiotemporal deficits. In agreement with Fuster (1980), Milner (1982), Pribram and Tubbs (1967) and Teuber (1969), he argues that the frontal regions "play a major role in the segmentation and organisation of ongoing experience into distinctive units that are discriminable from one another". This process he calls "contextual chunking". Schacter argues that "if an organism were unable to organise experience into temporally distinctive
chunks, then it would prove extremely difficult for it to
discriminate amongst recent experiences with respect...to
time and space. It would also be difficult for such an
organism to organise recent events sequentially, because
the component events would not be clearly differentiated"
(p. 32).

The obvious drawback with Schacter's view is that many
patients with cortical lesions apparently restricted to
the frontal lobes are no different in their presentation
than patients with posterior lesions (as discussed in the
section on space-occupying lesions above). As we have
seen, the empirical evidence for "spatiotemporal"
deficits in frontal lobe patients is not particularly
convincing, although his theory is more clearly
applicable to confabulation.

However, the question of confabulation raises a point
that will be examined further in the next section. This
is the fact that all cases of confabulation reported so
far (and where formal testing has been carried out) have
shown some degree of amnesia. Not only this, but the
severity of a classical amnesic's memory problem may
prevent proper analysis of their spatiotemporal
abilities. Clearly in order to "discriminate amongst
recent experiences with respect ...to time and space",
one first has to be able to remember something of those
experiences! Schacter therefore needs to explain the
relationship between his spatiotemporal hypothesis and
normal amnesia.

1.6(c) The "Partial Amnesia" Hypothesis

Warrington and her colleagues (Warrington, 1979;
Warington and Weiskrantz, 1982; McCarthy and Warrington,
1990) characterise amnesia as a loss of 'event memory'
which "can be documented and quantified using the
classical techniques of verbal learning experiments. Free
recall of word lists, paired associate learning, yes/no
recognition and forced choice recognition are all equally
and markedly impaired in patients with the amnesic syndrome." (1979, p. 163). For Warrington, amnesia is an "associative retrieval deficit" (1979, p. 163) which is due to a disconnection between a "cognitive mediational memory system" and a semantic memory system. The cognitive mediational system is held in the frontal lobes, the semantic system in the temporal lobes and they are disconnected by pathways in the fornix-mammillary body route (Warrington and Weiskrantz, 1982).

The term "cognitive mediation" refers to "cognitive elaboration, use of imagery, embellishment manipulation and organisation". The ability to perform these operations is not impaired "as such", but the amnesic is impaired "in those memory tasks in which the stored benefits of mediation are normally important" (1982 p. 242). The cognitive mediational memory system itself is "a dynamic...system, in which memoranda can be manipulated, inter-related and stored in a continually changing record of events" (p. 242). More specifically, they speculate that the hippocampus may be the structure in which inputs from the temporal lobe semantic systems and the frontal-lobe cognitive control mechanisms interact, and from which storage in the cognitive mediational memory system is initiated (p. 243). The idea was that if these two systems were disconnected, there could be no meaningful input into the mediational system - "it would be contentless, and indeed this is how the densely amnesic subject often describes his everyday memory".

At this stage, Warrington and her colleagues' theorising was aimed at giving an account of classical rather than frontal amnesia. Later this theory was expanded to include frontal amnesia. In McCarthy and Warrington (1990), the topic of frontal amnesia is not discussed as such, but the authors do discuss those phenomena which are considered to be symptomatic of frontal amnesia. Thus they describe paramnesia as a "subtype" of amnesia, which is qualitatively different from pure amnesia (p. 297) and suggest that confabulation can be "distinguished from
classical amnesia in that the patient may produce "memories" which have no basis in 'actual' events or occurrences." (p. 312-3). Confabulation, for McCarthy and Warrington, is "by no means a characteristic of a selective amnesia. Indeed, confabulation may arise in patients whose performance on standard tests of anterograde memory and learning is within the normal range..." (p. 313). In this way, McCarthy and Warrington describe these memory phenomena as not a part of classical amnesia whilst avoiding the need for an extended account of a specific non-classical syndrome. However later in the chapter they unequivocally ascribe paramnesic impairments (including confabulation) to damage to "those medial aspects of the frontal lobes which are closely linked anatomically to the limbic system and basal forebrain nuclei" (p. 318).

At the theoretical level, McCarthy and Warrington regard paramnesic phenomena to be a consequence of dysfunction of the cognitive mediational memory system. They argue that "the frontal memory impairment can perhaps be thought of as a failure of a system which is required to organise the search, modulation, editing and reconstruction of memories. In such cases, unlike in the pure global amnesic patient, cognitive mediational memory processing is itself disorganised or even degraded" (p. 327). What is not clear at this point is whether this system differs from the system mentioned earlier, which is "a dynamic...system, in which memoranda can be manipulated, inter-related and stored in a continually changing record of events (Warrington and Weiskrantz, 1982 p. 242). In many ways there are similarities to Shallice’s (1988) account in which confabulation and other paramnesic phenomena represent a failure of a system which is "concerned with formulating the description of any memories that might be required and of verifying that any candidate memories that have been retrieved are relevant" (1988 p. 378).

McCarthy and Warrington suggest that in the patient with a frontal memory impairment "unlike the pure global
amnesic patient" the "cognitive mediational memory system is iteslf disorganized or even degraded" (1990, p. 327). In contrast, the "pure global amnesic patient" has had a system (held frontally) disconnected. This is a system which is a "a dynamic system...in which memoranda can be manipulated, inter-related and stored in a continually changing record of events". So either there are two systems (the one defined in Warrington and Weiskrantz, 1982 and the one in McCarthy and Warrington, 1990) held frontally, one of which is disconnected in pure amnesia and the other which is degraded in frontal amnesia, or McCarthy and Warrington’s description is an expansion of the original specification. Certainly p. 327 seems to indicate the latter.

So then, for McCarthy and Warrington there is a system held frontally which is involved in the manipulation, "cross-referencing" and storage of memories of events, and also the search, modulation, editing and reconstruction of those memories for recall. Thus we have a system which operates at storage and retrieval (as agreed by McCarthy and Warrington on page 328). Implicit also in their model is the fact that autobiographical memories are held as "memories of events". An alternative view will be explored in chapter 7, but firstly there are some interesting points raised by Warrington and colleagues’ (especially McCarthy and Warrington, 1990) theory as it stands at present:

1) If memory storage and retrieval happens in the frontal lobes then why do not even massive frontal lobe lesions lead to amnesia (according to McCarthy and Warrington, 1990)?

2) If memory storage and retrieval (as above) is in the frontal lobes, then why do cortical lesions elsewhere lead to poor performance on memory tasks (even if the lesion is remote from the mamilliary-thalamic tract).

3) Much of the past studies of Warrington and colleagues have concerned themselves with demonstrating that it is
the method of testing retention that is crucial to amnesic’s recall, not the method of acquisition 
(Warrington and Weiskrantz, 1970; McCarthy and Warrington, 1990 p. 301). If the Cognitive Mediational Memory System is involved in storage, why is method of acquisition not an important factor?

4) The relationship between semantic memory and episodic memory is, in common with other theorists, left largely unexplained. This may be due to the difficulty with negotiating the inter-relationship between the process of recall and the state of knowledge. However, McCarthy and Warrington provide some of the best evidence available for this relationship. On page 322, they highlight the fact that memory for words can be severely impaired in the context of normal autobiographical memory functions as evidence against the argument that spared word retrieval (comprehension) in amnesia can be due to memory for words being "stronger" than memory for events¹. Later on in the paper they cite their patient RFR who was amnesic for periods in his life where he had acquired certain word meanings. The problem that such a patient presents for theories of amnesia is of course that the episodes in which RFR would have heard the word "AIDS", for instance, would form a part of an episodic record. If you present a normal subject with the word "AIDS" in a Crovitz-type paradigm, they may well be able to use this to recall an event in their life in which the word AIDS was used. Thus that component of the episodic record is recalled. If this were true of every component in that episodic record (and logically there is no reason why it shouldn’t be) why is RFR amnesic for the event? If he can recall all the individual features of the event, why cannot he remember it as a whole? One explanation at the retrieval level might be that the memory for the

¹ Given that the memory for the word was stronger, then if one were to have lost knowledge of the meaning of a word or "couldn’t remember it" in naming tasks, then one would necessarily need to be amnesic - since strong memories should be more resistant than weak (or episodic) memories. Thus McCarthy and Warrington conclude "...word knowledge and the ability to retrieve knowledge of events do not lie on a continuum".
individual items in the event might be stronger than the collective summation of all those items (as you may get assuming certain limited-capacity parameters to an episodic record). Another explanation at the storage level might be that the word will only come to be remembered with repetition (ie. trace strengths). But McCarthy and Warrington have already rejected trace strength (and therefore both possibilities) as an explanation. One way of dealing with this problem will presented in Chapter 7 of this thesis.

Returning to the application of the model to frontal amnesic syndromes, McCarthy and Warrington seem to be suggesting that those who confabulate and/or are paramnesic are suffering from what might be termed a "partial amnesia". That is to say, rather than the cognitive mediational memory system being completely disconnected, it is disorganised or degraded. This would be supported by the fact that there does often seem to be inter-test differences in frontal amnesics (eg. Delbecq-Derouesne et al, 1990; Moscovitch, 1989; Kapur and Coughlan, 1980)

However there is some evidence that not all amnesics are "equally and markedly impaired" on memory test. Warrington and Weiskrantz (1970) tested a group of amnesics for retention of an 8-word list under 4 different conditions (free recall, yes/no recognition, initial letters of the word - cued recall, fragmented form of the words - cued recall). Warrington and Weiskrantz report that the results:

"...provide the only firm evidence that the normal relationship between recall and recognition does not hold in amnesic patients. Though both recall and recognition were impaired in the amnesic group compared with the control subjects, retention tested by recognition was significantly better than recall in the control group but not in the amnesic group. Furthermore cued recall, either by the first three letters or by fragmented words, was not impaired in the amnesic
group, the small differences in performance between the experimental group and the control group on cued recall with initial letters not approaching significance. In no condition were the findings confounded by 'floor' or 'ceiling' effects" (Warrington, 1976).

The basis for McCarthy and Warrington's argument that paramnesic patients have a "degraded" rather than "disconnected" cognitive mediational memory system (CMMS) seems to be that "...confabulation may arise in patients where performance on standard tests of anterograde memory and learning is within the normal range...for these reasons confabulation can be considered a paramnesic disorder rather than a variant of a severe form of amnesia". In support of this argument they quote papers by Stuss et al (1978) and Kapur and Coughlan (1980). However it is not clear that these patients' memory test performances were indeed normal. The Stuss et al paper reports 5 cases (all confabulators) who had been referred to their department because of memory or behavioural problems. Stuss et al comment that "memory functions were sensitive to interference and a deficit in retrieval was prominent. A moderate-to-dense retrograde amnesia existed in all five subjects". Although in two patients Stuss et al suggest that the degree of amnesia was fairly mild (as measured by the WMS - both with MQs approximately one standard deviation below what they should be), there is no formal data on how they performed on the other two memory tests given them. However they conclude that "in that small subgroup of patients showing persistent, and spectacular and impulsive confabulation, it seems that, in addition to amnesia regardless of its etiology, a necessary deficit is frontal dysfunction" (p. 1171). The Kapur and Coughlan (1980) patient showed "poor memory and confabulation", and his recall of a short story was "nonexistant after an hours delay" (p. 462). Certainly his performance on Warrington's RMT was normal, but four weeks later his recall was still "slightly weak". Twelve weeks later, when the patient no longer showed spontaneous confabulation but his wife still complained that his "memory was still poor...He would
forget things she had told him several minutes earlier" (p. 462). On a story recall test, his immediate recall was good (as before) but "nonexistent...after an hour" and his RMT scores were 40/50 for words and 39/50 for faces. Both these scores are within the defective range for a man of his age. Whilst his paired associate learning and word list recall was only a little weak, his complex figure delayed recall was "markedly impaired". As Kapur and Coughlan put it "although our patients memory was certainly impaired, he was not globally amnesic".

The point is that whilst confabulation can certainly occur in the context of a relatively mild amnesia, there is no evidence that it can occur in patients whose memory performance on all memory tests is within the normal range. Some degree of memory problem is evident in every reported case of a patient who confabulates (as one might expect by definition). In some patients the amnesia can be very severe indeed (eg. Patient LE reported by Shallice et al, 1989). This is a point needing greater expansation since it is McCarthy and Warrington’s contention that paramnesic patients are not severely amnesic - which is the basis of the CMMS explanation of confabulation.

If McCarthy and Warrington are then correct about confabulation being due to a degraded CMMS, whereas a pure amnesic has had his/her CMMS completely disconnected, then the confabulator who is also densely amnesic must have a disconnected and degraded system. What is unclear is what additional effects would be seen when the system is degraded once it has already been disconnected.

An additional question that this raises is the question as to where exactly it is that the "memories" that amnesic confabulators produce, come from. This is an important question that will be considered in Chapter 7 of this thesis. For the moment, however, Warrington and colleagues’ theory presents the most clearly articlated view of the relationship between clasical and frontal
1.6(d) The "generally frontal" explanation

The clearest exponents of this view are Baddeley and Wilson (1986, 1988). They use as a starting point Shallice's (1982) development of the Norman and Shallice (1980) information processing model. However they come to rather different conclusions than Shallice (1988). Broadly speaking, it is their view that frontal amnesic symptoms are one manifestation of a much broader "dysexecutive syndrome" (Baddeley and Wilson 1986). The great advantage of their theory, from the start, is that they unequivocally equate amnesia with problems in autobiographical recall (and/or "recollection"), rather than providing a definition of amnesia in terms of performance on experimental tests of memory. But, as we shall see, the disadvantage is that on occasion the processes of problem solving and autobiographical recall are blurred together, without full explanation of how they might be related.

Their 1988 paper focusses on their patient RJ, who had suffered a severe head injury six months prior to their study. A CT showed haemorrhages in both frontal lobes.

By any (traditional neuropsychological test) standards, RJ was suffering from a severe amnesia. His performance was at chance on both the visual and verbal parts of Warrington's RMT, his recall of the WMS story was well below the 1st percentile for his age, as was his complex figure recall, and he achieved a score of 0/12 on the RBMT, which is really very impaired indeed. On a range of additional experimental memory tests (paired associates, jigsaw learning, reading transformed script) he fared no

2 Shallice's views on frontal lobe amnesia represent the starting point for the theory to be developed later in this thesis. It will therefore be discussed at that point.
better. He did however show good learning on the pursuit rotor task.

However RJ would not qualify as a pure amnesic as defined by Shallice (1988) or McCarthy and Warrington (1990) since his level of intellectual functioning (WAIS VIQ = 100, PIQ = 76) was considerably below his estimated premorbid IQ (NART FSIQ equivalent = 120). He also had a few other minor neuropsychological impairments with perceptual functioning, together with poor performance on the Mill Hill Vocabulary Scale and on Baddeley’s test of semantic processing.

Baddeley and Wilson contend that RJ showed a classical amnesic pattern in that he showed normal digit span and performance on anterograde memory tests. However, as Baddeley and Wilson point out, he did not show the classical amnesic pattern in that, for the most part, his performance on tests of procedural learning were not normal. Additionally, he showed variable patterns of primacy and recency on a test of free recall of word lists and his speed of "semantic processing" was not normal. He also confabulated freely, and his WCST (Milner, 1964), Cognitive Estimates (Shallice and Evans, 1978) and Verbal Fluency (Milner, 1964) performance was very poor.

Baddeley and Wilson (1988) present three possible explanations for RJ’s pattern of performance:

1) RJ’s memory problems are secondary to his attentional and motivation problems.

2) "...frontal amnesia constitutes a primary memory deficit, but one which is qualitatively different from the more classic amnesic syndrome" (P. 225).

3) "RJ is suffering from a combination of a classic amnesic memory deficit coupled with an overlay of problems induced by his dysexecutive syndrome" (p. 225).
They feel that the first argument is probably insufficient since RJ showed generalised poor memory test performance, and argue that if his difficulties stemmed from motivational or attentional difficulties his performance would be much more variable. The second interpretation is rejected on the basis of an argument that is less straightforward. They argue that since there has not been, as yet, a convincing demonstration that different types of amnesia exist (although they do accept that ECT-induced amnesia is different from classical amnesia) they cannot interpret RJ's amnesia as evidence for a different form of amnesia.

The third possibility is the one they explore most thoroughly, arguing that the pattern of performance that RJ shows on memory tests where he does not show a classical amnesic pattern is due to his dysexecutive problems. They explain his confabulation in the same light, suggesting that autobiographical recall is "a highly demanding and unstructured task" and his confabulations can be explained as "a classic dysexecutive deficit in attentional control coupled with a clouding of autobiographical memory...[that] also occurs in nonfrontal amnesics" (p. 227). Thus they conclude that RJ's amnesia is "consistent with the assumption that frontal amnesia consists of a combination of a classic pattern of amnesic deficit coupled with the secondary problems that might be expected to arise from the frontal dysexecutive syndrome" (p. 228).

However, if indeed confabulation is caused by a dysexecutive syndrome overlaid on a memory problem why then do patients exist who have poor memory and confabulate, but who are not dysexecutive - as defined by Baddeley and Wilson (eg. patient CA reported by Della Barba et al, 1990)? Other patients may have poor memory and be dysexecutive, but not confabulate (see case SJ reported by Burgess and Alderman, 1990).

One possibility that Baddeley and Wilson do not highlight at this point (although Baddeley (1988) hints at it
later) might be that the "frontal syndrome" can affect problem-solving type tasks. It can also affect memory. It may do so independently of one another, although the location of the necessary lesion makes it likely that they will often be affected at the same time. Nevertheless they are still associated deficits. When the patients have a dysexecutive syndrome affecting memory, they show "frontal amnesic" signs. When they have a dysexecutive syndrome affecting problem-solving, they show frontal problem-solving deficits. Very probably there are problem-solving components in memory just as there are memory components to problem-solving tasks. This is an argument that will be developed later in this thesis.

1.7 Conclusion

As pointed out by Mayes (1988 p. 119) and Stuss and Benson (1986), much of the existing empirical evidence for the role of the frontal lobes in memory is equivocal. In as much as current theories of frontal lobe memory functions are based on this empirical work, most of these can only be tentative. Nevertheless virtually all experimenters agree on a number of important points, which are perhaps more telling than their disagreements.

The first point of agreement relates to the memory test performance of patients with frontal lobe dysfunction. Most experimenters agree that some frontal lobe patients may perform memory tests poorly, but that in the main their performance is not as severe as those seen in the severe classical amnesia syndrome.

They also agree that frontal lobe patients may show executive problems which may not be seen in classically amnesic patients. Another point of agreement is that unusual paramnesic phenomena most often occur in patients who have signs of frontal lobe damage, and that this type of amnesia is not identical to the pure classical syndrome.
Most importantly, all investigators who have put forward a comprehensive theory of the role of the frontal lobes in memory agree that there is some structure or process damaged in patients who show frontal amnesic signs that is not damaged in pure classical amnesics. This structure or process serves a supervisory or executive function.

This thesis aims to define the relationship between executive functions and episodic memory, and (in doing so) to present a theory of the role of the frontal lobes in memory. All three of the investigative methods outlined above will be used, and most of the issues already raised will be considered.

Reflecting current investigation into frontal lobe memory problems (and like this introduction), the thesis falls into three main parts. The first section takes an experimental approach. Patients with isolated frontal lobe lesions will be tested on experimental memory paradigms aiming to answer the question of whether lesions in different parts of the brain produce different memory test performances (this is one of the issues that Mayes [1988] recommends as of key importance). To varying degrees, most of the issues dealt with in the earlier part of the introduction (release from PI; temporal sequencing; recall-recognition discrepancies;) will be examined, hopefully in such a way as to avoid the methodological flaws already outlined. In particular the aim is to answer the two questions which were left unresolved after the review of the experimental literature: a) Do frontals behave like posteriors on memory tests? b) What is the relationship between memory test performance and executive functions? The association and dissociation methodology will be used in attempting to discover whether dysexecutive patients necessarily fail memory tests (one of the points highlighted by Baddeley and Wilson [1986] as being of key importance). This requires first the development of new tests of executive function that are more sensitive than existing measures. The last part of the experimental section will
hopefully demonstrate that there are aspects of memory in which executive functions play an important role, and which have not yet be considered by mainstream neuropsychology.

The last section of the thesis will present the development of an empirically-based theory of the role of the frontal lobes in memory. Ideally, the aim is firstly for the theory to explain the experimental findings presented in the previous two sections of the thesis, and secondly for the theory to account for the role of the frontal lobes in memory more thoroughly than the existing theories.
2.1 Introduction

A considerable amount of research has been conducted into the cognitive effects of frontal lobe dysfunction, and there are now some features which have gained general acceptance (see Stuss and Benson, 1986). The most common of these are: impairments in establishing, changing and maintaining a set (Stuss and Benson, 1984); decreased monitoring of behaviour (Cicerone and Lazar, 1983; Stuss and Benson, 1986); rule-breaking behaviour (Canavan, 1983; Milner, 1964); impulsivity of responding (Miller, 1985); disorders of response preparation and inhibition (Perret, 1974; Verfaellie and Heilman, 1987); and problems in the maintenance and direction of attention (Salmaso and Denes, 1982; Wilkins, Shallice and McCarthy, 1987).

Experimentally, there are a number of concept-formation tasks that are traditionally held to be sensitive to frontal lobe dysfunction (see Wang, 1987 for a review). The problems that patients have with these tasks are generally twofold. Firstly they have a problem with initial set formation, and secondly they show a tendency to repeat a response when repetition of that response is not appropriate - ie. perseveration (eg. Nelson, 1976). However the tests are usually designed in such a way that it is difficult to understand the exact nature of the failure.

Consider, for example, a perseverative error on the Modified Wisconsin Card Sorting Test (MWCST) - generally considered perhaps the most cardinal feature of frontal lobe dysfunction (Goldberg, 1975, 1986; Goldberg and Tucker, 1979; Sandson and Albert, 1984). Any one of the problems listed above serves as a possible explanation of that error. For instance, decreased monitoring of one’s behaviour can lead to poor awareness of negative outcome,
which in turn may be one explanation of the cognitive basis of perseveration. Alternatively, it might be that the patient has a problem with response inhibition (the most common explanation of perseveration).

However a more interesting possibility exists which follows from the work of Miller (1985) which casts doubt as to whether these sorts of errors on the MWCST are perseverative at all.

Miller (1985) gave patients who had undergone temporal- or frontal-lobectomy a series of drawings of simple objects, which in each case a large proportion of the figure had been deleted. The patients were shown in turn different details from the same object, so that it was possible to build up "in the mind" a coherent whole, and thus identify the object. Miller found that whilst temporal and frontal-lobe excisioned patients were broadly similar in terms of their "visual synthesizing" impairment (ie. their ability to work out what the object was), their performance differed in two important ways.

Firstly the incorrect guesses were examined in order to determine whether there were differences between groups in the ability to make use of the information available in the clues. Judges were asked to decide whether the incorrect responses were possible target items, given the information available. The frontal-lobe group made more errors where the judges said that they were unable to imagine the guessed item as being constructed from the visual fragments.

Secondly, Miller found that the frontal-lobe patients were significantly more willing to make a guess as to what the answer might be, before they really knew. They would hazard a guess on the basis of less information than the posteriors. Miller characterised this as "cognitive risk-taking", and suggested that the test was an experimental measure of impulsivity.
Consider for a moment what the MWCST performance of such a patient would look like. Assume for the moment that the only pathological characteristic that this patient has is an exaggerated tendency to guess. And, following Miller, that this guess need not be particularly understandable (ie. is bizarre).

In the modified version of the WCST, in the vast majority of cases, any response to any trial is interpretable in terms of one of the criteria which is applied in the test. Thus "guessing" is unlikely to be detected (Drewe, 1974; VilkKi, 1980). If one were in fact to guess in a pseudo-random way, the apparent effect would be that approximately a third of all responses would appear to be "perseverations". If the subject were (additionally) only able to "get" two of the three possible criteria (colour and shape but not number, for instance) this number would rise to approximately 50%. This is of course exactly the criteria that Nelson (1976) suggests as the greatest discriminator of frontals from posteriors (ie. 50% of all errors being repetitions of the last response).

The same point can be made about other tests generally considered sensitive to frontal lobe dysfunction. Thus, for instance, in the Butters et al (1972) study, which used Money’s Road-Map Test, the patient has the choice of only two responses ("left" or "right") - this is also the case for the Chorover and Cole (1966) tests, and Semmes et al’s (19 ) Personal Orientation Test has only two dimensions on which the subject can fail.

In summary then, it seems that most concept-formation-type tasks that have been shown to be sensitive to frontal dysfunction have the characteristic where any response made by the subject can be interpretable in terms of that test. This means that "guessing" or a tendency toward bizarre or impulsive responding cannot be detected, yet this might provide an explanation of frontals’ performance on these tasks which is as equally compelling as those traditionally given. Clearly what is required is a task designed in such a way that the types
of errors that would arise from "cognitive risk-taking", perseveration, or concept-formation/detection difficulties would be separable.

2.2

METHOD

2.2(a) Subjects

A series of 126 patients referred to the Dept. of Clinical Neuropsychology at the National Hospital for routine neuropsychological assessment, were seen as part of the present study. Patients were then excluded if they did not fit all the following criteria:

1. Right-hand dominant (as assessed by admitting neurologist) and aged between 18 and 75.
2. Unilateral lesions restricted to one or two lobes (with the exception of bilateral frontal lesions with no posterior involvement, who were considered as a separate group).
3. No history of psychiatric problems, alcohol or other substance abuse, or previous neurological conditions.
4. No hydrocephalus or long-standing epilepsy.
5. First language English.
6. Capable of, and willing to undertake experimental procedure in addition to full neuropsychological assessment.

Forty-nine of the original 126 patients failed to meet these criteria and were excluded, leaving 77 patients. The remaining patients were classified according to site of lesion based on the radiographer's report of the CT scan. Any patient who had involvement of the frontal lobes was classified as "anterior", and those patients who had involvement elsewhere in the cortex, but not involving the frontal lobes, was classified as "posterior". Patients who had bilateral frontal lobe lesions with no posterior (ie. non-frontal) involvement
were considered separately as a "bifrontal" group. The ages of these patients, together with their, pre-morbid IQ estimates based on the NART, and current levels of intellectual functioning are shown in Table 5. There were no statistically significant differences between the groups. 3 bilateral frontals, 4 right and 4 left frontals and 6 left temporals included in this series were given the WAIS-R. To allow for comparison with the WAIS scores of the majority, these patients' scores were corrected by +8 points as recommended by Crawford et al (1990).

No significant laterality effects emerged at any point in the analyses, so the unilateral lesion groups were collapsed, yielding the groups shown in the bottom segment of Table 5.

<table>
<thead>
<tr>
<th>N</th>
<th>GROUP</th>
<th>AGE</th>
<th>WAIS</th>
<th>FSIQ</th>
<th>NART1</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>RP</td>
<td>48.5 (14.1)</td>
<td>108.8 (9.1)</td>
<td>111.5 (8.3)</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>LP</td>
<td>36.6 (14.3)</td>
<td>103.0 (17.4)</td>
<td>111.5 (11.7)</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>RA</td>
<td>48.7 (13.9)</td>
<td>108.4 (14.0)</td>
<td>114.3 (9.6)</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>LA</td>
<td>41.5 (13.6)</td>
<td>96.7 (12.5)</td>
<td>104.4 (9.8)</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Bifr</td>
<td>53.5 (13.6)</td>
<td>100.4 (9.4)</td>
<td>108.5 (9.8)</td>
<td></td>
</tr>
</tbody>
</table>

Collapsed to give:

<table>
<thead>
<tr>
<th></th>
<th>GROUP</th>
<th>AGE</th>
<th>WAIS</th>
<th>FSIQ</th>
<th>NART1</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>Co.</td>
<td>49.7 (13.7)</td>
<td>-</td>
<td>112.0 (12.9)</td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>Posts.</td>
<td>43.0 (15.2)</td>
<td>106.1 (13.5)</td>
<td>111.5 (9.8)</td>
<td></td>
</tr>
<tr>
<td>40</td>
<td>Ants.</td>
<td>45.1 (14.1)</td>
<td>102.6 (14.4)</td>
<td>109.3 (10.8)</td>
<td></td>
</tr>
</tbody>
</table>

1 National Adult Reading Test (Nelson and O’Connell, 1975).

Key: R = right  L = left  A = Anterior  P = Posterior

Posts = Unilateral Posteriors
Ants = Unilateral Anteriors  Co. = Controls
The patients were compared with a group of 20 age- and NART-matched controls.

2.2(b) Test Design

The Brixton test consists of a series of 55 A4 sized pages which are presented one at a time to the subject. On each page is one of the designs as shown in Appendix 1. Each page has the same basic template on it, the only difference being the position of the filled circle. The length of each "set" pattern is designed so that changes in pattern cannot be anticipated.

2.2(b)i Subject's instructions

The subject is shown page 1 and told "There are many pages here, all with the same basic design on them. There are 10 positions here and one of them is always coloured in (pointing to filled circle on page 1). However the coloured one moves around (showing subject a page later in the test with a different position). It moves around according to various patterns that come and go without warning. These numbers (pointing to numbers underneath the circles) are just here to refer to the position - there is nothing complicated and mathematical about this test".

"Now, as I turn the pages over, your job is to pick up on the pattern as best you can, and tell me each time where you think the coloured one is going to be on the next page. Its not just guess-work - you can work it out. For instance, imagine the filled one was here [pointing to position 6] and then when I turn the page it goes to 7, and then to 8, then to 9 - you might reasonably expect it next to go to ten. From time to time the pattern changes without warning, and then it is your job to pick up on the new pattern as best you can. Understand? [If subject does not understand then further explanation is given until subject is ready to continue].
The subject is then prompted to give a guess based on the stimulus at position 1 with the words: "Obviously the first time you have nothing to go on, so your first answer has to be a guess - have a guess as to where the filled one will be next".

Subjects are allowed to refer back one page only, if they so request (quite rare in practice). If a subject were to ask whether any given response was correct, they were told (but without further explanation).

The full sequence of the moves is shown in Appendix I §. A sequence of twelve different sets was piloted, with the task designed with nine of these: seven which discriminated best between a small group of frontals (n = 6) and posteriors (n = 5) plus two "sets" which proved easiest for all subjects, one of which was used as the first sequence, the second of which was the last. This was done in an attempt to make the test user-friendly, since even normals make a significant number of errors on this task, and it was desirable to minimise the chance of patients becoming discouraged.

2.2(b)ii Scoring

The response to the first test item is necessarily a guess, so this is not scored. Responses immediately preceding a change in "set" are scored as if the change in set had not occurred, since the subject has no way of knowing that the pattern is about to change. Responses to these items that would not have been correct had the set not changed are scored as errors.
TABLE 6

Sets (or "rules") Used in the Brixton Test

<table>
<thead>
<tr>
<th>Section</th>
<th>Rule</th>
<th>Rules so far</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>+1</td>
<td>+1</td>
</tr>
<tr>
<td>2</td>
<td>-1</td>
<td>+1, -1</td>
</tr>
<tr>
<td>3</td>
<td>Alternate (5-10)</td>
<td>+1, -1, 5-10</td>
</tr>
<tr>
<td>4</td>
<td>+1</td>
<td>&quot;</td>
</tr>
<tr>
<td>5</td>
<td>-1</td>
<td>&quot;</td>
</tr>
<tr>
<td>6</td>
<td>+1</td>
<td>&quot;</td>
</tr>
<tr>
<td>7</td>
<td>Alternate (10-4)</td>
<td>+1, -1, 5-10, 10-4</td>
</tr>
<tr>
<td>8</td>
<td>Stay the same</td>
<td>+1, -1, 5-10, 10-4, Stay</td>
</tr>
<tr>
<td>9</td>
<td>Alternate (8-9)</td>
<td>+1, -1, 5-10, 10-4, Stay, 8-9</td>
</tr>
</tbody>
</table>

RESULTS

2.3(a) Overall Task Performance

Differences in the overall number of errors made by the different subject groups are shown in Table 7. An unweighted oneway ANOVA revealed significant differences between the unilateral lesion groups (p = <.001) in the total number of errors made on the Brixton Task. Tukey-Kramer post-hoc comparisons showed that the difference was due to the fact that the Anteriors made significantly more errors on the test than either the Posteriors or the controls. The posterior lesioned patients were not significantly poorer at this test than the controls. The Bifrontal group were not compared with the unilateral for obvious reasons. However, as can be seen from Table 3, they were poorer still than the unilateral anteriors.
TABLE 7
Brixton Test: Total Errors across Groups

<table>
<thead>
<tr>
<th>GROUP</th>
<th>N</th>
<th>MEAN ERRORS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>20</td>
<td>18.15 (4.3)</td>
</tr>
<tr>
<td>Posteriors</td>
<td>24</td>
<td>18.25 (7.2)</td>
</tr>
<tr>
<td>Anteriors</td>
<td>40</td>
<td>24.50 (8.9)**1</td>
</tr>
<tr>
<td>Bifrontals</td>
<td>13</td>
<td>28.92 (14.4)</td>
</tr>
</tbody>
</table>

Posteriors vs Controls nsig.
Anteriors vs Controls $p = <0.01^2$
Anteriors vs Posteriors $p = <0.01^2$

1 $p = <0.001$ unweighted ANOVA
2 Tukey-Kramer post-hoc comparisons

2.3(b) Significant Factors Determining Overall Test Performance

Considering the unilateral lesion patients only, analysis of the overall number of errors on the task by multiple linear regression revealed a number of factors - other than lesion location - to be significant predictors of subjects' performance. While laterality of lesion and NART performance were not significant, the age and FSIQ of the patients were very important (Age: t-ratio 5.90 $p = <0.001$; FSIQ: t-ratio -3.35 $p = <0.001$ with age, FSIQ and Anterior/Posterior factors accounting for 48.4% of the total variance). Consequently an analysis of covariance, with age and FSIQ as covariates was performed on the number of total test errors according to lesion site (anterior/posterior). This actually served to increase the significance level of the anterior/posterior difference.
2.3(c) Error Classification

In addition to the subjects' performance on the test as measured by the overall number of errors they made, the nature of their errors was also considered in an attempt to find out whether the anteriors performed the test qualitatively as well as quantitatively different from the posteriors and controls.

Obviously, there may be many reasons why one might fail any particular trial of this task. For the purposes of classification, all errors were categorised as falling into one of three possible error types:

Type 1 Errors

The first error type were straightforward perseverations. These could be perseverations of either the last stimulus or the subject's last response.

Type 2 Errors

The second type of errors ("Type 2 error") were misapplications of strategies or sets. This category therefore included perseverations of set (since it is impossible to be sure when a subject produces a response of this type whether s/he is perseverating on set or merely deliberately using the last strategy for some reason). Other responses classified as Type 2 included incorrect applications of the current correct set and applications of a previous strategy to the last incorrect response. Often these cannot be distinguished from each other, although they can of course be distinguished from the other error types.

There is one type of strategy error that cannot be inferred from consideration of the stimulus-response relationship. This is where a subject produces a response based on an inference about the current "set". Subjects will occasionally see relationships between the stimuli that were not intended. Furthermore, these may be quite
rational. An example would be in the first sequence in the task where the stimulus moves from position 1 to position 2. Obviously the set here is stimulus + 1, but some normals - ignoring the task instructions - read the relationship to be stimulus X 2, which led them to produce the answer 4 when it should have been 3.

Whilst these sorts of errors are clearly misapplications of strategy in some sense and therefore are categorised as examples of a Type 2 Error, when considered in isolation it would clearly be possible to categorise any error as in some sense resulting from the application of a self-generated strategy. However these errors should be predictable if the subjects are behaving in a rational way, since there are only a limited number of reasonable possibilities for each stimulus/set combination.

In order to build up a corpus of these types of errors, the responses of 34 normal subjects were considered. If any patient made the same error as any of the 34 normal subjects had made on a particular trial, this was then assumed to be an application of a self-generated strategy and was classified as a type 2 error. Obviously this procedure assumes that normal subjects always act rationally. Equally obviously, this may not be the case. However this procedure is conservative in terms of identification of Type 3 errors below, and this was considered a priority for the reasons that will be described below.

Type 3 Errors

These are the type of errors that would be predicted to occur if the subjects' respond in a pseudo-random way, as is suggested by the hypothesis outlined in the Introduction. They therefore are uninterpretable in terms of the test stimuli, past or current responses, current or previous sets or the mistakes normal subjects make due to false assumptions made regarding the task.
There would seem to be three likely explanations for these errors. They either represent guessing, or are responses arrived at by consideration of factors which normals do not consider in doing the same items, or (if this can be considered a separate category) they are the product of bizarre behaviour or idiosyncratic responding. There is little understanding in the problem-solving literature of what constitutes a guess, although this type of behaviour has been noted in normal subjects whilst they are performing some concept-formation type tasks (Green, personal communication). However since a guess must constitute in some sense both an idiosyncratic response, and also a consideration of factors singular to that subject, it will be assumed here the Type 3 errors noted in our patients' errors represent guessing.

The decision-making process involved in the classificatory system used here is represented in schematic form in Figure 2.

2.3(d) **Statistical considerations of the classification technique**

The classificatory technique used here is conservative in the sense that it probably underestimates the real incidence of guessing behaviour. Calculation of the probability of a response being classified as a Type 3 error if one were in fact responding randomly requires consideration of a number of factors. One has to consider the subject's last response, the last stimulus and the rules currently or previously "active" in the test, and any possible response that might be interpreted in terms of a combination of these three. Thus the probability of correct Type 3 classification given random responding will change from trial to trial and subject to subject. Each trial from each subject would need to be considered independently and thus the task would be extraordinarily complex. However, one can at least estimate the parameters theoretically. Thus the highest probability of correctly classifying a Type 3 error given random responding, and using the classificatory mechanism
described here, varies from .8 (which would be, for instance, in the situation of test item 6 which no normal got wrong, and where there is only one currently - or previously - active rule and additionally where the patient has not yet made an error) to .1 (for instance in the situation on test item No. 42 where normals made a variety of errors, and where there are four previously active sets plus the current one, and where the subject has just made a mistake - in this case giving the response 5 to the last test item).

FIGURE 2.
Schematic Representation of Error Classification Criteria

<table>
<thead>
<tr>
<th>STAGE</th>
<th>DECISION</th>
<th>AFFIRMATIVE CONCLUSION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Is it correct?</td>
<td>Classification stops</td>
</tr>
<tr>
<td>2.</td>
<td>Is the response a misapplication of a strategy currently or previously active?</td>
<td>TYPE 2 ERROR&lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>a) Last response</td>
<td></td>
</tr>
<tr>
<td></td>
<td>b) Last stimulus</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Is it the same number as either the last response or the last stimulus?</td>
<td>TYPE 1 ERROR</td>
</tr>
<tr>
<td></td>
<td>a) Response</td>
<td></td>
</tr>
<tr>
<td></td>
<td>b) Stimulus</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Does the number appear in any of the normal subjects' responses to that trial?</td>
<td>TYPE 2 ERROR</td>
</tr>
<tr>
<td></td>
<td>Self-generated strategy (normal)</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>With reference to the subject's response to the previous trial, is the current response an application of a currently or previously occurring strategy?</td>
<td>TYPE 2 ERROR</td>
</tr>
<tr>
<td></td>
<td>Application of strategy to own incorrect response</td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>(If none of above correct)</td>
<td>TYPE 3 ERROR</td>
</tr>
</tbody>
</table>

1 The respective position of stages 2 and 3 is determined by the occurrence of the "stay the same" set (section 8 in table 2). Since the majority of errors are classified as Type 2, this was given priority.
2 Classification continues until one of the conditions is fulfilled. Classification then ceases at this point.

So the actual probability of the procedure used here correctly classifying a Type 3 error varies from trial to trial from between .8 and .1. As can be seen from the
above examples, however, the probability of correct type 3 classification decreases the poorer the subject is at the task. Thus if we accept that it is those subjects who are poorest at the test overall who are the most likely to guess (since they have been unable to establish set), it easy to see that this classification procedure is highly conservative.

2.3(e) Occurrence of error types amongst the lesion groups.

The Anteriors and Posteriors were not only different in the number of total errors they made on the Brixton test, but the number of each type of error expressed as a proportion of their total errors was also significantly different (see Table 8).

While the proportion of Type 1 errors (response/stimulus perseverations) made by the two lesion groups was very similar, this was not the case for the other two error types. Analysis by Kruskal-Wallis (2-tailed, adjusted or non-adjusted) showed that the Posteriors made a higher proportion of Type 2 errors (strategy or set misapplication), although the difference just failed to reach significance (p = 0.053). However, but the greatest difference between the lesion groups was in terms of their Type 3 errors. A significantly higher proportion of the Anteriors’ errors were classified as Type 3 (p = <0.02). Bifrontals made an even greater proportion of type 3 errors (mean proportion of Type 3 errors = 19.59 SD 19.53).

TABLE 8

<table>
<thead>
<tr>
<th>Error Types As a Percentage of Total Errors</th>
</tr>
</thead>
<tbody>
<tr>
<td>ERROR TYPE</td>
</tr>
<tr>
<td>------------</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
</tbody>
</table>

*Kruskal-Wallis, 2-tailed, adjusted or non-adjusted.
There are two possible explanations for this finding. The first is that Anteriors perform the task differently from Posteriors. The second is that the proportion of Type 3 errors that a subject will make may be related only to how well they can perform the task overall. If this were the case, then the Anteriors would show a significantly higher proportion of Type 3 errors since they were poorer than the Posteriors at the task overall.

In order to decide between these competing hypotheses, a multiple linear regression was performed examining the effects of age, overall score, lesion site and FSIQ on the percentage of Type 3 errors made by the patients. This analysis, which accounted for 46.7% of the total variance, showed that age and lesion site were not significant predictive factors, but that FSIQ and overall test score were highly significant (FSIQ: t-ratio -2.37 p = 0.021; score: t-ratio 4.30 p = <0.001). This was confirmed by an analysis of covariance, with overall score, age and FSIQ as covariates.

It seems therefore that the difference in qualitative test performance between the lesion groups is linked to their differing abilities to do the test, rather than lesion site per se. There are two possible explanations for this relationship. It may be that the Anteriors’ inability to perform the task leads them to have to resort to a different way of tackling the task. Alternatively the Anteriors may be too willing to guess, and this precludes proper consideration of the problem. Quite possibly the two factors are inextricably linked in some complex way. In any case, it is impossible to decide between these competing explanations.
In this study, the anteriorly-lesioned patients performed the task in a way both qualitatively and quantitatively different from the patients with lesions not involving the frontal lobes. Not only did the Anteriors make more errors on the task overall, but also a significantly higher proportion of their errors were of type 3. Moreover, these two findings do not seem to be independent of each other. Those that were worst at the test overall were also those that made most type 3 errors (as a proportion of those errors).

A second finding was that the anteriors, contrary to widely-held belief, did not show a tendency to perseverate. The anteriors' and posteriors' proportion of Type 1 errors (stimulus or response perseverations) were almost identical. Furthermore, the other type of perseveration - perseveration of set or strategy - was classified in this analysis as error Type 2, and in fact the posteriors made more of these errors as a proportion of their total errors than did the anteriors. It may be possible that there is an effect, but this has been masked by the inclusiveness of the classification procedure, but the majority by far of type 2 errors were perseverations of set or strategy, so this seems unlikely.

2.3(a) Relationship between overall test performance and Type 3 errors

It may of course be the case that the inability to perform the task (in the sense of establishing set) and the tendency toward giving type 3 responses are associated deficits. In other words, that the tendency to respond in a Type 3 way and the ability to do the task actually being independent factors theoretically, but that the lesions in the anterior group were so devastating that the cognitive skills underlying both were affected at the same time. However this does not seem plausible, since the anteriors and posteriors were
matched for both current level of general intellectual functioning, and also for NART performance. This suggests that the general level of intellectual deterioration in the two groups was similar. In any case, the mean NART-FSIQ discrepancy for both the anteriors and the posteriors was actually very small for lesion patients, suggesting that these patients’ general cognitive skills were relatively intact.

A second explanation is of course that there is a causal relationship between a subjects’ ability to do the task and the number of Type 3 errors they make. When they do not know the answer, the produce a Type 3 error more often than do patients who are generally better at the test. The converse may also need consideration – that type 3 errors are indicative of some other underlying pathological behaviour (such as impulsivity) which results in the patient not applying his/herself to the task sufficiently.

2.4(b) The nature of Type 3 errors

What then, are these Type 3 errors? They are responses which are not correct and are not understandable in terms of that subject’s past responses, the characteristics of the test, or the erroneous inferences that normal (unimpaired) subjects may make about the task. Where then do they come from? They must be formed from a preference not based on an understanding of the task, and which is novel in the sense that it is not based on previous responses (although clearly the process need not be novel). This would seem to be a reasonable definition of a "guess".

2.3(c) Theoretical explanations of the findings

There are a number of potential explanations for this guessing behaviour, which vary in their plausibility. One explanation might be that the patient is simply suffering from motivational problems (cf. Benson and Blumer, 1976). This has little face value since that patients, as part
of this investigation, had to complete a whole range of other cognitive tests, and since the anteriors and posteriors were matched on their performance on these tasks one would have to assume that there is something so different about the Brixton task that it leads to a task- and lesion-specific motivational deficit. This seems implausible. The same argument holds for an explanation based on notions of increased distractibility.

One characterisation, based on the Norman-Shallice model (Norman and Shallice, 1980, 1986; Shallice, 1988), is that the patients’ inability to abstract tasks, deactivate or decide between current schemata (due to SAS failure) might lead to spurious schemata development. However there was no detectable pattern in the patients’ type 3 error responding - they appeared as randomly in the response sequences as the responses were random themselves. A more plausible account, still based on the Norman-Shallice model is that the successful establishing of a set or schema (strategy) in the test is dependent upon the correct functioning of the SAS since (a) the task is novel and (b) task stimuli will not (unlike in the case of the WCST) activate existing schemata\(^1\). The result is therefore random responding. By this account, the posteriors, more able to achieve "set", would use this as default when the set changed - hence the greater proportion of set (or strategy) misapplications in their errors.

These findings are in agreement with those of Miller (1985) and Miller and Milner (1985) who suggested that anteriors showed increased "cognitive risk-taking". They suggest that this is an operational definition of "impulsivity". If we assume that impulsivity is the willingness to provide a response based on less information (or before the information has been properly considered) than is usual, then the connection with

\(^1\)All cards in the WCST have at least one of the attributes of colour, form or number, and these are concepts with which everyone is familiar even before attempting the test. This can hardly be said for the spatial set on the Brixton of 10-4, for instance.
guessing behaviour is obvious. Since the anteriors also showed no increased tendency toward perseverative responding, this suggests that some of the findings of increased perseverative responding in frontals on the MWCST might represent guessing behaviour. In this case a more sensitive measure of frontal dysfunction might be the number of times that a patient chooses an option for which there seems to be no good reason (as can happen on the full version of the WCST [Heaton, 1984]).

Overall, the findings are consistent with the hypothesis that anteriorly-lesioned patients have two deficits: a deficit in set formation (or strategy application) together with an increased tendency towards guessing. The complex and probably interactional relationship between the two is unresolved.
CHAPTER THREE

DEVELOPMENT OF THE HAYLING SENTENCE COMPLETION TEST

Introduction

It has been known for some time that frontal lobe lesions can cause deficits in response initiation and suppression (Kleist, 1934; Luria, 1970). These characteristics may be shown readily in the neuropsychological testing situation. For instance one of the most commonly used tests of frontal lobe functioning is the Verbal Fluency test (eg. Benton, 1968; Hecaen and Ruel, 1981; Miller, 1984; Milner, 1967; Pendleton, Heaton, Lehman and Hulihan, 1982), in which patients are required to produce as many words as they can that begin with a specified letter (or alternatively that belong to a particular semantic category eg. animals).

Typically, patients with frontal lobe lesions produce fewer words than patients with lesions elsewhere\(^1\). This reduced word fluency has been linked by some to a general aspontaneity of thought (eg. Kleist, 1934; Ramier and Hecaen, 1970) which may be a feature of frontal initiation problems. A more sophisticated interpretation of reduced verbal fluency is presented by Costello and Warrington (1989). In their patient ROH, reduced word fluency was one characteristic of the more general syndrome of dynamic aphasia, in which patients present with reduced spontaneous use of language in the context of normal confrontation naming, language comprehension and speech production. They suggest that dynamic aphasia represents a deficit in verbal planning at a stage prior to sentence construction.

\(^1\) It should be noted, however, that some studies have shown no anterior-posterior differences in verbal fluency performance (eg. Shallice, 1982).
Frontal lobe patients have also been shown to experience problems with response suppression in the testing situation. For instance, Verfaille and Heilman (1987) gave two patients with medial frontal lobe lesions a task derived from the experimental animal literature. The patients, with their eyes closed, have to respond to a touch of one hand by raising the contralateral hand. One of Verfaellie and Heilman’s patients consistently raised the ipsilateral hand in response to the touch. The authors interpreted this as an inability to inhibit an incorrect response since the patient showed self-corrections and was able to verbally describe the correct response when asked to do so.

Similarly, Drewe (1975) found that patients with frontal lobe lesions were significantly poorer than patients with lesions elsewhere at a go-no go task which required the subjects to withhold responses to one of two stimuli.

However the inability to suppress the most immediate response may also be a contributory factor in more complex cognitive deficits shown by patients with frontal lobe dysfunction. Shallice and Evans (1978) found that frontal patients produced more bizarre answers than posterior patients when asked to estimate the answers to novel questions such as "how fast does a racehorse gallop?". As Shallice and Evans point out, answering such questions requires the patient to select an appropriate "cognitive plan" (p. 295) and to check putative answers before responding. By implication, frontal patients seem less inclined to plan and check their answers, and so produce ill-considered (or impulsive) replies.

Similarly, Perret (1974) administered a form of the Stroop test to groups of patients with cortical lesions, and found that the patients whose lesion invaded the left frontal lobe were significantly poorer at suppressing the habitual response than patients with lesions elsewhere.

However, Perret presents an additional finding. Those patients who performed poorly on the Stroop Test were
also those who were most likely to achieve a poor word fluency score. This finding has three broad explanations. The first possibility is that the Stroop task and the VFT may actually make similar cognitive processing demands, despite their apparent differences in format. A second possible explanation is that when the "executive system" is damaged in frontal lobe patients, it necessarily affects both initiation and response suppression abilities. The third possibility is that the two processes are theoretically independent, but that since the cortical structures which subserve such processes are close in proximity in the brain, both processes tend to be affected in patients with cortical damage.

Perret favours the first explanation, arguing that, in reference to the Verbal Fluency Test (VFT), the "usual criterion in the search for words" (p. 323) is word meaning. By asking the patients to perform their search according to initial letter, one is asking them "to suppress the habit of using words according to their meaning" (p. 324). Thus for Perret, both the Stroop test and the VFT both require the subject to suppress the habitual response.

However, Perret also considers the third explanation. In the discussion, Perret makes the point that the correlation between the patients' performance on the two tests may be associated deficits caused by large lesions, with no actual causal relationship. Thus the question of the relationship between response initiation and inhibition remains open.

Clearly the difficulty in the interpretation of frontal patients' performances on many frontal tests arises from the differing characteristics of those tests. The present study, therefore, utilises a task in which the differing components of initiation and inhibition can be examined with minimal changes in the background characteristics of the task.
3.2(a) Subjects

A consecutive series of 108 patients referred to the National Hospital Dept. of Clinical Neuropsychology were assessed as part of the present study. The majority of the patients reported here also appeared in chapter 2, and criteria for acceptance in the study were as outlined in chapter 2. 17 patients failed to meet these criteria and were rejected. Classification of subjects according to lesion site was also as outlined in chapter 2. 63 patients were suffering from tumours (either meningiomas, gliomas, astrocytomas or oligodendrogliomas), 9 cases had other SOLs (cyst abscess or neoplasm), 21 cases were of vascular origin and there was one patient with known traumatic damage.

<table>
<thead>
<tr>
<th>GROUP</th>
<th>N</th>
<th>AGE</th>
<th>WAIS-IQ(^1)</th>
<th>NART(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>L Anterior</td>
<td>21</td>
<td>40.8 (13.2)</td>
<td>100.1 (13.8)</td>
<td>106.3 (10.5)</td>
</tr>
<tr>
<td>R Anterior</td>
<td>26</td>
<td>48.5 (13.3)</td>
<td>109.6 (13.4)</td>
<td>114.3 (8.8)</td>
</tr>
<tr>
<td>L Posterior</td>
<td>12</td>
<td>37.8 (14.3)</td>
<td>102.1 (15.8)</td>
<td>111.2 (11.6)</td>
</tr>
<tr>
<td>R Posterior</td>
<td>15</td>
<td>48.1 (14.0)</td>
<td>108.6 (9.5)</td>
<td>111.5 (9.8)</td>
</tr>
<tr>
<td>Bifrontal</td>
<td>17</td>
<td>51.4 (13.0)</td>
<td>100.6 (11.0)</td>
<td>107.2 (9.6)</td>
</tr>
</tbody>
</table>

Collapsed to give:

<table>
<thead>
<tr>
<th>GROUP</th>
<th>N</th>
<th>AGE</th>
<th>WAIS-IQ(^1)</th>
<th>NART(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anteriors</td>
<td>47</td>
<td>45.1 (13.7)</td>
<td>105.3 (14.3)</td>
<td>110.7 (10.3)</td>
</tr>
<tr>
<td>Posteriors</td>
<td>27</td>
<td>43.5 (14.8)</td>
<td>105.7 (12.8)</td>
<td>111.4 (10.4)</td>
</tr>
<tr>
<td>Bifrontal</td>
<td>17</td>
<td>51.4 (13.0)</td>
<td>100.6 (11.0)</td>
<td>107.2 (9.6)</td>
</tr>
<tr>
<td>Controls</td>
<td>20</td>
<td>49.7 (13.7)</td>
<td>-</td>
<td>112.0 (12.9)</td>
</tr>
</tbody>
</table>

1 Where subjects were given the WAIS-R a correction factor of +8 points was used (Crawford, Allan and Besson, 1990)
2 National Adult Reading Test (Nelson and O’Connell, 1975)
Assignment of patient according to lesion site was not conducted until after the subject had been seen, when the full results of investigations in the hospital were available. Details of the patients, along with a group of 20 normal subjects matched for age and NART are given in Table 9.

Significant laterality effects were not found at any point in the analysis so the unilateral patients were collapsed to give four groups: unilateral anterior or posteriors, a bifrontal group and the controls. The controls were gathered from many sources, but in the main were relatives accompanying patients to the hospital. None of the controls had experience of psychology or related disciplines.

3.2(b) Materials

The Hayling Test consists of 30 sentences selected from Bloom and Fischler (1980) in which the final word was omitted. Bloom and Fischler present norms for completion of the sentences, and the 30 chosen were those for which there was a particularly high probability of one given response. American terms in the text were translated into their British equivalents (see Appendix 2). The 30 sentences were pseudo-randomly assigned to one of two groups of 15 sentences to be used in either the first or second sections of the test. In addition four more sentences from Bloom and Fischler were selected to be used as examples for the subjects.

3.2(c) Task Instructions

The task consists of two sections (A and B), each consisting of 15 sentences. Subjects were tested with only the examiner present, in a quiet standard hospital office.
**Section A: Initiation**

The subject is given the following spoken instructions:

"I am going to read you a set of sentences, all of which have the last word missing from them. I want you to listen carefully to each sentence and when I have finished reading it, you tell me what you think the last word should be. The sentences are not difficult, and usually it is quite easy to think what the word should be. But first we’ll go through a couple of practice sentences so you can get the hang of it".

The two practice sentences are read to the subject and s/he is required to give a reasonable response. When the subject has satisfactorily completed the examples they are told the following:

"Now, the sentences don’t get any more difficult than the ones you’ve already done. What I am particularly interested in is how quick you can give me the answers. So when I’ve read the sentence to you, your job is to give me a word you think could fit at the end of the sentence as quickly as possible".

If the subject is still unsure about the task, further explanation may be given (in practice extremely rare). Sentences are read to the subject at a normal reading pace. Timing of response latency was by stop-watch operated by the examiner, started as soon as it was judged the last word of the sentence had been read, and timing stopped as soon as the subjects began their response.

**Section B: Response Suppression**

Section B was conducted immediately after Section A. Between the two tasks the subjects were told the following:
"Now the next section is a little different from the first. I will read you sentences which have the last word missing just like before, but this time I want you to give me a word which makes no sense at all in the context of the sentence. I want the word you give me to be unrelated to the sentence in every way. This is not always easy to do at first, so we will have a couple of practice trials first. Ready?

The two examples are then given to the subject. If s/he gives a word which is in fact related to the sentence, s/he is told that the word is too related, and they are required to give another. If they are unable to think of one, the example word "banana" (which is unrelated to either of the two practice items) is suggested to the subject.

If at any time during this stage of the test, the subjects gives a sentence completion rather than an unrelated word, s/he is told that the word is too related to the sentence, and the task instructions are repeated. In practice, with a few of the subjects who were very poor at the task, this resulted in their being reminded about the task instructions after each of the 15 trials. If the patient had not produced a word within 60 seconds, that trial was terminated and a response latency of 60 seconds was recorded.

3.2(d) Scoring

Three measures were considered: sum of the response latencies of section A (the quality of the answers was not considered since less that 1% of replies to this section were not one of those predicted by Bloom and Fischler (1980); sum of the response latencies of section B; and the quality of the replies in section B.
3.3 RESULTS

No significant laterality effects appeared in any of the measures (see table 10).

3.3(a) Section A

The response latencies (sum of latencies across 15 trials) for the unilateral anteriors, unilateral posteriors and controls are shown in Table 2. There was a significant difference between the unilateral anterior, posterior and control groups (Kruskal-Wallis H = 11.81, df = 2, p <0.005 adjusted or unadjusted). Post-hoc comparisons showed that the anteriors were significantly slower than the controls (p <0.002) and the posteriors (p <0.05) but that the posteriors were not significantly slower than the controls. As might be expected, the bilateral frontal group were slower than the unilateral anteriors.

Analysis of the patients’ times by multiple linear regression indicated that both age (t-ratio 3.27, p <0.005) and FSIQ (t-ratio -3.12, p< .005) to be significant predictors of performance on this test. An analysis of covariance (following log transformation) was therefore performed using age and FSIQ as covariates. Under these conditions the anterior-posterior difference in the section A times was significant at the <0.02 level.

3.3(b) Section B

While the overall difference in the response latencies between the unilateral anteriors, posteriors and controls was significant (Kruskal-Wallis H = 6.50, df = 2, p <0.05), none of the post-hoc between group comparisons were significant for a 2-tailed test.
TABLE 10.  

Response Latencies Across Groups

<table>
<thead>
<tr>
<th>GROUP</th>
<th>SECTION A</th>
<th>SECTION B</th>
<th>B-A</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anteriors</td>
<td>28.6 (34.5)</td>
<td>83.1 (106.8)</td>
<td>55.5 (78.5)</td>
</tr>
<tr>
<td>Posteriors</td>
<td>17.4 (14.1)</td>
<td>40.6 (38.9)</td>
<td>24.3 (27.8)</td>
</tr>
<tr>
<td>Controls</td>
<td>14.8 (12.5)</td>
<td>35.2 (31.2)</td>
<td>20.4 (24.3)</td>
</tr>
<tr>
<td>Bifrontals</td>
<td>35.8 (23.12)</td>
<td>140.5 (152.6)</td>
<td>109.0 (148.4)</td>
</tr>
<tr>
<td>LH (n=33)</td>
<td>26.2 (22.3)</td>
<td>59.5 (68.2)</td>
<td>33.3 (56.0)</td>
</tr>
<tr>
<td>RH (n=41)</td>
<td>23.2 (33.9)</td>
<td>74.1 (105.1)</td>
<td>50.9 (74.5)</td>
</tr>
</tbody>
</table>

Key: LH = all patients with (unilateral) left hemisphere lesions considered together; RH = all patients with (unilateral) right hemisphere lesions.

Analysis by multiple linear regression revealed that age, FSIQ and NART were all significant factors in determining performance amongst the patients (p <0.001 for all factors). An analysis of covariance (log transformed scores) was therefore performed, with age, FSIQ and NART as covariates. Under these conditions, the anterior-posterior difference in response latencies was significant at the <0.05 level. As with the performance on section A, the bifrontals were considerably slower than the unilateral anteriors on this section (see table 10).

The response latencies of all groups were significantly longer on section B, compared with section A (Paired T-Tests, all groups p <0.005).

One patient (a right frontal) was unable to produce a word within 60 seconds on 8 of the 15 trials. A number of other frontal patients failed to produce words within the time limit on single trials. No posterior or control ever showed this pattern.
3.3(c) B-A Times

As a measure of the difference that the experimental manipulation in section B made, the section B latencies minus the section A latencies were considered for each patient (see Table 2). This presumably represents the additional "thinking time" that is required in having to produce a novel word rather than a straightforward sentence completion. Obviously this serves to remove the possible confounding factor of initiation problems when considering the latencies in the response suppression condition.

The B-A times for the unilateral anteriors, posteriors and controls were not significantly different (Kruskal-Wallis H = 3.34, df = 2 p = 0.2). An alternative statistical procedure for controlling for initiation problems in considering section B scores is to covary for section A when considering section B times. When an analysis of covariance for section B times was performed, with section A times as a covariate, the differences between the groups still did not approach significance (F = 1.03, df = 2, p = 0.36).

3.3(d) Error Classification

Only the unilateral or bilateral anterior patients gave answers to section A which were not sensible completions of the sentence. However these responses were less then 1% of the total responses, and there were many more anterior patients in this study than posteriors or controls. Accordingly no conclusions will be drawn about the quality of the subjects' responses in section A.

However the responses to section B were quite different across groups. These were examined in detail.

Responses in Section B

In order to discover whether the groups differed in their ability to produce words unrelated to the stimulus
sentence in Section B, three raters, blind to the purpose of the study, rated each of the 1665 subject responses (111 subjects x 15 sentences). They were asked to classify each of the responses into one of the eight possible categories shown in Figure 3, following the instructions presented in Appendix 2.

These eight categories can be subdivided into 3 main groups. Firstly there are responses which are sensible completions of the sentence, thus clearly violating the task instructions (category C in figure 3).

Secondly, there are responses that are semantically connected to the sentence in some way (categories 0, SA, SB and SC in figure 3). The response may be semantically related to either the expected completion of the sentence (including opposites of what might constitute a sentence completion); it may be semantically related to the subject of the sentence, or may be a word, which whilst not entirely completing the sentence in a reasonable fashion, nevertheless renders some meaning to the sentence (socially inappropriate responses were included in this category).

Finally, there are those responses which are unrelated to the sentence, as required by the task instructions. Casual observation had suggested that there were two commonly used strategies when performing the task. The first method for producing these unrelated words was to look around the room, choosing objects within sight and naming these in response to the stimulus sentence. The second method was to self-generate a semantic category and choose members of that category as responses.

Accordingly, raters were asked to classify responses unrelated to the sentence into one of four additional categories. The words could be objects normally found in a standard hospital office. Alternatively, the words could be related to the last answer they gave. Obviously on occasions responses might satisfy both conditions, and so there was a category (URL) for such responses. If the
word was unrelated to the sentence, was not an object normally found in an office and was not semantically related to the last response, it was classified as U (see figure 3).

3.3(e) Inter-rater Agreement

Two or more raters agreed on 94.1% of occasions, which is quite satisfactory given that each response can be given any one of 8 different classifications. In the remaining 5.9% of responses, a fourth rater, blind to the purpose of the study, was asked to decide which one of the three raters was correct.

**FIGURE 3.**
Rater’s Classification Guide

<table>
<thead>
<tr>
<th>STEP</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. DOES THE WORD REASONABLY COMPLETE THE SENTENCE (IE. ITS A WORD YOU MIGHT GIVE --- &gt; C YOURSELF IF ASKED TO PROVIDE A WORD THAT WOULD FIT AT THE END).</td>
<td></td>
</tr>
<tr>
<td>2. IS THE WORD AN OPPOSITE OF WHAT YOU MIGHT --- &gt; O EXPECT AS AN ANSWER?</td>
<td></td>
</tr>
<tr>
<td>3. IS THE WORD OBVIOUSLY SEMANTICALLY --- &gt; SA RELATED TO THE SUBJECT OF THE SENTENCE?</td>
<td></td>
</tr>
<tr>
<td>4. IS THE WORD OBVIOUSLY SEMANTICALLY --- &gt; SB CONNECTED TO THE EXPECTED RESPONSE?</td>
<td></td>
</tr>
<tr>
<td>5. DOES THE WORD VAGUELY FIT AT THE END OF THE SENTENCE, BUT IN A WAY THAT MAKES THE --- &gt; SC SENSE OF THE SENTENCE LUDICROUS - OR IS THE WORD A SLANG &quot;SEMI-OBSCENITY&quot;?</td>
<td></td>
</tr>
<tr>
<td>6. IS THE WORD COMPLETELY --- &gt; EITHER U, UR, UL UNCONNECTED TO THE SENTENCE? OR URL (SEE BELOW)</td>
<td></td>
</tr>
<tr>
<td>7. IS THE WORD AN ITEM YOU MIGHT FIND --- &gt; UR IN A HOSPITAL OFFICE?</td>
<td></td>
</tr>
<tr>
<td>8. IS THE WORD SEMANTICALLY CONNECTED --- &gt; UL TO THE SUBJECT’S LAST RESPONSE?</td>
<td></td>
</tr>
<tr>
<td>9. IS THE WORD BOTH AN ITEM YOU MIGHT FIND IN AN OFFICE, AND IS ALSO RELATED --- &gt; URL TO THE LAST ANSWER?</td>
<td></td>
</tr>
<tr>
<td>10. ARE NONE OF THE ABOVE TRUE? --- &gt; U</td>
<td></td>
</tr>
</tbody>
</table>
3.3(f) Results of Error Classification

Completions of the Sentence

The percentage of responses belonging to each category across the groups is shown in Table 11. Analysis of variance showed that the number of responses that were straightforward sentence completions varied across the controls, posteriors and unilateral anteriors (F = 5.65, df = 93, p <0.005). Tukey-Kramer post-hoc comparisons showed that the unilateral anteriors completed the sentences significantly more often (Q = 4.02, df = 91, r = 3 p <0.05) than the posteriors or the controls (Q = 3.72, p <0.05). The posteriors did not differ significantly from the controls. The bilateral frontal group produced almost twice as many completions as even the unilateral frontals, with these responses accounting for nearly one third of all their answers.

Answers Semantically Related to the Sentence

The groups also differed in the number of responses that were judged to be semantically related in some way to the stimulus sentence (categories O, SA, SB and SC) (ANOVA F = 3.51, df = 93, p <0.05). Tukey-Kramer post-hoc comparisons revealed that the anteriors (Mean 3.9 SD 3.0) made significantly more of these responses than the controls (mean 2.0 SD 2.0; Q = 4.05, p <0.05), but the anterior-posterior difference failed to reach significance at the 0.05 level. The posteriors (mean 3.0 SD 2.5) did not differ significantly from the controls. The bifrontals made considerably more semantically related responses than even the unilateral anteriors (mean 4.9 SD 2.9).

Answers Unrelated to the Stimulus Sentence

The unilateral anteriors, posteriors and controls differed in the number of responses they gave which were judged to be unrelated to the sentence (i.e. fully met the
task instructions). ANOVA showed this difference to be significant at the p <0.001 level.

TABLE 11
Percentage Responses in each Category Across Groups

<table>
<thead>
<tr>
<th></th>
<th>C</th>
<th>O</th>
<th>SA</th>
<th>SB</th>
<th>SC</th>
<th>UR</th>
<th>UL</th>
<th>URL</th>
<th>U1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cont.</td>
<td>4.2</td>
<td>2.7</td>
<td>2.5</td>
<td>1.5</td>
<td>5.8</td>
<td>31.5</td>
<td>5.0</td>
<td>1.7</td>
<td>45.2</td>
</tr>
<tr>
<td>Posts.</td>
<td>4.7</td>
<td>3.7</td>
<td>2.7</td>
<td>3.7</td>
<td>9.6</td>
<td>27.2</td>
<td>8.9</td>
<td>1.7</td>
<td>37.5</td>
</tr>
<tr>
<td>Ants.</td>
<td>16.9</td>
<td>8.2</td>
<td>3.4</td>
<td>3.1</td>
<td>10.9</td>
<td>24.8</td>
<td>2.7</td>
<td>1.1</td>
<td>27.7</td>
</tr>
<tr>
<td>BiFr.</td>
<td>32.2</td>
<td>11.0</td>
<td>6.7</td>
<td>3.1</td>
<td>12.5</td>
<td>8.2</td>
<td>3.9</td>
<td>0.4</td>
<td>22.7</td>
</tr>
</tbody>
</table>

Section Totals

<table>
<thead>
<tr>
<th></th>
<th>Completions</th>
<th>Semantically Related</th>
<th>Unrelated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cont.</td>
<td>4.2</td>
<td>12.5</td>
<td>83.3</td>
</tr>
<tr>
<td>Posts.</td>
<td>4.7</td>
<td>19.8</td>
<td>75.3</td>
</tr>
<tr>
<td>Ants.</td>
<td>16.9</td>
<td>25.7</td>
<td>56.2</td>
</tr>
<tr>
<td>BiFr.</td>
<td>32.3</td>
<td>32.5</td>
<td>35.3</td>
</tr>
</tbody>
</table>

Key: Cont. = Controls; Posts. = Posteriors; Ants. = Unilateral Anteriors; BiFr. = Bifrontals.
1 See Figure 1 for explanation of categories.

Tukey-Kramer post-hoc comparisons showed that the anteriors gave significantly fewer unrelated words (categories U, UL, UR and URL) as answers than the posteriors (Q = 4.0, p <0.05) or the controls (Q = 4.9 p <0.01) whereas the posteriors did not differ significantly from the controls.

Analysis of the patients' scores by multiple linear regression revealed that both age and NART were significant predictors of performance on this measure (p <0.001 and p <0.05 respectively). Accordingly an analysis of covariance was performed, using age and NART as covariates. Under these conditions, the Anterior-Posterior difference increased in significance (F = 7.83, df = 72, p <0.01)
Responses Revealing the Use of Strategies

As explained above, three of the categories rated by the judges were intended to indicate the use of a strategy in producing answers to section B (UL, UR, URL). The unilateral anteriors, posteriors and controls differed in the proportion of their responses that were either semantically related to their last response, or were objects normally found in an office, or both (Kruskal-Wallis $H = 6.53$, df = 2, $p <0.05$). Multiple comparison procedures (following Daniel, 1990) showed that the anteriors’ responses contained a significantly smaller proportion of these types of response (at the $<0.05$ level) than either the posteriors or the controls, who were not significantly different from each other (see table 12). The proportion of the bifrontals responses that came from the strategy categories was, on average, less than half that of the unilateral anteriors.

The correlation between the B-A latencies (ie. suppression condition minus initiation condition latencies) and the proportion of responses revealing the use of strategies was $-0.45$, which is significantly different from zero ($p <0.01$ 2-tailed). In other words, those that showed longer thinking times tended to produce fewer strategy responses.

<table>
<thead>
<tr>
<th>Group</th>
<th>% Strategy Responses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>41.3 (28.0)</td>
</tr>
<tr>
<td>Posteriors</td>
<td>37.8 (22.8)</td>
</tr>
<tr>
<td>Anteriors</td>
<td>27.4 (29.2)</td>
</tr>
<tr>
<td>Bifrontals</td>
<td>12.6 (19.6)</td>
</tr>
</tbody>
</table>
Overall Semantic Relatedness

A measure of the overall semantic relatedness of the responses to the stimulus sentence, an error score was devised. Three points was given if the word was a straightforward completion of the sentence (category C), and 1 point for words that were semantically related to the sentence in some way (categories O, SA, SB and SC). Categories U, UR, UL and URL represent successful completion of the task requirements and so were given no score. A summary of the error score results are given in Table 13. Under these conditions, the differences in performance between the controls, unilateral anteriors and posteriors was significant at the <0.002 level (ANOVA), with Tukey-Kramer post-hoc comparisons showing the difference between the anteriors and controls significant at the <0.05 level ($Q = 4.1, r =3, df = 91$), and the difference between the anteriors and posteriors significant at the <0.01 level ($Q = 4.4, r =3, df = 91$).

Multiple linear regression showed that age and IQ were significant predictors of performance as measured by the error score ($p < 0.01$ and $p= < 0.001$ respectively), and so an analysis of covariance was performed on the patients' scores using age and FSIQ as covariates. Under these conditions, the anterior-posterior difference was significant at the <.005 level.

TABLE 13
Error Scores Across Groups

<table>
<thead>
<tr>
<th>GROUP</th>
<th>ERROR SCORE (Max 45)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>4.7 (5.5)</td>
</tr>
<tr>
<td>Posteriors</td>
<td>4.8 (4.8)</td>
</tr>
<tr>
<td>Anteriors</td>
<td>11.5 (11.3)</td>
</tr>
<tr>
<td>Bifrontals</td>
<td>19.4 (11.1)</td>
</tr>
</tbody>
</table>
3.3(g) Performance of Individual Cases

A very low correlation of 0.19 was found between performance on Section A (response latency in seconds) of the Hayling Test and Section B (error score) across all the unilateral patients. This correlation is not significantly different from zero. Moreover, removing the effects of age and FSIQ from both measures by partial correlation techniques (McNemar, 1969) reduced this figure to .07, which is very low indeed. Overall this suggests that a patient’s performance on one measure is a poor predictor of their performance on the other.

This was confirmed by consideration of the scores of individual patients. Thus using a strict criterion for a dissociation between tasks as being where the subject performs within 1 SD of the mean of the controls on one measure, but worse than 3 SD below the mean of the controls on another (see Shallice, 1988), 4 unilateral anteriors and one posterior showed this pattern for good performance on Section A with poor Section B performance, and one anterior patient showed the opposite pattern (poor A with good B performance). Thus it seems that performance on Section A may be independent of performance on Section B.

3.4 DISCUSSION

This study has two main findings. The first is that, compared with a group of age and IQ-matched patients with extra-frontal lesions (posteriors), patients with lesions that included the frontal lobes (anteriors) showed deficits on a measure of task initiation. The second finding is that the anteriors were also poorer than the posteriors on a task which involved the suppression of the habitual response.

One possible explanation for these findings might be that the anterior patients were suffering from a greater degree of generalised cognitive impairment than the
posteriors due, for instance, to the method of lesion site classification. However this can be rejected since the groups were matched for performance on a traditional neuropsychological measure of general intellectual functioning (the WAIS). If the anteriors had larger lesions than the posteriors, one would expect them to perform at a lower level on the WAIS. Moreover, not only were the groups matched for IQ, but in the analysis, where factors such as age, current FSIQ and levels of pre-morbid functioning were found to be significant predictors of performance on the measures, these factors were partialled out using ANCOVA. In many cases this actually increased the level of significance of the anterior-posterior difference.

A second possible explanation of the results might be that the anteriors performed the tasks badly due to distractibility or motivational problems. However for this to be the cases, one would have to assume that there is something specific to the design of the Hayling Test that makes it especially sensitive to these factors, since the anteriors did not perform other neuropsychological tests poorly (eg. WAIS). This seems implausible, which leaves an explanation of the results based upon the particular processing resources or abilities that may be damaged in patients with lesions in the frontal lobes. These will be discussed in turn.

De Lacy Costello and Warrington (1989) gave their patient ROH a similar paradigm to that used in Section A of the Hayling Test. ROH was asked to complete a series of 66 of the Bloom and Fischler (1980) sentences with a single word. De Lacy Costello and Warrington report that ROH was able to produce words to complete every sentence satisfactorily, but that his response latencies were long, with a mean of 7.9 secs, compared with a control group mean of 1.1 secs (which accords well with the control performance in the present study).

Two patients within this series performed at a similar level to patient ROH. This first, who had a mean latency
of 7.7 secs, had an abscess in the left frontal lobe, with his FSIQ commensurate with his NART estimated FSIQ. The second patient (mean latency 14.8 secs)\(^2\), was suffering from a right frontal AVM\(^3\), and his current FSIQ was 15 points lower than should be predicted by his NART. Thus neither patient could be said to be suffering from a severe global intellectual decline. Neither patient had CT scan evidence of extra-frontal involvement.

Of course it is not possible to say that these patients showed the same overall pattern of performance as ROH - the extensive investigations given to this patient were not administered to these patients. Nevertheless these findings confirm De Lacy and Warrington’s claim that this type of task performance is characteristic of frontal lobe impairment.

The performance of the groups on the response suppression (Section B) part of the Hayling Test requires simultaneous consideration of the subjects’ performance on Section A. All groups took longer to respond in Section B than Section A. Since all other characteristics of the task are the same in Section B excepting that the subject must produce a word which is unconnected to the meaning of the sentence, the extra time the subjects take presumably reflects the thinking time that this requires.

While the anteriors showed significantly longer response latencies than the posteriors in Section A, their response latencies in Section B were not significantly longer. Had the quality of their responses also been similar, one would conclude that there were no differences between the anteriors and posteriors on this measure. However this was not the case. In comparison with the patients with posterior lesions, the anterior

\(^2\) This included 8 failures to respond within the 60 secs permitted, which were scored as 60 sec latencies.

\(^3\) An unconfirmed report in this patient’s notes suggested a past history of a vascular abnormality in the left frontal lobe which was not noted on the present CT scan
lesioned patients made significantly more straightforward completions of the sentence, and those answers they gave which were not completions were nevertheless judged to be more semantically related to the sentence. It is clear then, that the end products of the anteriors’ thinking time less satisfactorily fulfilled the task requirements.

The key to what cognitive operations the normal and posterior subjects were involved in during this thinking time, is the nature of the responses. The controls and posteriors gave significantly more words (responses) that were either related to the last response they had made, or else were objects that one might normally expect to find in a hospital office.

This is unlikely to be a word frequency effect since if the anteriors were poorer at word retrieval one would expect them to produce words of higher frequency than the posteriors, and most office objects have high frequency names. The most plausible explanation is that the choice of these words reflects the use of response strategies in the posteriors and controls. It would seem that two commonly used strategies for producing a word unconnected to the stimulus sentence are either to pick objects from ones the surroundings, or else (and these are plainly not mutually exclusive) to generate a semantic category, and then choose exemplars of this category as responses. Given that the likelihood of the response fitting the sentence by chance must be relatively low, this may be quite an efficient strategy. One could either give the word immediately after hearing the sentence and take the risk that it might fit by chance, which would result in low response latencies at the risk of producing some poor quality responses, or alternatively, after hearing the sentence the subject may check the putative answer before responding. Either method would be likely to produce an efficient task performance.

One explanation of the poor performance of the anteriors on the response suppression task might be that they use strategies, but are less good at it. The prediction made
by this theory would be that those patients who take longer to reply would also be those who would show the greater number of "strategy" responses. However this was not the case. The significant negative correlation between B-A latencies and proportion of strategy errors suggests that those that took longer were also less likely to use strategies. Since the efficient use of a strategy would be expected to decrease response latencies in section B, we might suppose therefore, that somewhat longer (although not significantly so) times shown by the anteriors in section B represents the lack of use of such strategies. Instead it would appear that the anteriors tended to complete the sentence instead, showing a difficulty in suppressing the habitual response.

Perret (1974) suggested that the high correlation in his study between performance on the VFT and the Stroop task suggests that both Verbal Fluency and the Stroop task tap the same process – suppression of the habitual response. The alternative hypothesis was that Verbal Fluency requires good initiation abilities and the Stroop requires the suppression of the habitual response, but that either initiation and suppression are necessarily damaged together through being part of the same system or that they tend to be impaired together due to the anatomical closeness of the cortical areas subserving these processes.

However the findings of the present study suggest that initiation and response suppression difficulties may be impaired singly, and that overall correlations between performances on measures of these processes may be quite low. While poor test reliability could account for the low overall correlations\(^4\), this is unlikely to be a good explanation of the double dissociations noted within single cases (see McNemar, 1964, p.158). The double dissociations also militate against suggestions that response suppression and initiation problems occur due to

\(^4\) The problem of reliability of measures will be dealt with specifically in chapter 5.
damage to one system, but may occur singly where damage has occurred at differing levels within it.

Instead, the most likely explanation of the present results is that the two processes are orthogonal. This both supports Perret’s interpretations of his data, and also suggests that where both processes are impaired, they are so because the cortical areas which subserve such processes are damaged together.
4.1 Introduction

The Introduction to this thesis reviewed the experimental findings of memory deficits following frontal lobe involvement. While many of the patterns shown by these patients (e.g. defective release from PI, recall-recognition discrepancies, poorer cued than free recall, increased susceptibility to interference) have been discussed extensively by investigators, they have in the main not yet been incorporated into theoretical frameworks of the role of the frontal lobes in memory.

This is however not true of the findings of defective temporal functions\(^1\), many of which (e.g. Kimura, 1963; Milner, 1968; Petrides and Milner, 1982; Pribram and Tubbs, 1967; Prisko, 1973, reported in Milner, 1964; Smith and Milner, 1988) are extensively cited in support of frontal lobe memory theory (e.g. Milner, 1982; Schacter, 1987; Squire, 1987). As such, the issue of whether frontal lobe patients suffer from deficits in temporal functions is a key one.

The findings, however, remain contentious (Mayes, 1988). There are two reasons for this. The first is that many of the results from studies of temporal functions in frontal lobe patients are ambiguous due to methodological difficulties. These were examined in detail in the Introduction to the thesis, however they can be summarised as being either unusual patient selection procedures (e.g. Moscovitch, 1982), injudicious use of association methodology (e.g. Shimamura et al., 1990) or failure to compare directly frontal lobe patients with

\(^1\) Given that there is no agreed nomenclature for these functions (Schacter, 1987), "temporal functions" is here used to refer to the various findings of recency discrimination, frequency judgements, temporal order, spatiotemporal or contextual judgements and the like.
patients with posterior lesions (Eg. Petrides and Milner, 1982).

The second reason is perhaps more fundamental. Despite the considerable number of studies in the area, (see for instance Corsi, 1972; Damasio, 1979; Milner, 1963, 1971, 1974, 1982; Milner et al, 1985; Stuss et al, 1982), there is as yet no general consensus as to why it may be that frontal lobe patients fail tasks involving temporal functions.

Schacter (1987) favours an explanation which suggests that patients with frontal lobe lesions have deficits in "contextual chunking" - the ability to "organize experience into temporally distinctive chunks" (p. 32), although exactly what processes are involved in such an operation are not specified. Milner (Eg. Milner et al, 1985) provides more detail. She makes a distinction between recency discrimination, judgements of frequency of occurrence and self-ordered executive tasks. Milner suggests that recency discrimination involves some "direct encoding of temporal information" together with judgements based on memory trace strength, duration, intensity, familiarity and emotional significance.

In contrast, frequency of occurrence judgements require an overall evaluation of the representations of many items established over time. This is performed by searching back through memory for the instances of that particular stimulus, and comparison of these with instances of other stimuli.

Milner also uses the example of frontal lobe patients' performance on the Petrides and Milner (1982) self-ordered pointing task to highlight the prospective aspect of the temporal organisation of behaviour.

These accounts, valuable as they are, are difficult to test experimentally. This is however not the case for those explored by Mayes (1988). He proposes that most of the patterns of impaired memory performance following
frontal lobe lesions can be explained by suggesting that frontal lesions impair memory because they disturb effortful and elaborate processing of information. However this is less clearly the case for temporal functions, since some authors (Eg. Hasher and Zacks, 1979) suggest that spatiotemporal information is automatically encoded, whilst others (Eg. Jackson et Al, 1986) disagree, arguing that contextual memory is affected by effortful, semantic encoding of target items. Mayes (1988) concludes that more research is needed into the effects of effortful processing on contextual memory. This is the first of two issues addressed by the present study.

A second possible explanation of patients' inability to perform temporal order tasks is an inference based upon demonstrations that patients with frontal damage suffer from increased susceptibility to interference (see Mayes and Meudell, 1983 for a review). Specifically, in studies involving the use of A-B, A-C paired associate paradigms, amnesics typically demonstrate difficulty learning the A-C associates, and show many intrusions from the first list, even when they are first matched with controls for A-B recognition (Mayes, 1988, p. 169). By this account, patients who fail temporal order tasks may do so because they suffer from exaggerated inter-trial interference (Mayes, 1988, p. 174), which may be the root of the deficits in "contextual chunking" referred to by Schacter (1987). However, as pointed out by Mayes (1988), most of the demonstrations of increased interference in amnesics have used patients suffering from alcoholic Korsakoff syndrome, and as such it is unclear as to whether increased interference effects are characteristic of amnesia per se, or of the concomitant frontal lobe damage sometimes found in such patients. The present study will address this issue by studying interference effects in patients with localised anterior or posterior damage within the context of a measure of temporal order processing.
4.2 METHOD

4.2(a) Subjects

A consecutive series of 110 patients referred to the National Hospital Dept. of Neuropsychology were examined as part of the present study. Criteria for inclusion in the experiment were as outlined in Chapter 2. 13 patients failed to conform to these criteria and were excluded. Additionally 20 age- and NART-matched normal control subjects were tested. All but five of the patients reported here also appear in either of the series reported in chapters 2 and 3. The controls were the same group as appeared in these two chapters, the majority of whom were carers or relatives accompanying patients. Patients with unilateral lesions were classified as anterior or posterior in the manner described in chapter 2. In addition 17 patients with bilateral frontal involvement (but no posterior damage) were tested. Details of the ages and overall levels of intellectual functioning of the patients and controls are shown in Table 14.

<table>
<thead>
<tr>
<th>N</th>
<th>GROUP</th>
<th>AGE</th>
<th>FSIQ</th>
<th>NART</th>
</tr>
</thead>
<tbody>
<tr>
<td>26</td>
<td>L Ants</td>
<td>42.3 (13.6)</td>
<td>100.4 (7.5)</td>
<td>107.5 (10.6)</td>
</tr>
<tr>
<td>24</td>
<td>R Ants</td>
<td>48.7 (13.8)</td>
<td>108.0 (15.9)</td>
<td>113.7 (9.6)</td>
</tr>
<tr>
<td>16</td>
<td>L Posts</td>
<td>42.9 (15.4)</td>
<td>107.8 (9.7)</td>
<td>111.7 (10.6)</td>
</tr>
<tr>
<td>14</td>
<td>R Posts</td>
<td>43.0 (13.4)</td>
<td>104.2 (16.3)</td>
<td>110.1 (9.5)</td>
</tr>
<tr>
<td>17</td>
<td>Bifronts</td>
<td>52.8 (12.3)</td>
<td>101.8 (11.1)</td>
<td>106.9 (10.6)</td>
</tr>
</tbody>
</table>

Unilateral patients collapsed to give:

<table>
<thead>
<tr>
<th>N</th>
<th>GROUP</th>
<th>AGE</th>
<th>FSIQ</th>
<th>NART</th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>Anteriors</td>
<td>45.4 (13.9)</td>
<td>103.8 (15.1)</td>
<td>110.5 (10.5)</td>
</tr>
<tr>
<td>30</td>
<td>Posteriors</td>
<td>42.9 (14.2)</td>
<td>106.1 (13.1)</td>
<td>111.0 (9.9)</td>
</tr>
<tr>
<td>17</td>
<td>Bifrontal</td>
<td>52.8 (12.3)</td>
<td>101.8 (11.1)</td>
<td>106.9 (10.6)</td>
</tr>
<tr>
<td>20</td>
<td>Controls</td>
<td>49.7 (13.7)</td>
<td>—</td>
<td>112.0 (12.9)</td>
</tr>
</tbody>
</table>

2 One unilateral posterior patient was not given the NART, so the means are based on N = 29.
No significant hemispheric differences in test performance appeared at any time during the analysis, so the unilateral patient groups were collapsed (see table 14).

4.2(b) Procedure

Task Design: The Word Order Test

The Word Order Test (WOT) consists of three lists of 12 very high frequency words taken from Thorndike and Lorge (1944). Lists 1 and 2 are made up of same-frequency words, but neither list shares words with the other. The third list consists of the same words as appear in the second, but the words are presented to the subject in a different order (see Appendix 3). The lists are presented in order to the subject. Following each list presentation, the words that appeared in that list are read to the subject one-by-one, in a randomised order. The subject is required to make a judgement as to the position of that word in the presentation list, by saying either "beginning", "middle" or "end" in response to each word. The word lists and subsequent presentation order were the same for all subjects.

Task Instructions

Instructions are read to the subject before the verbal presentation of each of the lists, and the instructions are different for each list. Before the first list is read to the subject the following instructions are given:

"I am going to read you a list of words. I want you to listen carefully to them because I will be asking questions about them afterwards. Are you ready?"

The instructions may be repeated to the subject if necessary.
The words are read to the subject in order at the rate of one word each two seconds. After presentation the subject is given the following instructions:

"Now, I am going to give you the words that I have just read to you one by one, and I want you to tell me each time whether you think the word came at the beginning, middle or end of that list. Do you understand?"

Further explanation can be given as required. The words are given one-by-one to the subject who has to respond "beginning", "middle" or "end". No other responses are permitted except in the case where the subject correctly specifies the exact position of the word (Eg. "third from last" or "the very first word you gave me").

Following presentation of list one, and before presentation of list two, the following instructions are spoken to the subject:

"Now I am going to read you another list. The words are different in this list, but you will be doing the same thing afterwards - telling me whether you think each word came at the beginning, middle or end of the list. Before of course, I didn’t tell you that you would have to tell me where in the list the word was. But now you know what I am going to ask you afterwards. Do you understand?"

The instructions may be repeated as required. The second list is then read to the subject. This is followed by the presentation of the words singly and in random order for the subjects to judge their position.

This is followed by presentation of the third list, preceded by the following instructions:

"This time I’m going to read you the same words as were in the last list, but they are now in a different order. Afterwards I want you to tell me the position in which they appeared this time around. Is that clear?".
The instructions may be repeated as required, followed by presentation and temporal order judgements as before.

The changes in the task instructions and the nature of the lists has the effect of ensuring that the subject is unaware of the nature of the test when s/he attends to the first list. This is intended to be the "automatic encoding" condition. The instructions to the second list, however, is intended to draw the subjects' attention to the position of the stimuli within the list. This is intended to be the "effortful encoding" condition. The third list, as already described, consists of the same words as in list 2, presented in a different order. This is intended to be the "interference" condition.

4.2(c) Scoring

Since each list consists of 12 words, the list can be divided into three groups of four words which belong to either the "beginning, middle or end" categories. For those words which the subject correctly identified as belonging to the appropriate 4-word cluster, a zero was scored. Words the subject identified as belonging to the wrong cluster were scored according to the number of positions away from the nearest point where the subject would have been correct. For instance, if the word "ball" appeared in position nine of the presentation list (which would be correctly classified as "end"), but the subject responded "beginning", this response would be given an error score of 5, which is the number of positions away from the nearest segment of the "beginning" block.

As pointed out by Toglia and Kimble (1976), it is important to correct for subjects tendency to respond "middle" (central tendency) in serial order tasks. In addition, using the scoring method as described, it is possible for a subject to gain an error score below chance by responding "middle" to every word. Accordingly, all the results to be described below were also analysed according to an error score based on the following algorithm:
Corrected Score = Error score + \( (R\times4) - (C\times4) + 32 \)

where error score refers to the score as explained above, \( R = \) number of repeated responses above those that would be expected by chance responding (Ie. 4) and \( C = \) number of correct responses above those that would be expected by chance (again 4). Using this algorithm, subjects with extreme central tendency responding will achieve a score somewhat below chance levels, whilst still enabling those who achieve a perfect set of responses to gain an error score of zero. However the pattern of results achieved using this scoring procedure did not differ from those to be described below, and so for the sake of simplicity results based upon the uncorrected error scores will be described.

4.2(d) Control Tasks

In order to be sure that any differences between the subject groups on the WOT were due to poor memory for temporal order rather than merely generalised memory problems, three control tasks were used. Warrington’s (1984) RMT for words and faces was administered to all the patients (but not the controls), and all subjects were tested for free recall of a 12-word list. This was presented auditory-verbally at the rate of one word every two seconds. These words were taken from Coughlan and Hollows’s (1985) list learning paradigm and the subjects were required to free recall as many of the words as possible (one trial only).

4.3 RESULTS

Scores representing the serial position errors in judgement for the three experimental conditions are shown in Table 15. Performance on the individual measures is described first, with the interrelationships between the measures described later.
4.3(a) Trial 1: Automatic Encoding of Position

A oneway ANOVA revealed significant differences between the controls, unilateral anteriors and posteriors (F = 5.6 df = 97, p < 0.005). Post-hoc Tukey-Kramer comparisons showed that the unilateral anteriors were poorer than the controls at recalling the position of stimuli (q = 4.72 [3, 97], p <.01). However the unilateral posteriors were also poorer than the controls (q = 2.9 [3, 97], p < .05). The anteriors were worse than the posteriors overall, however this difference was not significant at the .05 level. The bifrontal group were poorest of all. However the bilateral frontal group also had the lowest IQs and were rather older than the other groups (see below).

Analysis of the unilateral patients' scores by multiple linear regression (backward elimination) using left/right hemisphere, anterior-posterior, NART, FSIQ and age as predictors, revealed that whilst group (anterior or posterior) was not a significant predictor of performance on this measure (nor were the other predictors), FSIQ and Age were highly significant factors in determining test performance (p <0.005 and <0.001 respectively), with these two factors together accounting for 20.4% of the total variance.

4.3(b) Trial 2: Effortful Encoding

Oneway ANOVA between the controls and unilateral lesion patients revealed no significant differences between the groups (F = 1.27, df 97, p = .3). Analysis of the unilateral patients' scores by multiple linear regression (using the same factors as above) showed that, as with the patients' performance on the automatic encoding condition, age and FSIQ were significant predictors of performance on this measure (t-ratio 2.11 p <0.5 and t-ratio -3.52, p <0.01 respectively) with these two factors contributing 16.9% of the total test variance.
There were no significant differences in performance between the controls and unilateral anteriors and posteriors ($F = 0.73$, df = 97, $p = 0.5$). Analysis of the unilateral patients' scores by multiple linear regression (backward elimination using the same factors as above) showed that, unlike conditions 1 and 2, age and FSIQ were not significant predictors of performance on this test (nor were any other factors). Remarkably, none of the factors was able to individually predict more than 1% of the total variance on this measure. This will be discussed below.

### TABLE 15.
WOT: Error Scores in Judging Serial Position by List

<table>
<thead>
<tr>
<th>Group</th>
<th>List 1</th>
<th>List 2</th>
<th>List 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>12.7 (7.0)</td>
<td>16.3 (10.3)</td>
<td>21.1 (11.3)</td>
</tr>
<tr>
<td>Posteriors</td>
<td>18.0 (9.3)</td>
<td>18.1 (10.5)</td>
<td>24.5 (11.1)</td>
</tr>
<tr>
<td>Anteriors</td>
<td>20.7 (9.6)</td>
<td>20.3 (9.5)</td>
<td>23.9 (9.6)</td>
</tr>
<tr>
<td>Bifrontals</td>
<td>22.6 (8.3)</td>
<td>20.5 (8.5)</td>
<td>25.1 (7.6)</td>
</tr>
</tbody>
</table>

#### 4.3(d) Inter-relationships Between Measures

**Automatic vs Effortful Encoding Conditions**

Knowledge of the purpose of the task had little effect upon subjects' performance, and no group showed statistically significant differences between the conditions. The normal controls were, as a group, actually slightly poorer at the task under the effortful encoding condition. However this difference did not approach statistical significance.

There was a modest positive correlation of .22 between the unilateral patients' scores on the two conditions. However multiple linear regression showed that this correlation was largely due to the effects of age and FSIQ on the two measures, and when these factors were
considered alongside condition 1 scores in predicting condition 2 performance, patients' condition 1 scores were not significantly predictive of performance in the effortful processing condition (t-ratio 0.86, p = 0.4).

Since there were no significant differences in the groups scores between condition 1 and 2, the scores across the conditions were combined to form an overall index of ability on this test. Under these conditions a one-way ANOVA between the controls and unilateral anteriors and posteriors showed significant differences between the groups (F= 5.34, df = 97, p <0.01) with Tukey-Kramer post-hoc comparisons showing that whilst the posteriors were not significantly poorer than the controls (q = 2.6, r = 97) the anteriors-control difference was significant at the 0.01 level (q = 4.59, df = 99). However the difference between the anteriors and posteriors was not significant (q = 2.01).

Interference Condition

Interference was expressed as the increase in error score between condition 2 and condition 3 as a percentage of individual patients' scores in condition 2 (condition 3 score - condition 2 score/condition 2 score * 100). However, considering first the raw score change between trials 2 and 3, as ready discussed, none of the factors right/left hemisphere, anterior or posterior, age, FSIQ or NART were significant predictors of unilateral patients' performance on condition 3. However analysis of the controls and unilateral patients' scores on conditions 2 and 3 showed that performance on condition 2 was a highly significant predictive factor of performance on the interference condition (t-ratio 3.63, p <0.001), with this alone accounting for 12% of the total variance. Overall correlation between the two measures was .35. This is of course unsurprising, since the degree to which the word positions were encoded on trial three should of course predict the degree of interference shown.

For this reason, a measure of interference perhaps more
sensitive than merely the raw scores on the trials is the percentage increase from condition 2 to 3. These are shown in Table 16.

**TABLE 16**

Interference Condition: Mean Increases in Error Scores as a Percentage of Encoding Condition (SD in brackets)

<table>
<thead>
<tr>
<th>GROUP</th>
<th>PERCENT INCREASE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>109.0 (182.8)</td>
</tr>
<tr>
<td>Posteriors</td>
<td>84.3 (137.3)</td>
</tr>
<tr>
<td>Anteriors</td>
<td>37.9 (85.5)</td>
</tr>
<tr>
<td>Bifrontals</td>
<td>65.3 (138.5)</td>
</tr>
</tbody>
</table>

Despite the apparent differences in the mean scores, the unilateral anteriors, posteriors and controls were not significantly different from each other on this measure (Kruskal-Wallis h = 2.7, df = 2, p = 0.26). Analysis of the unilateral patients’ scores by multiple linear regression (backward elimination) using 7 factors as predictors showed that of these seven, only performance on condition 2 was significantly predictive of performance on this measure (t-ratio -6.12, R\(^2\) = 32.4\% p <0.001). It would seem therefore that % increase over condition 2 score is an adequate measure of interference, and that the differences in the means (see Table 3) reflect the degree of encoding at condition 2. The anteriors were poorer at condition 2 and therefore tended to show less interference. The difference between the anteriors and posteriors however just failed to reach significance (t-ratio -1.98, R\(^2\) = 4.9\%, P = 0.051).

**Control Measures**

Performances of the patient groups on Warrington’s Recognition Memory Test (RMT) and on the free recall measure are shown in Table 17.

3 Anterior/posterior classification; Left/Right hemisphere; age, FSIQ, NART, Condition 1 score and Condition 2 score.
The data from the unilateral patients showed no significant effects of hemisphere, anterior/posterior classification (ANOVA) or interaction effects for any of the memory measures. Nor were the controls, anteriors and posteriors significantly different on the free recall measure, although this did approach significance ($F = 2.9$, df = 78, $p = 0.06$).

### TABLE 17
Performance of Subject Groups on Control Memory Measures

<table>
<thead>
<tr>
<th>GROUP</th>
<th>FCW$^4$</th>
<th>FCF$^5$</th>
<th>Free Recall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>NA</td>
<td>NA</td>
<td>6.5 (2.7)$^{13}$</td>
</tr>
<tr>
<td>Posteriors</td>
<td>43.3 (6.3)</td>
<td>41.0 (6.6)</td>
<td>11 5.6 (2.1)$^7$</td>
</tr>
<tr>
<td>Anteriors</td>
<td>41.6 (7.6)</td>
<td>40.8 (5.9)</td>
<td>10 5.1 (1.8)$^8$</td>
</tr>
<tr>
<td>Bifrontals</td>
<td>38.1 (6.8)</td>
<td>38.6 (5.5)</td>
<td>12 4.5 (1.6)$^9$</td>
</tr>
</tbody>
</table>

$^7$ N = 23 (data on 7 patients missing)
$^8$ N = 41 (data on 9 patients missing)
$^9$ N = 16 (data on 1 patient missing)
$^{10}$ N = 48 (data on 2 patients missing)
$^{11}$ N = 28 (data on 2 patients missing)
$^{12}$ N = 16 (data on 2 patients missing)
$^{13}$ N = 17 (data on 3 controls missing)

### Inter-relationships Between Measures

Correlations between the measures of age, current level of general intellectual functioning and the memory measures are given in Table 18.

---

4 Forced-Choice Words (Warrington, 1984)
5 Forced-Choice Faces (Warrington, 1984)
6 NA = Not Administered
TABLE 18
Correlations Between Measures

<table>
<thead>
<tr>
<th>AGE</th>
<th>FSIQ</th>
<th>NART</th>
<th>WOT:A</th>
<th>WOT:E</th>
<th>WOT:I</th>
<th>FCW</th>
<th>FCF</th>
</tr>
</thead>
<tbody>
<tr>
<td>FSIQ</td>
<td>0.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NART</td>
<td>0.16</td>
<td>0.64***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WOT:A</td>
<td>0.28*</td>
<td>-0.33**</td>
<td>-0.25*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WOT:E</td>
<td>0.19</td>
<td>-0.35**</td>
<td>-0.23*</td>
<td>0.32**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WOT:I</td>
<td>-0.09</td>
<td>0.30**</td>
<td>0.13</td>
<td>-0.22</td>
<td>-0.57***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FCW</td>
<td>-0.17</td>
<td>0.35**</td>
<td>0.26*</td>
<td>-0.31**</td>
<td>-0.27*</td>
<td>0.14</td>
<td></td>
</tr>
<tr>
<td>FCF</td>
<td>-0.31**</td>
<td>0.26*</td>
<td>0.01</td>
<td>-0.33**</td>
<td>-0.18</td>
<td>0.16</td>
<td>0.57***</td>
</tr>
<tr>
<td>RECALL</td>
<td>-0.39**</td>
<td>0.50***</td>
<td>0.31*</td>
<td>-0.27*</td>
<td>-0.38**</td>
<td>0.11</td>
<td>0.38**</td>
</tr>
</tbody>
</table>

* p < 0.05
** p < 0.01
*** p < 0.001
1 WOT: Automatic encoding condition
2 WOT: Effortful encoding condition
3 WOT: Interference condition
4 Free recall measure

-----

TABLE 19
Significant Predictors of Unilateral Patients' Performance on Test Measures

<table>
<thead>
<tr>
<th>MEASURE</th>
<th>PREDICTORS</th>
<th>t-RATIO</th>
<th>p</th>
<th>R-Sq2</th>
</tr>
</thead>
<tbody>
<tr>
<td>WOT:A</td>
<td>Age</td>
<td>3.02</td>
<td>&lt;0.005</td>
<td>20.4</td>
</tr>
<tr>
<td></td>
<td>FSIQ</td>
<td>-3.50</td>
<td>&lt;0.002</td>
<td></td>
</tr>
<tr>
<td>WOT:E</td>
<td>WOT:I</td>
<td>-5.41</td>
<td>&lt;0.001</td>
<td>42.3</td>
</tr>
<tr>
<td></td>
<td>Recall</td>
<td>-3.31</td>
<td>&lt;0.005</td>
<td></td>
</tr>
<tr>
<td>WOT:I</td>
<td>WOT:E</td>
<td>-6.12</td>
<td>&lt;0.001</td>
<td>32.4</td>
</tr>
<tr>
<td>FCW</td>
<td>Hemisphere</td>
<td>-2.46</td>
<td>&lt;0.02</td>
<td>45.4</td>
</tr>
<tr>
<td></td>
<td>NART</td>
<td>2.63</td>
<td>&lt;0.02</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FCF</td>
<td>6.77</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>FCF</td>
<td>Hemisphere</td>
<td>2.79</td>
<td>&lt;0.01</td>
<td>46.0</td>
</tr>
<tr>
<td></td>
<td>FSIQ</td>
<td>2.76</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td></td>
<td>NART</td>
<td>-2.70</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FCW</td>
<td>6.29</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Recall</td>
<td>Age</td>
<td>-4.50</td>
<td>&lt;0.001</td>
<td>43.8</td>
</tr>
<tr>
<td></td>
<td>FSIQ</td>
<td>5.58</td>
<td>&lt;0.001</td>
<td></td>
</tr>
</tbody>
</table>

2 R-Squared of combined predictors per table cell
The most obvious result of the correlational analysis is the degree to which FSIQ is apparently significantly correlated with all measures (except of course age). This raises the possibility that a number of the other correlations are due to mutual dependency upon FSIQ. This obviously holds, albeit to a lesser extent, for Age and pre-morbid IQ (NART).

An analysis by multiple linear regression (backward elimination) was therefore performed on each measure using all other measures as predictive factors\(^1\), with the addition of the variables anterior/posterior classification and hemisphere of lesion. The results are shown in Table 19. This analysis confirms the suspicion that many of the apparent correlations between memory measures are in fact due to mutual dependency on other, non-memory related cognitive abilities. Of especial note is the extent of the combined influences of age and FSIQ on the automatic encoding condition of the WOT, and in particular the free recall measure, where a considerable amount (almost 44%) of the total variance can be accounted for by these factors alone. Performance on the free recall measure is, however, not a significant predictor of performance on the WOT:automatic condition, and the overall correlation between the two was modestly significant (.27).

Moreover, the extent to which each Forced-Choice measure is predictive of performance on the other, but are not significantly predictive of any of the other memory measures suggests that performances on the temporal order task, free recall task and forced-choice tasks are orthogonal. However there are clearly a number of other factors not directly related to memory functioning per se which are significant influences upon memory test performance. This is particularly the case for the free recall measure.

An incidental finding was that the multiple regression analysis reveals hemispheric differences in the RMT measures which were not discovered by ANOVA. This was due to the

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1 From the factor pool: Anterior/Posterior; Left/Right hemisphere; Age; FSIQ; NART; WOT Automatic encoding; WOT: Effortful encoding; WOT: Interference; FCW; FCF; Free Recall.
influence of using the other forced-choice measure as a predictor, and did not appear when this factor was not present. Clearly there is a complex interrelationship at work.

DISCUSSION

The Introduction to this thesis made the point that whilst there is little doubt now that patients with frontal lobe lesions may perform test measuring temporal functions poorer than normal controls, the crucial point is whether frontal lobe patients perform the tasks poorer than patients with posterior lesions, and there is as yet little convincing evidence that this is the case. Accordingly, this study compared directly the performance of patients with anterior or posterior lesions on a test of memory for temporal information similar to those used before in the cognitive experimental psychology literature (eg. Hintzman and Block, 1971; Toglia and Kimble, 1976).

More specifically, this study investigated three possible factors which investigators have used as explanations of frontals’ failure on test of temporal functions. The first explanation, explored by Mayes (1988), is that frontals might suffer from poor contextual-temporal memory because these functions may disturb effortful processing (perhaps the most widely accepted view of frontal patients’ pattern of memory test performance). However Mayes makes the point that the issue may not be as simple as this, and quotes Hasher and Zacks’ (1979) evidence that some forms of temporal information are automatically encoded. Mayes concludes that if this is true, then it may be the case that frontal lesions impair automatic and effortful encoding. However the finding that patients were no better at remembering positional information when they were forewarned about the task supports the view that contextual-temporal information is processed automatically. Moreover, when anterior and posterior-lesioned patients’ overall test performance were compared directly, the differences between the groups was not significant, nor was the pattern of performance between the groups different.
A second explanation of the apparent deficits that frontal patients show on tests of temporal functions is that the failure is due to excessive inter-trial interference. However the results of this study do not support this view. In this study, by far the greatest predictor of interference was degree of initial encoding (unsurprisingly). The anterior patients were generally slightly poorer at the encoding condition than the posteriors, and so showed proportionally less interference. However the difference between the groups did not approach significance.

Negative findings in a study may of course arise for two main reasons. The first may be that there is indeed no significant effect to study, the second being that the experimental design was such that it did not adequately elicit the effect.

This second explanation seems an unlikely explanation of the negative findings reported here. The multiple regression analyses showed a number of highly significant effects upon WOT performance (especially age and FSIQ). So it seems unlikely that the data was so unreliable that an effect, should it be there, would not appear.

Moreover, the majority of the cases in this study also appeared in the development of the Brixton and Hayling tasks (chapters 2 and 3) which did show anterior-posterior lesion site effects. Of course it may be possible that the task used here is not a good measure of temporal functions. This is an argument which can hardly be refuted. However at least one WOT measure (averaged condition 1 and 2 scores) yeilded results similar to those in Petrides and Milner’s (1982) often-cited study, in that the posterior-control difference was not significant, yet the anterior-control difference was significant. In addition the task used was similar in many respects to other tasks that have been used (eg. Hintzman and Block, 1971). More obvious interpretations of the present results may be either that there is no specific role for the frontal lobes in contextual-temporal functions (at least, as will be argued later in the thesis, as regards traditional experimental memory test studies), or that the conditions
under which frontal deficits may be detected are very specific indeed.
5.1 Introduction

The introduction to this thesis explored the nature of the evidence regarding the role of the frontal lobes in human memory. The conclusion, broadly, was that whilst there is no good evidence that lesions restricted to the frontal lobes can lead to an amnesia of the classical type, some frontal lobe patients do show memory disturbances. This is no more than has been suggested by many others (eg. Mayes, 1988; Stuss and Benson, 1986; Shallice, 1988; McCarthy and Warrington, 1990).

The evidence regarding the precise nature of the memory disturbances that follow frontal lobe dysfunction was therefore reviewed. The conclusion was that there are few, if any, pertinent studies where the results are not made equivocal due to methodological difficulties. These methodological problems were addressed in chapter 4, which presented an analysis of frontal-lobe and extra-frontally lesioned patients on three memory tests.

The first was a traditional measure often used as an index of degree of amnesia of the classical type (Warrington's RMT). The second was a newly developed measure which tapped those memory processes that are often considered to be impaired in frontal lobe patients (WOT), and the third (free recall) was a test which has been shown to be either impaired or unimpaired relative to recognition memory in frontal lobe patients. However none of these measures showed significant anterior-posterior differences.

This is not, of course, to say that patients with frontal lobe lesions did not fail the memory tests. Indeed, individual cases with supposedly isolated unilateral frontal lesions performed at a level on, for instance, Warrington's RMT that would normally be
sufficient for that patient to be considered severely amnesic. 

And yet no author has suggested that a frontal lobe lesion, especially a unilateral lesions, can be a sufficient condition to cause a severe amnesia of the classical type. More usually authors explain the memory test performance of frontal lobe patients as being secondary to the particular cognitive deficits which these patients may show (eg. Mayes, 1988; Stuss and Benson, 1986). This will be referred to as the "dysexecutive explanation" of frontal lobe patient's memory test performance, and is contrasted with the "frontal amnesia" explanation which holds that frontal lobe patients show a particular type of memory disorder distinct from classical amnesia (eg, McCarthy and Warrington, 1990; Shallice, 1988).

Clearly the findings of chapter 4 cannot speak to the frontal amnesia hypothesis (except in a negative sense) since no anterior-posterior differences were found in memory test performance. However the results of chapters 2-4, when combined, may address directly the dysexecutive explanation.

There are two main predictions which follow from the hypothesis that frontal lobe patients who perform memory tests poorly do so because of their executive problems. These can be formalised as follows:

5.1(a) **Prediction 1:** Patients who fail "frontal lobe tasks" will tend to fail memory tests.

5.1(b) **Prediction 2:** Frontal lobe patients who fail memory tests will also have executive problems.

---

1 One patient with a left frontal lesion, aged 63 and with a FSIQ commensurate with his NART (105), scored 22/50 on FCW and 30/50 on FCF - performances at chance level.
Prediction 1 is required in order to secure the connection between memory test and executive test performance, and prediction 2 is required in order to secure the connection with frontal lobe dysfunction. If either (or both) of the predictions were shown not to hold, the dysexecutive explanation would be embarrassed. Accordingly, these predictions will be tested in the present study, using the data from previous chapters.

As we have seen, the dysexecutive explanation relies upon the (sometimes implicit) assumption of either an association between performances on memory tests and tests of executive function or some complex interactional relationship between them. However, a contention that was raised in the introduction to this thesis also potentially provides an explanation of such associations.

As already discussed, it has become increasingly common for authors to use both 'frontal' and 'memory' tests in the investigation of mixed pathology2 amnesics, ascribing the type of memory test performance shown by those patients who fail the frontal tests as being due to frontal pathology (eg. Schacter, 1987; Janowsky et al, 1989; Parkin et al, 1988; Squire, 1982).

In some cases authors use the performance on tests considered sensitive to frontal lobe dysfunction as an indicator of frontal lobe involvement, this evidence being accorded similar status as, for instance, evidence from radiological investigations (eg. Schacter, 1987). The idea is that the "frontal test" performance is a measure of what may be best described as "frontality", or the tendency to show those symptoms which are considered characteristic of frontal lobe dysfunction. In some cases this evidence is regarded as so strong that even within groups of frontal lobes patients, subgroups will be selected for study who show certain features which purportedly represent "frontality" (eg. Moscovitch,

2 ie. cases where pathology is unknown and probably widespread, or diffuse and uncertain.
Many of these studies have yielded information which is central to current understanding of the role of the frontal lobes in memory (eg. Leng and Parkin, 1988; Squire, 1982; Schacter, Harbluk and MacLachlin, 1984; Schacter et al, 1986).

Other studies use performance on frontal lobe tests as a measure of "executive abilities" rather than as a localisation tool, and regard associations in performance between the two as evidence for the possible role of executive functions in memory (eg. Baddeley and Wilson, 1988). The consensus from this school regards the memory problems experienced by patients with frontal lobe lesions as secondary to their executive problems (eg. Mayes, 1988; Stuss and Benson, 1986). Perhaps an example most pertinent to this thinking is Baddeley and Wilson's (1988) astute observation that there has yet to be a case reported who is severely dysexecutive but who performs reasonably on memory tests.

As already discussed in the introduction, a potential problem for both types of studies is where the association between executive and memory tests might be due to some other factor unrelated to the main point of these tests - for instance age, level of general intellectual functioning or pre-morbid IQ. This would certainly provide a compelling explanation of Baddeley and Wilson’s (1988) observation. If this were the case, the associations between the executive and memory tests would not actually be useful in our understanding of the role of the frontal lobes in memory.

A second issue is raised by those studies which use performance on tests considered sensitive to frontal lobe dysfunction as a kind of localisation tool. If the memory and frontal tests were to draw upon abilities secondary to the main aim of those tests, associations between tests may not reflect relationships between the abilities those tests are intended to measure. For instance (and for the sake of argument) let us suppose that performance on Warrington’s (1984) FCW test is influenced by a
subject's overall verbal abilities. Clearly this is, at least the case at extremes, since if a patient were unable to read, s/he would be unable to take the test in its standard form. Suppose also that the measure of "frontal abilities" being used is the Verbal Fluency Test (VFT), which has been shown to be highly correlated with measures of general verbal ability (see Miller, 1984). Thus one might get in a random group of patients with widespread damage, a correlation between performance on the FCW and VFT. In the sense that a) frontal lobe patients have been shown to perform the VFT poorer than posterior patients and b) in the random group of patients there was an association found between FCW and VFT performance, the experimenter may conclude, incorrectly, that 'frontal patients' fail FCW. However the association is incidental - not causal - and would exist in controls also.

These methodological points can be formalised into two predictions which, if supported by the data, may provide explanations of the associations between executive and memory test performance:

5.1(c) Prediction 3

This states that background factors such as age and FSIQ might affect both frontal and memory measures, which may lead to correlations between performances without these actually reflecting a relationship between executive and memory abilities. This is suggested by the results of chapters 2-4, which showed that while the patient groups (i.e. anterior or posterior classification) differed in their performance on the executive measures, they were not significantly different in their performance on the memory measures. If the executive and memory measures are significantly correlated, this may suggest some independent factor contributing to performance on both tests which may not be directly related to site of lesion.
The logic is as follows. Assume for the moment that an executive measure taps some general processes (G) plus a specifically executive one (E), where the G processes are age-corrected as in the WAIS FSIQ. Assume also that a memory measure taps those same general processes, but also a specifically memory one (M). Both measures (not processes) are similarly affected by age, independently of the G processes. Assume also that there is good evidence that patients may fail either/or executive or memory tests independently of age or general processing resources (as for instance, in the case of a pure amnesic). We might expect that in any population group there to be a relationship between memory test performance and executive test performance without this actually reflecting a relationship between the processes E and M. This may lead the unwary to conclude that there is some relationship between processes E and M when in fact there is not.

5.1(d) Prediction 4

This states that task-specific factors related to the demands made by particular tests might lead to correlations between performances on the memory and executive tests. These demands are not related to the intended purpose of the task - i.e. are not abilities generally regarded as either part of "memory" or "executive abilities".

Chapters 2 and 3 describe the development of two measures sensitive to frontal lobe involvement. Chapter 4 describes the development of a new memory test, together with the performance of anterior and posterior patients on traditional existing tests of memory. In addition the extent of the patients' pathology is known. Thus the data collected from chapters 2-4 is particularly relevant to studying the relationship between executive functioning (as measured by frontal tests) and memory test performance. Accordingly, these data will be used in an attempt to: a) test the validity of the dysexecutive explanation of frontal-lobe patients memory test
performance; and b) test the validity of the two methodological objections to the association methodology (predictions 3 and 4).

5.2 METHOD

The relationships between the memory and executive test scores of all unilateral patients from chapters 2-4 were examined. The following analyses do not include scores from the controls (except in the case of test validation) or the bifrontal patients.

5.3 RESULTS

5.3(a) Relationships Between Executive Measures

The relationships between the executive measures will be examined firstly from the point of view of the unilateral patients considered as a group, with the performances of particularly informative single cases considered afterwards. Overall correlations between the executive measures based on all the unilateral patients as a group, are shown in Table 20.

5.3(a)i Hayling Score and Brixton

The most striking correlation is that between Hayling Score and Brixton errors (0.478). However, as shown in chapters 2 and 3 (and reflected in the appropriate correlations in Table 20), both measures are significantly affected by age and FSIQ. Accordingly, the influence of both these factors upon both measures was removed from the correlation by partial correlation techniques (McNemar, 1949). This reduced the correlation to 0.324, which, whilst still significantly different from zero, indicates a rather low overall relationship between performance on the two tests. This was confirmed by construction of the 95% confidence intervals (.101; .573) which emphasise the overall weakness of the relationship.
5.3(a)ii Brixton and Hayling: Initiation Time

Likewise, the correlation between Brixton and Hayling I (ie. initiation condition) was a modest .271, which is significant. However, partialing out the effects of age and FSIQ upon both measures reduced this figure to .113, which is not significant at the 0.05 level for a one-tailed test. It would appear, therefore, that the rather weak overall correlation between the measures is for the greater part due to their mutual dependency upon background factors.

5.3(a)iii Hayling Initiation and Hayling Score

The correlation between Hayling I and Hayling Score was found to be .188, which is not significant. Moreover, partialing for age and FSIQ reduced this figure to .074. Clearly these two measures share little in common. This degree of independence is quite remarkable given that there are clearly common elements to each measure.

5.3(b) Dissociations Between Executive Measures

Test Reliability

While the overall correlations between the executive measures are low, the performance of individual patients is perhaps even more instructive. Table 20 shows the performance of 5 selected patients who showed the most striking dissociations between performances on the executive measures. Together, the patients suggest that double dissociations may exist between initiation, set formation and response suppression abilities.

However, consideration of the significance of correlations between measures requires firstly consideration of the reliability of those measures (McNemar, 1964). If both measures are unreliable, one would expect correlations between them to be low, giving
a false impression of orthogonality. This is particularly important in the consideration of individual difference scores (Anastasi, 1988).

Accordingly, split-half reliabilities were calculated for the control’s performance on the three measures, using the traditional odd-even method with Spearman-Brown estimates (Anastasi, 1988).

Under these circumstances, the split-half reliability of Hayling Initiation was found to be a modest 0.63, which is not particularly high but is comparable to the reliability of other traditional neuropsychological tests (eg. Coughlan and Hollows, 1985). The Hayling Score measure was somewhat more reliable at 0.78, which is rather better than, for instance, tests of verbal fluency (Anastasi and Drake, 1954).

Measurement of split-half reliability of the Brixton Test is somewhat more complex since individual trials cannot be seen as either equivalent or as independent of each other. The individual difficulty of any one trial will depend upon many factors, including the subject’s individual response history. These factors may tend to exaggerate the variability of a subject’s performance (when seen from the perspective of split-halves) and yet this tendency of the test is of course central to what is being measured. Accordingly, a traditional Spearman-Brown calculation may not be appropriate for this test, and may have the effect of underestimating the overall test reliability. Nevertheless, the split-half reliability of the Brixton calculated in this fashion was 0.65. This conservative estimate was used in the calculations of the significance of the individual differences reported in Table 20. However the Rulon (1939) method of calculating reliability may be more appropriate to this test since this requires only the variance of the differences between each person’s scores on the two half tests. Responses to items on the Brixton Test can hardly be considered as independent from each other, which would affect the Spearman-Brown coefficient, but not a
coefficient derived in this manner. Using Rulon's method, the split-half reliability of the Brixton Test was found to be 0.82, which is quite satisfactory.

5.3(c) **Significance of the Dissociations Between Executive Measures**

The significance of the differences in performance between the executive measures for the patients shown in Table 20 were calculated according to the method laid down by Anastasi (1988), using the reliability estimates outlined above.

Under these conditions, the differences in performance between all three executive measures were significant at the <0.01 level. Thus it would seem that double dissociations may exist between all three executive measures. Moreover, the only patients to show significant dissociations had frontal lesions (however the study includes considerably more frontal than non-frontal cases).

**Table 20**

**Patients Demonstrating Significant Individual Difference Scores on the Executive Measures**

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Age</th>
<th>FSIQ</th>
<th>NART</th>
<th>Brix</th>
<th>Hay:I</th>
<th>Hay:S</th>
<th>Diff$^1$</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF</td>
<td>47</td>
<td>108</td>
<td>107</td>
<td>62</td>
<td>-</td>
<td>2</td>
<td>60**</td>
</tr>
<tr>
<td>LF</td>
<td>28</td>
<td>89</td>
<td>95</td>
<td>76</td>
<td>8</td>
<td>-</td>
<td>68**</td>
</tr>
<tr>
<td>LF</td>
<td>49</td>
<td>104</td>
<td>111</td>
<td>0.1</td>
<td>-</td>
<td>82</td>
<td>&gt;82**</td>
</tr>
<tr>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>67</td>
<td>-</td>
<td>&gt;66**</td>
</tr>
<tr>
<td>RF</td>
<td>31</td>
<td>110</td>
<td>119</td>
<td>-</td>
<td>1</td>
<td>76</td>
<td>75**</td>
</tr>
<tr>
<td>LF</td>
<td>54</td>
<td>94</td>
<td>111</td>
<td>-</td>
<td>66</td>
<td>0.1</td>
<td>&gt;66**</td>
</tr>
</tbody>
</table>

Key: All executive measure scores are quoted as percentiles of the controls. Brix = Brixton Score (%ile), Hay:I = Hayling Initiation (%ile), Hay:S = Hayling Suppression Score (%ile).

$^1$ %ile differences between the scores.

$^2$ This patient is the same case as above.

** indicates differences between scores significant at the <0.01 level.
5.3(d) Relationships Between Memory Measures

The relationships between memory measures has been described in detail in chapter 4. In summary, both forced-choice measures were good predictors of performance on the other, but not of free recall or WOT performance. Free recall was heavily influenced by background factors (age and FSIQ) and the extent to which free recall was correlated with other memory measures was shown to be due to their mutual dependency on these background factors. WOT again was influenced considerably by age and IQ, and differing measures within the WOT were the best predictors of the other parts of the test.

While these analyses underline the danger of assuming that memory tests will be similarly impaired in any one patient, from the point of view of the later stages of the foregoing argument, only Warrington’s RMT performance will be considered. This is because it is widely accepted and cited, and performance on this measure (and a number of similar forced-choice measures) has been central to many of the single-case investigations of memory disturbances following frontal lobe damage. In addition, particular degrees of impairment on Warrington’s RMT have been cited as evidence of severity of amnesia (eg. Shallice, 1988) and some have considered this test as being accurate indicators of severity of amnesia in everyday life (eg. McCarthy and Warrington, 1990).3

5.3(e) Relationship Between Executive measures and Memory Test Performance

Correlations between measures are shown in Table 21. It is clear that there are some significant correlations.

---

3 It has been pointed out by some (eg. Coughlan and Hollows, 1985) that the link between memory test performance and severity of "real-life amnesia" has never been proven, and some contend that it is doubtful whether many experimental memory tasks are in fact tapping those processes that are damaged in amnesia (eg. Tulving, 1989). These issues will be explored in later chapters.
between certain of the memory and executive measures. For instance, Hayling Score is correlated with all four memory measures at the 0.01 level or better. Brixton performance is also significantly correlated with all memory measures, especially free recall. Hayling A, on the other hand, is not significantly correlated with either of the forced-choice measures, but is significantly correlated with WOT and Free Recall.

However many of these measures are also affected by the background factors (age, FSIQ and NART). Thus all the verbal memory measures (FCW, WOT and Free Recall) are significantly correlated with NART performance, all memory measures are significantly correlated with FSIQ; and FCF, WOT and Free Recall are correlated with age.

Similarly the background factors are obviously influential in patients' performance on the executive measures (as shown in chapters 2 and 3). For instance, the Brixton is highly correlated with age (.56). Neither Hayling measures are correlated significantly with FSIQ, but both are influenced by age.

Clearly then, some of the correlations between the executive and memory measures may be due to these background factors. However if the cognitive abilities measured by the executive tasks are also required in the memory tasks, and both sets of tests are similarly affected by the background factors, then executive test performance should be more predictive of performance on the memory tests than the background factors alone. In the case where the executive tests are sensitive to the background factors in a different way from the particular memory test in question, we might expect a combination of background factors and the executive tests to prove the best model of memory test performance.
### TABLE 21
Correlations Between Measures

<table>
<thead>
<tr>
<th></th>
<th>A/P</th>
<th>L/R</th>
<th>AGE</th>
<th>FSIQ</th>
<th>NART</th>
<th>BRIX</th>
</tr>
</thead>
<tbody>
<tr>
<td>L/R</td>
<td>0.04</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AGE</td>
<td>0.05</td>
<td>-0.21</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FSIQ</td>
<td>-0.08</td>
<td>-0.27*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NART</td>
<td>-0.03</td>
<td>-0.21</td>
<td>0.17</td>
<td>0.64***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BRIX</td>
<td>0.34***</td>
<td>0.02</td>
<td>0.56***</td>
<td>-0.29**</td>
<td>-0.07</td>
<td></td>
</tr>
<tr>
<td>HayA</td>
<td>0.11</td>
<td>-0.00</td>
<td>0.28*</td>
<td>-0.18</td>
<td>-0.10</td>
<td>0.27*</td>
</tr>
<tr>
<td>HayS</td>
<td>0.33***</td>
<td>-0.04</td>
<td>0.37***</td>
<td>-0.21</td>
<td>-0.14</td>
<td>0.48***</td>
</tr>
<tr>
<td>FCW</td>
<td>-0.11</td>
<td>-0.17</td>
<td>-0.16</td>
<td>0.36***</td>
<td>0.27*</td>
<td>-0.27*</td>
</tr>
<tr>
<td>FCF</td>
<td>0.02</td>
<td>0.12</td>
<td>-0.33***</td>
<td>0.24</td>
<td>0.03</td>
<td>-0.38***</td>
</tr>
<tr>
<td>WOT</td>
<td>0.13</td>
<td>0.03</td>
<td>0.29**</td>
<td>-0.38***</td>
<td>-0.27*</td>
<td>0.31***</td>
</tr>
<tr>
<td>Recall</td>
<td>-0.18</td>
<td>-0.11</td>
<td>-0.37***</td>
<td>0.52***</td>
<td>0.31***</td>
<td>-0.49***</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>HayA</th>
<th>HayS</th>
<th>FCW</th>
<th>FCF</th>
<th>WOT</th>
</tr>
</thead>
<tbody>
<tr>
<td>HayS</td>
<td>0.19</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FCW</td>
<td>-0.07</td>
<td>-0.42***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FCF</td>
<td>-0.09</td>
<td>-0.45***</td>
<td>0.56</td>
<td></td>
<td></td>
</tr>
<tr>
<td>WOT</td>
<td>0.35***</td>
<td>0.34***</td>
<td>-0.34</td>
<td>-0.30**</td>
<td></td>
</tr>
<tr>
<td>Recall</td>
<td>-0.37***</td>
<td>-0.44***</td>
<td>0.38</td>
<td>0.28*</td>
<td>-0.38***</td>
</tr>
</tbody>
</table>

Key: L/R = Left or right hemisphere; A/P = anterior or posterior lesion; FSIQ = Full Scale IQ (WAIS); NART = National Adult Reading Scale; Brix = Brixton Test Errors; HayA = Hayling A Initiation) response latency in secs.; HayS = Hayling Test Error Score; FCW = Forced-Choice Words; FCF = Forced-Choice Faces; WOT = Word Order Test combined trials 1+2; Recall = Free recall measure from Chapter 4.

* = p <0.05  
** = p <0.02  
*** = p <0.01

Accordingly, an analysis by stepwise regression (using the standard maximum F statistic procedure) was conducted for each of the memory measures. Each memory test was examined across all the unilateral patients for the

4 N for each correlation varies from n = 60 to n = 87 depending on number of missing data points.  
5 An analysis by best fit modelling was also conducted for all the analyses to be described here, but since the results were essentially the same the more conventional backward elimination results will be presented.  
6 Analyses were also performed for the anteriors and posteriors separately. However the results were remarkably similar for each group so the analyses
influence of the following factors: anterior/posterior classification; left/right hemisphere; age; FSIQ; NART, and the three executive test measures: Brixton, Hayling A, and Hayling Score. The results are given in Table 22.

### TABLE 22

**Factors Most Predictive of Performance on Memory Test Measures**

<table>
<thead>
<tr>
<th>Measure</th>
<th>Sig. Predictors</th>
<th>p</th>
<th>$R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>FCW</td>
<td>FSIQ</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hay. Score</td>
<td>&lt;0.005</td>
<td>25.2</td>
</tr>
<tr>
<td>FCF</td>
<td>Hay. Score</td>
<td>&lt;0.01</td>
<td>22.9</td>
</tr>
<tr>
<td>WOT</td>
<td>Hay. Score</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSIQ</td>
<td>&lt;0.001</td>
<td>31.1</td>
</tr>
<tr>
<td>Free Recall</td>
<td>AGE</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FSIQ</td>
<td>&lt;0.001</td>
<td>51.2</td>
</tr>
</tbody>
</table>

As shown in Table 22, the only executive measure to appear as significantly predictive of a measure of memory was the Hayling Score. This was more predictive of performance on the forced-choice measures than the other factors. Taken in isolation, this may appear to suggest that executive abilities (at least as measured by the Hayling) play a role in determining forced-choice memory test performance. However there are two points to be made about this finding. The first is related to hypothesis 1. This is that the correlations between FCW and FCF and the Hayling (-.416 and -.452 respectively) are not particularly high. Moreover, when the effects of FSIQ and age upon both measures was removed by partial

---

7 Of factors: anterior/posterior; left/right hemisphere; age; FSIQ; NART; Brixton score; Hayling A; Hayling score.

8 Note however that the Hayling (or any other executive measure) is never a better predictor of performance on a given memory test than is performance on at least one of the other memory measures (see chapter 4).

9 Note that while FCW is significantly correlated with FSIQ, FCF is not. In addition, FCF is significantly
correlation techniques, the correlation between FCW and Hayling Score fell to .36, which indicates little relationship between the measures, with only 12.6% shared variance. This is underlined by the construction of the 95% confidence intervals for this association (-.21, -.68) - were the figure of .36 to reflect an actual relationship as low as -.21, one would hardly regard this as remarkable.

Similarly, in the case of the correlation between Hayling Score and FCF (.45), removing the effects of age and FSIQ upon both measures by partial correlation reduced the correlation to -.35. Again, construction of the 95% confidence intervals for this figure (-.13, -.60) demonstrates that whilst significantly different from zero, one can hardly regard the magnitude of the correlation between the Hayling and FCF as suggesting, beyond doubt, a strong association.

Construction of partial correlations between other memory tests and executive measures demonstrated more clearly the contribution of factors such as age and FSIQ to the correlation coefficients shown in Table 21. Thus while the Brixton-Free Recall correlation is .51, suggesting quite a strong association between them, partialing for age and FSIQ reduced this figure to .17, which is not significantly different from zero. Likewise, the correlation between FCF and Brixton is .38, which is significant at the 0.01 level. However removing the effects of age and FSIQ upon both variables reduced this figure to .18, which is not significant.

Nevertheless, whilst the group data do not suggest a strong association between the executive process measured by the Hayling and the processes tapped by the forced-choice measures (and the WOT), the data is hardly correlated with age, whereas FCW is not. Obviously there are individual characteristics of these tasks which make their relationships with background variables quite complex.
unequivocal. Less equivocal, however, is the data from individual subjects which will now be considered.

5.3(f) The Dysexecutive Explanation: Testing Predictions 1 and 2.

The dysexecutive explanation makes two predictions which were tested with the data from the present study.

Dysexecutive Explanation - Prediction 1: Patients who fail executive tests will also fail memory tests

This was not strongly supported by the present data. As shown above (Table 22), and considering all the unilateral patients as a whole, performance on the executive tests was not generally a good predictor of memory test performance. However it may be the case that the relationship between memory test and executive test performance is not simply linear, and that some threshold needs to be exceeded before the executive impairment is of sufficient severity to affect the memory test performance. Accordingly, those individual cases who performed particularly poorly (ie. 2 standard deviations or more below the mean of the controls) on the various executive measures were considered with respect to their memory test performance.

It is clear that the data at the individual case level supports the overall regression analysis. Individual patients (all with frontal lesions, coincidentally) could be found who failed the executive tests but who performed Warrington’s RMT at a normal or near-normal level (see Table 23)
### TABLE 23

Selected Individual Cases Who Show Poor Executive Test Performance with Relatively Preserved Forced-Choice Memory Test Performance

<table>
<thead>
<tr>
<th>Site</th>
<th>Age</th>
<th>FSIQ</th>
<th>NART</th>
<th>Hay:I</th>
<th>Hay:S</th>
<th>Brix</th>
<th>FCW</th>
<th>FCF</th>
</tr>
</thead>
<tbody>
<tr>
<td>LF</td>
<td>54</td>
<td>115</td>
<td>119</td>
<td>&lt;0.1*</td>
<td>75</td>
<td>NA</td>
<td>90</td>
<td>&gt;95</td>
</tr>
<tr>
<td>RF</td>
<td>68</td>
<td>120</td>
<td>110</td>
<td>86</td>
<td>&lt;0.1*</td>
<td>&lt;5*</td>
<td>&gt;50</td>
<td>&gt;50</td>
</tr>
<tr>
<td>LF</td>
<td>49</td>
<td>104</td>
<td>111</td>
<td>66</td>
<td>82</td>
<td>&lt;0.1*</td>
<td>&gt;50</td>
<td>75</td>
</tr>
<tr>
<td>RF</td>
<td>59</td>
<td>98</td>
<td>122</td>
<td>&lt;1*</td>
<td>&lt;0.1*</td>
<td>&lt;0.1*</td>
<td>&gt;75</td>
<td>&gt;25</td>
</tr>
</tbody>
</table>

Scores for memory and executive tests are given in percentiles of the control performance. FCW + FCF percentiles are age-corrected.

Key: * indicates impaired performance. Site = site of lesion; R = right; L = Left; F = frontal; Hay:I = hayling Test, Initiation condition; Hay:S = Hayling Score; Brix = Brixton Test; FCW + FCF = Warrington’s (1984) Forced-choice words and faces tasks.

Table 23 shows three individual cases who failed, singly, one of the executive measures and yet performed normally on the forced-choice tasks. In each case the difference in scores between the impaired executive measure and both the forced-choice measures is significant at the <0.05 level or better.\(^1\)

In addition, individual cases were found who performed all three executive measures at or below 2 sd below the mean of the controls (only 22 of the 99 patients for whom data were available) and thus could presumably be described as "severely dysexecutive"\(^1\) (Baddeley and Wilson, 1988). One of these patients scored at a rather poor level on Warrington’s RMT (38/50 words; 42/50 faces), but the other performed quite creditably for his age (see Table 23).

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\(^1\) Using the reliability estimates for the executive tests as already given, and the reliability scores for the FCW and FCF taken from Coughlan and Hollows (1985)

\(^1\) Baddeley and Wilson do not give fully articulated criteria for the term "severely dysexecutive".

---
Dysexecutive Explanation - Prediction 2

Prediction 2 stated that frontal lobe patients who fail memory tests will also have executive problems.

TABLE 24

Executive Test Performances of Selected Frontal Lobe Cases Showing Severely Impaired Performance on Forced-Choice Measures

<table>
<thead>
<tr>
<th>Site</th>
<th>Age</th>
<th>FSIQ</th>
<th>NART</th>
<th>Brix</th>
<th>Hay:I</th>
<th>Hay:S</th>
<th>FCW</th>
<th>FCF</th>
</tr>
</thead>
<tbody>
<tr>
<td>LF</td>
<td>63</td>
<td>105</td>
<td>105</td>
<td>&lt;0.1*</td>
<td>62</td>
<td>7</td>
<td>&lt;5*</td>
<td>&lt;5*</td>
</tr>
<tr>
<td>LF</td>
<td>28</td>
<td>94</td>
<td>115</td>
<td>27</td>
<td>69</td>
<td>1</td>
<td>&lt;5*</td>
<td>&lt;5*</td>
</tr>
<tr>
<td>LF</td>
<td>50</td>
<td>102</td>
<td>110</td>
<td>5</td>
<td>66</td>
<td>&lt;0.1*</td>
<td>&lt;5*</td>
<td>&lt;5*</td>
</tr>
<tr>
<td>RF</td>
<td>45</td>
<td>111</td>
<td>110</td>
<td>50</td>
<td>14</td>
<td>2</td>
<td>&lt;5*</td>
<td>&lt;10</td>
</tr>
</tbody>
</table>

Key: As Table 20.

Accordingly, the executive test performances of those frontal patients who performed at or below the 5%ile for their age on the memory tasks was examined, and are shown in Table 24.

There was no example of a frontal patient who had a severely impaired memory test performance and who was normal on all three executive test measures. However 3/4 of these patients were impaired on only one of the three measures, and these differed amongst patients. It is certainly not the case, then, that memory-test impaired patients with frontal-lobe lesions are necessarily severely dysexecutive, and the relationship between executive test (or at least, the executive tests used here) and memory test performance is not at all straightforward. In fact there is good evidence, when Tables 23 and 24 are considered together, for double dissociations of function between these executive measures and forced-choice memory performance.
The Methodology Explanation: Testing Predictions 3 and 4

Methodology: Prediction 3

Prediction 3 stated that the association between memory and executive test performance is due to their mutual dependency upon background measures. This is largely confirmed by the analysis shown in Table 22. In the case of the Free Recall measure, none of the executive tests were significant predictors of performance at any time in the regression equation. This is supported by the basic correlations, which show that Free Recall is more highly correlated with FSIQ (for instance) than it is with any of the executive measures. In addition, the potential significance of the Hayling Score measure as a predictor of the forced-choice measures (cf. Table 21) is cast seriously into doubt by the patients reported in Tables 23 and 24 who show double dissociations between the measures.

As reported above, certain of the quite strong correlations between the executive measures and the memory tests (eg. FCF-Brixton; Free Recall-Brixton) could be shown to reduce to levels not significantly different from zero once factors such as age and FSIQ were taken into account. At least in those cases, then, background variables can be shown to be influential in the relationships between executive and memory measures. When one considers that there will be many other factors such as verbal comprehension skills, reading ability, or even perceptual abilities (cf. the Brixton-FCF correlation) which have not been measured here but which may affect associations between executive and memory measures, the already low $R^2$ values from the stepwise regression start to look even less meaningful in terms of a direct relationship between executive and memory processes.

One possible interpretation is that the modest correlations between these memory measures and the executive tests are indeed for the most part due to their
mutual dependency upon the background factors. The performances of the individual cases would be particularly difficult to explain if one were to suggest that, for instance, the Hayling taps some process which must be functioning in order for appropriate memory test performance to occur.

Methodology: Prediction 4

Prediction 4 stated that task-specific factors related to the demands made by the particular tasks might lead to correlations between performances on the memory and executive tasks which are not related to the way in which the executive test is being used (ie. as a measure of "frontality".

As shown in Table 22, the only executive measure to appear as significantly predictive of a measure of memory was the Hayling Score. This was more predictive of performance on the forced-choice measures than the other factors. Taken in isolation, this may appear to suggest that executive abilities (at least as measured by the Hayling) play a role in determining forced-choice memory test performance. However there are two points to be made about this finding.

The first is that the correlations between FCW and FCF and Hayling Score (.42 and .45 respectively) are not particularly high. Moreover when the effects of age and FSIQ upon both measures was removed by partial correlation techniques, the correlation between FCW and Hayling Score fell to .36, which indicates little relationship between the measures. This was underlined by the construction of the 95% confidence intervals for this measure (-.21; -.68) which emphasised the potential overall weakness of the relationship.

Similarly, in the case of the correlation between Hayling Score and FCF (.45), removing the effects of age (the most important background variable influencing both measures) by partial correlation reduced this figure
considerably, demonstrating that one can hardly regard the magnitude of the correlation between the Hayling Score and FCF as suggesting, beyond doubt, a meaningful association.

The second point regarding the Hayling-Forced-choice tests correlation is perhaps more directly pertinent to Prediction 4. This is that, as can be seen from Table 21, the point-biserial correlations between anterior-posterior classification and the forced-choice measures are very low (which is what would be expected from the data presented in chapter 4).

Applying this finding to use of the association methodology raises an interesting possibility. If one were to use the association methodology in the study of memory test performance of mixed pathology (ie. not merely frontal) patients or without data regarding the localisation of the pathology, one might be expected to find an association between "frontal symptoms" (measured, say, by the Hayling Score) and memory test performance. One might be tempted to draw the conclusion that these associations reflect the role of the frontal lobes in memory. However as can be seen from the data here the association between executive test performance and memory test performance may actually be significant when the correlation between actual lesion site and memory test performance is far from significant.

5.4 DISCUSSION

Three broad topics are raised by the foregoing results. The first is the degree to which the present data supports the objections to the association methodology raised in the Introduction to this thesis. The second is the issue of localisation and memory - the question of

12 Just such a conclusion is reached by such authors as Leng and Parkin (1988), Squire (1982), Schacter, Harbluk and MacLachlin (1984) and Schacter (1986), using this experimental design.
whether patients differ in their memory performance according to the site of their lesion. The last topic is that of the inter-relationship between executive functions and memory test performance. These will be dealt with in turn.

5.4(a) Methodological Factors in Considering Relationships Between Executive and Memory Measures.

The results presented in the current chapter support the view that apparent relationships between memory and executive test performance (at least in patients with unilateral lesions) may appear for two reasons.

Firstly, general factors influencing test performance (eg. current overall IQ, age, pre-morbid IQ levels) may lead to correlations in test performance. When these factors are removed, however, it becomes clear that these apparent associations are weaker than at first appears.

Secondly, the influence of task-specific factors may lead to correlations between memory and executive measures. That is to say that any neuropsychological test will make demands upon processing systems which are incidental to the main focus of the task.

Consider the correlations found between the Hayling Task and the Forced-Choice measures. It may be the case that the RMT taps processes which are held anteriorly, plus some process(es) held posteriorly. If the anterior process(es) required for satisfactory RMT performance were the same as those tapped by the Hayling, then one might indeed obtain the overall group results shown here. However one would not expect to find statistically significant dissociations in performance between the measures in individual cases unless the anterior process contributes negligibly to RMT performance. If this were the case, one would not expect the strength of association between the tasks shown in the group data.
Additionally, while Hayling Score was a significant predictor of forced-choice memory performance, and the point-biserial correlations between the Hayling Score and anterior/posterior classification was significant, the point-biserial correlations with the forced-choice tasks was not significant. Moreover, the actual correlation between Hayling Score and the forced-choice tasks was rather low once the effects of age and FSIQ upon both measures was taken into account.

The most plausible account of these findings is that task-specific factors (which were in the main not tested here) may lead to correlations between executive and memory measures, where these factors are incidental to the main "purpose" of those tasks.

The pattern of correlations between anterior-posterior classifications, the executive measures and the memory tests does, however, demonstrate a final and perhaps crucial point regarding the legitimacy of inferences made about correlations between measures. Specifically, if X (in this case, say, performance on an executive task) is correlated with Y (in this case anterior/posterior classification) and X is correlated with Z (ie. memory test performance), it does not necessarily follow that Y is correlated with Z\(^{13}\) (Jonckheere, personal communication). The relationship between Y and Z must be demonstrated directly.

The danger with index correlations of these sort is not obscure. Consider for instance the dummy data presented in Table 25. Here the memory test "scores" and the MRI classification (which is not shown as an index correlation for the sake of clarity, but is intended to represent some measure of extent of damage to the frontal lobes measured by, say, MRI scan) have been taken from a table of random numbers. It is hardly surprising that the low correlation between them therefore does not approach

\(^{13}\) In fact if \(r_{12}\) and \(r_{13}\) are known, the limits for \(r_{23}\) may be calculated as \(r_{12}r_{13}/\sqrt{1-r_{12}^2-r_{13}^2+r_{12}^2r_{13}^2}\) (McNemar, 1969).
significance (t=0.95 df=14). However the executive measure "scores" and the measure of frontal lobe damage are highly (and significantly) correlated, as are the scores on the memory test and the executive test. This dummy data illustrates clearly the logical flaw with the association methodology.

Table 25

Fictional Data to Illustrate the Logical Flaw Inherent in the Association Methodology

<table>
<thead>
<tr>
<th>MRI &quot;Frontality&quot;</th>
<th>Memory Score</th>
<th>Executive Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>9</td>
<td>11</td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>10</td>
</tr>
<tr>
<td>6</td>
<td>4</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>1</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>1</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>6</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>6</td>
<td>11</td>
</tr>
<tr>
<td>1</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
<td>8</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>6</td>
</tr>
</tbody>
</table>

Correlation Matrix for Data Above:

<table>
<thead>
<tr>
<th></th>
<th>Frontality</th>
<th>Memory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Memory</td>
<td></td>
<td>0.25</td>
</tr>
<tr>
<td>Executive</td>
<td>0.71**</td>
<td>0.85**</td>
</tr>
</tbody>
</table>

** p<0.01

Together, these objections mean that it is not legitimate to assume that because frontal patients fail executive tests and show a particular pattern of memory performance, that other types of patients who show similar executive and memory test performance do so because they have frontal involvement. Nor even would it be appropriate to assume, where a high correlation exists between executive and memory tests, that frontal lobe patients who fail the memory tests do so because of their "frontality". The problem quite obviously lies with the
use of executive tests as measures of "frontality" (a concept which was discussed in the thesis introduction).

The case obviously gets weaker still where the relationship between executive and memory test performance cannot be consistently demonstrated in frontal patients - or even as was the case here, that executive and memory test performances can be seen to dissociate. Yet this is exactly the sort of inference made, without qualification, by many of those who use the association methodology. For instance, Parkin and Walter (Psychobiology, in press) administered a Brown-Peterson Task and a recognition memory task to a group of normal young and a group of normal old subjects. They also gave their subjects the WCST and a Verbal Fluency (FAS) task. Parkin and Walter found significant (but modest) correlations between the elderly subjects’ performances on the Brown-Peterson paradigm and their performance on the WCST. They concluded that their age-related differences in BP performances reflected a deficit in acquisition plus "a retrieval deficit stemming from the frontal atrophy known to be associated with normal aging". This is quite obviously not a legitimate conclusion to draw from their results. However it is not uncommon for researchers using such an association methodology to draw similar conclusions (see for instance Kopelman, 1991; Robbins, 1989; Leng and Parkin (1988); Squire (1982); Schacter, Harbluk and MacLachlin (1984); and Schacter (1986)).

5.4(b) Localisation and Memory

As reported in chapter 4, no significant differences in memory performance were noted between the anteriors and posteriors. This was despite the fact that, using the same group of patients, anterior-posterior differences did emerge on the executive measures. Of particular note was the replication of Warrington’s (1984) finding of no significant within-hemisphere differences in forced-choice memory performance. This suggests that the argument given by Mayes (1988) that the lack of an
anterior-posterior forced-choice memory effect was due to a small group of frontals having additional posterior involvement, is an unlikely explanation of Warrington’s findings. For the moment, the best evidence is that, within hemispheres, there is no specific region which can be unequivocally associated with a given performance on these measures.

An interesting finding at the single-case level was that a few patients in this series performed both FCW and FCF at chance levels. The available evidence suggested that their lesions were unilateral (and frontal). If Shallice’s criteria for classification of severity of amnesia were applied to these patients, they would be classed as severely amnesic. Severe amnesia is, of course, generally regarded - in cortical cases at least - as only following from bilateral lesions. This suggests that either these cases had more widespread damage than was detected by their radiographical investigations, or else that memory test performance is not always a reliable measure of amnesia (see Tulving, 1990). This is of course the same conclusion as might be drawn from Delbecq-Derouesne et al’s case, whose recognition memory performance was essentially at chance, and yet was not amnesic in everyday life. Quite obviously the situation is not as simple as McCarthy and Warrington (1990) suggest when they say that (in reference to Warrington’s [1974] RMT test) "there is now no dispute that these quantitative measures of memory function provide an index of the impairment of event memory which corresponds with the difficulties which the patients experience in everyday life" (p. 299). The fact that most cases who are amnesic in everyday life perform memory tests poorly does not mean that all people who perform memory tests badly are amnesic. Instead, as Coughlan and Hollow’s (1985) point out, the validity of memory tests as an indicator of amnesia has yet to be proved. In any case it is instructive to note that in certain cases patients with apparently circumscribed unilateral cortical lesions may show a pattern of performance on traditional memory tests
which is usually associated with severe amnesia of the classical type.

5.4(c) Relationship between executive test and memory test performances

At the group level, performance on the executive measures was found to be a rather weak predictor of performance on the memory tests. While certain executive measures (e.g., Hayling Score) variables were significant predictors in the regression analyses (albeit only when other memory test scores were not entered) the amount of variances which they explained were typically very low. It should be remembered that this chapter presented the data using only the background variables plus the executive measures as predictors of memory test performance. However, chapter 4 showed that the best actual predictors of performance on a memory test were generally performance on other memory tests. The variances explained by the regression equations presented in chapter 4 were considerably higher than those obtained here.

In addition, consideration of individual cases showed that all those with very poor memory were impaired on at least one of the executive measures, but not all patients with very poor executive functions had similarly impaired memory test performance. The relationship between these measures, if any does in fact exist (and the dissociations between individual measures suggests that it does not), is obviously straightforward.

At both the group and single case level the data does not support the suggestion that frontal patients fail memory tests due to their impaired executive skills - at least not those measured by the Brixton and Hayling tests. The interpretation most parsimonious with the results presented here is that where memory and executive tests are impaired together in the same case, this is either due to that patient having both executive and memory problems, or else is due to the mutual influence of
background factors upon both executive and memory measures.

5.4(d) The "dysexecutive explanation" vs the "methodology explanation".

These two explanations of frontal patients' memory test failures in frontal patients can now be evaluated. The dysexecutive explanation suggests that patients with frontal lobe lesions fail memory tests because their executive problems somehow interfere with their realising their "potential" on memory tests. As we have seen, this explanation makes two predictions, both of which were not supported by the present data. There are two possible conclusions. Either the dysexecutive explanation holds true, but the Hayling and Brixton tasks do not tap those abilities to which advocates of this explanation are referring, or else the dysexecutive explanation is inadequate.

The criticism that the executive tests used here are not measuring similar processes as such tests as, for instance, the WCST or Cognitive Estimates or the Tower of London cannot of course be directly refuted. However advocates of the dysexecutive explanation (eg. Stuss and Benson, 1986) generally refer to a number of different "frontal tests" or broad references to dysexecutive symptoms without specifying particular tests or processes. It is hard to see why it should be that all other "frontal tests" measure those processes which affect memory tests but that, by pure coincidence, the Brixton and hayling do not, especially in view of their prima facie similarity, and given that the two tests used here are greater in their discriminatory power than most (if not all) existing "frontal" measures (see the Introduction to the thesis). Overall, the dysexecutive explanation does not look a convincing explanation of frontal lobe patients' memory test failures.

The methodology explanation also made two predictions (3 and 4). The first was that background factors might
affect both executive and memory measures, leading to "spurious" correlations between them. This was supported by the present data, at least as regards certain of the memory measures.

The other prediction (prediction 3) was that task specific factors not central to the abilities purported to be measured by the executive or memory tasks would lead to associations between executive and memory measures without these actually reflecting an executive/memory co-relation. This prediction could not be formally demonstrated in the data, but nor could it be discounted. It remains a possibility.

5.4(e) Conclusion

The analyses presented in this chapter in many ways raise as many questions as they answer, and serve overall to demonstrate the complexity of the theoretical and psychometric questions that are involved in exploring the relationships between test measures. However certain findings remain clear.

There was no suggestion that anteriors who fail memory tests show different neuropsychological profiles (at least with the measures used here) from posteriors. In addition, single cases may fail the executive measures used here and still be able to perform the RMT satisfactorily. One case performed all three executive indices below the 1%ile, and yet still managed a high average forced-choice words performance (although his faces performance was low average). This is of particular note since Baddeley and Wilson (1988) suggest that such a case would be evidence contrary to the dysexecutive hypothesis of frontal amnesia.

Moreover, single cases may perform both RMT measures at or below the 5%ile for their age, and yet still be able to achieve average, or above average performance on any of the executive measures used here. However no case was found who performed the RMT very poorly in the context of
above-average performance on all three executive measures. Nevertheless if one were to characterise the processes tapped by the executive measures as being initiation, response suppression and rule detection, one would either have to accept that these processes are at best only marginally required for successful memory test (especially forced-choice) performance, or postulate a very complex argument about the relationships of memory and these executive processes. As yet there would be no direct empirical support for such an argument.

Overall there was some relationship between performances on certain of the executive and memory measures in the patients seen as part of this study. Some of these inter-relationships were seen to be the direct result of the effects of "background variables" upon both types of measure. Other correlations could not be so easily dismissed. However it remains a possibility that task-specific factors not examined here were at work in these cases. Certainly it is not legitimate to suggest that since there was some association between, say, the forced-choice tasks and Hayling Score, and that there is an association between frontal pathology and Hayling Score, that frontal (or any other patients) who fail forced-choice measures do so because of their pathology.

Additionally this study found little relationship between one measure and another, whether they be executive or memory tests. These results, and the results of previous chapters, suggest that the dysexecutive explanation of frontal lobe patients' memory test performance is inadequate in many respects.

The numerous arguments presented here and in the Introduction to the thesis suggest that the use of the association methodology is inadvisable. If these criticisms are accepted, this means that many of the recent studies which have used such methods, and have advocated that frontal lobe dysfunction lead to particular patterns of memory test performance, should be regarded with considerable caution. In all probability
the only way to ascertain with any degree of certainty that frontal lobe dysfunction can lead to certain types of memory test impairment is to test patients who are known to have frontal lobe damage, and to compare them directly with a non-frontal lesion group matched scrupulously for background variables. Few studies fulfil these criteria.

Yet it is an undeniable fact that some frontally-damaged patients show certain types of memory disorder that do not occur in patients with exclusively posterior damage (eg. paramnesias, confabulation). If the dysexecutive explanation can be disregarded, this leaves explanations which are rather more complex in terms of their articulation of the relationship between memory tests, amnesia and the cognitive symptoms shown by frontal lobe patients. These vary from the fully-articulated "frontal amnesia" hypothesis of Shallice (1988) to the rather more cautious approach taken by Mayes (1988). These ideas will be developed in the remaining sections of the thesis.
6.1 Introduction

Patients who have suffered damage to the frontal lobes may, as we have seen, show a number of forms of cognitive deficit. However many frontal lobe patients may perform normally on standard psychometric tests (Teuber, 1964; Stuss and Benson, 1986). This does not of course necessarily mean that they have no deficit, only that they performed normally on those tests which they were given.

While some of these "psychometrically normal" patients make good recoveries from their illnesses and return to a life situation not appreciably different from their pre-morbid state, other patients with apparently similar psychometric profiles find themselves unable to return to their life as it used to be (Brickner, 1936; Ackerly and Benton, 1947).

For instance, Eslinger and Damasio (1985) describe the case of a previously successful accountant (EVR) who, aged 35, suffered a large orbitofrontal meningioma, which was removed surgically. He made an uneventful recovery and was discharged 2 weeks after his operation.

Three months later he returned to accounting and bookkeeping for a small home construction business. However he soon became involved in a business partnership which collapsed, leaving him bankrupt. He then drifted through several jobs which should have been well within his capabilities, but was fired from each. His wife left him, taking their children, and EVR moved in with his parents.

Subsequently his life situation did not improve. Moreover, he was unable to make even the simplest decision, and choosing a restaurant would require travelling around checking all the possibilities and
considering the seating plan, menu, atmosphere and so forth in great detail. Even then he was often not able to come to a decision.

Eslinger and Damasio observe that "As he awoke, there was no evidence that an internal automatic program was ready to propel him into the routine daily activities of self-care and feeding, let alone those of travelling to a job and discharging the assignments of a given day. It was as if he "forgot to remember" short and intermediate-term goals\(^1\). If these goals were presented externally and repeatedly, they triggered the expected actions. But when the external recall mechanism provided by relatives or friends failed, or when the environment failed to challenge him with situations that demanded a response, he resumed his relatively goal-less, unpressured existence (pp. 1738-40).

However, despite these obvious everyday difficulties, EVR performed normally on a wide range of psychometric tests, including those known to be sensitive to frontal lobe lesions. In addition, EVR had no detectable psychiatric condition.

It is clear, therefore, that these psychometric tests were not tapping whatever processes are damaged in patients such as EVR. Eslinger and Damasio's (1985) characterisation of EVR’s problems suggests that he had little difficulty with situations in which goals were presented to him "externally and repeatedly" by the environment. The problem arose when he had to set himself goals and remember to carry them out. In these situations he "forgot to remember" such goals.

If we consider the format of traditional executive tests, it is apparent that they require little from the patient in terms of these processes of goal-setting and their subsequent spontaneous realisation. Thus the start and end of traditional frontal lobe tasks are clearly

\(^1\) Present writer's emphasis
indicated, what constitutes success or failure is apparent, and psychometric tests generally do not require the patient to remember to carry out intentions at a future time. It is possible, then, that the processes involved in such goal-setting and goal-realisation are subserved by the frontal lobes, and that these are the processes selectively damaged in patient EVR.

However, the creation of an intention clearly requires some preliminary processing or planning. At the very least this must consist of cursory consideration of future contingencies. This type of "forward thinking" is self-evidently a routine part of many everyday activities.

Consider for example the organisation and planning that is required by a straightforward task such as preparing a meal. Planning is required at the preliminary stages, such as purchasing the food and ensuring that one has all the necessary ingredients. Then within the preparation of the meal itself, the cooking times of the different foodstuffs need to be considered in order to ensure the correct progression of cooking stages, and many intentions need to be created and realised at the appropriate (future) moment. If one considers other factors such as cost, tastes and the quantity of food required, it becomes apparent that even such a commonplace activity requires much in the way of executive skills.

If it is the case that traditional neuropsychological tests do not tap the particular executive processes involved in such activities, then it should be possible to construct tests which do. We would then expect patients with similar profiles to EVR to perform particularly poorly on these tests, despite normal or near-normal traditional neuropsychological test performance.
6.2(a) Case 1.

A.P., a right-handed man, was involved in a road traffic accident at the age of 23 years, approximately 4.5 yrs before the present investigation. He sustained a serious open head injury involving a complicated anterior fossa fracture and an open wound in the forehead. He was admitted to the Centre Hospitalier Regional et Universitaire de Marseille in a state of semi-consciousness but without focal neurological signs. A CT scan showed evidence of extensive bifrontal damage. Following an operation by M. Guillerman his state of consciousness improved. A second CT scan showed evidence of bifrontal contusions. He was transferred to the Kent and Sussex Hospital where he was noted to be leaking CSF from the left nostril. He was demanding and difficult, had a bilateral anosmia but was fully conscious with a PTA estimated at two and a half weeks. He was transferred to the Brook Hospital and had a second operation carried out by Mr J. R. Bartlett to stem the leak in the CSF. His condition gradually improved but he had concentration problems.

After approximately a year recovering, he returned to college to complete his training. However his work was consistently late, incomplete and disorganised and he was soon forced to leave college. The following year he was admitted to St. Andrew’s hospital, Northampton, for rehabilitation. He was well-motivated and keen but could not carry out even the simplest activity because of an inability to keep his mind on the task in hand. For example, on one occasion he was discovered on the local golf course having originally stepped outside the therapy room to fetch some coffee. At night he commonly drove 6-8 hours until daybreak having originally intended to use his car to drive the short distance to a local cafe. At these times he maintained that he "completely forgot" whatever it was that he was supposed to be doing. He was
unable to shop for himself because he would buy one item at a time returning to his car after every purchase.

Rehabilitation on a day-patient basis had limited success, and after three months AP was transferred to another rehabilitation centre as an in-patient. There he was treated with behavioural methods describe by Wood and Burgess (1988) with a slow but progressive improvement in his ability to organise his activities of daily living. He remained in rehabilitation for approximately a year and then returned home to live with his parents.

In response to a clinical interview, he said that before the accident his room was immaculate ('5/5 tidiness') but now it is untidy with 'hotchpotch piles of magazines' on the floor. He had an efficient filing system but had abandoned it. Shopping, cleaning and laundry are done for him. His social life is very rarely organised in advance. When questioned he said that he had nothing planned for the coming weekend, and was unable to give any example of consciously organising an activity beforehand.

6.2(a)i Clinical Neuropsychological Investigations

A.P was tested at St. Andrew's Hospital and then at the National Hospital on a wide range of neuropsychological tests. On almost all he performed satisfactorily. At St. Andrews Hospital he obtained a Verbal IQ of 129 and a Performance IQ of 128, which corresponds well with the estimate of 124 for his pre-morbid IQ as measured by the NART.

There was one exception - the Digit Span subtest where he scored only in the Dull Normal range; however on a repeat testing two years later he scored in the Superior range on the subtest. His performance on other subtests was slightly better, which is probably a practice effect (see table 1). His spontaneous speech was not at all aphasic and on all perceptual, language and arithmetic tests carried out he performed satisfactorily (see table 1).
On memory tests he performed well on the Warrington and Wechsler batteries (Warrington, 1984; Wechsler, 1955), on complex figure copy and recall (Osterreith and Rey, 1944) (92%) and on Rey’s (1964) Auditory Verbal Learning (all 15 items recalled after 3 trials). The one memory test where his performance was below average was on the Petrides and Milner (1982) self-ordered pointing. On the picture version he made 7 errors by comparison with a mean for their control subjects - who were of comparable age - of 3 errors (however he was within one SD of the mean of a posterior lesion control group (see table 2)). On all of a sizeable group of tests thought sensitive to frontal lesions he performed very satisfactorily indeed (see table 2).

In summary, AP performed very well on a wide range of perceptual, memory, language and frontal lobe tests. There was one borderline exception - his performance on Self-Ordered Memory.

6.2(b) Case 2: DN

DN, a 49 year-old right-handed ex-university lecturer, had sustained a serious head injury in a car crash in 1964, involving a depressed skull fracture and an intracerebral haematoma treated at operation. He still has a right frontal depression of the bone and a severe left-sided weakness. He says that consciousness was reduced or altered for three months. A CT scan carried out in 1987 showed an extensive low attenuation area in the right medial insular cortex. Both lateral ventricles were enlarged, the right more so than the left. There were additional changes in the left frontal lobe. On leaving hospital after a stay of 6 months he returned to his previous employment but was unable to prepare or give lectures and so left to take an MA. He failed this, but obtained a teacher’s certificate at a Further Education College. For the next five years he held a succession of jobs in teaching and the Civil Service from most of which he was dismissed.
His response in a clinical interview and his wife's testimony were in agreement over his domestic behaviour. He is untidy. He only bathes if going somewhere important. Shaving, changing his clothes or undergarments, washing his hair and having his hair cut are only carried out when his wife tells him. He does not spontaneously tackle any domestic chores such as laundry, cleaning, cooking, making repairs or paying bills. Occasionally he uses a vacuum cleaner without being asked. If his wife is out he normally leaves the preparation of a meal to his 10 year-old son. When he shops he never makes out a list himself and also usually comes home without all the items on the list his wife prepares. If he is sent out to go to six different places, his wife says he will complete three of the tasks but forget to carry out the other three. When she gives him a task she has to specify exactly what is required and even so he might do some parts only and then start reading a newspaper. In addition his wife organises all trips, outings and social contacts with relatives. Occasionally DN will telephone a friend. When asked what activities he had planned for the coming weekend he said he had to take clothes to a Methodist mission; however this was not a spontaneous piece of organisation as he was on a rota organised by a church he attends. His wife said he was occasionally irresponsible over money; for instance even though they were in financial difficulties he was out of work, he would buy gadgets they did not really need including a sophisticated music system, costing £500.

6.2(b)i Clinical Neuropsychological Tests

DN was tested at the National Hospital on a wide range of neuropsychological tests. On the WAIS, DN obtained a Verbal IQ of 126 and a Performance IQ of 112, which may well be a little below his premorbid level but in fact corresponds well with the results of the NART (Nelson, 119) IQ equivalent of 119. On all verbal subtests he performed in the superior range and on the Performance
subtests in the average or bright average ranges (see table 1).

On memory tests he performed well with verbal material, but rather poorly with visual material (see Table 2). On tests sensitive to frontal lobe lesions his performance was generally satisfactory. However his performance on the picture version of the Self-Ordered memory test was rather weak (see table 3).

In summary, DN performed well on a wide range of perceptual, language and frontal lobe tests. However, while his performance on verbal memory tests was well within the normal range, his performance on visual memory tasks was impaired.

6.2(c) Case 3: FS

FS, a 55 year-old right-handed woman, employed as a librarian, had earlier sustained two separate head injuries. Thirty years before she had been thrown from a horse, had fractured her skull and had been left with a post-traumatic amnesia of unknown length. She was also aphasic at the time. The accident had left her with a slight left-sided facial paralysis. Two years before being tested she had been knocked off her bicycle by a car and hit her head on the road; she did not lose consciousness but as a result has had a permanent loss of the senses of smell and taste.

A CT scan carried out at the Middlesex Hospital in 1986 showed an extensive lesion to the left frontal lobe with atrophy causing enlargement of the frontal horn of the lateral ventricle. There was also some atrophy in the left frontal lobe.

In the period between her two accidents she had first done clerical work and translating for two publishing companies. For the past twenty-five years she had worked as a librarian and she continues to hold the same
position. She lives by herself in a single room. Her responses in a clinical interview show that she undertakes virtually no inessential or novel activities. She is very untidy, never putting things away. She washes up only when plates or cutlery are needed. She cleans her room only when the floor is "crumby and dirty-looking", her room being covered in books and records in piles 18 inches high. She selects what she will wear at the last moment and this shows little variation from one day to the next. She very seldom goes out in the evening, and virtually never travels out of London. Others always make arrangements when any joint activity is to be carried out. She is said by her sister never to organises anything. She shops every day buying only a few things on any occasion and never visits supermarkets. She had no activity planned for the following weekend and could give no example where anyone had relied on her to do anything. Her sister confirmed that these behaviours were characteristic.

6.2(c)i Clinical Neuropsychological Tests

FS was tested at the National Hospital on a large range of neuropsychological tests. On the WAIS she obtained a Verbal IQ of 135 and a Performance IQ of 114, which is comparable to the estimate of her optimal premorbid level of functioning of 127 derived from the NART. Verbal subtests were all carried out at the Bright Normal level except for Picture Arrangement which was at the average level. She had no aphasic difficulties and performed within the normal range on language and perceptual tests (see table 26).

Her performance on memory tests was mixed. On some she performed well; these include Recognition Memory (Warrington, 1984) for both words and faces, and list learning. On free recall of 10 word lists she obtained an average of 6.1 correct, better than any of the six similarly aged controls of Baddeley and Warrington (1970). On Wechsler Logical Memory she was in the normal
range but not high within it. However on certain other tests she performed poorly - Wechsler Paired Associates, Immediate and Delayed Recall of both the Rey and the Coughlan and Hollows figures and on the Self-ordered Pointing Task (see table 27). She was also unable to learn span+1 with either digits or spatial positions (see Milner, 1971) in 12 trials; on a second attempt she did learn span+1 in 4 trials but still failed the Corsi span+1.

On tests considered sensitive to frontal lobe dysfunction her performance was variable (see Table 28). On some her performance was entirely satisfactory (Weigl, Wisconsin, Proverb Explanation; Tower of London; Stroop). Cognitive Estimation was performed in the normal range but not at a high level within it. However on four others her performance was impaired (Alternation; Trail-Making; Personal Orientation Test; Self-Ordered Pointing). Verbal Fluency, when first tested, was very poor (5 words in 2 mins.) but when tested a second time, her performance was within the normal range (see Table 3).

In summary, FS performed well on a wide range of perceptual, language and general cognitive tasks, but performed poorly on some memory and some frontal tasks.

Overall the three patients perform well and close to their premorbid level on a wide range of perceptual, language and general cognitive tests. One (AP) has no problem with memory tasks but one (DN) has a primary visual memory impairment and a third (FS) has difficulties on certain tasks which have been suggested as being sensitive to frontal lobe dysfunction (Signoret and Lhermitte, 1976). Two of the patients perform very well on a wide range of tasks thought sensitive to frontal lobe lesions, but one shows impairments on some frontal tasks. However all show severe organisational difficulties in everyday life.
TABLE 26

Performance on Tests of General Cognitive Functioning, Language and Perception (Scaled Scores unless otherwise indicated)

<table>
<thead>
<tr>
<th>Cases</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>WAIS Verbal Subtests</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arithmetic</td>
<td>13</td>
<td>14</td>
<td>15</td>
</tr>
<tr>
<td>Similarities</td>
<td>15</td>
<td>14</td>
<td>15</td>
</tr>
<tr>
<td>Digit Span</td>
<td>15</td>
<td>14</td>
<td>15</td>
</tr>
<tr>
<td>Vocabulary</td>
<td>16</td>
<td>15</td>
<td>17</td>
</tr>
<tr>
<td><strong>Verbal IQ</strong></td>
<td>128</td>
<td>126</td>
<td>135</td>
</tr>
<tr>
<td><strong>WAIS Performance Subtests</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Picture Completion</td>
<td>14</td>
<td>12</td>
<td>13</td>
</tr>
<tr>
<td>Block Design</td>
<td>15</td>
<td>12</td>
<td>13</td>
</tr>
<tr>
<td>Picture Arrangement</td>
<td>13</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td><strong>Performance IQ</strong></td>
<td>129</td>
<td>112</td>
<td>114</td>
</tr>
<tr>
<td><strong>WAIS Full Scale IQ</strong></td>
<td>130</td>
<td>121</td>
<td>127</td>
</tr>
<tr>
<td><strong>NART WAIS FSIQ Equivalent</strong></td>
<td>124</td>
<td>119</td>
<td>127</td>
</tr>
<tr>
<td><strong>Language</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stringent Graded Naming Test&lt;sup&gt;1&lt;/sup&gt;</td>
<td>14</td>
<td>14</td>
<td>14</td>
</tr>
<tr>
<td><strong>Perception (%iles)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cube Analysis&lt;sup&gt;2&lt;/sup&gt;</td>
<td>50</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>Dot Centre&lt;sup&gt;3&lt;/sup&gt;</td>
<td>50</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>Unconventional Views&lt;sup&gt;4&lt;/sup&gt;</td>
<td>50</td>
<td>25</td>
<td>10</td>
</tr>
<tr>
<td>Conventional Views&lt;sup&gt;5&lt;/sup&gt;</td>
<td>50</td>
<td>50</td>
<td>50</td>
</tr>
</tbody>
</table>

---

1. McKenna and Warrington, 1983.
2. Warrington and James, 1988.
### TABLE 27
Performance on Memory Tests (Scaled Scores)

<table>
<thead>
<tr>
<th>TEST CASES</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Auditory Verbal Learning(^1)</td>
<td>12(^2)</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>Complex Figure Recall(^1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Immediate</td>
<td>NT*</td>
<td>NT</td>
<td>6</td>
</tr>
<tr>
<td>Delayed</td>
<td>17(^3)</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Digit Span(^4)</td>
<td>15</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>Paired Associates (Sum)(^5)</td>
<td>11</td>
<td>NT</td>
<td>7</td>
</tr>
<tr>
<td>Recognition Memory(^6)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Words</td>
<td>10</td>
<td>10</td>
<td>15</td>
</tr>
<tr>
<td>Faces</td>
<td>14</td>
<td>4</td>
<td>11</td>
</tr>
<tr>
<td>Story Recall(^7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Immediate</td>
<td>13(^5)</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>Delayed</td>
<td>14(^5)</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>Visual Reproduction(^5)</td>
<td>13</td>
<td>14</td>
<td>NT</td>
</tr>
</tbody>
</table>

* NT = Not Tested
1 Coughlan and Hollows (1985)
2 Score on worst trial of Rey Auditory Verbal Learning Test (Rey, 1964).
3 Osterrieth (1944-5).
4 Wechsler (1955).
5 Wechsler (1945).

### TABLE 28.
Performance on Executive Tests

<table>
<thead>
<tr>
<th>TEST CASES</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alternation Task(^1)</td>
<td></td>
<td>90%ile</td>
<td>75%ile</td>
</tr>
<tr>
<td>Bilateral Hand Movements</td>
<td>Good</td>
<td>Good</td>
<td>Good</td>
</tr>
<tr>
<td>Cognitive Estimates(^2) (error score)</td>
<td>2</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Money's Road Map Test (errors)(^3)</td>
<td>0</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Personal Orientation Test (errors)(^4)</td>
<td>0</td>
<td>4</td>
<td>14*</td>
</tr>
<tr>
<td>Proverb Interpretation</td>
<td>Good</td>
<td>Good</td>
<td>Good</td>
</tr>
<tr>
<td>Self-Ordered Pointing (errors)(^5)</td>
<td>7</td>
<td>10</td>
<td>9</td>
</tr>
<tr>
<td>Stroop(^6) (time)</td>
<td>20.1</td>
<td>17.3</td>
<td>21.0</td>
</tr>
<tr>
<td>Tower of London(^7) (score)</td>
<td>33</td>
<td>23</td>
<td>24</td>
</tr>
<tr>
<td>Trail-Making(^8) (completion time)</td>
<td>15.7</td>
<td>25.6</td>
<td>49.0*</td>
</tr>
<tr>
<td>Verbal Fluency(^9) (FAS 60 secs)</td>
<td>70</td>
<td>34</td>
<td>33</td>
</tr>
<tr>
<td>Modified Wisconsin(^10) (categories)</td>
<td>6</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>(total errors)</td>
<td>3</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

* indicates performance more than 2 SD poorer than controls.
1 Chorover and Cole (1966); patients compared with nonlesion patient controls \([n = 24, \text{mean FSIQ} = 114.9, \text{SD 12.7}]\) from Shallice, Warrington, Watson and Lewis (unpublished study).
2 Shallice and Evans (1978); mean of controls = 3.0 SD
2.6 (data from study above)
Butters et al (1972); mean of left frontal group 11.9; normals 1.7.
Semmes et al (1963); mean of 10 right posterior lesion patients [mean FSIQ 108.2, SD 9.8, mean age 45.7 SD 14.0] = 4.1, SD = 3.5; mean of 10 left posterior lesion patients [mean FSIQ 108.1, SD 12.2, mean age 41.8 SD 14.0] = 2.4, SD = 2.7 (Burgess and Shallice, unpublished data).
Petrides and Milner (1982); Cases 2 and 3 > 1 SD worse than a lesion control group (8 right posterior lesion patients [mean FSIQ = 107.1, SD 8.9, mean age 45.5 SD 14.8 and mean FC faces performance of 7.25, SD 4.8 - dull average].
Perret (1974) controls mean = 23.3, SD = 11.0 (Shallice Warrington, Watson and Lewis, as above)
Shallice and McCarthy (see Shallice, 1982). Controls mean = 24.9, SD 4.5 (Shallice et al, as above).
Reitan (1958). Controls mean = 29.6 SD 4.5 (Shallice et al, as above)
Miller (1984). Controls mean 34.8, SD 14.3 (n= 30; Miller's data). Note that if Miller’s formula is used, Cases 2 and 3 both fall below the expected range.
Nelson (1976). Controls mean = 9.2 SD 8.5 (n = 46; mean age 45 yrs SD 14.0)

6.3 METHOD

6.3(a) Organisational executive tasks

Two tests were developed to measure the ability of a subject to organise and carry out a series of subtasks under the situation where the demands are open-ended (thus requiring planning and goal-setting) and there are a number of rules which force the subject to form intentions to carry out certain operations later (thus loading upon prospective memory).

The Six Element Test (SE)

This test, which was carried out in a standard hospital office, assessed the ability of the patients to carry out six subtasks whilst following certain rules which dictated the order in which these could be done. The six tasks divide into two sets of three - dictating routes, arithmetic problems and writing down the names of approximately 200 simple line drawings of common objects.
Each set was of comparable difficulty to its counterpart, and the individual items of the tasks were designed to be well within the patients' capabilities. Each set of arithmetic problems consisted of 35 sums which yielded whole numbers and where the items were of increasing difficulty.

The patients were presented with the instructions written on a card (see Figure 4 for full details of the SE task).

**Subject’s Instructions**

The E first read through the instructions with the S, explaining which stack of cards were A or B, and so forth. Having read through the task items, the E gave the following instructions:

"But before you go on, there are a number of important rules you must follow which govern the way you are allowed to do these tasks, and may influence the way in which you decide to organise the way you do the test".

The five rules are read through with the S and explained as fully as necessary. The instruction sheet is then turned over and the S is "tested" on his/her recall of the rules. Any errors are corrected. If the S makes any mistake, the entire rules are read through again and the S is tested again. This is repeated until the S can recite all the rules correctly without looking at the instruction sheet. The sheet is then given to the S (face upwards) and it is explained that s/he is free to refer to it at any time during the test.

Two additional tasks were carried out. In the first, the subjects, whose watches had been removed, were required to carry out the Digit Symbol subtest of the WAIS, stopping themselves after 1 minute and 15 seconds. A stop-watch was visible at all times. If the S continued after the allotted time, the E said nothing until the S stopped of his/her own freewill.
In the second task, Rey’s (1964) Tangled Lines test had to be carried out with the subjects instructed to stop themselves as close as possible to 2 mins 30 secs after starting. This time the stop-watch was covered by a small cardboard box. The subject could remove the box at any time to inspect the stop-watch, but the box then had to be replaced directly. As before, the E gave no indication as to when the S should stop.

FIGURE 4
The Six Element Test

In the next 15 minutes we would like you to carry out three different tasks, each of which is in two parts.

The tasks are:-

1. To dictate into the tape recorder a brief account of two journeys:
   a) your journey to here.
   b) your intended journey from here.

2. To write down the names of as many pictures as you can (in order)
   a) those in the left pile.
   b) those in the right pile.

3. To solve two sets of arithmetic problems (as many as you can, in order).
   a) Set I
   b) Set II

   But You are not allowed to do the two sub-tasks (a) and (b) of the same type one after the other.

Each of the six sub-tasks is given equal weight.

Within sub-tasks 2 and 3, points will be given for correct answers.

Earlier pictures/problems will be given more points than later ones in each sub-task, and errors/omissions will be heavily penalised.

6.3(b) Results

Three measures of performance on the SE were taken. Firstly the number of subtasks attempted, secondly the
maximum time spent on any one subtask, and lastly the
mean time taken to do one item in the written object
naming subtest. This last measure was used as an estimate
of the work speed of the subjects (since it may be the
case that the patients would attempt fewer subtasks
merely because they were slower than the controls).

The performance of the three patients was compared with
that of 10 controls, mean age 41 (range 25-62) of similar
educational background to the patients and with a mean
NART IQ of 120.2 SD 4.28 (range 113-127). The results are
shown in tables 29 and 30. Two different attempts were
made by each patient, at least three weeks apart. On
their first attempt AP and FS tackled less subtasks than
any control and all three spent more time on their
subtask they attempted for longest. The work-rates of the
three patients, as assessed by mean speed of written
object naming, were comparable to the controls (see table
30).

TABLE 29

Performance on the Six Element Task

<table>
<thead>
<tr>
<th>Case</th>
<th>No. of Subtasks Tackled</th>
<th>Max. Time on any subtask</th>
</tr>
</thead>
<tbody>
<tr>
<td>AP (1)</td>
<td>2</td>
<td>7 mins 30 secs</td>
</tr>
<tr>
<td>AP (2)</td>
<td>4</td>
<td>6 mins 19 secs</td>
</tr>
<tr>
<td>DN (1)</td>
<td>5</td>
<td>10 mins 11 secs</td>
</tr>
<tr>
<td>DN (2)</td>
<td>3 (+ 2 incorrect)</td>
<td>6 mins 22 secs</td>
</tr>
<tr>
<td>FS (1)</td>
<td>3</td>
<td>7 mins 18 secs</td>
</tr>
<tr>
<td>FS (2)</td>
<td>2</td>
<td>9 mins 30 secs</td>
</tr>
<tr>
<td>Controls</td>
<td></td>
<td>5.7 SD 0.5</td>
</tr>
</tbody>
</table>

TABLE 30

Written Object Naming: Mean Time Taken to do One Item

<table>
<thead>
<tr>
<th></th>
<th>AP</th>
<th>DN</th>
<th>FS</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>5.6</td>
<td>6.6</td>
<td>4.8</td>
<td>5.1 SD 2.5</td>
</tr>
<tr>
<td>II</td>
<td>4.9</td>
<td>NA</td>
<td>6.3</td>
<td></td>
</tr>
</tbody>
</table>

Qualitatively the behaviour of the patients was also
atypical. On his first attempt AP made notes for over 4
minutes to help with the dictation task, but in fact never dictated at all. He only attempted two subtasks, the second of these occupying the last 7.5 minutes. On his second attempt he tackled only 4 of the subtasks claiming that he did not dictate his journey back as he had not decided where he would go when he left the hospital.

DN succeeded in tackling 5 subtasks but spent much longer than any normal subject (over 10 minutes in three separate periods) on one of the subtasks (Maths I) without ever trying its complementary task (Maths II). On his second attempt he behaved bizarrely, changing task 63 times (mean task changes of controls 5.7 SD 2.19) and dictating the two sets of picture names (spending only 14 secs and 20 secs on them) instead of writing them down; no normal subject carried out a subtask incorrectly. Also on six occasions he carried out a subtask immediately after the complementary one, so breaking one of the rules (one control also made this error. She said she had mistaken the rule as not doing part 1 of a pair of subtasks after another part 1; this she correctly applied).

On FS’s first attempt she tackled only 3 subtasks. On the second she wrote her journey instead of dictating it, attempted only two other subtasks and spent the last nine minutes on just one of them. During this time she looked at the stop-watch on 7 occasions but did not switch tasks.

On the two timing tasks, MO performed well on both (Task I stopped 1 min 17 secs; Task 2 stopped 2 mins 28 secs). However DN failed the first, stopping at 1 min 38 secs although being fully aware that he should stop at 1 min. 15 secs. On the second task his performance was very poor on both testing occasions. On the first attempt he stopped after 1 min. 37 secs., saying that he thought 2 mins 45 secs had elapsed. and on his second he failed to stop until 3 mins 26 secs. had elapsed. FS performed well on Task 1 (stopping at exactly the correct time). However
on Task 2 she did not stop until 3 mins 20 secs had elapsed despite having looked at the watch at 2 mins and 38 secs. No control was more than 1.0 sec out on the first task or more than 5 secs out on the second (mean time out for task 1 = 0.1 sec SD 0.3; Task 2 mean = 2.9 secs SD 2.0)

Multiple Errands Test (ME)

The purpose of this test, which is carried out in a pedestrian precinct near the hospital previously unknown to the patients, is for them to carry out a number of tasks in situations where minor unforeseen events can occur. The subtasks the patients carried out were basically very simple, except for one which had subcomponents designed to be reasonably demanding for someone of the IQ level and cultural background of the patients. While still inside the hospital, the patients (tested separately) were given a card with 8 tasks written on it. Six of these are quite straightforward (eg. buy a brown loaf, buy a bar of soap), a seventh requires the subject to be at a certain place 15 minutes after starting the test, and the eighth is more demanding - four sets of information have to be obtained and written on a postcard, which is then posted:
1. The name of the shop in the street likely to have the most expensive item.
2. The price of a pound of tomatoes.
3. The name of the coldest place in Britain yesterday.
4. The exchange rate of the French franc yesterday.

The card also contains the following instructions:-
You are to spend as little money as possible (within reason) and take as little time as possible (without rushing excessively).
No shop should be entered other than to buy something.
Please tell one or other of the experimenters when you leave a shop what you have bought.
You are not to use anything not bought on the street (other than a watch) to assist you.
You may do the tasks in any order.
This second part of the instructions - the "rules" was then read aloud to the patient who was asked to repeat them. If the rules cannot all be repeated satisfactorily the procedure was repeated until they could. Subjects were then taken a short distance to a local pedestrian shopping precinct and then tested again on the rules. If there was any failure to reproduce the gist of any instruction the subject is reminded of it. It was then indicated to the patient where the limits of the test area were; in practice these were very clearly demarcated by a large street ending the pedestrian precinct at one end and a set of traffic lights at the other. The behaviour of the subjects was monitored by two observers and they are also debriefed after the task had been completed.

6.3(d) Results

The patients' performance was compared with that of 9 normal controls matched for age (mean 40, range 24-63 and NART IQ [mean 122 range 113-127]). The number of errors produced by each of the three patients was at least two standard deviations more than that of the controls. Table 31 also shows the errors broken down into categories:

i) **inefficiencies** - where a more effective strategy could have been applied eg. entering the same shop more than once.

ii) **rule breaks** - where a specific rule (either social or explicitly mentioned in the task) is broken eg. going outside the boundaries or leaving a shop with a newspaper without paying for it.

iii) **interpretation failure** - where the requirements of a particular task are misunderstood eg. assuming that the information must be written on the birthday card rather than the postcard.

iv) **task failure** - a task not carried out or completed satisfactorily.
All three patients performed at the 5% level or worse – as estimated from the control data – on both the number of inefficiencies and on number of rule breaks. There was no difference overall in the rate of errors of the other two types, although patient FS produced more task failures. Thus two normal controls as well as the patients failed to provide the required information on the postcard, probably because the information required was placed on the back of the instruction sheet.

The qualitative aspects of the patients’ performances were perhaps even more instructive, and the patients produced types of errors that were not produced by any control. For instance, FS broke a rule (entering a shop without purchasing any thing in it) because after she had gone in the shop to buy the soap, she discovered that they did not sell a particular (quite expensive) brand she favoured. However the shop sold many other cheaper brands which would have been more adequate given that she was supposed to spend as little money as possible. She also failed to take note of the time when she started the task and so had no way of knowing when the 15 minutes were up, which led her to fail the formal prospective memory component of the task.

AP and DN both became involved in awkward social situations whilst performing the test. AP made the interpretative error of deciding that he needed yesterday’s paper in order to discover the coldest place in Britain yesterday. So he went to a newsagents and asked if they had a copy of yesterday’s paper. They gave him one, and he just walked out without paying for it. The shopkeeper, clearly irate, pursued him into the street. AP explained that he thought that since a previous day’s newspaper is generally regarded as worthless, he thought he could have it for free.

DN produced a number of complex string of errors. One of these began when he entered one shop merely to ask the whereabouts of another (thus breaking the buying rule). He was referred to a shop outside the allowed area, so
went to that shop (breaking the limit rule) and then became involved in a heated argument with the shop assistant as he asked her to give him a birthday card for free (which would have, had he succeeded, broken the buying rule).

### Table 31

<table>
<thead>
<tr>
<th>Errors on Multiple Errands task</th>
<th>AP</th>
<th>DN</th>
<th>FS</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inefficiencies</td>
<td>6*</td>
<td>9*</td>
<td>5*</td>
<td>1.4 (1.1)</td>
</tr>
<tr>
<td>Rule Breaks</td>
<td>5*</td>
<td>8*</td>
<td>8*</td>
<td>1.6 (1.3)</td>
</tr>
<tr>
<td>Interpretation Errors</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0.4 (0.7)</td>
</tr>
<tr>
<td>Task Failures</td>
<td>0</td>
<td>5*</td>
<td>4*</td>
<td>1.1 (1.4)</td>
</tr>
<tr>
<td>Total Errors</td>
<td>12*</td>
<td>23*</td>
<td>17*</td>
<td>4.6 (2.1)</td>
</tr>
</tbody>
</table>

6.4 DISCUSSION

Three patients have been described whose level of dysfunction in everyday life is at odds with their relatively intact neuropsychological test performance. All three cases were intact as far as general levels of intellectual functioning were concerned, and one of the cases had no difficulties in performing a wide range of other tests, including those traditionally considered sensitive to frontal lobe dysfunction. The other two cases had mild difficulties on certain frontal and memory tests. However all three showed severe and debilitating impairments in their ability to perform two tests which require the sequencing and organisation of multiple sub-goals. In addition, in commonplace activities of daily living which - on the face of it - require these skills, all three patients showed lasting impairment as described both by the patients and their relatives.

Before accepting, however, an explanation of the patients' impairments in terms of neuropsychological theory, a number of other possible explanations of the findings must be considered.
One such possible explanation would be that the patients' difficulties stem from memory problems: they were unable successfully to comply with task rules simply because they forgot them. There are two problems with this account. The first is that one of the patients had no difficulty with a wide range of standard memory tests, and yet was still impaired on the Multiple Errands and Six Element tasks. The second is that the memory demands of both tasks were kept deliberately low - the patients had task instructions, to which they could refer, with them at all times. This is especially the case with the Six Element Task, where all the rules are presented on the same sheet as the task items. Moreover, it would be very difficult to perform this test without reference to this sheet at some point(s) in order to remind oneself of the task items. At these times, if one were unsure of the task rules, one would naturally consult them.

A second putative explanation which needs to be considered is the suggestion that the patients' performance on any given task might be affected by the degree of social facilitation inherent in the task. That is to say that when the patients are required to perform a task on their own, they do less well than when there is close involvement of the examiner, as is the case with the majority of traditional neuropsychological tasks. However this seems implausible since AP was given the entire AH6 followed by the Watson-Glaser Critical Thinking Appraisal (Watson and Glaser, 1980) and was left to work through them on his own. He scored at the 50th and 80th percentiles respectively, the total of the task times being two hours. Both DN and FS carried out the long form of the Raven’s Matrices, working through the test on their own. This took them 31 and 42 minutes respectively - less than the 45 mins that Raven et al (1977) give as the upper limit of the normal range and yet longer than the 2 multiple-subgoal scheduling tests would normally take. They scored at the 95th and 80th percentiles, respectively, for their ages.
A third possible explanation might be that the patients' were poorly motivated to perform the Multiple Errands and Six Element Tests because they found them boring. Apart from the fact that it is in any case difficult to see how or why the patients should find the organisational tasks much duller to perform than such tasks as the AH6 or Raven's Matrices, the explanation has, in any case, little face validity. Thus if the patients' poor performance on the timing tasks (where they had to stop themselves in the middle of performing a relatively simple task) was because the task were boring, it is not easy to see why this should lead them to continue doing the test beyond the point where they could stop. In addition, the "boredom" explanation should predict that the patient's work-rate on the most mundane tasks should be slower than the controls. Yet this was not the case (see Table 30).

This leaves an explanation of the patients' experimental task performance which centres upon the specific demands made by these tasks, and the way in which they differ from traditional neuropsychological tests.

Traditional neuropsychological tests require little, if any, planning before the task is started\(^1\) since tasks generally consist of a series of items, which may differ in their difficulty, where the subject starts at a given point and progressively works through the items. The subject is not usually required to decide in advance what order to do the tasks in, or to apportion priorities to competing tasks. The contingencies involved in the task are usually explained quite clearly, and from this the subject can deduce what will represent a satisfactory performance.

However, the two experimental tasks described here made quite different demands. The actual task items were quite straightforward, and certainly within the capabilities of these quite intellectually sophisticated patients. The

\(^1\) Certain tests may however require planning skills once the task has been started (eg. Tower of London).
crucial elements of the tests were that they were required to organise their efforts themselves, bearing in mind a series of rules or constraints which had to be followed. There were a number of similarly suitable methods of approaching the tasks which the patient had to decide upon themselves. This obviously requires some forethought. But then this plan of action needs to be realised.

The issue of the setting of goals and their subsequent realisation is an area of psychology where clearly worked out and detailed theories are few. However it is often argued that dealing successfully with situations which are in some sense novel, or where the series of actions required to achieve a goal are not routine, generally requires a minimum of four stages which may be called recursively (de Groot, 1965; Ben-Yishay and Diller, 1983). Firstly a goal has to be set, then a course of action needs to be drawn up followed by the carrying out of this putative solution, and lastly the outcome needs to be checked against the original goal.

However these processes do not necessarily follow directly from each other in time. A goal to, for instance, have prepared and delivered a letter by the end of the week, may require the various actions or stages involved to be performed at different times during that week. During the intervening periods the intention to perform the given action needs to be stored in some sense, and then resurrected at the appropriate moment. This will involve prospective memory processes (Meacham and Leiman, 1982; Kvavilashvili, 1987). How then do these intentions come to be realised at the appropriate time?

It is proposed that one consequence of the analysis of the stages involved in achieving a goal (planning) is that temporal markers are created. A marker is no more than a message to treat a certain situation as non-routine, in that some action either additional to, or different from the routine actions normally performed in that situation is required. Retrieving the actual
required course of action at this point would be the
domain of more traditional retrospective memory. If the
planned (or considered) situation or event does occur
later, the marker is activated, and the subject will
become aware that some action needs to be performed
(without necessarily knowing, of course what that action
might be: cf. Kvavilashvili, 1987). Thus a task rule,
such as (in the Multiple Errands Test) "do not enter a
shop unless something is to be bought there" would be
realised through a marker being created when the task
instructions are considered and understood. This marker
would then be activated on approach to a shop, or when
thinking about doing so. Of course many of these markers
may be set up at any one planning "session", and exist,
until their realisation, simultaneously.

FIGURE 5
Simple Scenario to Illustrate the Component Processes in
the Solution of a Practical Problem

7.45 a.m. SITUATION: need to take car for MOT test.
7.50 a.m. Goal Articulation: need to have car for
tomorrow so car must be tested and returned today.
8.00 a.m. Plan Formulation: Telephone garage at 9 am when
opens, take car over on way to work, collect car having
left work.

Marker Creation 1 - Before leaving for work->
phone garage (if garage too busy then
: Plan Modification*.
Marker Creation 2 - On way to work-> stop at
garage.
Marker Creation 3 - When leaving work ->
phone garage and then pick car up.
9.00 a.m. (leaving for work) Marker Trigger 1 ->
Telephone garage
9.15 a.m. (en route to work) Marker Trigger 2 -> Stop
at garage
5.00pm (leaving work) Marker Trigger 3 -> Phone garage
and then pick car up

* If not possible to take car to garage, plan
modification occurs and new markers are set up.
According to this view, there are five executive processes which are involved in carrying out many everyday activities, or tasks such as those described here: 1) goal articulation; 2) provisional plan formulation; 3) marker creation; 4) marker triggering and 5) action evaluation. This last process may of course occur at any stage, and would lead to further goal articulation and marker set-up. These processes, at work within a relatively simple everyday scenario, are illustrated in Figure 5.

Since there are possible overlaps between these processes, and it is not always easy to distinguish between types of process, these 'strategy application' processes may be grouped into three broad types: 1) Those involving planning (plan formulation or modification; 2) Those involving markers (marker creation or triggering); 3) Those involving goals (evaluation and goal articulation). Table 32 shows examples of types of errors in the patients’ performances on the experimental tasks which correspond to failures in these processes.

Many examples of the patients’ experimental task lapses cannot be assigned exclusively to one category: since these actions are complex and happen over time, often there may be a number of factors which variously contribute to the behaviour. This is even more the case as regards the patients’ everyday behaviour. Nevertheless the experimental task lapses do seem to involve the same processes as those real-life situations with which they have difficulty.
TABLE 32.

Examples of the Three Types of Lapse Made by the Patients on the Experimental Tasks

Plan Formulation or Modification
1. Going to Post Office before all relevant information obtained (Cases 1-3).
2. No plan for SE (Cases 1-3).
3. Failure to look at watch at start of ME (Case 3).
4. No knowing quite how he would get to X and therefore not tackling SE. journey back subtask.
5. Generation of inappropriate criteria in an ME subtask (Case 3).

Marker Creation or Triggering
1. A very extended period spent on a single SE subtask without a break (Case 1, 3).
2. Going out of bounds in ME (Cases 2, 3).
3. Attempt not to pay for item in shop (Case 1, 2).
4. Looking in irrelevant shops (Cases 1, 2).
5. Not using the notes made for 4 min. about journey at start of SE).

Evaluation and Goal Articulation
1. Posting postcard without all information (Cases 2, 3).
2. Failure to keep a check of money in ME (Cases 2, 3).
3. Thinking finished ME when had not (Case 1).
4. Ending SE attempt 1 min. early when far from having completed tasks.
5. Carrying out SE subtasks incorrectly (Cases 2, 3).

The processes of plan formulation and evaluation have often been suggested as being subsumed by the frontal lobes (Penfield and Evans, 1935; Luria, 1966), as have goal articulation (Duncan, 1986). If one adds to these the elements of marker creation and triggering, then it is easy to see how patients such as Damasio and Eslinger’s EVR might show the characteristics of living a "goal-less existence" with "no evidence that an internal automatic program was ready to propel him into the routine daily activities" (goal articulation and plan formation), with one of his main problems being that he often "forgot to remember short and intermediate-term goals" (marker triggering).

The obvious interaction between goal formation/plan formulation and prospective memory (marker creation and triggering) highlights the difficulty of demonstrating
fractionation of these processes. Thus, for instance, given that marker creation is the product of plan formulation and goal articulation, should these preliminary processes not occur it is theoretically improbable that one would be able to demonstrate deficits in marker triggering. It may therefore be impossible to detect prospective memory deficits in patients whose planning/goal articulation abilities are severely impaired. However the three cases reported here could all perform the Tower-of-London task within normal limits, so their ability to plan (or show anticipatory processing (McCarthy and Warrington, 1990) may have been preserved in the short-term at least.

Likewise, the question of whether our patients could demonstrate marker triggering but not marker creation deficits is a debatable one. There is some evidence that this might happen - our patients could usually, when asked, recall a rule which they had just broken. However this is hardly a conclusive argument, and the question rests to a large extent upon the inter-relationship of prospective and retrospective memory, and this is an area of psychology which is at present in its infancy.

Nevertheless, our patients did show differing ratios of strategy application errors, with AP one showing marker processes most affected and FS showing mainly goal articulation and plan formulation/evaluation errors. It seems plausible therefore that future patients may show clearer evidence for the fractionation of these processes.

As regards the present thesis, this study has demonstrated prospective memory deficits in patients with frontal lobe damage. Moreover, these deficits occurred in one of the patients whose retrospective memory functioning - at least as measured by traditional neuropsychological tests, was normal. This supports the distinction between the two forms of memory as suggested by Meacham and Leiman (1982) and Kvavilashvili (1987). Whether prospective memory deficits can occur as a
consequence of non-frontal lesions is as yet unproved. However there is a wealth of evidence that the likelihood of realising an intention is dependent (at least to some degree) upon the use of internal and external strategies (see Harris, 1983 for a review). To this extent, then, should prospective memory functions be shown eventually not to be subserved exclusively by frontal structures, it still seems likely that patients with frontal lobe damage will be those who, in everyday life, show problems with "remembering to remember".
7.1 Introduction

The Introduction to this thesis suggested that confabulation (and other forms of paramnesia) is the one phenomena which seems incontrovertibly linked to frontal lobe dysfunction. There is as yet no reported case of a confabulating patient in which frontal lobe involvement can be ruled out. However, as discussed in the Introduction, it is less certain whether additional posterior involvement can be excluded as a necessary predisposing factor (see Moscovitch, 1989). Nevertheless the level of agreement amongst investigators that confabulation is a memory phenomena secondary to frontal lobe dysfunction is quite remarkable, and these days is virtually taken for granted. This is despite the fact that few cases of patients with apparently isolated frontal cortical involvement have actually been described (see Kapur and Coughlan, 1980): the majority of cases have shown direct evidence of more widespread damage. Most cases have either suffered a ruptured anterior communicating artery (eg. Delbecq-Derouesne et al, 1990; Kapur and Coughlan, 1980; Luria, 1976; Moscovitch, 1989; Shapiro et al, 1981; Stuss et al, 1978; Sweet, Talland and Ballantyne, 1966; Vilkki, 1985), or are suffering from Korsakoff’s syndrome (eg. Butters, 1985; Talland, 1965; Talland and Ekdahl, 1959; Victor, Herman and White, 1959). As already discussed, both these disorders generally lead to involvement of structures outside the prefrontal cortex.

Chapter five argued that there was little incontrovertible evidence for differing patterns of memory test performance between patients with anterior or posterior lesions. Although there have been several interesting claims that frontal lobe patients show
particular patterns of behaviour on memory tests, there is considerable conflicting evidence as to what this pattern might be. The same is true of studies of confabulators. For instance, Delbecq-Derouesne et al (1990) suggest that their patient's free recall/recognition paradigm performance discrepancy is instructive as regards understanding the processes that are damaged in confabulators. Specifically they suggest that their patient's recognition test difficulties stem "from his inability to distinguish his experience of remembering from another kind of misleading subjective experience triggered by the distractors" (p. 1069), and suggest that this may also be the root cause of his confabulation. However, (and as they go on to point out) the confabulating patients reported by Kapur and Coughlan (1980) and Moscovitch (1989) showed the opposite pattern of memory test performance (preserved recognition and poor recall), so it seems unlikely for the moment that the isolated study of memory test patterns in confabulators will be directly instructive about the specific mechanisms that are damaged in confabulators. This chapter will therefore concentrate upon study of the phenomena of confabulation directly.

7.1(a) Characteristics of Confabulation

As shown in the Introduction, the characteristics of classical amnesia have been well investigated, and there is now general agreement as to its defining characteristics. This is however not true of the memory disorder of which confabulation is the symptom, which is reflected in the varying characterisations of

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1 Additionally, in the Introduction to this thesis it was argued that neuropsychological theories of amnesia are weakest when they are used to explain paramnesic phenomena since those theories have been for the most part based upon studies of amnesics' performances on experimental memory test paradigms. Since there is an ill-defined relationship between amnesia and confabulation (eg. Kapur and Coughlan, 1980) the application of theories of classical amnesia to paramnesic phenomena has led to similarly ill-defined explanations of confabulation.
confabulation as "honest lying" (Moscovitch, 1989); "the recall of incorrect, sometimes bizarre, information in response to standard questions" (Mayes, 1988, p. 111); "pseudoreminiscence which involves fabrication" (Henderson and Gillespie, 1937); "making up a lively story" (Cameron and Magaret, 1951); and "free inventions taken as experiences" (Bleuler, 1916). As Kapur (1988) points out, confabulation is a rather loosely defined term, which reflects, at least in part, the lack of detailed study of patients' actual confabulations; studies have for the most part concentrated upon the neuropsychological characteristics of the patients rather than examples of the phenomena itself.

Talland (1965) is one of the few exceptions in that he provided a analysis of confabulation which attempted to define its characteristics, based on an examination of transcripts of his patients' recall. According to Talland, confabulation (at least as found in the early stages of the Wernicke-Korsakoff syndrome) is:

a) Typically, but not exclusively, an account, more or less coherent and internally consistent, concerning the patient.

b) This account is false in the context named and often false in details within its own context.

c) Its content is drawn fully or principally from the patient's recollection of his actual experiences, including his thoughts in the past.

d) Confabulation reconstructs this content, modifies and recombines its elements, employing the mechanisms of normal remembering.

e) This material is presented without awareness of its distortions or of its inappropriateness.

f) Serves no other purpose, is motivated in no other way than factual information based on genuine data (p. 49-50).

Moscovitch (1989) adds two further characteristics to Talland's list, one taken from Talland himself, and one from a recent study by McGlynn and Schacter:
g) The readiness to confabulate may be determined by the patient's "personality structure, his traits evolved in dealing with the environment and in monitoring his self-image" (Talland, 1965; p. 44).

h) "All confabulating patients seem to suffer from anosognosia, an unawareness of their memory deficit or, at best, a profound lack of concern and lack of appreciation of its severity and extent (McGlynn and Schacter, in press)" (Moscovitch, 1989, p. 134).

Characteristic (a) is of doubtful validity. Thus patient LE reported by Shallice et al (1989) suggested that Harold Wilson was one of the examiner’s friends, and patient RJ’s (Baddeley and Wilson, 1986) confabulations included details about his mother in which he played no direct part. Moreover, patients also confabulate in experimental settings (eg. Kopelman, 1987; Wyke and Warrington, 1960) on tasks which do not require the giving of "an account...involving the patient". Rather, the spirit of Talland’s characteristic (a) is perhaps that confabulation is often most noticeable when a patient is asked to recount autobiographical experiences (see Talland, 1965, pp. 41-2). This suggests that the processes damaged in confabulators are most taxed when autobiographical recollection is required, and is reflected in characteristic (c).

Characteristic (b) is in all probability axiomatic to confabulation, and in any case is generally accepted. Characteristic (d) is more interesting, suggesting that confabulation is a reconstruction of past thoughts and experiences. This is to a large extent a logical requirement given that one accepts characteristics a-c, and perhaps (f); if confabulation is a false autobiographical account, the content of which is drawn from actual experiences, it must be that those experiences are false in their relation to each other. They quite obviously couldn’t be real experiences and correct in relation to each other: this would not be confabulation.
Characteristic (d) is however particularly interesting from another viewpoint. The autobiographical recollection of normal subjects is these days accepted by many authors as being an active reconstruction of events rather than merely the recall of some discrete, finite record (eg. Baddeley, 1990; Brewer, 1986; Reiser, Black and Kalamidas, 1987). In this way, confabulators do indeed appear to be "employing the mechanisms of normal remembering". However the products of that remembering are not normal. This suggest two possible explanations given that one presupposes a distinction between a memory store, network or database\(^2\) and the retrieval processes that act upon that database. Either the remembering processes are normal in confabulators and the database is not\(^3\), or else the converse is true, since if both the store and the processes were inoperative there could be no recall at all. These possibilities will be examined later.

Characteristic (e) is inextricably linked with (f). If it were not the case that confabulators are unaware of the inaccuracy of their recall, their confabulations could be considered lies. This would suggest, certainly at least in patients who are not suffering from psychiatric disorder, some motivation for their lying behaviour. Clearly however confabulators are not aware of their confabulations, and this is accepted by most, if not all, current theorists, and is expanded in characteristic (h). It is difficult to see how a patient might continue confabulating once s/he has become aware of their memory deficit, which is a point supported by observations that the degree of a patient’s confabulations decreases as

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2 This assumption is examined further in a later section. For the moment it will be taken as characteristic of most current theories of autobiographical memory.

3 The possibility that confabulation is an encoding deficit is dismissed for the reason that patients may confabulate about events that have happened before their illness. This is not a characteristic of amnesia per se, since classical amnesic patients do not show this pattern.
7.1(b) Theories of confabulation

There are a number of theories as to the mechanisms that are impaired in confabulation. However, as pointed out by Stuss et al (1978) few are satisfactory in providing a comprehensive explanation of the phenomenon, although some do well in explaining certain aspects. Stuss et al (1978) summarise the arguments against the older positions. Thus confabulation cannot only be a consequence of memory loss (as proposed by Barbizet, 1963), since many dense amnesics do not confabulate. Nor does it seem to be a consequence of increased suggestibility (one mechanism proposed by Berlyne, 1972) since experimental investigation has discounted this as a factor (Mercer et al, 1977). Vanderhorst (see Berlyne, 1972) suggested that confabulation was a consequence of a disorder of chronology, but many amnesic patients may have disordered chronology and yet not confabulate (Stuss et al, 1978). Other theories have emphasised the role of psychological defense mechanisms and/or personality changes as pathognomonic of confabulation. However, this again has not been supported by close investigation (Talland, 1965).

More recent accounts of confabulation range from those that concern themselves largely with anatomical considerations, through to theoretical accounts couched in information processing terms. Some present both (eg. McCarthy and Warrington, 1990). Thus Stuss et al (1978) and Kapur and Coughlan, (1980) emphasise the role of "frontal lobe dysfunction" in addition to a memory disorder. Kapur and Coughlan suggest that the differing forms of confabulation can be explained by the degree of frontal lobe dysfunction, which is akin to the view taken by Baddeley and Wilson (1986, 1988) who suggest confabulation to be a consequence of a general
"dysexecutive syndrome" overlaid on an amnesic disorder of the classical type, the severity of the confabulation probably representing the severity of the dysexecutive disorder. Kopelman (1987) copes with the differing manifestations of confabulation by suggesting that there are two forms of confabulation, the first of which ("provoked confabulation") "represents a normal response to a faulty memory", and the second ("spontaneous") which results from "the superimposition of frontal lobe pathology on an organic amnesia" (p. 1486).

Moscovitch's information-processing account is more detailed. He suggests that "pre- and post-ecphoric strategic retrieval functions are mediated by the frontal lobes and related structures". These functions serve to "guide, organize and evaluate the essentially modular, ecphoric processes mediated by the hippocampus and its related limbic structures" (p. 157). He contends that "The essentially irrational, shallow, and fragmented output of the hippocampally mediated system is most obvious in the confabulating patient...having severely damaged the frontal system...[the confabulator]...haphazardly combines information from disparate events, jumbles their sequence, and essentially accepts as veridical whatever the ecphoric process delivers to consciousness" (p. 155).

That confabulators accept as "veridical" whatever is recalled is a point developed by Delbecq-Derouesne et al (1990). They present an account which emphasises the subjective experience of remembering, and suggest that their confabulating patient's difficulty was that he suffered from an "inability to distinguish his experience of remembering from another kind of misleading subjective experience". This led to "too wide a range of experiences [being] accepted by him as memories" (p. 1069).

There are recurrent themes in all these accounts, and these seem to be reducible to three main points. Firstly that confabulation occurs in the context of some degree
of amnesia. McCarthy and Warrington (199) make the point that some confabulators can perform normally on some standard tests of memory (pp. 313). However a patient has yet to be reported who performed normally on all tests of memory, and that at least some degree of amnesia is present in confabulators is now generally accepted. Secondly most modern investigators emphasise the role of "frontal lobe dysfunction" or a "dysexecutive syndrome" in the development of confabulatory disorders. Thirdly there is a component of confabulation which characterised by haphazard recall, with events recalled out of context and (sometimes) interspersed with fictitious events or details that either have their basis in external events, or more perplexing, seem to be self-generated and idiosyncratic. Any adequate theory of confabulation should account for all three aspects. However, as outlined in the Introduction to this thesis, existing neuropsychological theories of frontal amnesia (and therefore confabulation) are for the most part inadequate (see Stuss et al, 1978) due either to under-specification, or their inability to explain simultaneously these basic characteristics of confabulation, amnesia and executive disorder4.

7.1(c) Confabulation and Traditional Experimental Theories of Memory

Some have considered "frontal amnesia", including confabulation, from the perspective of normal memory theory (eg. Schacter and Tulving, 1982; Shallice, 1988; Moscovitch, 1989; Delbecq-Derouesne et al, 1990). All agree that confabulation is primarily a phenomenon of retrieval rather than of encoding or storage.

Traditional human memory theorists have discussed the processes involved in retrieval in considerable depth. For the most part they accept that recall involves at

4 In the interests of space the arguments regarding theories of frontal amnesic phenomena presented in the Introduction will not be repeated. However these arguments are assumed in the following discussion.
least two stages, although there is some debate as to what these two processes are. Usually these two processes are the formation of some kind of "description" (Norman and Bobrow, 1979) of the to-be-remembered material followed by the accessing of a memory "store". Often the agreement that a process akin to Norman and Bobrow's is involved in memory is made tacitly. Thus, for instance, Mandler (1979) distinguishes between schema-relevant and schema-irrelevant content. Rumelhart and Norman (1973) emphasise that "retrieval of an experience from memory is usually a reconstruction which is heavily biased by the person's general knowledge of the world" (p. 450) and Tulving and Thomson (1973) describe the process of remembering as "a joint product of information stored in the past and information present in the immediate cognitive environment of the rememberer" (p. 352)\(^5\). All three views accept that recall of an event comes about through an interaction of currently perceived information and stored information, and in this way at least, they are similar to Norman and Bobrow's explanation of the description process. Even those that take a less "reconstructive" (Brewer, 1986) view of memory (eg. Morton, Hammersley and Bekerian, 1985) still distinguish between a memory store and the way of accessing it. There is, then, general agreement that there exists some sort of retrieval "code" which is used to access a memory store. This emphasises recall as a process which is performed upon a store and therefore is an active cognitive task. It also emphasises the existence of a logically separable store, which has its own characteristics. So it appears, if we are to accept these characterisations, that the memory system consists of at least two components.

However Norman and Bobrow discuss a third stage, although it is not entirely clear how this relates to the description process. This is a process of evaluation

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\(^5\) Tulving and Thomson's quote is perhaps particularly apposite given that it is a common occurrence for a confabulator's recall to be heavily influenced by his/her perception of the current environment, as will be discussed later.
where "whatever memory records are yielded through matching [ie. of the description to the candidate records]...then.. evaluated for suitability according to the verification criteria established by the retrieval specification" (p. 116). That some kind of evaluation process exists is self-evident; if there were not, we should never know when we have made a mistake unless external contrary evidence suggested it. That memory mistakes occur (and may be self-corrected) in everyday life has been generally accepted by reconstructive theorists (eg. James, 1890; Neisser, 1981; see also Conway, 1987) and the probability of correct recall seems to be highly influenced by the retrieval cue or, if this can be considered different, the question that is being addressed (see for instance Rubin, 1982). An adequate theory of autobiographical recall must, therefore, at least account for the way in which retrieved memories are evaluated, even if this is not postulated as a separate process in itself but is considered a by-product of other processes.

Whilst there is then some general agreement amongst investigators that the study of retrieval requires explanation of the store characteristics, and the methods of addressing the store and evaluating retrieved material, the question of how these processes and structures are represented is where those investigators most differ.

Associative network theorists (eg. Anderson, 1980; Anderson and Bower, 1973; Collins and Quillian, 1972; Kolodner, 1985; Ross and Bower, 1981) place most emphasis upon the structural representation of memories. Although access to the representations is discussed inasmuch as it relates to the properties of the network, the secondary processes of description and verification (especially description) are not made explicit. Baddeley (1990) points out that much of recollection of experiences

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6 Baddeley makes a useful distinction between recollection and autobiographical recall since autobiographical recall may include the retrieval of
requires the active use of strategies. The use of such strategies are relatively commonplace in recall and must serve some purpose as regards content addressing. However these aspects are not discussed by network theorists, and yet it is within this domain that most investigators agree that confabulators have their deficits.

Schema theorists (eg. Abelson, 1981; Mandler, 1979; Rumelhart, 1980; Schank, 1980; Schank and Abelson, 1977), in contrast to network advocates, explain at some length the reconstructive nature of memory. However this is at the expense of close specification of how descriptions address the store; It is usually assumed that access of memories is achieved through memorial fragments (eg. Rumelhart, 1980) which trigger activation of certain schemata. How these fragments might be created and modified in the absence of environmental triggers is not closely specified, and thus secondary cognitive processing in memory retrieval is largely unexplored. Additionally, despite schema theorists’ emphasis upon the structure of representations, the exact nature of schemata is unclear (see Morton and Bekerian, 1986 for a full critique).

Other singular theories are in some ways more complete, but still have problems in explaining confabulation. The headed records model (Morton, Hammersley and Bekerian, 1985; Morton and Bekerian, 1986) takes Norman and Bobrow’s model as a starting point, and similarly postulates three devices (encoder, describer and evaluator). However whereas the Norman and Bobrow model is limited in its specification (as the authors admit themselves), the headed records model is limited by its specification, at least as regards confabulation. For instance, Talland’s characteristic (d), that confabulation is a misconstruction of memories is difficult to explain if one supposes that i) memory records are discrete structures that have no direct relationship with each other (Assumption KA2, Morton and
Bekerian, 1986 p. 48) and ii) search with any one description will only lead to the retrieval of one record (Assumption KA6, p. 49). In fact the headed records framework has difficulty in the types of explaining memory errors that occur in autobiographical recall by normal subjects (see Conway, 1987) let alone the more florid and fantastic examples shown by some confabulators, and in this respect it loses out to reconstructive theories (see Neiser, 1986).

Perhaps the most influential single theory derived from human experimental work as regards confabulation is Tulving’s framework (Tulving, 1983). However this framework is motivated by findings from experimental studies which typically use sets of words or lists (in any case, discrete items) as stimuli, and recall rarely requires exposition of the relationships between elements in the same way that autobiographical recall does. Thus Tulving is able to concentrate on retrieval mechanisms without specifying the structure of the store, and in this sense the theory is incomplete. Moreover, Tulving’s encoding specificity encounters problems in the case of confabulation; if a memory is manifestly incorrect and fantastic, it is difficult to understand the relationship that a retrieval specification might have with it, given that the probability of recall depends directly upon that specification having been encoded at the time of the event. In this respect Tulving’s model demonstrates (as argued in the Introduction to this thesis) the limitations of a model in explaining autobiographical recall phenomenon when that model has been derived from experimental data not directly concerning autobiographical recall. This was one of the points made about two theories of frontal lobe amnesia which are clear developments of Tulving’s ideas – Moscovitch (1989) and Schacter (1987) in Chapter One of this thesis (although this is less clearly true of Moscovitch’s model, which is similar in many respects to the view taken here). Nevertheless, both developments still fail to address the detail missing in Tulving’s theory as regards the nature of the network or store and thereby
similarly limit their usefulness. Moreover whilst both notions succeed in explaining confabulation of the provoked variety, neither are able to address fantastic confabulation, as Moscovitch admits. Interestingly, Tulving (1989) has argued that the experience of remembering has been largely ignored by experimental psychology, yet it is self-evidently this domain in which confabulators have difficulty.

The present study articulates a position derived by combining aspects of the theories of Tulving and of Raajimakers and Shiffrin with assumptions derived from Norman and Bobrow’s theory. This is applied in an attempt to explain neuropsychological phenomena, in particular the properties of confabulation. Some of the assumptions made are theoretically motivated by the arguments presented in this section. The key aspects of the approach as far as the explanation of confabulation is concerned derive from Norman & Bobrow’s paper. However any theory derived from the ideas presented by Norman and Bobrow needs a more secure empirical basis in normal memory retrieval than that they provided. In particular it needs to be shown that memory lapses are a standard part of the normal autobiographical memory retrieval process so that mechanisms that guard against them are required. The following empirical investigation aims to address these issues.

7.2 Protocol Evidence

To what degree does the type of memory question which is used to elicit confabulations in patients, require the operation of memory control processes such as retrieval specification and verification in normal subjects? What is the nature and extent of errors in naturalistic recall? A lower bound can be obtained from the processes of this type of which subjects are, in some way, consciously aware. Given the success of protocol analysis in developing a data-base for theories of the higher mental processes in general (see eg. Newell & Simon,
1972; Ericssen & Simon, 1985) it seems appropriate to apply it to the interface between memory and thought.

However where related procedures have been used by the autobiographical memory theorists, they are open to two criticisms. First the veridicality of the memory - a critical issue when considering a process like confabulation - has not normally been assessed (but see Brewer, 1986). Second, the procedure generally used has been to ask subjects to write down the contents of their thoughts. But such a slow means of obtaining a record of the thought processes means that subjects must compromise between giving a brief account of any part of the thought process so as not to disrupt the overall flow, and giving an account adequate to the complexity of the part. The subtle parts of the thought process are likely to be lost.

In this study, two of the standard protocol procedures were combined. Subjects were asked first to produce a verbal protocol just giving brief labels for each element of their thought process. This was tape-recorded. Immediately afterwards, the tape was replayed to them and they were asked to provide a commentary on any label which had stood for a more elaborate thought. This procedure seeks to combine optimum procedures for thinking-aloud and for retrospective recall (immediate cued recall) (see Ericsson & Simon, 1985). In addition, in the commentary subjects spontaneously commented on errors in the initial report giving us a lower bound for the actual number of errors in the initial report. In the analysis the original report is divided into elements defined by being simple ideas expressed at most by simple main verbs in the protocol (or if less by a phrase segmented by clear pauses from the rest of the response). Each element of the original report was then categorised using the commentary as an aid. The categorisation was concerned primarily with the elements that were not just memories.
As the study concentrates upon the types of situation in which patients typically confabulate, the subjects were asked to remember recent events in which they had been involved.

7.3 METHOD

Questions (see Appendix 1) were divided equally into two types - those where we expected the subjects to have previously described the experience, where (technically) explicit summarisation was likely to have occurred and those where it seemed unlikely the subject would have previously thought about the experience.

7.3(a) Procedure

Each of the 8 subjects (4m, 4f), blind to the purpose of the experiment, was tested separately, in a quiet room on their own with the experimenter. They were shown the two tape-recorders used and it was explained to them that one would run throughout the complete session, the other being used to record the initial recall string for playback. The subjects were given the following instructions, type written:

"We are interested in the way people recall particular everyday events. We will be asking you about events that have happened to you recently. We want you to tell us as much as you can about what is passing through your mind as you are trying to remember - so you’ll be giving us a ‘running commentary’ of your recall processes.

Tell us what you can remember in the order that things come to mind, not necessarily in the order that they actually happened. As far as you can, say something about every distinct idea you have. However we don’t want you to side-track your thinking by extensive descriptions, so just give
any sort of label or key-word you can for each thought. Immediately after the 1 minute attempt to recall we will go over what you said and you can expand each remark in order to describe better what you were thinking. We will not be asking you to explain why you thought what you did, but merely giving an opportunity for you to describe what you were thinking.

You can think of the initial labelling as an aide memoire for the later explanation. When you're expanding, only include things you are confident occurred to you during the initial presentation and do not include thoughts you believe you must have, or might have thought but don't actually remember.

Sometimes, especially when you are trying to initially remember an event, other events will come to mind. Please mention these even though they may not be directly relevant to the question. However your main job is to try to remember the particular event asked of you.

So you have 2 tasks:

1. To try to remember what happened.
2. To say what goes through your mind while you are trying to remember

If by chance something happened on the occasion we ask you about, which you would prefer not to discuss, please feel free not to answer. If you find difficulty in remembering please persevere and still try to carry out the task, even though it may mean that you have to think (and describe what you are thinking) for quite some time."
Subjects were also given a transcription of a protocol taken from the pilot studies to demonstrate what they should be doing.

The 14 questions put to the subjects were designed to be as close as possible to the types of questions the subjects might face in everyday life. There were three types of question, each of which the experimenters intuitively felt were likely to be tapping material that the subject would either have been very likely to have discussed before, or tapping material that the subject probably had not rehearsed in such a way. The subjects of the three types of question either referred to a particular time of an event (eg. "Describe the first thing that comes to mind that happened to you in the month before last"), an activity (eg. "When did you last clean your car") or an object (eg. Describe a present you have recently given or received"). However these factors were only considered in an effort to make the questions dissimilar, and were not justified empirically or used in the analyses. The full set of the questions used are shown in appendix 4

Subjects were told that they were permitted one minute for initial recall (later expansion was not time-restricted), however no effort was made to enforce this since it was felt that such restrictions were not naturalistic, and might alter the structure of their recall protocol. In practice, there was considerable variation in the amount of time subjects took for initial recall (approx. 45 seconds-3 minutes). If the subject became unsure of the original question, the question was not repeated to them, and the subject had to continue with the best information they had. Complete testing of each subject took approximately 1.5 hrs. Each session was then transcribed word-for-word for analysis. This yielded 111 recall protocols, each with their consequent expansion (one question answer was lost due to technical reasons).
7.4 RESULTS

7.4(a) Basic Findings - Protocol Elements

From pilot studies a method was developed for categorising memory protocols. First the protocols were divided into putative minimum elements, then each element was categorised into one of 25 different types. Both judgements were carried out independently by the two authors who had developed the set of categories in the pilot studies (the complexity of the categorisation process meant that it would have been inappropriate to use naive judges).

As far as the process of dividing the protocols into elements was concerned, if the subject was using the labelling procedure then an element would correspond to a label. Where however the protocol contained continuous speech, an element was counted as the smallest number of clauses that both contained any information different from that in the immediately preceding element and was separately interpretable (except in the case of elements clearly left incomplete (see Appendix 5 for an example).

The two judges concurred about element boundaries on 2279 occasions. They both made a single boundary between an element with an agreed beginning and one with an agreed end, but placed it in a substantively different position on 479 occasions. On 263 occasions judge I made a division where judge II did not and on 216 occasions judge II made a division where judge I did not. Overall there was a 82.7% agreement between the two judges. For the element-categorising process which followed that of making boundary, where one judge had made a boundary which the other did not, then it was included. Where they both made a boundary but differed in its position, one of the positions was selected at random. In either case the judge whose boundaries was not accepted, rated the changed elements again. Only when an agreed set of
boundaries was obtained, were the ratings of the elements compared.

The twenty-five categories used in the element rating procedure are shown in appendix 5. An overall agreement rate in element classification of 76% was noted, with 51.3% of all responses classified as memories.

The concordance between the raters is on the whole highly satisfactory, given the following potential causes of disagreement between ratings which exist with this procedure:-

i. Some dichotomies are in fact continua (eg. that between Recall Specification and Conscious Memory Search) so the setting of a boundary must be to a certain extent arbitrary.

ii. Many elements have aspects which fit with more than one category. Consider the situation where the subject has just recalled a detail, but goes on to say "...but I'm just guessing, really...". It is clear that this is notification that the subject is not confident that the detail is correct (and therefore would be classified as a "correction possibility"). But it also has some of the characteristics of a "hypothesis" in that it may be a best estimate of what the answer might be, used for further recall, or indeed may indicate that the subject has decided that the correct detail will never be recalled, and has given up trying (which would make it a "failed recall"). These kinds of difficulties occur particularly often with "Explanations" and "Problem Solving".
iii. Even with the help of the commentary certain elements in the protocol are ambiguous.

iv. With 25 categories from which to select, errors by raters are difficult to avoid!

It is clear that certain categories cannot be at all unambiguously assigned – for instance answers (which were extensively confused with memories and explanations) problem-solving, task demand analyses and unlinked images (of which there were virtually none).

Those categories for which there are examples that judges agree on in more than 10% of the question protocols will be considered, i.e. examples in the mainstream rather than the exotica of the phenomenology of memory. They may be divided into five groups:

1. First-level Memory Control Processes
   These provide information that the memory process has been directed but gives no indication of how it should develop. These include corrections, familiarity impressions, yet unavailable/no appropriate event.

2. Strategic Retrieval Processes
   These not only indicate that retrieval has been directed but also are orientated towards directing the future course of the retrieval process – correction possibilities, failed recall, hypotheses, recall specifications, success points, verification analyses.

3. The Application of Standard Problem Solving Procedures to the Memory Task.
   These include problem-solving and task analysis.

4. Voluntary Metamemory Processes
   These appear to perform a function similar to that of meta-level processes in normal problem-solving. This include demand setters and statements.
5. Elements Arising From the Protocol Procedure Itself

These include comments, conclusions, explanations, incomplete elements and repetitions

Some of these elements - in particular recall specification and verification analyses - would appear to correspond well with the description and verification stages of Norman and Bobrow’s (1979) analyses. However it is clear that in addition a variety of other forms of supervisory memory process also exist.

The relation between the elements other than memories was clarified by an analysis carried out of the sequential structure of the protocol elements. Tables 33a and 33b give the sequential dependencies between successive elements for the two raters. Of the 625 cells, for 26 both raters had a higher number of occurrence than would be expected by chance, for 40 this was so for rater A but not rater B and 19 for rater B only. The only cells where significantly less observations occurred than would be expected by chance involved memories. Of the 49 cells involving memories, for 1 (Memory -> Memory) both raters gave highly significantly more occurrences than would be expected by chance (z >12, p<0.0 for both raters), for 23 both raters had less occurrences than would be expected by chance, for 5 this was the case for rater A but not rater B and in 4 this was the case for rater B but not rater A. Figure 6 shows those pairs where both raters had significantly more occurrences than would be expected by chance (for nonmemories) or both did not have significantly fewer than chance for pairs involving memories.
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Diagrammatic Representation of Protocol Element Clusters

Note: Dotted lines represent paths to and from memories, full lines represent pathways between other cognitive processes.
Key: 1= Answers; 2= Comments; 3= Conclusions; 4= Conscious memory search; 5= Corrections; 6= Correction Possibility; 7= Demand setters; 8= Explanations; 9= Failed recall; 10 = Familiarity Impression; 11 = Furtherances; 12 = Hypotheses; 13 = Incomplete; 14= Memories; 15 = Metamemory insights; 16= Multiple memories; 17= Problem solving; 18= Recall specifications; 19= Repetitions; 20= Statements; 21= Success points; 22= Task Demand Analysis; 23= unlinked images; 24 = Verification analysis; 25 = As yet Unavailable.
From figure 6 it can be seen that the most likely sequential relations form a coherent pattern of the mapping from general cognitive onto specifically memory processes. The elements most distant from memories in the structure generally correspond to the more purely cognitive processes, eg. demand setters (4), task demand analysis (3), metamemory insights (5), problem-solving (7), hypothesis (3). In contrast the control elements more tightly linked to the memory process lie at an intermediate distance - recall specification (18) and metamemory insights (6).

7.4(b) Basic findings - Errors

The most basic finding of the research is that errors are a frequent part of the retrieval process, when this is viewed on a moment-to-moment basis, given that one accepts the subject’s commentary as an accurate record. In other words, subjects commonly make errors whilst in the process of recalling, but far fewer erroneous errors are normally reported since these are often subsequently corrected. In total, 41 admitted errors were made in the protocols. Some were very minor (eg. 2 small pictures (instead of 3)), and a large number (17) were the giving of an event which did not in fact fit the specification of the question.

Other types of error were more interesting:

(i) Incorrect insertion of information from personal semantic memory - or at least more generic memory - occurs. Thus in answer to Q3 concerning his latest visit to the coast JS said ‘I went to the coast down to Plymouth to catch a ferry ... No..’ and in the commentary said he went to Spain most summers and therefore presumed that he would have gone the last summer. In fact he had gone elsewhere.

(ii) In certain protocols it is apparent that rival candidate memories are being assessed for which is the most appropriate answer to the question and that those
can become conflated. Thus in answer to Q (5) SX had four different memories of similar incidents involving reporting to the police break-ins or vandalism to her car and to a friend’s. She tried to disentangle them but they became conflated. Thus she said "...which is the last visit to the Police station which was when John’s car got broken in". The car in question was vandalised but not broken into. It was her car that had been broken into.

(iii) There are memories which are in fact incomplete but are claimed to be complete. Thus subject IB in answer to Q4 said that for dinner the previous night he had "soup.. er, spicy soup" and later " [That is] in fact all I had". Later he remembered separately having both pitta bread and an orange. It is noteworthy that all three examples here were corrected later in the protocol and it is noteworthy that a large percentage of the errors were corrected in the protocol (44%) often after doubt was expressed first and sometimes after more detailed cross-checking was carried out.

7.4(c) Additional Findings:– Memories

A number of aspects of the retrieval process are relevant to memories:–

i. Relevant aspects are often produced in an unbroken stream of labels or discourse as the high rate of immediate succession of one memory by another in tables 33a and 33b indicates. However, it also frequently occurs that part only of an episode may be accessible with other parts being recalled at a later time in the protocol. Thus SX in answer to Q2, when she had last cleaned her car, was able to say that it was cold, night was drawing in and it was about 3.30 (and therefore November) but did not recall that two people were present until later. All-or-none access is not a property of many of the scenes retrieved in the study.
ii. Personal semantics are frequently used in retrieval. The example used above, where the subject assumed that his last visit abroad must have been Spain (in fact it was not), is a good case. The same subject produced another example. When asked when he had last cleaned his car, he produced one answer, but in the commentary he revealed that in fact this was not the last time at all, and that he had recalled an episode based on the knowledge that he generally only used one particular car-wash.

iii. At times a strong memory keeps recurring even when it has been explicitly rejected. Thus SX when trying to remember when she had last been in a certain area of London said later in the protocol "what keeps coming to mind was the Guy Fawkes night" (a night of bonfires and fireworks) which she had retrieved first and rejected. She commented 'it just can't be the last time'... 'about 23 years ago'. In the commentary she said she kept remembering it because it was 'memorable' with 'everyone going crazy'.

iv. On many occasions subjects give a list of memories associated with the question but do not directly answer the question even though they can recall the question when asked. For instance, when asked "When was the last time you had dealings with the police", subject GM gave an extensive description of an argument she had had with a policeman who had pulled her over for questioning. However she did not say when this had occurred, and later in the commentary, she admitted that she had not actually answered the question itself.

v. At times a memory occurs which appears to be unrelated to the question but is in fact linked to it by an inferential process which probably occurs contemporaneously. For instance, when asked what the weather was like yesterday morning, one subject said that although she couldn't immediately remember what
it was like, she could remember putting her sunglasses in the car "...so it must have been sunny".

vi. The order in which memories are retrieved frequently differs from the temporal sequence in the event itself. For example, one subject recalled the details of a visit to the seaside before she recalled the travelling involved in getting there. In the commentary, she commented that "I couldn’t do it the other way round".

7.4(d) Additional Findings:– Recall Specifications

i. Precise specification of what is required occasionally occur. Thus subject IB, when asked to describe a present he had recently given or received, conjured the image of the Christmas Tree he had in his front room, then going on to "search" the image for the presents that had been placed at the foot of the tree, for one that fitted the recall criteria.

ii. Restricting the area of memory search occurs frequently. For instance, when asked for the last time he had cleaned his car, one subject based his memory search on the personal semantic knowledge that he generally only cleans his car when he is at a particular location. He then went on to search recent memories associated with that location in order to recall instances of car-cleaning.

7.4(e) Verification

This can take various forms:

i. Assessing whether the solution fits the question requirements. For instance, in response to the question "When was the last time you went to the coast", subject JS recalled his holiday the year before. However he then remembered that he had crossed the Forth Bridge on his
way to Scotland more recently than that, and went on to say

"...I mean, that was a river estuary so that could technically be the coast. But, er, but other than that it was definitely last summer".

Similarly, subject NA recalled first (when asked for his last dealings with the police) an episode when he had been waved over to the side of the road by the police who were conducting a roadside poll. However he goes on to say that:

"there was no specific interaction between me and the police and I don’t know whether you would include that".

He then went on to recall another incident in which he had actually talked to a policeman.

ii. Assessing whether more information should be obtained about a solution or question. Thus in response to the question "describe what you were doing during the two hours before lunch last Sunday", subject DHB started her recall protocol with:

"...last Sunday? Do you mean yesterday last Sunday? Or the week before that? Because [when] some people say last Sunday they mean, you know, as immediate as yesterday, and other people interpret it as the week before that...".

iii. Considering whether a putative solution is better than one previously obtained. Subject NA recalled a present that he had been given by his wife a few weeks earlier, but then went on to recall that he had given her some flowers more recently, commenting that:

"...and I suppose, you know, that’s, giving her a present". To the next question (describe the last meeting etc. that you went to) he immediately
recalled two events, one the day before, and one that morning. He went on to say "I'm not sure whether that [ie. the second event] falls in your criteria..."

iv. Checking to see whether an additional element retrieved fits with previous elements. Consider the following example, where NA is trying to describe what the weather was like yesterday morning:

"...I think I have an impression of the weather being stormy. But somehow that doesn't ring true. What I do remember is driving to Aylesbury yesterday and driving back, and on both occasions the windows of the car were down because it was hot and sunny. So why I think it was stormy in the morning I don't know. I think that its probably because I have a better recollection of the weather being stormy the day before when it definitely was stormy".

v. Reaccessing of a candidate solution so as to check explicitly if more information is available which fits with other elements of the memory. Subject DB first remembered a dinner party she had been to, then, in case the gathering was too small to fit the criteria "party", she went on to describe another (larger) party she had been to. However, she then went back to describing the original dinner party, saying "Well, the dinner, if, if its a dinner party and ten counts as a party, then, um, I could remember that very distinctly" and going on to describe this in some detail. In the commentary she confirmed that she re-remembered the dinner party and gave details of that party since she was trying to make sure that her answer fitted the requirements of the question.

vi. Checking if there are any other memories which might compete as a candidate, eg: (Subject KN) "..I'm just checking back to see if there are any other dealings with police..er..um..no that's probably it".
7.5 Discussion and Theoretical Assumptions

The analysis of the memory protocols supports a number of conclusions. Errors are a frequent part of the retrieval process when it is viewed on a microscopic level. Linton's (1987) account of the accuracy of long-term memory for key events does not correspond to the moment-to-moment retrieval of the humdrum happenings of daily life. Moreover errors take a number of different forms.

Looking in more detail at the protocols, it is possible to identify with satisfactory reliability a variety of different types of elements in the retrieval of a memory episode. Recall specifications and verifications which correspond to Norman and Bobrow's (1979) description and verification stages are indeed parts of the retrieval process. However the statistics of the sequential dependencies between successive elements indicates that the organisation of the episodic retrieval process is that of layers of control lying between general problem-solving and specific memory retrieval.

In order to discuss confabulations a set of theoretical assumptions will be made, based on the model illustrated in figure 7. They are motivated primarily by the need to account for the characteristics of the different layers of control shown in the retrieval protocols, and by the considerations raised in the Introduction. These assumptions, which are those primarily involved in the account that is given of confabulation, are related to those articulated by Norman and Bobrow. However, as explained in section 8.1(c), this particular theory dealing with the outer setting of the memory process is incomplete, in the sense that it provides no account of the basic ecphoric process - the inner motor of memory retrieval. The lacuna is filled on the present approach by three assumptions (labelled*) which are derived from discussions in more classical memory theory and to which the evidence gathered in the protocol study does not speak.
FIGURE 7
Schematic Representation of the Modulatory Memory Processes

Analysis of Task Demands (other cognitive systems) → Locatory Templates → Posterior Memory Systems

Descriptor

Mediator → Verifier → OUT
A network approach is adopted and indeed one couched in connectionist language. However, alternatives using other conceptual framework might also have been used.

The findings that some elements of an episode can be retrieved separately from the rest (finding 8.4(c)) together with evidence of the conflation of memories (finding 8.4(b)) argue against a view of storage as consisting of discrete records. Instead a fairly standard associative net view related to that put forward by Raajimakers and Shiffrin (1981) combined with positions put forward by Barsalou (1988) and Schacter (1989) will be adopted. A rather more novel view taken here is that a position developed from that of Norman and Bobrow (1979) is linked to the associative net view.

The Overall Function of Episodic Memory

1. Episodic memory is assumed to be intimately linked with processes involved in coping with novelty (Schank, 1982; Shallice, 1988) and therefore to show many characteristics in common with problem-solving. The main way that this assumption manifests itself in the theory is through the properties of the description and verification procedures (see below). Other types of protocol element which fit with this characterisation are the voluntary metamemory processes which appear to function in an analogous fashion to the meta-level processes that occur in normal problem-solving (see De Groot, 1966), and the Task Analyses which in most circumstances will correspond to means-end analysis.

In addition explicit problem-solving episodes occur in the protocol (see Results, section (a)). Moreover, the phenomenon of apparently irrelevant memories being produced at the same time as an inferential process which links it to the solution to the question (Finding 8.4(c)) corresponds in this domain to the insightful flashes that occur in normal problem-solving.
An additional assumption made is that the memory control processes are part of what Norman and Shallice (1980) called the Supervisory System and therefore information available to systems at that level from the memory store/associative net correspond to what the subject is aware of and what is available in the protocol.

The Ecphoric Aspects of the Process

Properties of the Store

2. The memory store may be considered to be a set of domains each of which is realised by a system containing representations of a particular type (memory elements). Co-occurrence of activated representations in different domains leads to the formation of associative links between the representations. These can be strong, if the co-occurrence occurs frequently, or individually weak but potentially mutually reinforcing if multiple concurrently activated set of representations are reactivated (for speculation on how this latter process which relates to encoding specificity might occur see Crick, 1984). New representations are formed when the associative link between two elements becomes so strong that this can then be considered a new element in itself. Thus the storage space is in a state of continuous evolution. However the empirical study provides no evidence relevant to this assumption.

The representation of a typical event will form as Neisser (1982) has pointed out a complex nested structure. It will contain many links of differing strengths between different types of element and forming a set of overlapping attractors within the semantic space. A single element within an event structure (eg. "the car we saw last Saturday" as opposed to "the car we saw last Sunday") is represented by its central semantic characteristics and the activation of additional contextual information (in the example, the day on which the car was seen). This makes specific predictions regarding retrieval failure which will be examined later.
7.5(a)ii 3. Properties of the Retrieval Process

Retrieval of linked representations normally requires two different types of input (from (i) the Locatory Templates and (ii) the Descriptor (see figure 7). The first of these (stimulus-type) corresponds to the 'stimulus' of a classic encoding specificity account, the second (the control type) to the 'description' of Norman and Bobrow. The first involves the transient activations of certain representations in the memory system. The second makes the effect of the relevant input longer lasting, and explores the associative links between the representations. Thus remembering an event is a reconstruction of a pattern. A region of the memory space remains activated by the description until it succeeds or is explicitly withdrawn. During this activation, other regions are inhibited.

The phenomenological correspondence for the second of these types of input would be the recall specification elements of the protocols. For the explicit withdrawal it would be the failed recall type of element. Thus it is assumed that memory retrieval has a control input very similar to that which occurs in problem solving. It differs from the position of Norman and Bobrow in that retrieval is not controlled solely through the Description system. Other controlling inputs (eg. motivational ones) and transiently active inputs from the Locatory Templates are also present. Hence memory protocols can illustrate a failure to follow what would be the most appropriate development from a problem-solving perspective (see Results section (d) (iii) and (iv)).

4. The production of an element in the retrieval process occurs when the activation of a stored representation reaches threshold. This will happen due to the combined influence of the activation of other associated representations plus the determining tendency. This is basically a spreading activation assumption for which the present study provides no additional evidence. Given
the variety of influences postulated, the possibility of inappropriate episodic retrieval is continuously present (see section 8.5(a)i). Additionally, the way different memories are represented by different patterns of activation in the same net - not in different records - allows memory conflation to occur (see finding 8.4(b)). (Such memory "blends" will be few in number because of the attractor structure of the representations).

5. Not all the activated representations potentially available need to be within the explicitly available set required to give rise to an above threshold activation of a further representation. This assumption is required to prevent any incorrect recall blocking subsequent correct recall.

The overall consequences of these assumptions is that retrieval of a small part of an event would require the reinstituting of some of the co-occurring elements (as in encoding specificity) together with the appropriate determining tendency. Retrieval of different parts of the event will proceed by the recovery of a series of the substructures, the order of retrieval being dependent upon the relative strength of connections and the specific retrieval conditions which as Moscovitch has pointed out is not identical in order to that occurring in the event itself (finding 8.4(c). Thus one can have the temporal gaps observed between retrieval of different parts of an overall event (finding 8.4(d)).

7.5(a)iii Control Inputs

6. Different levels of control-type input can be simultaneously present. It is presupposed that in the representation of the problem, multiple levels of recall specification can be activating, simultaneously, regions nested within the representation space to different degrees (see finding 8.4 (d)). These correspond to Norman and Bobrow’s descriptions.
7. An episode once retrieved is subject to a verification procedure in which a number of subprocesses can occur. This can involve both a Verification Unit and a Mediator. This corresponds to the verification stage of Norman and Bobrow but also to the assessment or evaluation stages that occur in problem-solving (eg. De Groot, 1966). Many examples of explicit verification and error correction exist in the protocols (see Results sections (a) and (e)). Moreover the frequency of such protocol element errors indicates that such a process is essential.

It is assumed that there are two basic types of verification process. The first occurs when an element of the memory is retrieved and is seen to contradict one or more of the elements that have previously been retrieved. For instance, subject JS, when asked to describe the first thing that had happened to him in the month before last recalled the following:

"...Something that happened in January? Er, I comp...completed a major sale. No! I didn't complete a major sale in January at all. I didn't sell anything at all in January because I remember looking at the board and that was blank [ie. the sales figures board]."

This process does not need to be initiated by any awareness of the possible inadequacy of the previously retrieved elements. Instead what is essential is the operation of a process which detects a contradiction between separately retrieved elements of the memory. This occurs in cases of Correction Possibility eg. JS recalled that the last time he had cleaned his car was at a carwash near to where he works. He continues:

"...I seem to remember it costing ridiculous amounts of money as well. Oh no, hang on a second! [NB: correction possibility] Maybe I might have wa...might have, er, might have been on Upper Brook Street, theres a good car wash on there...In fact
it was because I was handing it over to one of the directors for a week or two so I thought I had better clean it first..."

This process may require re-entering outputs into the net controlled by a Verification Unit which is capable of detecting and signalling contradiction.

The second type of verification process occurs due to an awareness of the possible failure of previously retrieved elements to fit the overall requirements of the memory. An explicit process of checking and problem-solving can then occur - which would correspond to Verification Analysis. It is held that the Verifier will signal possible error but that any problem-solving processes would be initiated by a Mediator.

8. Outputs can be re-input as Locatory Templates. As part of the second type of verification procedure, output can be re-input to see whether further related elements can be retrieved, and whether they do or do not fit with the preceding elements. Subject DT, for instance, when trying to remember the last time she had had dealings with the police, remembered being in the local police station reporting an incident where someone had broken one of her car windows. However she also remembered being in the same police station on another occasion when her partner's car had been vandalised. In trying to decide which was the more recent of the two, she activated in turn the memories she had in order to try to discover a key piece of evidence that would help her. This key evidence was which part of the car was damaged. In the commentary, she reported

"...I had an image of standing in Kilburn police station, um...and I went there two, two periods quite close together, once to report the broken window in - the second broken window in my car and once either before it or after it to report that John's aerial
had been damaged and the car had been scratched and at the time that, that I had that image, um, I, I, initially remembered it as being going down for John's car and then I was trying to think was that the case or was it when I went down for my car?"

7.5(b) More Specific Assumptions.

9. Retrieval of a subset of the elements of the complex structure that represents a complete event corresponds to 'summarisation' of the memory trace. In the context of the occurrence of related events with overlapping representations, repeated application of this process leads to 'schematisation' of the memory trace (Barclay, 1987) and the development of generic memories. In classical memory theory it is well accepted that memory retrieval acts as a learning trial (Tulving, 1986). In a connectionist account of memory representations, the reactivation of some of the elements in a memory representation will strengthen the links between those elements at the expense of other links which are not reactivated. Retrieval will therefore produce the summarisation described in normal recall (De Jong, 1983; Kahneman & Miller, 1986; Barsalou, 1988). If the process occurs sufficiently often and in particular if it occurs for many similar events then what the autobiographical memory theorist Barclay (1987) called schematisation will occur, namely generalisation across events. Eventually when such schematic representations are sufficiently activated they would be accessible without the need for Descriptions. They would then correspond to 'generic' memories or 'personal semantic memories', which could be accessed by routines similar to those by which knowledge is accessed in the on-line carrying out of cognitive skills, which do not require novel problem-solving (the schema of contention scheduling on the Norman/shallice theory). Thus when specific events are asked for, generic representations can be retrieved - (Reiser et al 1987; Pillemer, 1988; Williams & Dritschel, 1988; see also Results 2(i), 3(ii))
9. Some of the domains of representation are more abstract in that they represent an activity which takes place over a sizable time (unlike the potentially second-to-second changes in other representations.) These correspond to the MOPs of Schank (1982) and Kolodner (1985) and in certain respects the 'headings' of Morton, Hammersley and Bekerian (1985). Typically, a subject in an experiment when the episode is retrieved fairly easily begins their response with a general statement and then goes on to describe the details of the event. For example, subject IB, when asked when he had last visited the coast, said:

"The last time I went to the coast? Crikey! Um, last weekend in fact. Yes, I can remember it. Drove down, had a meeting in Reading. Got out fairly early. Whizzed down to the parents' house [ie. on the coast]. Remember I had a headache..."

It is presupposed that the general statement reflects access to the more abstract representation. These more abstract representations include certain other temporal units eg. the stages of a day-routine (Ellis, 1988). However, it is assumed that there is not a continuous, available "time-line".

7.6 The Explanation of Confabulation.

How does the theory of retrieval presented above explain confabulations? What, first, requires explanation? A large number of clinical accounts of patients who confabulate and of their confabulations exist in the neuropsychological literature. However, in most of these, quantitative results are limited to background neuropsychological tests. In addition there are a very small number of group studies where experimental manipulations of the confabulations have been carried out (eg. Mercer et al, 1977; Shapiro et al, 1981) and an almost equally small number of single case studies where this is done (eg. Delbecq-Derouesne et al, 1991;
Moscovitch, 1989). There are a few existing accounts of the mechanisms damaged in confabulators (eg. McCarthy and Warrington, 1990; Moscovitch, 1989; Delbecq-Derouesne et al, 1990) which are not, as reviewed in the introduction to the thesis, based on empirical study. All three types of available evidence will be utilised in the following account of the mechanisms damaged in confabulation, which is based on the empirical data already presented.

7.6(a) Experimental Studies of Confabulation

There are few experimental investigations of confabulation. However those that have been carried out have yielded some useful findings which either directly or indirectly support the view taken here.

Mercer et al's (1977) innovative study does both. They investigated 11 patients, 5 of whom had Korsakoff's Syndrome, 3 cases of encephalopathy, a case with normal pressure hydrocephalus and 2 cases with dementia. Four of these patients were "nonconfabulators", but had been "identified by the attending physicians as possible confabulators" (p. 429), two showed a severe confabulatory disorder and four showed mild confabulation. The degree of confabulation showed by the remaining patient changed throughout the study period.

These patients were asked a series of 41 questions which were divided into four broad categories: 1) questions probing "overlearned issues associated with remote memory"; 2) recent memory; 3) questions that could be answered by drawing upon cues available in the room (eg. present weather); 4) questions that frequently drew an appropriate "I don’t know" response reply from a nonneurological control group (eg. the winner of the Superbowl last year).

On the basis of their patients’ replies to these questions, Mercer et al conclude that there are quantitative and qualitative differences among patients having different degrees of confabulation. Severe
confabulators have grossly impaired recent memory, are comparatively successful with long-term memory questions, and are generally aware when an answer to a question is not expected. Mild confabulators, on the other hand, differ from severe confabulators chiefly in their response on questions involving recent memory. Their overall success on such questions is far superior, and there is a greater tendency toward verbal self-correction and latencies before responses. They are alert to environmental cues and can usually draw on them to arrive at correct answers. Importantly, and in contrast to the emphasis laid by some authors (eg. McCarthy and Warrington, 1990), Mercer et al emphasise that a necessary (though not sufficient) prerequisite for confabulation is impaired memory function. They suggest that the replies given by confabulators "reflect use of those aspects of memory that are most strongly entrenched - overlearned long-term memory topics" (p. 433). As discussed above, this may be due, in part at least, to the fact that they have a memory problem. Mercer et al differ from some (eg. Moscovitch, 1989; Schacter, 1987) by tackling the differences between spontaneous and provoked confabulators directly; they suggest that the severe confabulator differs from the non-confabulator or the mild confabulator primarily in their decreased ability to monitor and correct answers.

They conclude that confabulations occur due to the "coincidence of four factors": a) the patient believes that a response is required; b) an accurate memory of the answer is lacking; c) an overlearned and affectively significant response is available; and d) The ability to monitor or self-correct is defective.

Factor C is reminiscent of Talland's characteristic (g), and factor D here is in many ways subsumed by Talland's characteristic (e). These similarities are primarily of interest because of the differences in the methods of derivation: Talland's characteristics were derived from his (untested) observations of confabulators, whereas Mercer's are the result of experimental manipulation.
Of course the bulk of Mercer’s conclusions 2, 3 and 5 can be explained by the fact that the severe confabulators have poorer recent memory - one can hardly be expected to confabulate in response to a question to which one easily knows the answer. Additionally a memory problem is more likely to affect knowledge of recent memory than overlearned (personal semantic) remote memory items.

However there are some additionally interesting points to be made about Mercer et al’s findings. One of the things that they did not look at was the proportion of responses that were incorrect, that were also confabulations. Since the severe confabulators were poorer at answering the recent memory questions (perhaps, because of weaker memory functioning), the apparent difference between the mild and the severe confabulators might be only one of degree.

If their data is considered in this way an additional, important point becomes obvious: The greatest difference in the occurrences of confabulations between the two groups of confabulators showed itself in response to the "I don’t know" questions. Thus it seems that one of the clearest differences between mild and severe confabulators is that severe confabulators are willing to give an answer to a question where mild confabulators will say "I don’t know". The other differences are more a matter of degree than representing a qualitatively distinct performance. Of course a deficit in "ability to monitor and self-correct" might lead a patient to produce a confabulatory reply, then to check it and reject it, but an equally plausible interpretation is that the severe confabulators’ retrieval system, having suffered greater damage, produces erroneous recall whereas the mild confabulators’ system is sufficiently intact for this not to happen as often. This is to say that one function of the normal description/verificatory system is to facilitate failed recall as appeared in our normal subjects’ protocols. The failure of this function has its consequences in Talland’s characteristic (e).
Mercer et al assume that when a normal subject is given a question that s/he cannot answer, the retrieval system first recalls some candidate, for this to be rejected post recall if inappropriate. A problem for this account is normal subjects' ability to almost instantly know that they do not know the answer to a question, or, at a lesser extreme, their sense of the difficulty of the recall task ahead. Our normal subjects often expressed first some estimate of the difficulty of the recall task they had been set, even before any actual recall had begun. This presumably represents a first-pass (or early-pass) failure at the descriptor level.

Thus an additional, interesting question raised by Mercer et al.'s data is why it is that confabulators produce a memory in the first place when others do not, and then, secondly why do they then not realise that it is (possibly) in competition with what is logically plausible given their surroundings, other information they have and so forth.

Whilst it is difficult to disagree with Mercer et al regarding the breakdown of a post-recall monitoring system, there needs to be an additional postulate. On the account given here, this is that confabulators show a breakdown in a descriptor process which is involved in specifying the requirements of the to-be-recalled memory. If this specification were too noisy (as would be the result of a compromised system) this would result in a greater probability of an inappropriate memory fitting the criteria and thus being recalled. An additional consequence would be an occasional inability to recall an event or detail on one occasion, on the next occasion (possibly) being able to do so. Examples of these types of occurrences in the protocols of confabulators will be given below.
7.6(a)i Description or Verification?

Are the processes of description and verification dissociable in the damaged system? There is some evidence that they may be. Thus Dalla Barba, Cipolotti and Denes (1990) gave a confabulating patient suffering from Korsakoff’s syndrome a series of different types of questions. In this way, this part of their study was similar to Mercer et al (1977). However their questions were rather different. They were divided into six categories, two tapping autobiographical memory, and four probing semantic memory:

1) Remote autobiographical memory (ABM) questions.
2) Questions probing recent ABM.
3) Questions on famous facts.
4) Questions about famous people.
5) Questions about mythological stories (ie. Snow White).
6) Questions constructed in such a way as to elicit an "I don’t know" response from normal subjects.

Their patient showed a different pattern to Mercer et al’s patients in that she did not confabulate on the "I don’t know" questions. Nor did she confabulate on any of the questions tapping semantic memory. Significantly, the content of her confabulations was never fantastic. Dalla Barba et al comment that the content of her confabulatory responses "...was never markedly bizarre, but either consisted of real memories framed in a wrong temporal context or of answers which were clearly false but still plausible" (p. 9).

The account given here suggests that the pattern demonstrated by this patient would be the consequence of descriptor malfunction in the context of an adequate verificatory system. The normal subjects in this study occasionally produced memories which were particularly salient for them, and which might have been appropriate given another context. These memories were usually corrected by a post-recall verification process. They
came to realise that these memories were not in fact what was required either as a consequence of a post-recall problem-solving analysis, or by recall of some additional memory (or detail) which was incompatible with the original (see findings 6(iii) and 7). The model presented here would make the prediction that if this process were inoperative in the context of intact descriptor functions, a consequence would be the production of memories which are plausible in themselves, but for which there may be an alternative (which is not considered). This would lead to memories being recalled "out of context" (Talland's characteristic (d)). Additionally, and critically, one would still be able to know that one did not know something (if there were no memory which clearly fitted the criteria).

Whether the complementary pattern of deficit exists (intact verification process with descriptor dysfunction) is harder to determine. The essentially serial arrangement of the processes as depicted in figure 7 would predict not - if one cannot produce memories of sufficient coherence in the first instance, potential incompatibilities would not be detected by the post-recall verification process.

7.6aii Relationship Between Description and Verification

Shapiro et al (1981)’s study speaks to this question. In addition to asking their confabulators questions in a Mercer et al-style paradigm, they also administered a modified version of the Visual-Verbal Test (Feldman and Drasgow, 1959) under varying conditions to see how these conditions affected the confabulators’ performances. The Visual-Verbal Test requires the subject to detect two different possible groupings of four stimuli on each card. Dimensions along which these groupings can be made are colour, shape, size or orientation, and the subject is asked to "find three items (on each card) that are alike in some way". Once this has been achieved, the subject is asked to "find three items on the same card
which are alike in another way". The conditions under which the test was administered were as follows:

1. Motivation - the patient received verbal feedback from the examiner after each trial designed to heighten the patients’ concern about their performance.

2. Inhibition A - the patient was required to wait and think for 10 seconds before replying. They also had to perform an oral spelling task for one minute between cards. This aimed to improve the subjects’ self-monitoring and inhibit perseverative tendencies between cards.

3. Inhibition B - identical to Inhibition A except the oral spelling was performed between trials on each card rather than between cards. This aimed to improve self-monitoring, ability to inhibit between-trial perseveration and the ability to shift response sets.

4. Modeling - the patient was shown a list of the four possible dimensions along which they could "group" the stimuli. After they had used one of these dimensions, that option was covered up on the list, and the patient had to choose their second dimension from the remaining three. This aimed primarily to examine the patient’s ability to make good use of "environmental cues".

The results were very interesting. The two major findings were that there was an increase in errors from trial 1 to trial 2 across all experimental conditions, but that while on trial 1 the experimental conditions (irrespective of what they were) decreased the number of errors made, this did not hold for trial two. Shapiro et al drew three main conclusions from these results.

Firstly they maintain that "confabulators have difficulty monitoring the accuracy of performance" (pp. 1073-4). They based this upon the findings from conditions 1-3, suggesting that all confabulators improved performance when they were directed towards monitoring responses (irrespective of the method used), but that this monitoring was nevertheless not sufficient to prevent errors on trial two.
Secondly, Shapiro et al contend that "confabulators have difficulty shifting response sets and inhibiting perseverative responses" (p. 1074). This is largely self-explanatory, the one particularly interesting feature here being that severe confabulators in particular, showed a tendency to not only perseverate on their choices, but that the perseveration also extended to their explanations about their choices. The third conclusion that Shapiro et al draw, is that "confabulators have difficulty using cues to aid performance" (p. 1075). This is supported by their finding that even with the benefit of the cues in condition (4) above, severe confabulators in particular did not improve their performance.

While the first and second conclusions are in agreement with previous work (eg. Berlyne, 1972; Pick, 1903; Stuss et al, 1978; Talland, 1965; Mercer et al, 1977) it is the last conclusion that is particularly important from the point of view of the present study. Shapiro et al found that whilst confabulators could not benefit from the use of cues in a problem-solving task, this was not the case when the patients were given a memory task.

Shapiro et al showed their confabulators 20 black and white drawings, each of which contained three major components (eg. a man in a boat with a fishing rod). After a period of distraction with an unrelated task, the patient was asked to describe the picture. If the patient could not give a complete description, they were given a "cue" of one of the items from the picture (either verbally or visually). Failure still to describe the whole picture accurately resulted in giving the patient two of the three items from the original. Recall on each occasion was scored as correct, incomplete, "I don't know" or confabulatory. Under these conditions, Shapiro et al discovered that "despite the structured nature of this task, many confabulatory responses were elicited" (p. 1073). However the number of confabulatory responses "showed a marked decrease from the initial condition...to cue conditions" (p. 1073). In addition, Shapiro et al
noted that mild confabulators tended to reply more frequently with "I don't know" responses, echoing the findings of Mercer et al (1977). Interestingly, the patients confabulations showed marked consistency in their confabulations. Shapiro et al record that "One patient tended to perseverate elements from one picture to the next even when several days had elapsed between picture presentations and even when he denied remembering earlier pictures. Another patient tended to perseverate one particular feature across several consecutive pictures, although the source of this feature did not come from any of the pictures. The source of the confabulatory answers that were not perseverations could not be determined" (p. 1073).

This consistency in confabulation has also been noted by Dalla Barba et al (1990), and suggests summarisation or schematisation (see above). However Shapiro's discovery that confabulators can produce more accurate replies when given cues needs to be discussed.

If retrieval specifications were not influential in confabulators' recall, then Shapiro et al's patients should have been expected to show no benefit from cues. The chances of their producing a confabulatory recall would not have been affected by their manipulation. But this was not the case. In terms of the model presented here, it could be argued that giving the patients "cues" is in a sense subsuming the function of the (damaged) descriptor process, allowing the patients to gain a more accurate recall specification. Shapiro et al report that the "nature of the confabulatory responses was not altered when reduced in frequency" (p. 1076). Thus it was not the case that providing the cues somehow altered the test situation to one in which confabulations were not elicited. Obviously the cues helped the patients recall more accurately. This process of providing "cues" or new recall specifications was often internally driven in our normals in a way that confabulators seem unable to do.
However the consistency of the confabulatory responses requires additional explanation. Assuming that one of the functions of the descriptor was the potentiation of certain traces in respect to others would predict some consistency in confabulation. However the active schemata could be "seeded" by externally-provided recall specifications. Confabulations are clearly not a mere jumble of recall, but follow a general recall "trend" dictated by the currently activated schemata (Talland's characteristic (c) and possibly (g)). New incoming information tends to be incorporated in the context of this schemata. Apparent inconsistencies are not noticed (since there is a verification analysis failure) but merely become a part of the recall string. Case examples of such recall from patients will be given in the next section.

7.6(b) Phenomenological accounts of confabulation

The findings from our normal subjects show that the process of autobiographical recollection requires considerable involvement of memory control processes. This raises the issue of what the consequences might be for a patient who has suffered damage to these processes. One would of course expect to see a reduction in autobiographical recall, of the relevant characteristics isolated from the protocols of the normal subjects, together with those features of recall which would be the consequence of their absence. A number of investigators have reported accounts of the confabulations of patients they have studied which are detailed enough to allow close analysis.

7.6(b)i Descriptor Process Dysfunction

As explained above, the account given here predicts a number of consequences of descriptor process breakdown. The first is that those confabulators with descriptor failure would be expected to give a different response, on different occasions to the same question. In other words on some occasions a confabulator might appear to be
able to remember something, on other occasions not being able to remember the same thing. Alternatively, what they remember in response to the same question might be different. There may be two effects here. The first is due to the way that part of the descriptor process is involved in specification of recall. The second is that, in the absence of this ability currently activated representations would prevail.

Consider first the process of recall specification. Large parts of our normal subjects’ protocols were devoted to recursive recall specification and modification. Problem-solving and hypothesis formation were used to develop new specifications should the products of the original prove not satisfactory. This process was fallible in our normal subjects, but not often so. Consider what would happen with pathological damage at this level. This would result in the patient not being able to initiate a self-restricted memory search accurately, and within an isolated event that is being explored, not being able to "home in" on the particular detail if it is not central to that event. This would mean that the likelihood of the patient being able to retrieve an event accurately would depend upon the degree of match between the given specification (ie. the precision or characteristics of the request) and the representation of the event(s) of which the required information is a part. Thus one might expect that a person suffering from such a disorder would apparently show an ability to retrieve a particular event on one occasion, not necessarily being able to recall it the next given that the recall request, some detail inherent in the situation of that request, or the currently activated representations differ. Supplying the patient with further information, a modification of the original request, or otherwise altering the recall specification might be expected to increase the likelihood of successful recall in a way that would not happen in classical amnesia.

Consider the Korsakoff confabulator, Helen, reported by Talland (1965). Talland reports that on some 40
occasions, spanning 6 years, she had been asked how long she had been in hospital. She always replied that she had been admitted "yesterday". Talland continues "...Still, if questioned how she spent her days in the hospital, she would often describe her work in the sewing room, some of which manifestly spread over several days. If asked to talk about her daily routine, without specific reference to the hospital, her answer would be drawn from an earlier setting of her life" (p. 24)

An even more dramatic example is provided by case 1 from Damasio et al (1985). They report that:

"...at one point the patient described himself as a college senior, but when told he had completed college eight years earlier, he was able to describe his current circumstances accurately...A similar effect occurred when the patient was questioned about his wife and family. He initially denied he was ever married, but helped by successive cues, he gradually described his wife, her name, his children, their ages, and their names" (p. 263).

This is a good example of the type of apparent "inconsistency" which clinicians often meet in frontal amnesics. In this case the request by the experimenter which included the word "hospital" would enable her to access information regarding activities at the hospital. If however the key "hospital" reference was omitted, she was unable to access that information, falling back in favour upon more stock information - those memory representations that are most primed.

Interestingly, Damasio et al's patient provided some excellent insights into this condition, commenting on his inability to remember things one minute, but instantly remembering it once sufficiently cued. In response to such a situation, he remarked "...Now why didn't I recall that? This is the problem with the surgery [ie. his operation to clip an anterior cerebral artery aneurysm]. Somebody mentions something and it comes flying out of
"your head. It's like flashes of knowledge that unleash from the dark side of your head" (p. 264).

The second consequence of that descriptor breakdown, "intrusion" from currently activated representations is also illustrated by the example above. However, this is only part of the picture. An additional consequence of reliance upon already activated schemata is that one would predict that events recalled would be heavily influenced by on-going template activation - thus recalled events would be expected to contain detail which either belongs to other events, or which were never part of the event in the first place.

Case 1 from Damasio et al (1985) also showed this second consequence of descriptor breakdown in that he believed he was a spaceship commander at the time of the Columbia space shuttle mission. In addition, during that period, he occasionally became a "space pirate". Damasio et al make the point that the visually based descriptions the patient provided appeared to come out of "Star Wars" or some television science fiction program (p. 264).

A further example was shown by a patient LE previously reported by the present authors (Shallice et al, 1989). LE was undergoing neuropsychological assessment in a standard hospital office. When asked where he thought was, he replied that he was in an office at his place of work (he worked in the building trade). Immediately afterwards he was given the famous faces test (REF) which contains a number of former Prime Ministers of Britain. At the end of this test he described how he had met Harold Wilson the day before whilst walking down the High Street, and had discussed with him a building job that they were working on. He thought that Harold Wilson was the current Prime Minister and that he (ie. HW) was a friend of the examiner.

This highlights also the schematisation involved in confabulation. Damasio et al's patient commented on his belief that he was a spaceship commander, once he had
come to believe that this was not true. When asked what he believed at that time, he remarked "I was a pirate and I commanded a spaceship (embarrassed laughter). Now I realise it’s not true. I didn’t dream that; it was a total part of consciousness. To me, it was reality at that time. It’s embarrassing now. At times, even reality is a dream. It was continuous throughout the day. I did believe it!" (p. 265).

The implausibility of these confabulations require, however, some additional explanation in terms of other supervisory processes, which will be dealt with in the section on mediator failure. However, as already noted, not all confabulation contains implausible material. An intermediate failure at the descriptor level is where the patient is showing recall which is based on events in which they were involved, but in which the context, or details within the memory are confused. An extreme example is give by Damasio et al (1985). Their case 4 "...described her recent graduation from a technical institute: 'I just got out of there’. In fact, it was her daughter who had just graduated from the school". (p. 268). It is clear here that there was some general activation of memories surrounding a graduation. However, on the account here, the recall description was insufficiently precise for the potential incompatibilities of her recall to activate verification procedures.

Examples of this type in confabulators are also documented by a number of other authors (eg. Baddeley and Wilson, 1986; Berlyne, 1972; Delbecq-Derouesne et al, 1990; Kapur and Coughlan, 1980; Moscovitch 1989; Shapiro et al, 1981; Stuss et al, 1978). On occasion, the normal subjects in this study also recalled information incorrectly - conflating details and recalling out of context. Yet these "memories" only relatively infrequently were accepted in their entirety. Often they were corrected post-recall. The inconsistency which occurs between competing memories recalled concurrently rarely goes unnoticed in normal subjects, whereas (as we
have seen) it necessarily does in confabulators. What then is the nature of this verification process?

7.6(b)ii Dysfunction of the Verification Process.

The (working) verification process was noted in the protocols of our normal subjects as the elements called "Correction Possibility" and "Corrections". These elements appeared at the point where the subject has realised that some aspect of their recall was incorrect, and typically originate from the recall of some new detail which conflicts with the memory as already recalled. The "Correction Possibility is the 'switching point' of the verification process, with the correction itself often taking place after some recursive use of the other memory control processes. In other words, at the point where the subject has come to realise that the detail s/he has just recalled is incorrect, s/he is not necessarily aware of the 'correct' detail. Ascertaining this requires further reconstructive autobiographical recall using the full range of control processes such as hypothesis testing, problem-solving and respecification of recall descriptors.

These processes do not seem to occur in florid confabulators. In Mercer et al's experimental study of confabulation, their confabulators paused before responding to a question on only 10% of trials. Their nonconfabulating neurological group did so on 59% of the trials. In addition, "verbal checking" (where patients voices aloud the fact that they are correcting the answer they have just given) occurred on only four occasions in the case of their severe confabulators, whereas their mild confabulators corrected themselves 21 times. Evidence for the importance of this process in accurate recall comes from their finding that nonconfabulators were more likely to answer correctly those questions on which a significant latency was shown, whereas the confabulators not only showed fewer latencies, but also were no more accurate in their answers when they did pause before answering. Mercer et al concluded that their
findings were consistent with the hypothesis that the latency of responding represented "an internalization of a self-correction process", and that confabulators tended not to show this self-correction.

It could be argued that decreased response latencies may reflect impulsivity of responding rather than an actual inability to verify memories. However this does not seem to be the case, since confabulators are renowned for producing conflicting information in recall, without apparently being aware of the conflict. If the conflict is pointed out to them, this provides a new cue (locatory template) for further (erroneous) recall, rather than their changing their original recall in the light of the new information (as would a normal subject). Consider, for instance the confabulator - patient RJ - reported by Baddeley and Wilson (1986). RJ was describing an event where he sent a letter to his great-aunt, telling her that his younger brother had been killed. He was asked about the wording of the letter, and replied:

RJ: Dear Auntie Bertha, I am sorry to tell you that Martin has been killed in a car accident..."
E: Have you got just one brother?
RJ: I've got three now; I've got two actually, one older and one younger.
E: What are they called
RJ: Martin and John.
E: Which one was killed, then?
RJ: Martin
E: So did you have two Martins?
RJ: We had actually in those days one Martin, then mother had another one and we called it Martin as well. I think she felt a bit sort of morbid about it so she called it Martin so we had two, I suppose, yes, or what would have been two. (Baddeley and Wilson, 1986; p. 244)

A similar example is given by Barbizet (1970), who describes a confabulating patient who had suffered a cranial trauma as a result of an accident in his car. However, when asked about his accident (nearly two years
post-trauma), he reported that he had been piloting a plane which had crashed into a car which had strayed onto the runway. When he was then shown a photograph of his car, he denied that he had had an accident in it, claiming that since at that time he was piloting his aircraft, he could not have been in his car at the same time. Barbizet reports "This [ie. showing him the photograph] started him off again, entirely convinced of the story he had just told, with another recital, which, although enriched with a few additional details, remained perfectly coherent and in conformity with the initial account. This delirious conviction persisted for long months, but little by little he began to acknowledge that he really had had a car accident, without in any way denying his airplane accident. When he was seen again...he told us once more and with great precision of detail all about his aircraft accident, then, linking the two together, he said: 'Would you believe my luck? On leaving the police station I had a second accident, in which my car was completely wrecked!'" (p. 50)."

7.6(b)iii Dysfunction of the Mediator process

That the frontal lobes are involved in certain types of problem-solving, particularly in novel situations, is now generally accepted (See Stuss and Benson, 1986 for a review). Equally, many investigators have suggested that the presence of confabulation in a patient may be related to the degree of dysexecutive-style problem-solving deficits that the patient shows, probably more so than the degree of memory dysfunction per se (Baddeley and Wilson, 1988; Kapur and Coughlan, 1980; McCarthy and Warrington, 1991; Mercer et al, 1977).

It is clear from the protocols of the normal subjects that at certain times (most typically when the memory required is not immediately available) correct autobiographical recall requires some hypothesis testing and frank reasoning (see also Baddeley and Wilson, 1986). The products of this problem-solving approach are then used to set up new descriptors for recall.
Consider the implications for memory performance of a patient who has problems on Shallice and Evans' (1978) Cognitive Estimates test. This test requires patients to produce estimated answers to a set of questions which they are unlikely to be able to answer purely from knowledge alone, but where reasonable estimates of what the answers might be can be given after some reasoning. For instance, one of the questions asks the length of the average man's spine. Clearly there are a number of ways in which one can arrive at a reasonable estimate. One might be to think of the spine as approximately half the length of a man's total height, and so answer the question by dividing an average male height in half. There are many other ways of solving this problem, of course, but the critical aspect is that it requires some novel problem-solving. Shallice and Evans' study used a measure of the bizarreness of response (ie. the degree to which the answer differed from that given by normals) and demonstrated that patients with anteriorly located lesions were significantly poorer at performing this test than patients with posterior lesions. It is of course patients with anteriorly located damage that are generally considered to be the most likely to confabulate (McCarthy and Warrington, 1991; Shallice, 1988), and it has been suggested that the presence of confabulation might be related to the degree of frontal lobe dysfunction (Berlyne, 1972; Kapur and Coughlan, 1980). Given that accurate recall under certain circumstances, requires some problem-solving ability, then one might expect that a patient who has a dysexecutive syndrome would be likely to be inaccurate (on occasion) in recall, and that this inaccuracy might be related to the severity of the deficit.

While there is little definitive evidence for the case that problem-solving or "supervisory" deficits necessarily play a part in confabulation, there is some evidence that this might be the case, at least for confabulators who display "spontaneous" confabulation (Kopelman, 1987). Thus Kapur and Coughlan's (1980)
patient who, at least in the early stages of his illness, showed spontaneous confabulation, also performed below the first percentile on Shallice and Evans' Cognitive Estimates test. Other cases of severe confabulation are also reported who were severely "dysexecutive" in their presentation (e.g. Baddeley and Wilson, 1986) whilst others where the content of their confabulations was qualitatively less bizarre performed better on tests considered sensitive to frontal lobe lesions (e.g. Dalla Barba, Cipolotti and Denes, 1990; Delbecq-Derouesne et al, 1990; Moscovitch, 1989). Thus on this account, the bizarreness of (or the more "fantastic") the confabulation is directly related to the degree of damage to the mediator process. Those whose confabulations are more sensible have primary dysfunction in the other memory control processes.

7.7 Conclusion and Summary

Talland's Properties of Confabulation.

The introduction listed eight properties of confabulations, of which (b) is essentially a characterisation of confabulation. All except possibly (e) follow from the model. Thus confabulations would be expected to occur in whatever types of situation the Description-Verification processes fail. It is also implicitly assumed that these would primarily involve episodic recall from which property (c) would follow. It should, however, be noted that Norman & Bobrow (1979) give an example from the attempt of a subject to retrieve the meanings of imperfectly learned commands in programming languages, which would imply that certain specific tasks might also use these processes, which are more of a semantic memory type.

Properties (c) and (d) are essentially the same as the third prediction that was held to follow from the assumption of a damaged Description process. Characteristic (h), that patients should at times act on the basis of their confabulations, is to be expected if
the Description/Verification system is evolutionarily part of problem-solving and would therefore come into play when novel situations occur.

This leaves (e) - the lack of awareness of the deficit. The domain of the model includes issues concerning the awareness of memories, but does not relate to the more abstract issue of anosognosia. As no generally accepted theory of anosognosia exists in neuropsychology to which the model can be related or with which it might be in conflict, this seems as acceptable lacuna for the model at present. Indeed if verification processes are impaired, the patient would not seem to have the raw material available on which on awareness of the deficit could be based.

More Specific Questions.

Different degrees of damage would be expected to lead to different degrees of severity in the confabulators. Might any qualitative difference be expected? Three issues will be considered - the possibility of the restriction of confabulations to episodic memory only, the occurrence of bizarre confabulations and the persistence of individual confabulations.

Dalla Barba et al's (1990) patient confabulated to autobiographical memory questions only and not to semantic memory questions, while the patients described by Moscovitch (1989), and Delbecq-Derouesne et al (1990) confabulated in both types of situation. To a certain extent the evidence is not directly comparable since the semantic memory tasks on which the latter two patients confabulated - ordering historical events in time and geographical locations in space - were not given to Della Barba et al's patients. However, as we have seen, that patient did show a different pattern from Mercer et al's severe confabulators in not confabulating on questions designed to produce an 'I don't know' response in normal subjects (eg. "What did Marilyn Monroe's father do?"). This could be explained if the description mechanism was
functioning in the sense that if a description specification is not elicited by the question - as it would not be by a semantic memory question (see assumption 9) - an inappropriate description is not produced. If, however, the description functions are severely impaired the inhibitory control of the memory store is lost and confabulatory responses can occur to virtually any stimulus.

Why might spontaneous and bizarre confabulators occur? For these to occur requires three deficits. First the inhibitory Description function just discussed has to be lost so that virtually any activity of the memory store can be produced as a memory. Second the content has to be seeded by locatory templates. Third, the ability of coping with novel situations and showing general problem-solving skills must be lost.

The third component, that spontaneous confabulation requires the loss of the second type of verification procedure involving general problem-solving abilities - controlled by the Mediator as in figure 7 is related to suggestions made by Mercer et al, 1972, and Kopelman, 1987. Thus Kapur and Coughlan's (1980) patient who, at least in the early stages of his illness, showed spontaneous confabulation, performed below the first percentile on Shallice and Evans' (1978) Cognitive Estimation task, a test of "frontal" problem solving. Other cases of severe confabulation are also reported who were severely "dysexecutive" in their presentation (eg. Baddeley and Wilson, 1986) whilst others where the content of their confabulations was qualitatively less bizarre and who did not show spontaneous confabulation performed better on tests of coping with novelty (Eg. Della Barba, Cipolotti and Denes, 1990; Delbecq-Derouesne et al, 1990; Moscovitch, 1989). Thus on this account, the bizarreness of the confabulation is directly related to the degree of damage to the mediator process. Those whose confabulations are more sensible have primary dysfunction in the other memory control processes.
Finally why might a confabulation such as that shown by Damasio et al’s case I have become fixed, as can also occur in the Capgras syndrome and reduplicative paramnesia? The obvious mechanism produced by the present model is schematisation (assumption 9). If when the confabulator retrieves memory elements from the memory store, which are a conflation of existing memories (as occurred in normal subjects - see results 2(ii)) or involve excessive Locatory Template activation, then it is presumed that their links will be strengthened. Verification processes will not delete the connections between the erroneously linked element. This reactivation of the erroneous combination can easily occur. The production of false belief could therefore occur in an analogous fashion to the way schematisation of (valid) memories occurs in normal subjects.

This chapter presents a theoretically based account of the neuropsychological literature on confabulation, and also a study of normal memory lapses and a model of normal memory triggered by an interest in confabulators. The aim was to show that some at least of the phenomena of confabulation occur also in normal memory retrieval, if at a much reduced rate (see also Kopelman, 1989), and that a memory model, which when damaged gives rise to certain properties of confabulation can be tested in normal subjects. The account given of confabulation within the context of the model incorporates many of the elements presented by other investigators of frontal amnesic phenomenon, as illustrated in the introduction to this thesis. However, hopefully by expressing them within the confines of a model of normal functioning and by using the analogy with memory lapses in normal subjects this study will stimulate the cross-talk between studies of normal subjects and neurological patients which has been so fruitful in other areas of cognitive neuropsychology.
8.1 Introduction

There have been three main approaches to the investigation of the role of the frontal lobes in memory. The first selects patients with known or putative damage to the frontal lobes and examines these patients' memory characteristics. This will be referred to as the localisation approach. The second, more data-driven method is to select first patients for study who show particular characteristics of memory functioning, and then to examine their neurological condition. This will be referred to, for convenience only, as the phenomenological approach. The third method has two forms. One uses two groups of patients, neither of which have anterior-only lesions, but compares patients with pathology generally thought to involve posterior and anterior areas, and compares them with patients with posterior-only damage (eg. Leng and Parkin, 1988, but see chapter one for a review). The other form studies a group of patient with suspected frontal damage and correlates their performance on certain memory tests with their performance on a range of neuropsychological tests considered sensitive to frontal lobe lesions (eg. Kopelman, 1991; Robbins, 1989; Leng and Parkin, 1988; Squire, 1982; Schacter, Harbluk and MacLachlin, 1984; Schacter, 1986). These two forms are parts of a methodology which will be referred to as the deductive approach.

8.2 The Localisation Approach

These methods were examined in turn in the Introduction to this thesis. The overview of the localisation approach examined the evidence from group studies of patients with putative frontal dysfunction resulting from a number of
neurological conditions (eg. Korsakoff's syndrome; space-occupying lesions; surgically-induced lesions). The broad conclusion (and one in general agreement with Kapur, 1990 and Stuss and Benson, 1986) was that there was little in the literature that is not equivocal. Indeed some of the best studies from a methodological view (eg. Warrington, 1984) failed to find any significant differences between patients with damage to the frontal lobes and damage elsewhere in the cortex.

8.3 The Phenomenological Approach

The phenomenological approach, in the main, fared little better in terms of making the link between particular patterns of memory behaviour and the frontal lobes. The characteristics most commonly considered as resulting from frontal lobe dysfunction were summarised and each of these proposed characteristics was examined in turn, with close reference to the studies key to each.

As with the localisation approach, the studies which have used a more phenomenological approach have also yielded little that is not equivocal. There were many reasons for this, but the main ones were a) weak effects; b) lack of replication; and c) poorly chosen control groups and/or analysis. However, the exception was paramnesic phenomena: the involvement of the frontal cortex as at least a part requirement of paramnesic disorders at present seems quite secure. There is as yet no reported case of a patient who exhibits florid paramnesic signs in which frontal dysfunction can be ruled out.

8.4 The Deductive Approach

Chapter one made the contention that much of the current momentum in the literature linking the frontal lobes to particular memory test patterns is a direct result of studies which have used the deductive approach.
As already mentioned, this approach has two strands. The first – the subtraction methodology – uses a group of patients with widespread cortical involvement and contrasts their performance with patients with posterior-only damage. The logic behind this approach is that those characteristics shown by the widespread-involvement patients but not the posterior-only patients will be due to the involvement of the frontal cortex in the former patients. This approach was, however, shown to be both of doubtful validity on both neuropsychological and neurological grounds: differing pathologies have their own characteristics and there are a number of reasons (unrelated to memory functions) why patients with differing neuropsychological profiles might perform memory tests in different ways.

The second strand of the deductive approach was the association methodology. This approach uses a series of patients with putative frontal dysfunction, where often no attempt is even made to prove frontal involvement. They are tested on a small series (or even only one) of tests which have been shown to be performed poorly by patients with frontal lesions. They are also tested on a series of memory tests. Associations are made between memory test and "frontal test" performances, with the conclusion being that the memory test characteristics shown by patients with poor frontal lobe test performance are due to frontal lobe dysfunction.

This approach was also shown to be seriously flawed; patients may fail both executive and memory tests only because their lesion is bigger or their general cognitive resources are weaker. Moreover, at the single-case level the information one can gain from such a methodology is limited. Thus a patient fails both memory and executive tests, this may be explained by general cognitive problems, and if s/he passes both memory and executive tests it may just be that the patients lesion has not affected his cognitive abilities in any domain. The other two possibilities – dissociations one way or the other between executive and memory tests severely test the
association hypothesis. Thus dissociations within this design of study are of limited value, and associations can be misleading. These points can be coupled with those made in chapters 4 and 5, which highlight the complex set of psychometric issues (reliability, detection rate, individual differences etc.) surrounding the use of the association approach. Moreover, and perhaps most seriously, the logical flaw in the use of the association methodology (see chapter 5) led to the rejection of this form of investigation as a potentially useful source of evidence.

A modified form was considered, however, and was used in the thesis. This is where the localisation evidence for the lesion is known. This then enables one to separate out process and localisation issues, thereby avoiding the greater pitfalls of the deductive approach.

This thesis used all three approaches (phenomenological, localisation and deductive) in the investigation of the role of the frontal lobes in human memory. The first two chapters describe the development of two new measures which are sensitive to frontal lobe lesions: by this it is meant that the processes which are tapped by these measures seem to be more affected by anterior than posterior lesions. The development of these tests was necessary for three reasons. Firstly the poor predictive validity of most existing measures and their dependence upon general levels of intellectual functioning meant that these would be poor measures of "frontality"\(^1\). Secondly many existing executive tests are not particularly "clean" measures in that they require a number of processes for successful operation, only some of which are traditionally accepted as being supported by the frontal lobes. Thirdly, examination of the notion that frontal lobe patients fail memory tests because of executive disorder rather than memory dysfunction (the

\(^{1}\) The issues surrounding the use of tests as measures of "frontality" were described in chapter 5, with the overall conclusion being that this is a method which requires a set of precarious assumptions which should be clearly articulated in any study which uses it.
dysexecutive explanation) requires that the subject should have data on both memory and executive tests.

The Frontal Lobes and Temporal Order Memory

Chapter 4 compared the performance of patients with anterior or posterior lesions (and controls) on a newly developed test of temporal order memory, which is held by some to be a function of the frontal lobes. More specifically this study tested the theories that frontal lobe patients fail temporal order tasks because of either exaggerated interference effects or because they are poor at tests which require effortful (as contrasted with automatic) encoding.

The results were essentially negative. Whilst both groups of patients (anterior and posterior) were poorer than the controls at the test overall, the anteriors were not worse than the posteriors at the task. Moreover no group showed an effortful-automatic encoding effect, and the anteriors actually showed less interference than the posteriors (although this just failed to reach significance).

It could of course be argued that the test used here was not a measure of temporal order memory (despite its similarity to other measures of temporal functions). It was obviously, however, a measure of something in that Trial 2 performance was very highly predictive of Trial 3 performance - it was not the case that the test was so unreliable that the scores were meaningless. Additionally, since the task asked for information regarding temporal order, it would be difficult to argue that the test measured some other aspect of memory. The results could of course be criticised from the point of view of aspects of methodological design - such as the groups being too small. However these criticisms, as we have seen, may at least equally be levelled at those studies which have suggested that frontal lobe patients do show poor temporal order memory.
This study went on to show no significant differences between the anteriors and posteriors on both the free recall measure or either of the forms of Warrington’s RMT. Again, the lack of demonstrable effect could not be due to the poor reliability of the measures, since factors such as age, IQ, and premorbid levels of functioning were significant predictors of performance on the measures. This emphasises the importance of meticulously matched controls and comparison lesion groups in studies of lesion site and memory test performance.

Instead it appears that despite the fact that the groups of patients in the memory test study were the same as those used in the development of the executive measures – where an anterior-posterior effect was shown – these patient groups did not differ in their ability to perform three different types of memory test (supporting the findings of Warrington’s [1984] study).

8.4(a) Background Variables and Test Performances

Chapter 5 went on to investigate the relationship between patients’ ability to perform tests of executive function and tests of memory. Broadly speaking, the findings were that memory tests and executive tests were mutually affected by certain background factors\(^2\). There was therefore some modest correlation between performance on the various measures. However when these effects were removed statistically, it became apparent that a patient’s executive test performance was not a good predictor of their memory test performance. This was particularly the case for the anteriors, where the greatest dissociations existed. These results did not support the idea that there is a major dysexecutive

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2 Whether the executive tests can be considered dissociable from IQ measures conceptually is a complex question. For the moment it is assumed that the WAIS measures a number of processes, at least some of which are not shared by the executive measures.
component to the memory test performance of the anterior group.

Further analysis of the effects of background variables such as age, current level of intellectual functioning and premorbid ability showed that these were important predictors of memory test performance. Moreover, differing forms of memory test differed in the extent to which they relied upon any one - or a combination of - these variables. This suggests that apparent discrepancies in memory test performance - say, recognition memory versus free recall, might occur as a consequence of general background pathology rather than revealing anything about memory processes per se. For instance current IQ and age as factors affect free recall tasks more than they do recognition tasks; premorbid levels of functioning (or, more probably, verbal abilities) affect verbal memory tests, but do not appear to be influential in visual tasks (eg. forced-choice faces); forced-choice words is quite highly associated with FSIQ, forced-choice faces is much less so, and so forth. Thus, for instance, we might expect an older patient with depressed current IQ but very high premorbid abilities to score poorer on free recall tests than would be predicted from a verbal forced-choice test (using prediction equations based upon a large population), with a young patient with intact current IQ but low pre-morbid verbal abilities showing the converse pattern. Quite clearly the significance of patterns of memory test performance should always be considered in the light of an understanding of the individual demands of the test and the overall neuropsychological profile of the patient.

Overall, different memory test measures no doubt differ along a general dimension of "degree of resource demand" in Shallice’s (1988) sense. However individual patients will also differ in their baseline abilities to perform those tasks, leading to complex interactions between the characteristics of the task and those of the individual patient.
Additionally, crucial of course to the interpretation of patterns of patient groups' (or individuals) memory test performances is an understanding of such factors as reliability and discriminating power of the tests. Some (eg. Kapur, 1990) acknowledge the importance of such factors, but for the most part these considerations are ignored in neuropsychological studies.

8.4(b) Factors Influencing Test Performance

Overall the findings of chapters four and five make three general points less closely related to the direct experimentation. The first is that many variables which are not memory-related affect a patient's memory test performance. One might suppose that there are a minimum number of factors which affect performance on any one test. But even consideration of a hypothetical minimum number demonstrates how complex the task rapidly becomes. For instance we might suppose that the determination of a score on any memory test might be represented by the equation:-

\[
\text{Score} = \text{Constant} + aP + bI + cT + dT + eA + fF - (gP - I)
\]

where \( P \) = premorbid ability; \( I \) = current IQ; \( P-I \) = deterioration index; \( A \) = age; \( T \) = task-specific factors; \( R \) = reliability of test; and \( F \) = factor under study [Note however that the sign of each factor would depend upon the type of measure (eg. number of errors, or number correct) under consideration, and so may not always be as shown here]. However many of these factors will interact, and factors such as education, daily fluctuations in performance, patients' past experience of testing, anxiety, as well as test reliability and so forth are all ignored.

Since the weights for each factor will be different according to the particular test under consideration, one must be very careful about drawing conclusions regarding
the significance of discrepancies in memory test performances. And yet it is patterns of this sort that for a large part make up our present understanding of the effects of frontal lobe lesions upon memory functioning, as reviewed in chapter one of this thesis: preserved recall versus impaired recognition or vice-versa; poorer cued recall than should be predicted from recognition memory tests; poorer "spatio-temporal context" knowledge than should be predicted by recall or recognition of the items and so forth. It is perhaps hardly surprising that there has yet to be a convincing replication of a study which centres upon the role of the frontal lobes in human memory. Overall, the results from the empirical studies presented in this thesis support the arguments made by Stuss and Benson (1986) and Mayes (1988) regarding the fragility of frontal lobe memory effects.

8.5 Discrepancies in Memory Test Performances

The second conclusion of chapters 4 and 5 is that correlations in performance on memory tests are generally quite low (see also Wilson, Cockburn and Baddeley, 1988). Where the correlations were significantly different from zero, this was shown in many cases to be largely a consequence of their mutual dependency upon the background factors. While there have been, as we have seen, many studies looking at discrepancies in performances in patients with putative frontal damage, there have been few if any which study such discrepancies in patients with known posterior-only damage. The data presented in chapter 5 suggests that discrepancies in memory test (but not executive test) performance may occur in any patient and are not characteristic of patients with exclusively frontal involvement.

8.6 Dissociations between Executive Processes

The third conclusion of chapters 4 and 5 was that correlations between "frontal measures" can be very low.
In fact certain frontal patients were found who demonstrated, singly, problems with either response suppression, initiation, or rule detection. It would appear that the processes underlying such functions may doubly dissociate. Even if it were not the case that large memory test performance discrepancies could occur, this would still be enough to cast serious doubt upon the use of the deductive approach. For it means that it makes little sense to use tasks shown to be sensitive to frontal lobe lesions as a measure of "frontality". Frontal functions do not break down uniformly, and the case where a patient is uniformly impaired on traditional frontal measures in the context of preserved intellectual functions can be considered highly unusual. It is more usually the case to see relatively selective breakdown of the processes subsumed by the frontal lobes, at least in cases with well localised pathology.

8.7 The Frontal Lobes and Prospective Memory

Chapter six took a less experimental approach. For many years it has been known that certain frontal patients show a deficit in what might best be described as "remembering to remember". This is currently a poorly understood area of psychology. Whilst there has been some traditional experimental psychology work in the area, it has as yet received scant attention from neuropsychology. Currently there is enough work in both fields to suggest that prospective memory functions dissociate from retrospective ones (eg. Kvavilashvilli, 1989; Wilson, Cockburn and Baddeley, 1988) but little more.

This pattern was broadly shown by the patients reported here. Retrospective memory functions were generally good in two of the patients (as assessed by a range of tests) and excellent in the third. And yet all three showed severe deficits in planning and organisation in everyday life. It was hypothesised that one of the steps in carrying out an intention is the formation of "temporal markers". These markers are signals to treat an activity
as novel, and carry no actual information in themselves. Markers can be "attached" to cues such as activities or times and are triggered when the person becomes aware of that activity or time. The recall of the actual operation required at the point of the marker triggering falls within the domain of traditional retrospective memory. This demonstrated itself operationally when the patients contravened task rules or requirements which they were later able to articulate. It was not that they could not remember what they were supposed to do (or not do), but that at the point where that knowledge should have been "activated", it was not.

These patients did not show a general impairment in executive skills. On the contrary, their performance on a wide range of tests traditionally considered sensitive to frontal lobe dysfunction was quite satisfactory; excellent in one case. It would appear that prospective memory skills may dissociate from traditional executive ones in the same way that chapter 5 demonstrated dissociations between the executive processes of response suppression, initiation and set detection and formation. What is less clear is whether prospective memory skills will dissociate from planning. It is reasonable to suppose that marker creation may be a different process from marker triggering. However if marker creation is a product of planning (or at least the creation of an intention at some level) the dissociation may necessarily only go one way. This awaits further investigation, as does the question of whether the strategy application disorder syndrome may occur in patients whose dysfunction lies outside the frontal lobes. The three patients reported in chapter 6 were selected because they showed the syndrome, not because of the localisation of their lesions. They all happened to have damage, to varying degrees, to the frontal lobes. This does not of course mean that all cases will have such damage. However Goldstein et al (in submission) report a case with the same symptoms as those reported here, and who was tested on the same measures as those reported here, with broadly the same results, at least on the ME task. This patient
had an isolated, surgically induced left frontal lesion, with no damage extending beyond that lobe. It seems increasingly likely, therefore, that a frontal lobe lesion may be sufficient condition in itself for the development of the strategy application disorder, and the consequent prospective memory deficits.

8.8 The Frontal Lobes and Confabulation

Chapter 7 took a similarly phenomenon-centred approach to the previous chapter, but returned to that characteristic that was shown in chapter one to be most securely linked to frontal lobe dysfunction - confabulation. This study attempted to demonstrate the processes that are at work when a normal subject attempts to recall episodes that have happened to them. Since confabulation is principally a disorder of autobiographical recall, it was proposed that those processes which are damaged in confabulators should be able to be demonstrated at work within normal subjects’ recall. Studying confabulations themselves, whilst quite obviously valuable, would have its limitations in that they would show only the consequences of the breakdown of processes - it would still be speculation as to what those processes were before they were damaged.

This study demonstrated, amongst other things, that a number of control processes are required for successful autobiographical recall, especially for episodes which are unrehearsed (and therefore bear the least resemblance to the personal semantic recall referred to as autobiographical recall by, for instance, Kopelman, Wilson and Baddeley (1990)). In this way, the study concurred with Baddeley (1990) who makes the distinction between autobiographical recall and recollection, making the point that recollection is a effortful process. It is argued that it is the operation of the secondary control processes which make the task "effortful".
The study demonstrated that normals routinely make errors in recall. Were these errors to remain uncorrected, they would bear close resemblance to confabulations of the momentary type. Yet in the main they were corrected by post-recall verification process. This process required, on occasion, frank problem-solving. It was hypothesised that patients who have suffered problem-solving deficits of the type seen, for instance in failures on Shallice and Evans's Cognitive Estimates test might be unable to operationalise this problem-solving, and the result would be confabulations of the fantastic type. Thus the degree of executive disorder will affect the degree to which the confabulations are fantastic.

However an executive disorder per se is not a sufficient condition alone for confabulation. The normals in the present study were shown to spend considerable amounts of time in addressing the correct memory: answers such as "When did you last clean your car?" could often not be answered quickly. Often many candidate memories were retrieved, and there followed a recursive operation of specification and re-specification which served the purpose of honing the description of the to-be-retrieved memory thereby refining its characteristics. In this respect the account given here bears some resemblance to the associative recall elements of Moscovitch’s (1989) strategic recall hypothesis outlined in the thesis Introduction. It was proposed that a disorder in this description process is central to confabulation, although there needs to be concomitant modulatory system disorder.

8.9 General Conclusion

One particular criticism remains to be answered, which was first raised in chapter one. This is the question of whether the experimental memory data presented in this thesis is not at odds with the hypothesis about the role of the frontal lobes developed later (particularly in chapter 7). More specifically, one of the arguments followed in chapters 1-5 was that the evidence from
traditional experimental psychology paradigms regarding the role of the frontal lobes in human memory is largely equivocal. However the main thesis presented in chapters 6 and 7 (especially chapter 7) was that the frontal lobes are crucially involved in some aspects of memory. On the face of it these two contentions would seem to be at odds with each other. If it were the case that the frontal lobes play such a vital part in memory, why are frontal lobe effects so difficult to elicit using experimental paradigms?

From the point of view of prospective memory (chapter 6), the answer is relatively straightforward; the thesis that prospective memory functions may be critically supported in some way by the frontal lobes has simply not been tested. Chapter 7, however, discusses notions which require further explanation from this point of view.

If one accepts that the frontal lobes play a key element in the modulatory memory elements (description, verification and mediation), what would be the consequence of damage to such systems as regards traditional laboratory-based experimental paradigms? Presumably those which require the operation of such functions would be impaired, and those that do not, would not. Few experimental paradigms do test these functions, however. Moreover many of these methods would not elicit the type of information that would enable one to suppose that there are such structures.

A typical forced-choice recognition paradigm, for instance, yields only all-or-nothing information about a patient's recognition of an item. In the case where a patient "recognises" an item which was not in the stimuli set, we can only know that the patient did not, for some reason feel a greater sense of recognition for the target than the chosen foil. The experimental design does not give any further information. Thus it may be the case that where a patient "misrecognises" foils this is due to faulty descriptive processes, or of course it may not. The data simply does not speak sufficiently to the
question. Similarly, a free recall task generally only provides all-or-nothing information. An exception here is where the patient produces intrusions. And of course some have attributed high intrusion rates to frontal lobe dysfunction (eg. Kapur, 1985). A similar point may be made about the data from the various types of interference paradigms (see Stuss, 1991).

It may well be the case that failures on these experimental memory paradigms result from the failure of the same processes that were underlying autobiographical recall in the normals in chapter 7. But whereas the procedure used in chapter 7 provided a wealth of information about the reasons behind erroneous recall, most tightly constrained experimental paradigms do not. Moreover, the requirements of the task seem quite different. More specifically, the operation of (for instance) choosing which of two recently presented words seems the most familiar to you may be quite different from the operation of recalling a rich sequence of events that happened weeks ago. One requires the all-or-nothing choice between two discrete stimuli, the other a construction of a complex recall chain where elements vital to further recall have to be sought and separated from other candidates and meshed together in a sensible sequence.

This leads to perhaps the main point regarding the differences between the requirements of traditional experimental and naturalistic autobiographical recall. Typically in free recall or forced-choice tasks, a failure of recall (or misrecall) in response to one stimuli has little consequence as regards recall in response to the next. This is hardly the case for autobiographical recall, where as we have seen in chapter 7, the recall of details out of sequence or not properly belonging to the required recall structure, may influence further recall enormously, and lead the subject "up the garden path". An exception, as regards experimental paradigms, is perhaps the recall of stories as used in (for instance) the Wechsler, Coughlan and Hollows AMIPB
and RBMT batteries. It is perhaps unsurprising that authors have used these tests most widely in trying to elicit confabulatory behaviour in normals (eg. Kopelman, 1990).

The resolution of the experimental findings of this thesis with the hypothesis being presented in chapter 7 is perhaps, therefore, that the operations required by many experimental paradigms are different from those required by naturalistic autobiographical recollection (although clearly some may be the same). In short, whilst amnesia is an inability to recall events that have happened to you, most neuropsychological tests do not require you to recall autobiographical events.

In summary, the operations required by typical experimental paradigms may be qualitatively different in many respects from those required in recalling a string of events that have happened to you. The experience of recalling the events surrounding your last visit to Trafalgar Square may be quite different from that of being presented with two words and having to choose which one had seen before. This is the point made so eloquently by Tulving (1989), who argues that neuropsychology has largely ignored these differences, and perhaps the answer lies as much with his argument as it does with complex discussions about the probably interactive nature of poorly understood processes occurring at sites remote from each other in the brain. Quite clearly many of the these notions, and the hypotheses about the localisation of structures subserving the processes demonstrated in chapter 7, could be tested by using procedures similar to those developed here with neurological patients, and this remains an interesting possibility for future research.

3 Baddeley (1990) makes a distinction between automatic components of retrieval, and an "active process of retrieval" termed recollection (p. 293). It is assumed here that the recall of autobiographical memories which have not the status of "generic memories" or "personal semantic memories" (see chapter 7) corresponds largely to this process of recollection.


Additional Reference


Kleist, K. (1934) Gehirnpathologie. Leipzig: Barth


Appendix IA: Basic Template for the Brixton Task (Stimulus Position 10).
### Spatial Anticipation Task

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Appendix 1B: The Brixton Test Scoresheet - Complete Sequence

Positions Used in the Test.
The sheets you have been given have two sets of 15 sentences on them. We are only interested in scoring the SECOND SET of sentences.

Each subject had each sentence read out to them in turn, and the response the subject made is written at the end of that sentence. We would like you to rate each of these words according to the "decision tree" below.

Examples:

C rating: (a straightforward Completion of the sentence)

The captain wanted to stay with the sinking BOAT or PLANE
He bought them in the sweet SHOP or STORE

O rating: (an Opposite of what might be expected)

They went as far as they COULDN'T
The whole town came to hear the major REMAIN SILENT

SA rating: (a word obviously semantically connected to the subject of the sentence)

Most sharks attack very close to FISHES or SEA or BITE
She called the husband at his PHONE or WIFE or DIAL

SB rating: (a word showing an obvious semantic connection to the word(s) that you would expect to appear at the end of the sentence.

None of the books made any UNDERSTANDING
Most cats see very well at DAWN or DUSK or WINTER

SC rating: (a word which makes vague sense at the end of the sentence but which makes the sentence ludicrous. Also swear-words, obscenities or other inappropriate words).

The whole town came to hear the mayor FART
The dog chased our cat up the TOWER OF PISA

UR rating: (a word that is completely Unconnected to the sentence and which might reasonably be expected to be found in a normal office Room-includes office fixtures and fittings and clothes that someone in an office might wear)

The dough was put in the hot COATHANGER/PAPERCLIP
Jean was glad the affair was DESK/CHAIR/WINDOW/DOOR
Most cats see very well at TALK (where previous answer was CONVERSATION)
The whole town came to hear the mayor sandcastle (where previous response was SEA)

URL rating:
- this is where both UL and UR are true.

U rating: (a word that is Unconnected to the sentence and which isn’t connected to the subject’s response to the previous sentence and you would not generally expect to find in an office)
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| River Foot Animal School | Work Money          |
| Sun Plane Money Food   | Girl Girl              |
| Fish Boy Letter Road   | Food Snow              |

| Ball Sun Road Hand     | School Hand            |
| Boat Home Rain Work    | Rain Food              |
| Foot River Hand Grass  | Animal School           |
| Plane Fish Snow Girl   | Letter Grass            |

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Appendix 3: Word Order Test Scoresheet
APPENDIX 4
Chapter 7: Autobiographical Memory Questions

1. Describe the first thing that come to mind that has happened to you in the month before last.

2. When did you last clean your car?

3. When was the last time you went to the coast?

4. Describe your main course for dinner last night.

5. Describe the last time you had dealings with the police.

6. What was the weather like yesterday morning?

7. When was the last time you saw one of your relatives you don’t live with?

8. Describe what you were doing during the two hours before lunch last Sunday.

9. Describe what you did the last evening you went out to a party, the theatre, cinema restaurant or similar.

10. When was the last time you were in Trafalgar Square?

11. Describe an object you have recently given or received.

12. Describe the last meeting, seminar or case conference you went to.

13. Describe the state of your house/flat/room when you moved into it.

14. Describe the last time you were introduced to someone you had not previously met.
APPENDIX 5

Chapter 7: ELEMENT ANALYSIS OF AUTOBIOGRAPHICAL MEMORY PROTOCOLS

The Elements used in Analysis (in alphabetical order only):

1. Answers (ANS)
2. Comments (COM)
3. Conclusions (CON)
4. Conscious Memory Search (CMS)
5. Corrections (COR)
6. Correction Possibility (COP)
7. Demand Setters (DS)
8. Explanations (EX)
9. Failed Recall (FR)
10. Familiarity Impression (FIM)
11. Furtherances (FU)
12. Hypotheses (HYP)
13. Incomplete
14. Memories (MEM), incorrect memories = 14E
15. Metamemory Insights (INS)
16. Multiple Memories
17. Problem Solving (PROB) (3 types) -
   A: uses semantic knowledge
   B: uses autobiographical knowledge
   C: Contextual Personal Semantics
18. Recall specifications (SPEC)
19. Repetitions (REP)
20. Statements (STAT)
21. Success Points (SUP)
22. Task Demand Analysis (TDA)
23. Unlinked Image (UNI)
24. Verification Analysis (VER)
25. (as) Yet Unavailable

These are defined as the following:

1. **ANSWERS**

   Statements which contain no suggestion of any additional memory content, are not part of a reasoning process and directly answer the set (stimulus) question are known as answers. Answers can be positive when based on a memory. Answers are different from conclusions because they don't appear to have followed from any reasoning process - it appears that the S has the answer to the question readily to hand without having to have thought about it. They are distinct from FAILED RECALL because a FAILED RECALL does not answer a question, but merely informs the listener that the sought-after memory is not available and the search is being discontinued. An
example of an FR (for contrast’s sake) would be "I can’t remember".

Example: When responding to the question ‘What did you have for dinner last night?’ "Fried squid and mustard". Another example might be where the S was asked when s/he had last seen one of his relatives and merely responded "Last Tuesday". An answer in the negative can also be an answer - like responding to above question with "I didn’t have dinner last night".

2. COMMENTS (COM)

Rhetorical remarks, comments or statements, aimed at the listener, that do not convey a memory, are not part of problem solving or anything specifically to do with the recall process, but which summarise a state for the subject (or are a throwaway comment not intended to be germane to the task, but conveying some comment upon something recalled), for instance "...which was fascinating.." or "That was that", etc. Parts of discourse that could be removed without altering the significance or message from the point of view of understanding the recall processes. Speaking "to camera" is generally also a comment.

Examples:
"That’s about it.."
"So that’s your lot, I’m afraid".
"So there’s your answer.."
"..if you can call it that"
"..not that I’d have wanted to anyway..."
"all pretty meaningless to me really"
"..what an idiot..."
"that’s something I’d rather forget.."

3. CONCLUSIONS (CON)

Summaries of what has gone before which usually come towards the end of a recall structure. They add no new memory elements and their purpose seems to be to sum-up for the listener all the different strands of memories and reasoning so as to make a sensible ‘answer’. However they are not ANSWERS because they are the conclusion of various wranglings whereas ANSWERS are where the S just answers the set question without (apparently) having to sort anything out in her own mind. They are not COMMENTS because they are an attempt to make sensible a recall structure whereas COMMENTS are just throwaway remarks which serve no elucidatory function.

Example: "So, yes, as I’ve already said the visit to Towcester must have come before the last ditch attempt in
Maidenhead and that is where I was last introduced to someone...and that’s the answer (CONCLUSION).

4. CONSCIOUS MEMORY SEARCH (CMS)

Where the subject has identified a topic for recall and claims to be trying to recall detail/s pertinent to it, but not in a problem-solving way eg. "...thinking about the dummy again, in Brixton, trying to think when that was...". This does not include the situation where a S merely identifies a topic s/he is thinking about (eg. "thinking about the shop window..." [a MEMORY or MEM]). It is distinct from a PROBLEM-SOLVING (PROB) routine because there is no evidence of a reasoning process occurring. It is distinct from a RECALL SPECIFICATION (SPEC) process because aspects of the event/episode/situation have already been recalled whereas a RECALL SPECIFICATION is where the S is homing in on the first concrete aspects of the event. CONSCIOUS MEMORY SEARCH (CMS) is searching a memory whilst RECALL SPECIFICATION is getting there in the first place. Only CMS if can be demonstrated that the S is thinking about one thing only - no competing possibilities (a period of time is unlikely to be a CMS because many things may have happened during that time).

Examples: "There was something I was reading this morning (MEM)...now what on earth was it?...(CMS)...oh yes! (SUCCESS POINT), it was about that peculiar professor...".

"Trying to remember the last time I went up there......"

"Thinking about my last meetings with the professor, trying to identify the time when I was told about the operation (SPEC)...Ah yes, I’ve got it (SUCCESS POINT)...it was the meeting last Thursday (MEM)...now who was there? (CMS)...".

5. CORRECTIONS (COR)

Obviously usually follow at some point from CORRECTION POSSIBILITIES, they are where the S has realised that some element of what s/he is saying is not correct. CORs start with a statement announcing the fact that the S has realised what it is that is inaccurate (not the realisation that something is not right but not knowing what - that is a COP) and end when the S starts to produce memories again (ie. once the correction has been made). Perhaps an obvious string would be where a COP is followed by a problem solving routine (PROB) resulting in a SUP followed by a COR.

Examples: "..removed odd scraps from the floor (MEM), took out...-wait a minute (COP), it wasn’t before I took
6. CORRECTION POSSIBILITY (COP)

Comments which indicate that the S has just become aware that what they've just may not be correct. This can result either from either the S realising that 2 memories, or details within a particular memory, are incompatible or may result from a reasoning routine (PROB). Thus COPs can result from conflicting episodic or semantic material. Also refer to recalled material that the subject is saying may well not be correct eg. "Nick Sales...or was it Nick Sainsbury? - something like that (COP)

Examples:

"Hang on a minute, that's not right, lets see.."
"No, that can't be right...."
"Actually, I don't know that I'm right there..."
"Maybe that wasn't the occasion after all.."
"Oh, there's an element of doubt in my mind now..."
"No, that is certainly wrong.."

7. DEMAND SETTERS (DS)

Comments which refer (directly or indirectly) to the ease with which the person expects to be able to recall the required material. They contain no memories in themselves and are usually self-contained.

Examples:

"Ah! Straightforward!"
"..really difficult to remember now..."
"Oh Christ, this is going to be difficult..."
"That's easy......."
"This is difficult..."
"I immediately thought I hadn't had a meeting with my bank manager for ages"

NB: occur before recall has started - a "Well, I found that very difficult to remember" would be a statement (see STATEMENTS)

8. EXPLANATIONS (EXs)

Explanations of hypotheses or other protocol elements that have already been formed. They are usually self-contained. Explanations can appear in relation to many other protocol elements - for instance PROBS. They clearly follow an element in time: "I know that because..." (if not a metamemory insight). And do not convey episodic memories. Background information about states or
situations used to situate/explain why the event in mind occurred are also Explanations.

Explanations of a memory can exist as well - For instance "I remember asking for a red one (MEM) because red is my favourite colour, you see (EX)

Examples:

"There were two papers that needed doing (MEM) and I’m trying to remember which one of them was the more important (CMS)… Presumably the one closest to the submission deadline would be the most crucial (PROB) so it must have been the African Toads project, I would have thought (HYPOTHESES or HYPO)… That’s how I’d reason it, anyway (EX).

"thinking about talking to my niece about it afterwards (MEM) - because she was there the same night (EX)".

Note:

"I doubt that I would have found it interesting (HYPO) since I find these meetings very boring (PROB)".

BUT: "I was feeling very tired (MEM), and felt even more tired by the end (MEM) - I find these meetings very boring you see (EX)".

Comments on explanations which extend the understanding but are not actually new explanations in themselves are called furtherances (FU)

9. FAILED RECALL (FR)

The point where a S notifies the listener that the attempted recall has been unsuccessful and will not be continued any further - where the S has come to the conclusion that s/he cannot remember some sought-after detail. It is a conclusion and needs to be distinguished from an on-going CMS: so in the example: "Can’t remember what we were… Ah! we were talking about my leg…" the ‘can’t remember what we were’ is actually a CMS because it is followed by a recall. If the phrase had been only "can’t remember what were talking about" followed by a shift to another topic then this would have been a FR.

Examples: "I can’t remember the number (FR), but I can remember it was light in colour (MEM)".

"I’ll have to give up on that one… no, I can’t remember what it was, let’s go on"

"I forget. So anyway....".
10. **FAMILIARITY IMPRESSION**

A feeling of familiarity without specified detailed and precise content. Be careful not to confuse this with a HYPO. It is a rough idea of a time, area or detail that has no attached reasoning (so can't be a HYPO) based on a trace that is so weak that no details can be accurately recalled. Just an vague impression of something.

Examples:

"somewhere round here..."
"some time ago, now..."
"...oh, years ago now."
"pretty recent I think.."
"something vaguely to do with work, I think..." (without any attached reason why it should be to do with work).
"must have been a month ago"

11. **FURTHERANCES**

These are extensions to explanations (see above) which extend one's understanding of what it is that is being explained, but which are not new explanations in themselves.

Example: "because I always go there because they serve enormous burgers (an EX)....other places' burgers just don't fill me up, you see (FU).

12. **HYPOTHESES (HYPOs)**

Usually formed from PROBS, they are hypotheses about what the answer might be which are fed into the memory system to facilitate recall.

Example: S is asked when he last cleaned his car and responds:
"...Must have been in November I would have thought....in Towcester... [a PROB]...this is difficult...yes, probably early November [HYPO]...now lets see, what happened in November? (CMS)...".

13. **INCOMPLETE (INCOM)**

Where an element of a protocol is so incomplete that it is impossible to decide what it is/was/might/was intended to be. Also includes isolated and non-directed expletives and uninterpretable utterances.

Examples:

"...because..." followed immediately by a new line of thought.
"but turned out to be (INCOM) - I was still half an hour late" (MEM).

14. MEMORIES. Must refer to a specific event in which the subject was personally involved an apparently to an evoked image or other autonoetic memory experience (Tulving, 1989). They refer only to autobiographical material. Memories that are factually 'incorrect' (this being discovered either later in the protocol or in the commentary) are designated with an "E", whether they are later corrected or not.

"I'm thinking about the tree [unqualified]" is a MEM, not a CMS or SPEC.

What are memories?

NOT memories:
1. Knowledge of facts.
2. Knowledge of events in which the S was not personally involved.
3. Knowledge of one's own personal characteristics, behaviours or abilities (contextual personal semantics) expressed independently from the events in which they might have been learned.
4. Knowledge of any information about other people outside the activities that the S has personally witnessed.
5. Any other information that is event-independent.

Memories are:
1. Autonoetic experience of any form as expressed by the S that are representations of particular episodes in which that S was personally involved.
2. [In most part included in definition above] Recollections of states of mind, reasoning processes, and other mental activity which existed at a particular time, linked with a personal episode to which the S currently has access.
3. Generic memories: These are images evoked by an S that are specific to that S but which are event-independent. They are images derived from repeated exposure to a concrete stimulus - the repeated exposure has ensured that the S cannot tell from which episode that image derives. Hypothetically we assume that it is a culmination of different images of the same thing taken from all the episodes and represents a memory 'half-way' between the transition from autobiographical memory to semantic memory. Imagining a situation is also classified as a generic memory.

15. METAMEMORY INSIGHTS (INS)

When S tells you why it was that s/he thought of what s/he did. They are not comments on how difficult/easy
recall is/will prove (these are DEMAND SETTERS) but tell, post-recall, which memory led to which and why.

Example: "...and I remember thinking I about the trees outside and that sparked the memory of the state of the garden...".

"..one could elaborate...[then another element].." [ie. noting that there would be more to recall if one had the time].

"now that association has triggered off.."

16. MULTIPLE MEMORIES (MM)

Where the subject gives notification of the recall of more than one memory in response to one stimulus. These memories are recalled either simultaneously or in such quick succession that recall appears simultaneous to the subject.

Examples:

"Well, initially thought of two things..."
"Ah, well, two things come through very strongly.."

17. PROBLEM SOLVING (PROB)

There are three types of problem solving (which occurs because memories are unavailable). There must be evidence of reasoning going on, otherwise it might be a CMS - where a subject is scouring a memory for some associated detail - that is not problem solving. For instance ".trying to think what time I left." is a CMS, but "Let me see, what time did I leave? - Well, it was 12am when I saw the patient and the interview took 2 hrs (PROB) so it must have been about 2pm (HYPO)". PROB can include many different types of elements, but does not include hypotheses. It can be in brief or extended form:

A. Problem solving based on non-autobiographical semantic knowledge (but includes common semantic event knowledge).

B. Problem solving based on autobiographical knowledge (or personal semantics). They are the elements of one’s knowledge about oneself which can be independent of any specific event (ie. I went to Manchester Grammar School) or specific to an event (ie. I was born on...) but do not rely on knowledge of the details of events themselves. They are the elements of one’s knowledge about oneself that do not change over time.

C. Problem solving based on contextual personal semantic knowledge (CSK). Contextual semantic
information is knowledge of what one personally does in a given situation, one’s preferences, prejudices and the like i.e it is information that one has about oneself, independent of events themselves, but necessarily bound up with one’s life situation at the/that time. One’s current age would therefore fall into this category.

Example: "..Well it was 8.30 when I left because I can remember looking at the clock as I went out of the door (MEM) and I remember arriving at the station with only 2 mins to spare before the train left (MEM). So if I left home at 8.30 and the train left at 11.30, the journey to the station must have taken me 3 hours (PROB) ".

"..I know that the standard price for that kind of text is £40 and that discount is usually 25% (PROB) so it must have cost me about £30 (HYPO) ...Yes I remember now (SUP) making out the check (MEM) ..".

"..I can’t quite remember what kind of restaurant it was..er..um..(CMS) ..well it wouldn’t have been an Indonesian because I hate peanut butter and it’s unlikely to have been the chicken place because my company was vegetarian (PROB) so it must have been either the Pizza place or the Indian (HYPO)......yes! (SUP) it was in fact the Indian (MEM) ..".

18. RECALL SPECIFICATIONS (SPEC)

Needs to be carefully distinguished from a CMS. A SPEC, as opposed to a CMS, is where the S is giving notice of the fact that they are trying to recall something. It should indicate that the S, already having an idea in mind about what he wants to recall, is trying to home in on the particular example s/he requires using ever more precise descriptions of what is required. It is not the same as a CMS because the S has not yet arrived at the memory which he then wants to go on and explore - he is in the process of getting there. A SPEC has to show evidence of actively trying to get a handle on one particular exemplar of a number of possible candidates. Just "I’m thinking about..." is not sufficient. Conversely, the conscious attempt to deactivate a memory is also a SPEC eg. "I keep thinking of that incident...trying to think of another time I’ve had dealings with the police..".

Examples: "thinking now along the lines of work (MEM) ...trying to trace back to that week.. (SPEC)"

"Thinking now about meetings I have been to recently (SPEC)......yes (SUP), I can remember the one now where that was discussed (MEM) - now who was there? (CMS)"
19. **REPETITIONS (REPs)**

Repetition can be either of a memory or a non-memory. If the repetition includes all of the old elements but also introduces some new, then these new elements are categorised as to what they should be independent of the repetition - however, obviously everything around them will be repetitions.

20. **STATEMENTS (STAT)**

General statements about a remembering process or a particular memory element which provide information (after retrieval has occurred) about how well the process has gone or the element has been remembered such as "so that's quite memorable" or "so that's about all I can remember really". They do not add any new memory elements or other content information. The fact that they convey information about states/patterns of retrieval differentiates them from RETs.

Examples: "Yes (SUP). I can now remember (STAT)...."

**Note:**

Distinction between statements and explanations: statements refer to memory retrieval, explanations exist to explain something.

"Oh that's easy [before recall] = Demand Setter.

"No, I can't remember" or "I don't know" = Failed Recall

21. **SUCCESS POINT (SUP)**

Clear expression of the sudden accessing of either a sought-after memory or memory element or an associated but not sought-after memory strand. To be distinguished from a COP as no suggestion of any need to correct error. These criteria distinguish between SUPs and COPs because a SUP happens before any manipulation is performed on a memory that comes to mind. A COP that results from the realisation that two memories are not compatible is obviously not the same as the point at which the incompatible memory itself is recalled. However the S may not express both stages of the process and at these times the COP has priority.

For instance: "Um, now when was it....Ah yes! (SUP) I remember now (STAT) It was at Hereford at the car wash (MEM).....Oh, (SUP) there's another car wash at Windsor..."
(MEM)... hang on, was it that one then? (COP)...Yes it was (COR)..."

as opposed to:

"Um, now when was it?...Ah yes! (SUP) I remember now (STAT). It was at Hereford at the car wash (MEM)....er, I’m not sure about that actually (COP), there’s another car-wash at Windsor (MEM)....In fact it was that one, not Hereford (COR)...".

Examples:
"Ah, got it!"
"Oh I know, yes that’s it..."
"Yes, I’ve got it now..."
"Oh, there’s something else..."
"Ah!...
"Wait a minute..."

22. TASK DEMAND ANALYSIS (TDA)

Where the stimulus question is being analysed in order to try to think what is required. Pre-recall.

Examples: "The month before last....so you don’t want last month then, do you..."

23. UNLINKED IMAGE

Where the S recalls some image which does not appear to be related to the subject of the recall and notifies the E that they do not consider it relevant to the main purpose of their efforts.

Examples: ".I just got an image of my house at home...don’t know where that came from" [followed by unconnected recall]. ".for a moment I thought of Croyde Bay, but I can’t think why..."

24. VERIFICATION ANALYSIS (VER)

A) Where a retrieved memory is being compared with the criteria established by analysis of the task. eg. "...that was my initial thought - thinking well, could you call him ‘a relative I don’t live with?’.

B) Where potential incompatibility of 2 memories is assessed (see also COPs; CORs and PROBs).

Examples: ".oh, hang on, I don’t know whether that would constitute a ‘main meal’...lets see...". 
This has two forms:
A) Where the S gives notice that, for the moment at least, they cannot recall a particular detail, although they do believe that they will be able to recall it soon.
B) Where the S, after having 'checked' for a particular detail, has come to the conclusion that they do not know (and have never known) the sought-after detail.

Distinguished from Failed Recall in the case of A) by the fact that the S has given notice of an intent to recall rather than a giving notice of the end of an attempt to recall, and B) in that an inability to recall something that one has never known cannot be considered a failure in the recall process.

Examples: "..Can't think of it for the moment.."
"I don't think I could ever have told you that..
"I've never been able to remember that...."