Memory Deficits

In Parkinson's Disease

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Abstract

Twenty-two Parkinson's Disease (PD) patients and 22 age-matched and gender-matched comparison participants (aged 48-83 years) were tested on the California Verbal Learning Test (CVLT), a shopping list memory task with items divided into four semantic categories.. Results supported other research in showing verbal memory deficits in PD. The PD group performance was lower on all recall trials of the CVLT. In addition, ability to discriminate between old and new items was impaired in the PD group. Participants who scored highly on total recall measures also showed a strong ability to use semantic categories in recall. A hierarchical cluster analysis (Ward's method) was used to explore the nature of the memory deficits found. Results support the existence of distinct stages of memory decline in PD, with the differences between subgroups identified showing significance when subjected to an analysis of variance (ANOVA). These results suggest that the memory deterioration which occurs in PD is initially associated with aspects of retrieval. However, as the disease progresses, encoding processes become compromised with more severe effects on memory. An interpretation, based on neural network models of memory, is discussed to suggest reasons why memory processes in PD may fail. These include activation failure, inefficiency of gating mechanisms in encoding and retrieval operations and inability to access semantic memory at the encoding stage of a memory task.

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Introduction

Overview

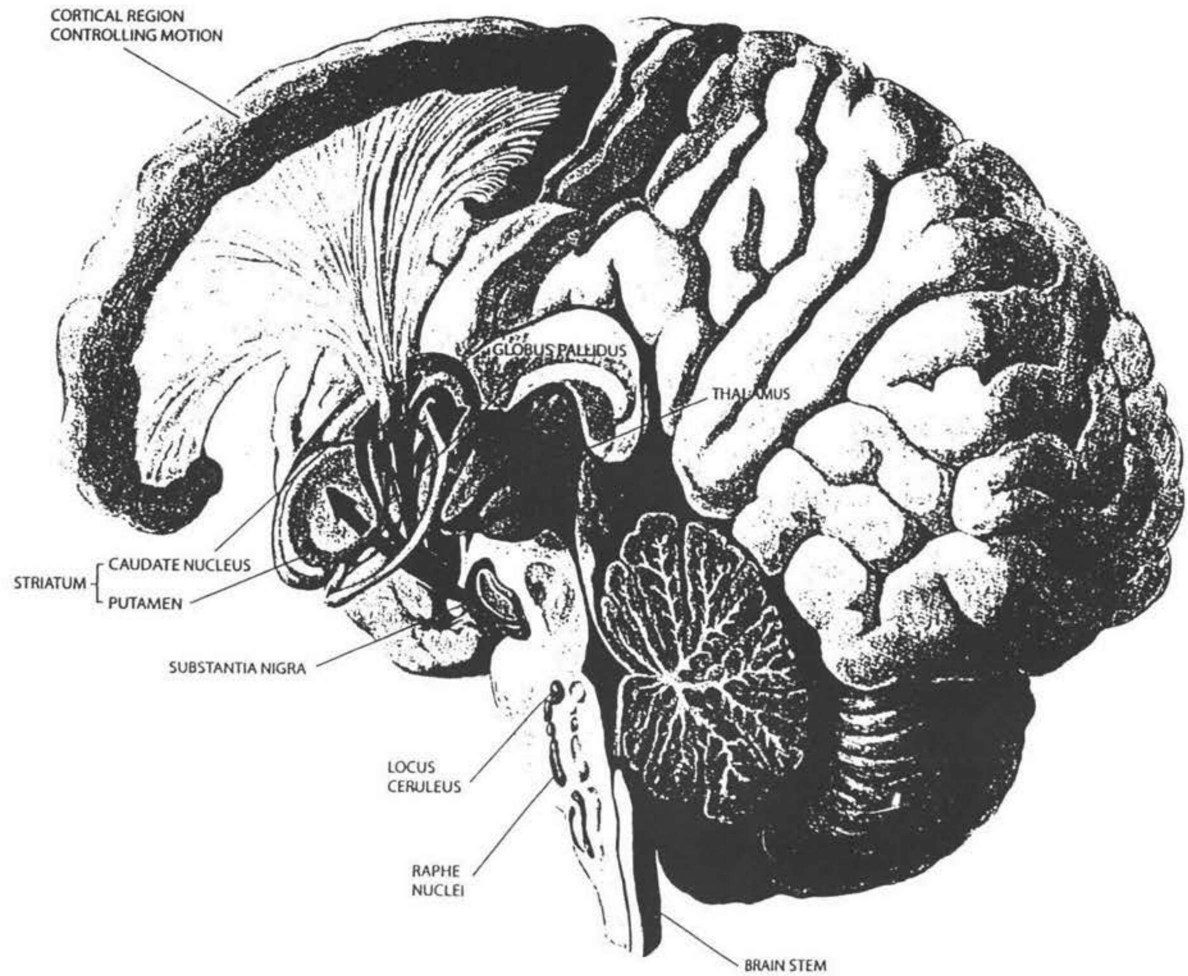
In 1817, James Parkinson outlined his observations of a distinctive nervous disorder which now bears his name. In his original work, "An Essay on the Shaking Palsy", Parkinson stated that the intellectual capacity of patients with what we now know as Parkinson's disease was "uninjured" (Parkinson, 1817).

Parkinson's Disease (PD) is characterised by motor disturbances which include tremor, rigidity, akinesia and changes in posture. These symptoms may occur in different parts of the body and in different combinations. The distinctive tremor associated with PD is a resting tremor which is absent during voluntary movements or sleep. Commonly, the hands are affected, but tremor may also be present in other limbs, jaw and tongue (Lezak, 1995). Muscular rigidity affects extensor and flexor muscles simultaneously. When pressure is exerted on a rigid joint, the muscles yield for a short distance, then become rigid again. Assisted extension of the joint, therefore, occurs in a series of steps known as cogwheel rigidity. Akinesia refers to a poverty or absence of movement which may cause difficulty in initiating movement or be reflected in a "masked" facial expression, absence of eye-blinking, or loss of arm movement while walking. The PD patient may sit motionless for long periods and this akinesia can occur in the absence of rigidity (Kolb & Whishaw, 1990; Lezak, 1995).

Changes in posture may lead to difficulty in maintaining a normal standing position. The PD patient's head may incline forward or knees may be abnormally bent. Maintaining equilibrium and balance can also be difficult. Falls frequently occur, as once the postural equilibrium is disrupted, the PD patient may fall passively without attempting to regain balance. Gait is characterised by small, shuffling steps. Festination is common, with increasingly faster steps being taken, often resulting in a forward fall. Other symptoms may include aphagia (difficulty in chewing or swallowing) and speech disturbances.

The particular constellation of symptoms experienced by individual PD patients may vary considerably. The symptoms, however, all arise as a result of dopamine loss in the brain. The regions of the brain affected by dopamine loss are illustrated in Figure 1.1.

Figure 1.1: *Brain Regions Affected Physically or Functionally by PD*



From "Understanding Parkinson's Disease" by M.B. Youdim and P. Riederer, (January, 1997), *Scientific American*, p 39.

Neurons in the substantia nigra normally release a chemical messenger, the neurotransmitter dopamine, into the striatum. Striatal neurons, in turn, relay the chemical messages to the motor cortex as well as areas of the prefrontal cortex. In PD, a loss of neurons in the substantia nigra results in a marked reduction in dopamine levels with a corresponding loss of motor function. The condition is progressive, and as more neurons die out, motor functioning deteriorates. In the normally functioning brain, the amino acid tyrosine is converted to levodopa which is then converted into dopamine. Treatment for PD normally involves prescribing a synthetic form of levodopa, which the brain converts to dopamine to replace most, but not all, of the dopamine loss (Youdim & Riederer, 1997).

Despite Parkinson's original 1817 disclaimer, a growing body of evidence over the past decade has established the existence of cognitive deficits in PD. While motor difficulties and tremor remain the most visible characteristics of the disease, a number of subtle but identifiable cognitive deficits are also common manifestations of the condition. Dubois, Boller, Pillon, and Agid (1991) in a review of cognitive deficits in PD have noted evidence for cognitive disorders which may be isolated and occur without global intellectual impairment. These include word-finding difficulties in naming and word fluency tasks along with visuospatial disorders thought to be the result of impaired internal representation. Impaired functional use of memory processes has been demonstrated with impaired performance in delayed response tasks, recency discrimination, temporal ordering and associative learning. In addition, a slowing in the processing of complex cognitive tasks has been found, as well as behavioral regulation deficits and disorders of programming, planning and strategy (Dubois et al., 1991).

The neuropsychology of these cognitive deficits has been difficult to establish. The heterogeneity of symptoms found in PD has led many researchers to conclude that different parts of the brain are differentially affected in individual cases (Dubois et al., 1991; Caparros-Lefebvre, Pecheux, Petit, Duhamel, & Petit, 1994). The specific features of any cognitive deficit are thought to depend, in part, on the distribution of brain lesions, their severity and the order in which lesions develop during the course of the disease.

Despite the difficulties in applying fixed criteria, PD is usually accompanied by cognitive disorders which worsen as the disease progresses (Pillon, Dubois, Ploska, & Agid, 1991). Bradley, Welch, and Dick (1989) found that reaction times of PD patients were significantly slower on visuospatial tasks than on verbal tasks while response time of Controls was approximately the same on both tasks. However, task difficulty was also found to be a contributing factor in this study. PD patients performed at slower rates relative to Controls when they were required to generate a response in the process of carrying out a complex "mental mapping" task, but not when given a multiple choice task (Rensmayr, Schmidhuber-Eiler, Karamat, Engler-Plorer, Poewe, & Leidlmair, 1987, cited in Dubois et al., 1991).

Dubois et al. (1991) stress that cognitive slowing has only been demonstrated on tests which require high levels of processing and that the greater time required may indicate disturbances of cognitive strategy rather than a true slowing of central processing. The authors point out that in an analysis of performance on the 15-Objects Test, a visual discrimination task of 15 superimposed images (Pillon, Dubois, Bonnet, Esteguy, Guimaraes, Vigouret, Lhermitte, & Agid, 1989, cited in Dubois et al., 1991), "performance slowing" increased as the task became more difficult. The slowest patients extracted details without reference to any global shape, identified objects randomly, repeated identifications and showed poor set maintenance. This response pattern appeared to result from a defective response strategy, more indicative of frontal lobe dysfunction than reduced processing capacity (Pillon et al., 1989, cited in Dubois et al., 1991; Petrides, 1995).

Some caution is required, however, in comparing performance of patients with frontal lobe lesions with that of patients with PD. Owen, Sahakian, Hodges, Summers, Polkey, and Robbins (1995) noted subtle but important differences in frontal lobe dysfunction in PD patients compared with frontal lobe lesion patients. Accuracy appeared preserved, but latency of response increased in milder cases of PD, while the frontal lobe lesion group showed not only less response latency but were also less accurate. Planning functions appeared to deteriorate in PD as disease severity increased. The authors concluded that both accuracy and efficiency of planning are dependent on frontostriatial systems, while speed of thinking may be mediated by neurotransmitter systems of subcortical origin which innervate the cortex.

Much of the evidence presented for the frontal nature of cognitive deficits in PD has centered around studies which have examined the ability of PD patients to initiate, change and maintain a mental set. A mental set has been defined by Flowers and Robertson (1985) as:

> "*a state of brain activity which predisposes a subject to respond in one way when several alternatives are available*".

Maintaining a set, therefore, involves maintaining a preferred response or strategy against 1 or more competing alternatives. Also associated with set maintenance is the ability to change strategy when circumstances change. The ability to initiate, maintain and change a mental set appears disrupted with both frontal cortical lesions and basal ganglia lesions in human and animal studies (Flowers & Robertson, 1985). In addition, Petrides (1985) found patients with frontal lobe lesions were able to learn a series of response strategies but could not apply them to the task when required. Similar results have been obtained with studies of PD patients (Flowers & Robertson, 1985).

Gotham, Brown, and Marsden (1988) have proposed that an explanation for this pattern of impairment lies in the close association of the striatum with the frontal cortex. Postmortem studies and animal studies have provided evidence that the striatum is linked by neuronal loops which extend through the thalamus to specific regions of the frontal cortex. Delong, Georgopoulus, Crutcher, Mitchell, Richardson, and Alexander (1984, cited in Gotham, 1988) describe 2 such loops. The first, a motor loop, links the putamen (part of the striatum) with the motor cortex, while the second, referred to as the "complex loop", links the caudate (another striatal component) with the prefrontal cortex. It is this complex loop which is considered to play an important role in cognitive function. Lesions to nuclei in the caudate-prefrontal loop in monkeys have produced a marked deficit in cognitive functioning, unlike lesions to the motor loop (Canavan, 1988, cited in Gotham et al., 1988).

To ascribe cognitive function to this loop alone is somewhat simplistic, however. There are 3 other circuits which have been identified as making a possible contribution to cognition. The dorsolateral prefrontal circuit, comprising the dorsolateral portion of the head of the caudate and the rostrocaudal section extending to the tail, receives input from the dorsolateral prefrontal cortex, posterior parietal cortex and premotor area. Output from the caudate to the prefrontal cortex may be by 2 routes, either through the globus pallidus and thalamus to the caudal prefrontal region or through the substantia nigra and thalamus to the dorsolateral prefrontal cortex. The lateral orbitofrontal circuit is an area of prefrontal cortex which projects to the ventromedial sector of the caudate. Again, output can take 1 of 2 routes, involving both the globus pallidus and substantia nigra which converge on the lateral orbitofrontal cortex (Gotham et al., 1988).

Agid, Agid, and Ruberg (1987) in examining dopamine depletion in the basal ganglia and projection areas, found the putamen reduced to only 5% of normal levels. The caudate appears the next most seriously affected area with 15-20% of normal levels. Dopamine levels in the projection areas were least affected with the nucleus accumbens, hippocampus and frontal cortex at approximately 40 percent of normal levels while levels in the cingulate cortex, amygdala and hypothalamus were approximately 50 percent of normal. Little is known, however, of the levels of dopamine depletion in the caudatocortical loops.

Gotham et al. (1988) have proposed that these circuits may be differentially or selectively depleted of dopamine, which would, in part, explain the heterogeneous cognitive profiles seen in PD patients. Circuits may not be affected in the same way in all patients. Taylor, Saint-Cyr, and Lang (1986) consider that PD essentially disconnects the basal ganglia from its cortical input. It also disconnects premotor and prefrontal regions from the thalamically relayed basal ganglia input. These disconnections may initially affect the supplementary motor area, then the dorsolateral prefrontal cortex.

Middleton and Strick (1994) have presented anatomical evidence for cerebellar and basal ganglia involvement in higher cognitive functions. Their study examined outputs from the basal ganglia and cerebellum to Walker's area (Brodman's area 46) of the dorsolateral prefrontal cortex. This area was regarded by the authors as representing a distinct non-motor region of the frontal lobe. They also cited evidence for the involvement of this area in spatial working memory (Goldman-Rakic, Lidow, Smiley, & Williams, 1992) and planning of future action (Fuster, 1985).

Walker's area (Brodman's area 46) in 3 cebus monkeys was injected with a strain of herpes simplex virus with activity confined to the cerebello-thalamocortical and pallido-thalamocortical pathways of these animals. Results showed that the dorsolateral prefrontal cortex is the cortical target of a pallido-thalamocortical pathway and that this pathway is distinct from motor areas of the cortex. A similar input to Walker's area was identified from the cerebello-thalamocortical pathway. The authors concluded that the cerebellum and basal ganglia should not be viewed as purely motor structures and that functional descriptions should be widened to include such cognitive processes as working memory, rule-based learning and planning for the future.

Establishing dopamine levels within the basal ganglia and prefrontal cortex affected by PD is difficult. Postmortem evidence provides an essentially worst case scenario, with the disease process usually in advanced stages at death. Moreover, absolute levels of dopamine give little indication of functional significance. Adaptations such as receptor sensitivity may allow significantly depleted dopamine levels to produce relatively normal activity. Thus, wide variations between individual PD patients occur.

In summary, it appears that the cognitive deficits occuring in PD are subtle, selective and show wide variation between individuals who are affected. While many of the deficits observed in PD appear to be functions under the control of the frontal lobes, care should be taken in comparing patients with PD patients with those suffering frontal lobe damage. Complex connections are thought to exist between the prefrontal cortex and basal ganglia structures. The cognitive deficits which arise in PD may be the result of dopamine depletion in these caudatocortical loops which disconnect the basal ganglia from cortical input. In turn, it is likely that the prefrontal cortex plays a predominant role in sequencing, planning and remembering output from the basal ganglia.

The Effects of Aging

PD is usually considered to be a disease affecting older people, since onset before the age of 30 is rare. Incidence in the general population is estimated at 0.02%, which rises to 1% in people over the age of 50 (Lezak, 1995). Thus, age appears to be a factor in onset of the disease. Conversely, normal aging processes impact on the severity of PD. Neurodegenerative changes with age have been noted. More specifically, neuronal changes in the substantia nigra and loss of dopamine cells can be attributed to normal aging (Cruz-Sanchez, 1995). McGear, Tooyama, and Klegeris (1994) have established that there is a slow reduction in dopaminergic neurons throughout normal life, but this loss is accelerated in PD. Thus, the loss of dopaminergic neurons in PD should be viewed as a matter of degree, rather than as a distinct and separable neurodegenerative process.

The aging process alone, however, does not explain the selectivity of many deficits observed. Neither does it explain the normal performance of PD patients on many tasks of memory and intelligence, alongside impaired performance on specific tasks, such as the Tower of London (Owen et al., 1995). In addition, younger PD patients can show evidence of cognitive deficits (Hietanen & Teravainen, 1988). Thus, while it may be tempting to explain the cognitive changes of PD in terms of aging, age alone cannot account for the findings made.

It should be acknowledged that the relationship between age and PD is a complex one. Reid, Broe, Hely, Morris, Williamson, O'Sullivan, Rail, Genge, and Moss (1989) have presented evidence that PD patients with late-onset symptoms (beyond 70 years) show more evidence of cognitive impairment. Many early-onset PD patients with a predominantly tremulous form of the disease appear to reach a plateau where cognitive deficits remain mild for 10 years or more (Koller, Silver, & Lieberman, 1994). Determining the exact onset of the disease is also difficult. PD patients may experience mild motor difficulties or tremor for many years before seeking medical advice. Thus, for example, a PD patient may be described as having late-onset PD at diagnosis, where, in fact, they have had mild symptoms of the disease for approximately 5 years.

Gender Differences In Verbal Learning

While age appears to be a factor in the onset of PD, studies of gender differences have not identified differences in the etiology and progression of the disease in males and females (Diamond, Markham, Hoehn, McDowell, & Muenter, 1990). However, males and females may have pre-morbid differences in cognitive abilities which mediate the severity of any subsequent deficits attributable to PD. Evidence has been presented to support the existence of gender differences in verbal learning ability within normal populations.

Gender-related differences in cognitive functioning have been noted across a broad spectrum of tasks. Maccoby and Jacklin (1974), in a general review of the subject, concluded that females have superior verbal skills, particularly in adulthood, and that males tend to perform better on mathematical and spatial tasks. Kramer, Delis, and Daniel (1988), in an examination of gender differences in verbal memory, cite a number of studies in support of a gender advantage for females. These include female superiority on measures of oral verbal fluency (Benton & Hamsher, 1978; Fuld, 1977; Gaddes & Crockett, 1975; Kolb & Whishaw, 1990), written word fluency (Yendall, Fromm, Reddon, & Stefanyk, 1986) and naming tasks (Benbow & Stanley, 1983). (All studies in previous sentence cited in Kramer et al., 1988).

Historically, assessment measures have attempted to control for gender differences by eliminating items empirically found to favor one gender over the other. However, this has proved difficult to achieve in a domain as pervasive as verbal memory. Females have been found to consistently outperform males on the Wechsler Memory Scale, particularly the more verbal subtests of logical memory and paired associates (Ivison, 1990). They also outperform men on the Rey Auditory Verbal Learning Test (RAVLT: Rey, 1964), (Bolla-Wilson & Bleeker, 1996, cited in Kramer et al., 1988). Despite the gender advantage established on the WMS, only a single set of norms is provided for this widely used memory test.

Kramer et al. (1988) examined gender differences in 136 neurologically intact adults (68 males and 68 females) who were closely matched for age (Mean: M 47.01, F 46.93), and years of education (Mean: M 14.22, F 13.84). All participants were administered the California Verbal Learning Test (CVLT: Delis, Kramer, Kaplan, & Ober, 1987). The results showed that women had higher levels of immediate free recall, delayed free recall, and semantic clustering. In contrast, men showed a greater tendency to use serial recall as a strategy and recalled more from the primary and recency regions of the list. Gender explained over 10% of the variance on the immediate free recall and semantic clustering measures. A second comparison was carried out matching 36 males and 36 females for verbal intelligence. Comparable differences were found on free recall and semantic clustering for this group, suggesting that the gender differences found could not be explained by differences in verbal intelligence.

Results on the recognition trials showed no significant differences between the 2 gender groups. In interpreting this finding, the authors suggest that males are able to encode information as accurately as females and show a sufficient specificity to be able to discriminate target and non-target items with accuracy. They do not semantically organise the information to the same extent, however, tending to adopt more serial learning strategies and this may affect the way in which the information is encoded. Women, in contrast, used more semantic categories in organising the list which lead to more organised encoding and subsequently more efficient retrieval. Such efficiencies are not thought to be as applicable to the less effortful process of recognition. Hence, in the recognition task, men and women performed equally well.

Kramer et al. (1988) conclude that the gender differences established in this verbal learning task highlight the need for research design which takes account of gender differences by matching clinical and Control groups by gender and the need for separate norms by gender on measures of verbal learning and memory. Thus, while disease progression in PD may follow a similar course in males and females with respect to motor symptoms, the higher performance of women generally on verbal learning tasks needs to be taken into account.

Evidence for Verbal Memory Deficits in PD

The existence of verbal memory deficits in PD has been well documented. Dubois et al. (1991), in their general review of cognitive deficits in PD, cite a number of studies which show impaired performance on verbal tasks. Deficient performance on paired associate recall, immediately and following a delay, as well as impaired story recall have been noted (Bowen, Burns, & Yahr, 1976; Stern, Mayeux, & Rosen, 1984).

Such deficits appear more marked when material is not semantically organised, as demonstrated by performance on the Rey Auditory Verbal Learning Test (Taylor, Saint Cyr, & Lang, 1986; Caltagirone, Carlesimo, Nocentini, & Vicari,1989), and the Buschke Selective Reminding Test (Della Salla, Di Lorenzo, Giordano, & Spinnler, 1986; Halkala, Laulumaa, Soininen, & Riekkinen,1989). PD patients' visual recall of verbal memory is well below that of Control groups (Boller, Passafiume, Keefe, Rogers, Morrow, & Kim, 1984; Sullivan, Sagar, Gabrieli, Corkin, & Growden,1989) while recognition memory for the PD group as a whole has been found to be largely intact (Ruberg & Agid, 1988). Dubois et al. (1991) conclude that the ability to learn verbal material is preserved in PD, but impairments in recall suggest that either functional use of memory stores or the processes of retrieval are deficient.

PD patients have shown impaired performance in naming and word fluency tasks. Dubois et al. (1991) consider impaired semantic memory may be a possible explanation for these findings. These include impaired performance on confrontation naming (Globus, Mildworf, & Melamed, 1985), verbal fluency (Cools, Van der Bercken, Horstink, Van Spaendonck, & Berger, 1984; Stern et al., 1984; Pillon, Dubois, Lhermitte, & Agid, 1986), and synonym detection (Tweedy, Langer, & McDowell, 1982). Vocabulary generally remains intact (Matison, Mayeux, Rosen, & Fahn, 1982; Lees & Smith, 1983).

More recently, Massman, Delis, Butters, Levin, and Salmon (1990) compared the verbal learning abilities of PD patients with those of a Huntington's disease (HD) group. They considered that many earlier studies were constrained by methodological flaws, citing a failure to control for current verbal intellectual functioning and lack of statistical power as important constraints. Accordingly, a larger sample was used (57) with 19 participants in each group and matching PD, HD, and Control groups on Wechsler Adult Intelligence Scale - Revised (WAIS-R) (Wechsler, 1987) vocabulary scores. The CVLT was administered to the 3 groups. It should be noted that the groups were not matched by gender or age, with the PD group being 12 years older on average than the HD group and 4 years older than the Control group.

Massman et al. (1990) found a consistent profile of impaired performance within the PD group relative to normal Controls. This profile, which was also consistent for the HD group, included impaired immediate memory span, inconsistency of recall across trials, deficient use of semantic clustering, elevated intrusion rates on delayed recall and impaired recognition memory. In contrast to earlier studies, which were largely restricted to an examination of global scores, this study found evidence of impaired recognition memory. Both HD and PD groups showed difficulty in discrimination of previously included items from distractor items on the recognition trials of the CVLT. Thus, in contrast to earlier studies, the authors concluded that PD is associated with mildly deficient encoding as well as recall deficits. Massman et al. considered that the pathology characteristic of PD and HD interferes with the ability to utilise active, efficient encoding and retrieval strategies on an unstructured learning and memory task, such as the CVLT.

Kramer, Levin, Brandt, and Delis (1989) examined the profiles of Alzheimer's (AD), Huntington's (HD) and PD patients on the CVLT. Participants were classified into 5 groups representing high and low PD, high and low HD, and AD. A cutoff score of 30 words correctly recalled on the 5 learning trials of the CVLT was used as the basis for high or low classification. Again, participants were not matched for gender or age, with age ranging from a mean of 45.7 for the low HD group to 71.2 for the low PD group. A stepwise discriminant function analysis was used to establish whether memory performance would correctly classify the patients into the 5 groups. Results showed that 75% of PD, HD and AD patients sub-grouped according to memory functioning on the CVLT. A notable feature of the PD group was that it exhibited a faster rate of forgetting than the HD group during the short delay interval. The authors note that futures studies of verbal learning in PD need to take account of age and education factors to reduce potential error variance.

Delis, Levin, and Kramer (1987) investigated verbal learning strategies in PD, AD and normal Control groups, also administering the CVLT. PD participants showed impaired levels of recall and recognition accuracy relative to normal Controls, but better performance on these 2 measures than the AD group. Both PD and AD groups did not differ on what the authors termed "strategies of learning". Both groups showed impaired utilisation of semantic categories, larger recency effects, inconsistency of recall across trials, and impaired ability to discriminate relevant from irrelevant responses on the recognition task. It was concluded that PD and AD patients differed only in the degree of impairment, with AD patients showing a greater deficit.

Sullivan et al. (1989) examined the cognitive profiles of patients with PD and AD against those of normal Controls over a wide range of cognitive tasks. These included picture arrangement, vocabulary, mental status, an estimate of I.Q., and the WMS. The PD, HD and Control groups consisted of 15 patients each, with the PD group being further divided into high and low PD based on scores on the Blessed Dementia Scale (Blessed, Tomlinson, & Roth, 1968). This classification resulted in a total of 8 high PD and 7 low PD participants. Once again, age and gender were not controlled, with 8 females in the Control group compared with 1 female in the PD group. The Control group showed an age advantage of between 3-5 years. The small sample sizes combined with the gender differences are potential sources of error variance and have the potential to reduce statistical power.

With specific reference to verbal memory, the most striking result of this study was the recall scores for the PD group on the immediate and delayed recall trials of the WMS. The low PD group recall showed a mean movement of 15.2 items on immediate recall to 11.1 items on delayed recall, while the high PD group moved from 13.2 to 6.2 items. The authors noted that memory impairment for the low PD group was either mild or nonexistent over a range of tasks and comparable to normal Controls. This pattern of performance raises the possibility of 2 distinct sub-types of cognitive deficit in PD. The authors, however, favor the alternative explanation that memory deficit is a continuous process related to overall disease severity.

Additional studies provide further evidence that distinct sub-types of PD do exist. Reid, Broe, et al. (1989) examined the characteristics of early-onset and late-onset PD in 100 *de novo* patients at Sydney's Royal North Shore Hospital. They found that patients with late-onset PD have more widespread cognitive impairment than those with early-onset PD. This finding was established by administering a range of tasks including perceptuomotor speed and decision time tasks, a modified Austin maze, vocabulary and block design (Wechsler, 1987), Ravens Progressive Matrices (Raven, 1965), the RAVLT (Ray, 1964), and the Benton Visual Retention Test (Benton, 1974). (All test authors are cited in Reid, Broe, et al., 1989). Of specific interest to this discussion of verbal memory was performance on the RAVLT. The early-onset PD group recalled a mean 10.80 items, compared to 8.50 for the late-onset group and 11.96 for Controls.

Reid, Broe et al. (1989) provide some evidence that PD can be divided into sub-types based on motor symptoms. These sub-types consist of the akinetic-rigid group, the tremor-dominant group and those that have uniformity in akinesia, rigidity and tremor. They argue that, in patients with PD consisting predominantly of tremor, the disease follows a more benign course than those with akinetic-rigidity difficulties as the predominant symptoms. This latter group show more cognitive decline (Mortimer, Hansch, Pirozzolo, & Webster, 1982; Ransmayr, Poewe, Plorer, Birbamer & Gerstenbrand,1986; Rinne, 1980, all cited in Reid, Broe, et al., 1989).

Later studies have also established the existence of cognitive deficits of greater severity in late-onset patients (Biggins, Boyd, Harrop, Madeley, Mindham, Randall, & Spokes, 1992). Thus, the evidence suggests that there are specific constellations of symptoms and etiology related to onset which influence cognitive decline in PD. These specific clusters of symptoms may represent specific sub-types of PD.

Buytenhuijs, Berger, Van Spaendonck, Horstink, Borm, and Cools (1994) examined verbal learning strategies in PD by administering the CVLT to 59 PD and 30 Control participants. The authors argued that PD patients perform poorly relative to Control participants on tasks which require internal planning, such as semantic categorisation. Where external cues such as category prompts are provided, however, no substantial difference in task performance between PD and Control participants is found. Once again participants were not matched for age or gender. In addition, the average duration of PD was only 5 years. Results showed that recall in the PD patients was deficient, whereas recognition memory remained intact. In addition, PD patients showed a tendency toward serial recall rather than organising the list into semantic categories. Buytenhuijs et al. concluded from these results that PD patients adhere to a strategy which is externally imposed by the experimenter and have difficulty with internally generated semantic categorisation.

The 5 year average duration of PD in this study may explain the finding that recognition memory was intact. Furthermore, the authors ignore a number of studies which establish the existence of impaired recognition memory in PD and opt instead for a consensus opinion that recall generally is affected, while recognition remains intact. The conclusions reached in this study are open to question on two grounds. Firstly, they are based on the erroneous assumption that recognition remains intact in PD. Secondly, the authors do not present any results for the cued recall trials of the CVLT as evidence that externally guided recall is not impaired. Without these results, it is not possible to establish that PD patients receive any benefit from the provision of an external structure, as the authors claim. If participants in this study did not receive any benefit from cueing in recall, then the argument that recognition remains intact because it is externally guided does not hold. It is more likely that recognition remained intact because of disease duration in these participants.

Halkala et al. (1989) carried out an examination of delayed memory in PD and AD using the Buschke Selective Reminding Test as modified by Fuld (1980, cited in Halkala et al., 1989), and a story recall test according to Lauria's homogenous interference method (Christenson, 1975, cited in Halkala et al., 1989). The PD group recalled more items following interference and after a 30 min delay. The PD group benefited from rehearsal of the stories more than AD patients and were able to recall more information. On the list learning test both AD and PD were equally impaired on recall, but PD patients showed better recognition memory on a 30 min delay. The authors concluded that recognition memory is better preserved in PD than in AD and that PD patients appear to be able to bind more information into long term storage than AD patients. Other studies have demonstrated that memory for remote events is relatively well preserved in PD (Sagar, Cohen, Sullivan, Corkin, & Growden, 1988; Newman, Weingartner, Smallberg, & Caine, 1984).

Tweedy et al. (1982) compared the performance of 35 PD participants on a series of verbal memory tasks with 21 normal Control group members and 23 right hemisphere (RH) stroke patients. Controls were age advantaged with a mean age 3 years below the PD group. In addition, 33% of Controls were female, while the number of females in the PD and RH groups was not stated. The groups were administered a verbal learning task with recall similar to the CVLT and a recognition task which required the participants to signal using hand movements, either the repetition of a previously presented word or the presence of a synonym. In the recognition task, participants were reduced to 16 for the PD group and 18 for the right hemisphere stroke group. As expected, the PD group showed impaired performance compared to the RH and normal Control groups. This difference was particularly marked for the initial free recall trials. While the PD group did not benefit from the provision of semantic cues as an aid to recall at the end of the free recall trial, a cluster analysis revealed that use of semantic categories during the initial recall trial itself was identical to that of the Control group.

These results suggest that ability to utilise semantic categories was preserved in the PD group. However, only 1 free recall trial was used to substantiate this. It is likely that normal Controls would require more than 1 trial to uncover the semantic clustering strategy as a means of efficiently recalling the list.

The finding that semantic memory is unimpaired in PD is not borne out by a number of other studies (e.g., Gurd & Ward, 1989; Massman et al., 1990). In the recognition task, PD participants recognised fewer repetitions and synonyms and showed higher false positive rates (that is, responded that an item was a repetition or a synonym when it was not) than the RH or Control groups.

Tweedy et al. (1982) conclude, somewhat optimistically in view of the data, that these results constitute evidence of a partial sparing of recognition in PD. They suggest that this finding indicates a selective impairment of retrieval operations since recognition constitutes less effortful retrieval than recall. However, their results could also be due to the relatively homogenous sample used. All PD patients had mild to moderate symptoms, and all were outpatients with a good response to medication.

Studies which confidently assert that semantic and recognition memory are intact in PD using participants with a mean duration of PD less than 5 years (Buytenhuijs et al.,1994; Sullivan et al., 1989; Gabrieli, Singh, Stebbins, & Goetz, 1996) do not appear to take account of the neurodegenerative aspects of the condition, or a number of studies which link memory deficits to disease severity (Hietenan & Teravainen, 1988; Mohr, Junos, Cox, Litvan, Fedio, & Chase, 1990).

From an examination of the research on cognitive deficits in PD, it appears that the existence of such deficits has been well established. These include difficulties in word-finding, visuospatial tasks, judgments of recency, temporal ordering and associative learning. Slowing of response times on complex cognitive tasks have been noted, along with disorders of programming, planning and strategy. A number of studies have identified impaired verbal memory performance in PD patients relative to normal Controls. Impaired recall and recognition memory have been demonstrated, along with poor semantic organisation and utilisation of semantic categories. Larger recency effects have been found, along with inconsistency of recall across trials, high intrusion rates and discrimination difficulties on recognition tasks. Impaired recall is thought to be associated with retrieval deficits, while impaired recognition appears related to encoding deficits.

Impaired recognition memory has not been established in all studies, however, and, while these different findings may be due to methodological issues, the subject remains contentious. Onset of symptoms in PD has been found to be a factor in the severity of cognitive decline. Patients with late-onset PD have more widespread cognitive impairment than those with early-onset PD. In addition, patients with tremor as the predominant symptom appear to have less cognitive impairment than those with predominantly akinesia and rigidity.

The Effects of Levodopa Treatment

Dopamine is a neurotransmitter which is synthesized from the amino acid, tyrosine, found in food. In the naturally occuring process of dopamine production, which occurs in the substantia nigra, tyrosine is first converted to dopa and then to dopamine. Administered externally, however, dopamine does not cross the blood-brain barrier. In order to increase dopamine in the brain, therefore, dopa, in the form of levodopa, is prescribed. Dopamine's principle role as a neurotransmitter is thought to be in the control of movement (Kolb & Whishaw, 1990), having a modulator effect on motor control pathways (Mahurin, Feher, Nance, Levy, & Pirozzolo, 1993). Overt physical symptoms of PD do not occur until approximately 80% of dopamine reserves have been depleted (Riederer & Wuketich,1976, cited in Mahurin et al., 1993)

Levodopa treatment can, selectively, have an adverse effect on cognitive function (Koller, et al., 1994). Therefore, the effects of this treatment on verbal memory tasks should be considered. The early state of well-being which usually follows initial treatment with levodopa is often followed by a decline. In some cases, a confusional state which is drug-related can occur. Such confusional states often arise where medication is overprescribed. However, where optimal levels of levodopa can be achieved, such as by intravenous infusion, improvement in multiple choice reaction time and delayed verbal memory has been noted (Saint Cyr, Taylor, & Lang, 1993).

The "on-off" phenomenon which commonly occur as the disease progresses, however, can introduce a number of complex changes. Tests of verbal fluency tend to show impairment when patients are in the "off" state with associative learning impaired in the "on" state, the latter being attributed to a confusional state (Saint Cyr et al., 1993). Gotham et al. (1988) examined the impact of fluctuations in brain dopamine levels on frontal cognitive function in patients with PD, testing both "on" and "off " states in levodopa treatment. Verbal fluency was also impaired in the "off " state while patients on medication showed normalised performance on verbal fluency tests. Conditional learning tests also showed impairment in the "on" state in this study.

Thus, the general picture which emerges is one of preserved verbal memory in response to levodopa treatment with associative and conditional learning tests showing impairment where a confusional state exists. Provided that optimal levels of levodopa are administered, verbal memory can be aided by levodopa treatment.

Memory

"It is the nature of dynamic systems, that no one can ever achieve complete knowledge and control of all the relevant variables that interact in them at any given time. This is true for social systems, economic systems, ecological systems and, of course, for the brain, which is the most complex system of all".

(Fuster, 1995: "Memory in the Cerebral Cortex")

In an attempt to arrive at a clearer understanding of memory, many different classifications have been introduced. In 1890, William James, in his book "Principles of Psychology" stressed the active nature of the human mind. He distinguished 2 types of memory, primary and secondary. Primary memory was equated with events which were the current contents of consciousness and secondary memory consisted of memory of the past, which required a process of bringing information back into consciousness. Following the resurgence of interest in cognitive processes which began in the 1970's, this concept of memory consisting of 2 distinct processes received wide acceptance and has been a feature of memory research over the past 30 years (Ratcliffe, 1978; Matlin, 1989; Conway & Engle, 1994).

In recent times, however, many models of memory have assumed that short term memory, equivalent to James's primary memory, is simply the activated portion of long term memory and that such distinctions of memory, based on processes, do not exist (Cowan, 1988; McClelland & Rumelhart, 1986). This subject is one of debate and has yet to be resolved.

Conway and Engle (1994) have found functional differences between processes involved in retrieval from primary and secondary memory. In a series of experiments designed to investigate these processes of retrieval, the authors found that retrieval from these two states was qualitatively different, with retrieval from primary memory a function of set-size, whereas retrieval from secondary memory was not, with large sets of words retrieved from secondary memory as quickly as small sets. The authors concluded that the two states showed qualitative and discrete distinctions in the level of activation which did not support the concept of primary memory at one end of a continuum of activation with secondary memory at the other. Thus, for the purposes of the present study, it is assumed that there are qualitative differences between short and long term memory processes.

Working Memory

Working memory has been described as a "table-top" memory, where events and information that form the current focus of attention are "laid-out" and processed. It incorporates what is widely regarded as short-term memory and is closely aligned to what James (1890) termed "primary memory". The concept of working memory is generally attributed to Baddeley (1986,) who posited the existence of 2 short term stores for visual and verbal information which were in turn managed by a central executive responsible for allocation of processing tasks. Baddeley's (1986) definition of working memory, however, is somewhat restrictive and refers to;

> "*the temporary storage of information that is being processed in any of a range of cognitive tasks*".
>
> (Baddeley, 1986)

This definition encompasses information which is held for a short time only without enhancement by rehearsal or reactivation.

Central to the concept of working memory is the concept of executive function, which has evolved from Baddeley's (1986) definition of a central executive. Executive function refers to functions, usually assumed to originate in the frontal lobe, which are involved in the regulation of problem solving strategies (Baddeley, 1990). The existence of such functions has been challenged, however (David, 1993; Stuss, 1993, both cited in Spaendonck, Berger, Horstink, Buytenhuijs, & Cools, 1996). Both David and Stuss have concluded that the concept of executive function lacks specificity and an empirical basis. Spaendonck et al. (1996) found a firm relationship between cognitive shifting ability in PD and motor rigidity. Word fluency showed no significant association with severity of motor symptoms. As both cognitive tasks are regarded as executive functions, it was expected that both tasks would show an association with severity of motor symptoms. The authors concluded, therefore, that the concept of executive function is too heterogeneous for empirical studies.

Other aspects of working memory have also been challenged and the area, as a theoretical issue, has been the subject of much debate. Critical issues surrounding the validity of working memory concern questions such as whether the limited capacity of working memory demonstrated in the laboratory can also account for the vastly expanded working memory of experts or skilled performers. The concept of working memory does not appear to account for the fact that skilled activities can be interrupted and later resumed without impaired performance (Ericson & Kintsch, 1995).

Procedural Memory

A further categorisation of memory relates to how information contained in memory is acquired. Procedural memory involves knowledge about how to perform cognitive activities (Matlin, 1989). This knowledge is generally implicit and not consciously recalled; hence, it is often referred to as "implicit memory". The more overlearned the skill, the less likely the processes involved will be remembered. For example, if one were to ask a professional tennis player to describe the steps involved in serving a ball, they would be unlikely to remember them. If asked to think about the steps while actually serving a ball, performance would be disrupted. These skills are automatic sequences previously learned that appear to be no longer under the control of conscious processes.

Declarative Memory

Declarative memory is another broad-based distinction which involves knowledge about facts and things (Matlin, 1989). It encompasses both James' definition of secondary memory and what other theorists have defined as long term memory and memory for temporal judgments related to dates and times (Cohen, 1988). Semantic and episodic memory are also included in this definition, the latter concerned with events of an autobiographical nature.

A Neural Network Model of Memory

While numerous accounts and taxonomies of memory take a cognitive approach, concentrated on the analysis of higher cognitive functions, perhaps a more appropriate discourse for a discussion on the memory deficits associated with PD is the neuroscience approach. As PD is classified as a neurodegenerative condition, a discussion which has at its focus the structure of the nervous system and the changes which result from brain lesions is a logical approach. Thus, in this examination of the memory processes which are impaired in PD, the neuropsychology of memory will be highlighted. The following discussion draws extensively on the memory research of Fuster (1985, 1989, 1995). The reader is referred to Fuster's (1995) book, "Memory In The Cerebral Cortex" for further detail.

Central to any examination of memory functioning within the nervous system is the concept of a neural network. In this context, memory ***is*** the network of cortical neurons and the connections between networks. The network is formed by experience as a result of concurrent activation of groups of neurons that represent aspects of the internal and external environment and motor action. By the process of activation, these groups of neurons become the "nodes" of the network. Networks may vary in size and type of grouping. They are modifiable by experience and have the capacity to grow throughout life. Each neuronal group may be part of multiple memory representations. The network is most defined by the type of groupings which form it. Relatedness is the essential component and, therefore, all memory is associative.

A key feature of many models of neural memory networks is the concept of activation. This concept is not unique to neural network models and follows on from Hebb's (1949) dual-trace memory mechanism. Hebb proposed that activity continued for a period of time in memory cell groupings by a process of reverberation within the neuronal circuits. The term activation itself, however, is more directly attributable to Anderson's (1983) Adaptive Control of Thought (ACT) model (Matlin, 1989). The ACT model assumes that primary memory consists of information in secondary memory that has been activated above a critical threshold. As the activation level of a concept increases, so does accessibility. Thus, the ACT model states that the 2 states of primary and secondary memory are distinguishable only by the level of activation. Access to the neural network can be achieved by more than one process of activation. Processing can thus be carried out in parallel (Conway & Engle, 1994). The ACT model and other associative models of neural memory base the networks in the cerebral cortex and conform to most of the constraints and requirements imposed by neural structure and function as presently known (Fuster, 1995).

These earlier models of neural networks contained a number of limitations. Fuster (1995) argues that, in human memory, variance is the norm. No certain output comes from a given input. Therefore, the early computational-based models of memory, such as the algorithm models of McClelland and Rumelhart (1986), contained a certain rigidity which the human brain does not exemplify. It has been difficult to reconcile these models with the redundancies, dynamics and plasticity of the brain. In addition, the brain is robust and has the capacity to sustain injury without any loss of long term function. Fuster (1995) concludes that memory is robust but not probabilistic. Neural memory models are required to be less rigid, algorithmic and probabilistic, and more dynamic to closely proximate the properties of cortical memory.

Fuster (1995) presents an alternative model of neural memory networks which is highly dynamic, open to change and assumes the following properties. Firstly, a level of spontaneous discharge above a certain maintenance level is necessary for effective activation or inhibition of a network. Activation is extremely fast, with all the elements of a large cortical network capable of being activated within 100-300 ms. It occurs through the arrival of impulses which are convergent, divergent and parallel. Reactivation can also be continuous.

Activation involves all levels of the network from the lowest to highest. There is a hierarchy of representation and, further up the hierarchy, activation is less dependent on the current stimulus and more dependent on past history and experience. In this way, large networks can be reactivated by simple stimuli, to recreate elaborate memories. Thus, we need only see a small part of a picture to recreate the whole image in our mind.

Memory is dispersed. Recall involves the activation of distinct areas of neocortex that are also vast, due to the multimodel and heterogeneous nature of the contents. The hippocampus is thought to participate as a convergence zone which facilitates activation of outlying cortical areas. Activation spreads between distant areas via corticocortical connections.

Encoding Processes

Central to the model is the notion that activation is a requirement for the acquisition of new memory as well as the short term retention of reactivated memory, old or new. New memory involves the opening of new synapses, while old memory does not. Adding new memory to an old network is thought to involve splitting the old network and reclassifying it to form a discrimination (Fuster, 1995).

Retrieval Processes

The traditionally held view of retrieval from memory is that it may fail for essentially two reasons, decay or interference (Klatzky, 1975; Tulving, 1983). However, within the framework of a neural network explanation, it is also possible that the failure of a network to activate or a failure in the process of continuous reactivation may result in non-retrieval of a previously learned item.

Attention

Within the neural network model proposed by Fuster (1995), attention is a part of memory and memory is a part of attention. Single cell studies (Fuster, 1990) show cells which fire in attention tasks belong to established cortical networks. These cells and their networks continue to be activated after retrieval, if behavior requires the active remembering of stimuli which are being attended to. Thus, the model does not make distinctions which allow the separation of attentional deficits from memory deficits.

Inhibition

Many of the mechanisms which activate neural networks and subsequently deactivate them when retention is no longer required, remain unclear. One of these mechanisms is thought to be the process of inhibition. Once again, with reference to single cell studies, it has been shown that specific cells may be inhibited during the delayed period of a delayed matching task (Miller, Li, & Desimone, 1993). This suggests a gating process into short term memory, with reciprocal inhibition of some cells because they are part of networks which represent alternative cues and memories. The gating mechanism appears to operate as a load signal and is thought to come from either limbic structures such as the amygdala or the prefrontal cortex, signaling what is important to be retained for subsequent action. When a gate is open, input is allowed into the network and held at its original level of activation. In this way the input is maintained until unloaded (Fuster, 1995).

The Structure of Neural Networks: Anatomy and Organisation

One of the difficulties in attempting to account for memory by an examination of neural organisation is the fact that memory is not a localised function. One cannot simply point to a localised structure, such as the hippocampus, and say 'here is the neural location of memory". It is widely distributed throughout the cortex with links to limbic and basal ganglia structures which form a complex interactive system.

Perceptual memory, which encompasses declarative, episodic and semantic memory, is based mainly in the neural networks of the posterior cortex. This encompasses the large area of neocortex of the parietal, temporal and occipital lobes, from the rolandic fissure and the temporal pole to the occipital pole. Unimodal perceptual memory is based in primary and associative areas of what has been called the sensory cortex. This encompasses the occipital lobe for vision, the superior temporal cortex for audition and the posterior parietal cortex along with Brodman's area 3b for touch and memory for somatosensory information. Gustatory and olfactory memory is based in the paralimbic areas of the orbitofrontal cortex.

Declarative memory is found in wide networks which interlink with areas of multisensory convergence. The networks of semantic memory are also widely connected, with nodes that appear prevalent in the lateral and inferior regions of the parietotemporal lobes. Conceptual knowledge is thought to be so broadly based in the posterior and frontal cortex that localisation is not possible within the confines of our present knowledge (Fuster, 1995).

Within the posterior cortex, Wernicke's area, in the posterior extremity of the superior temporal gyrus, is thought to be a critical focus for semantic language representation. Language, in turn, requires continuous interactive cooperation between the sensory memory networks of the prefrontal cortex and Wernicke's area (Fuster, 1995). Neocortical links also exist between the frontal cortex and basal ganglia structures. These areas are thought to incorporate a number of intermediate representational layers of the motor hierarchy responsible for the execution of actions and habits which have become learned to the point of being automatic (Marsden, 1982). Basal ganglia structures are connected to the action plans of the prefrontal cortex.

Motor memory networks which input from the thalamus and cerebellum represent relatively concrete vectors of striatal muscle movement. At the dorsolateral-prefrontal cortex they represent in a more general and conceptual sense, what has been called "the schemata of action". They have an essentially implicit nature (beyond consciousness) which ceases when the network becomes operational. More direct motor responses are represented in the motor cortex where learning has a lesser role. Lesions of this area generally result in paralysis (Fuster, 1995).

When perceptual memory is activated in the posterior cortex, and that memory has implications for action, the prefrontal cortex is activated at the same time. This frontal involvement is accomplished by long corticocortical connections from the posterior to the anterior cortex. The frontal hierarchy in neural networks for motor memory appears to be a mirror image of the posterior cortical hierarchy for perceptual memory. The 2 areas connect in the dorsolateral-prefrontal cortex at similar hierarchical levels, with the processing of action in the frontal cortex following a similar hierarchical progression from highest (prefrontal) to lowest (motor).

Thus, it is in the dorsolateral-prefrontal cortex that the perceptual and motor memory systems meet. The 2 types of neurons in the prefrontal cortex related to motor and perceptual organisation are intermingled and in close proximity (Fuster, 1982, cited in Fuster, 1995). They represent 2 complementary and interactive systems; one retrospective, the short term memory of the stimulus, and the other prospective, the short term memory of the forthcoming response, or what has been termed "memory for the future" (Fuster, 1995).

In application, the complex motor plans held in the prefrontal cortex often involve monitoring a sequence of actions and carrying information across time. To achieve this, the dorsolateral prefrontal cortex involves 2 complimentary memory sets; short term active memory and sort term motor set. The first keeps the memory of recent events active and the second sets motor systems to act.

Thus, motor and perceptual memory systems are complimentary, interactive, and in close proximity to each other. It is possible, therefore, that these systems may also share some similarities in pathology when affected by conditions such as PD. Single cell studies of motor neurons show that the cell exhibits a burst of neural activity in preparedness for a motor act (Fuster, 1995; Iansek, 1997). It is thought that this initial burst of activity is absent in PD (Iansek, 1997) and it is also possible that it is absent in the neurons of cortical memory networks affected by PD.

Supporting Evidence from Positron Emission Topography and Magnetic Resonance Imaging:

Fuster's (1995) model is supported by a number of studies which have used Positron Emission Topography (PET) and Functional Magnetic Resonance Imaging (MRI) to examine the neural activity associated with memory functioning. These techniques measure the changes in microvasculature surrounding metabolically active brain tissue. The images obtained are thus able to quantify the activation associated with various memory tasks.

PET studies have revealed increased activation in the right prefrontal cortex during performance of a spatial memory task, and increased cerebral blood flow to both left and right mesial prefrontal cortex during performance of a modified version of the Tower of London task (Jonides, Smith, Koeppe, Awh, Minoshima, & Mintun, 1993; Rezai, Andreasen, Allinger, Cohen, Swayze, & O'Leary, 1993, both cited in Owen et al., 1995).

Gabrieli, Desmond, Demb, Wagner, Stone, Vaidya, and Glover (1996) examined frontal-lobe activation associated with encoding and retrieving from semantic memory. Semantic encoding was required to judge whether words had abstract or concrete meanings. Perceptual encoding was required to judge whether words appeared in upper or lowercase letters. Results showed that participants recalled 6 times more words after semantic than after perceptual encoding. Greater activation of the left inferior prefrontal cortex was found for semantic encoding. The authors concluded that the left prefrontal cortex mediates semantic working memory. A region in the left inferior prefrontal cortex appears to access semantic information stored in the more posterior language cortex. In the inferior prefrontal cortex, activation increases as the demands on semantic working memory increase. The authors argue that the processes measured in the left inferior prefrontal cortex may be best regarded as "a search for meaning".

Awh, Jonides, Smith, Schumacher, Koeppe, and Katz (1996) examined storage and rehearsal components of verbal working memory. Participants performed an item recognition test and a "2-back" memory task which involved continuous maintenance of a verbal working memory load, with the participant indicating whether a letter presented was identical to one presented 2 letters previously. Results indicated that the rehearsal and storage components of verbal working memory are separate processes.

Both tasks accessed speech planning and execution areas in frontal lobe regions (although no overt speech was required during the task) along with posterior parietal regions associated with storage of verbal information. In addition, when rehearsal-based activation was subtracted, the bulk of frontal brain activation was subtracted out, while posterior parietal regions remained active. The authors concluded that rehearsal in verbal memory is a process drawing on a frontal mechanism similar to the one used for overt speech. Thus, the study supports the concept of frontal mechanisms mediating verbal memory, with rehearsal processes appearing to be dependent on the integrity of the frontal lobes.

Memory and Pathology

Neural Networks in Parkinson's Disease

In consideration of the neural network model of memory (Fuster, 1995) presented in the previous section, there are a number of points within the active processes of the model where memory, as affected by PD, may fail. These cognitive failures may encompass aspects of encoding and retrieval mechanisms, inhibition and gating systems, and the interactive components of verbal memory. In addition, the corticocortical loops from the prefrontal to the posterior cortex and caudatocortical loops from the basal ganglia may be impaired.

Retrieval Processes

A central component of Fuster's (1995) model is the assumption that activation is a requirement for the acquisition of new memory as well as for the short term retention of reactivated memory, old or new. Retrieval processes may fail in PD as a direct result of a loss of dopamine receptors which normally facilitate the activation of a neural network. Neurochemical studies have indicated that the activation of the prefrontal cortex during a delayed memory task is mediated by dopamine receptors. Sawaguchi and Goldman-Rakic (1991) demonstrated that localised injection of dopamine agonists in the prefrontal cortex of monkeys blocked activation in a manual delayed response task. These localised injections produced reversible deficits in delayed response performance of 3 monkeys at a total of 17 sites in the principal sulcal region of the dorsolateral prefrontal cortex. The authors concluded that activation of dopamine receptors is critical for the memory processes mediated by the prefrontal cortex.

While many of the examples given by Goldman-Rakic and Fuster to illustrate memory processes rely on memory tasks carried out by monkeys, other studies have established that the dopaminergic organisation of the frontal cortex is similar in humans (Goldman-Rakic et al., 1992).

Activation levels determine how available information will be and this in turn will impact on the probability and speed of successful retrieval (Anderson, 1983; Anderson, Reder, & Lebiere, 1996). If activation levels in the prefrontal cortex are low, due to a loss of dopamine receptors, this limits the ability of activation to spread to perceptual memory networks, so that information can be retrieved or reliably discriminated from other distractor items.

Inhibition mechanisms may also be affected in retrieval operations in PD. Conway and Engle (1994) measured how individual differences affect retrieval from primary and secondary memory. They found that participants with low levels of recall were unable to inhibit activation of irrelevant information, allowing it to come into an active state. It also seems likely that when working memory and selective attention are working normally, inhibitory mechanisms serve to limit access to working memory to information that is directly relevant to the goals of the task.

Thus, inhibitory mechanisms that are inefficient allow information that is off the "goal path" to enter working memory (Hasher & Zacks, 1988, cited in Conway & Engle, 1994). This processing deficit is age-related and may be accelerated in PD due to the loss of dopamine receptors in the prefrontal cortex. In neural network terms, gating mechanisms may remain open when they should be closed, allowing more input into the neural network than is necessary and making inhibition of alternative responses difficult.

This difficulty in inhibiting alternative responses has been well established in PD (Flowers & Robertson, 1985; Sullivan et al., 1989). A similar phenomenon has been observed in studies of patients with lesions to the prefrontal cortex. These patients were able to learn a series of strategies effectively but were unable to apply them, suggesting difficulties in choosing between alternative responses (Petrides, 1985).

Thus, evidence suggests that activation of dopamine receptors is critical for memory processes involving the prefrontal cortex. Where these activation levels are low, this, in turn can limit the spread of activation to perceptual memory networks. In addition, if inhibitory mechanisms are inefficient, information which is irrelevant to the memory task in hand may be retrieved, resulting in difficulty in choosing between alternative responses.

Encoding Processes

"The richest source of new plans is our old plans, transformed to meet new situations"

(Millar & Chomsky, 1963, cited in Fuster, 1995)

In Fuster's model, activation is a necessary condition for the acquisition of new memories, with access to existing neural networks being necessary to add new information to them or recategorise old information to account for new information. Within this model, encoding processes may also fail due to a failure of the neural network to activate. Again, this may be due to a loss of dopamine receptors in the prefrontal cortex. Inefficiencies in inhibition and gating mechanisms may also result in encoding difficulties. If neural gates remain open when they should be closed during the encoding phase of a learning task, then correct categorisation and assignment of semantic meaning to the new item may be impaired. Ability to discriminate between old and new items may also be compromised.

Encoding processes in PD may also be affected by the level of difficulty of the task. In a verbal learning task, for example, the participant may be required to uncover a semantic encoding strategy as a means of efficiently retaining information on a list. The participant is not generally told which strategy to use. In order to successfully complete the task, the participant appears to employ a dual-encoding strategy. Firstly, the list seems to be held in working memory for several minutes while the participant reorders the list into its respective categories. It is likely that the list is initially encoded either in a random or serial order and then encoded again elaboratively, with a semantic structure imposed. Thus, in a neural network framework, if this strategy is used then activation must be maintained and refreshed over a considerable period of time until the task is completed. Given the pathology present in PD, this task could prove to be extremely difficult, if not impossible.

In determining the viability of encoding processes in PD participants, a number of memory processes may be involved. As outlined earlier by Kramer et al. (1988), a lack of organisation of material to be encoded may also lead to lower recall overall. However, core encoding processes are shown to be most directly affected in recognition memory, probably because it is not possible to recognise an item which has not been previously encoded. Encoding processes are also thought to be more easily disrupted by interference (Haut & Shutty, 1992).

Verbal Memory

Difficulty in encoding and retrieving verbal information is inextricably associated with the complex organisation of verbal memory itself, within the neural network. In contrast to Broca's aphasia, which is concerned with elementary syntax and synthesis of speech, language disorders resulting from lesions to the prefrontal area, outside of Broca's area, are subtle, often difficult to define and more abstract. Prefrontal lesions appear to produce an impoverishment of speech which contains stereotypical expressions and little creativity. These disorders thus appear of a higher order and, therefore, further up the neural hierarchy (Fuster, 1995). Thus, it could be interpreted that damage to the prefrontal area results in a disruption of the cortical links which serve Wernicke's area and, in turn, to a loss of a certain richness of semantic relations along with a diminished ability to organise new speech plans. This loss results in semantically impoverished speech and a willingness to rely on ready-made and stereotypical expressions.

As numerous studies of Traumatic Brain Injury (TBI) have shown, semantic memory is more resistant to damage than declarative or episodic memory (Squire, 1987; Levin, 1989). It is thought to be anchored in larger neural networks with numerous associations. Widespread posterior damage may be a necessary condition before semantic memory is affected (Fuster, 1995). Thus, the difficulties which people with PD encounter with verbal learning tasks are unlikely to be the result of any localised lesion site. Instead, the deficit more properly resides in the links between the large associative areas of the posterior cortex and the prefrontal cortex. These corticocortical connections are, in turn, thought to be disrupted by the absence of activation in the prefrontal cortex, due to a loss of dopamine receptors, which effectively stops the spread of activation to the posterior cortex.

Semantic memory is robust. The brain has a substantial capacity to compensate for damage incurred as a result of injury or degeneration (Luria, 1973). When several of the systems within the neural network fail, however, the ability to encode and retrieve semantic information is severely compromised.

In conclusion, there is a wide body of evidence supporting the existence of cognitive impairment in PD and in verbal memory processes in particular. Evidence has also been presented to support the involvement of frontal lobe processes in memory tasks and how these processes may be disrupted in PD. An outline of a neural memory network has been presented to suggest ways in which memory processes may break down where PD is present. These include the failure of the neural network to activate and inefficient inhibition mechanisms. While the mechanisms which cause neural networks to activate (and fail to activate, as may be the case in PD) remain unclear, the neural network model goes some way toward explaining why verbal learning deficits in PD may occur. In support of the neural network interpretation, Sawaguchi and Goldman-Rakic (1991) have presented evidence to support dopamine activity as critical to the memory processes undertaken by the prefrontal cortex.

The organisation of semantic memory was discussed with reference to its substantial role in verbal learning tasks. It is likely that deficits in semantic processing in PD are the result of disruptions in corticocortical connections, rather than widespread damage to the posterior cortex.

Many studies of verbal memory in PD have produced disparate findings with respect to recall and recognition performance along with differing interpretations of the memory processes involved. The aim of the present study was firstly, to establish the existence of verbal memory deficits in the PD group under study, and secondly, to explore the nature of the memory processes affected by PD, with emphasis on encoding and retrieval processes.

The Present Study

Background

During the latter part of the 1980's, a number of clinicians observed what appeared to be distinct sub-types of memory deficit among head-injured patients. This lead to a series of experiments to determine whether such sub-types did in fact exist. Most notable of these were the studies carried out by Crosson, Trennery, Novack, and Craig (1989), and Haut and Shutty (1992). They examined verbal learning processes using the CVLT and then analysed the data to ascertain whether distinct sub-groups of memory deficit would emerge.

Crosson et al. (1989) identified one group which was impaired on the number of correct recognition responses and had higher responses that an item was on the list when it was not. Difficulties with semantic organisation and a failure to improve from short to long delay recall trials were also noted. This group benefited from cueing. A further group showed intact recognition and a normal number of false positive responses, improvement from short to long delay trials, but had lower recall overall and was more susceptible to interference. The authors tentatively suggested that these groups represented encoding and retrieval deficits respectively.

In the study conducted by Haut and Shutty (1992) a clustering procedure was employed with a specific hypothesis in mind. The cluster analysis was carried out on the 8 recall trails of the CVLT. The clusters formed from this procedure were then subjected to an analysis of variance (ANOVA) which confirmed that the data conformed to 3 distinct clusters. It was found that participants in cluster 1 showed relatively intact acquisition and delayed recall with no evidence of interference. Those in cluster 2 had problems with both acquisition and delayed recall and were more vulnerable to interference, while participants in cluster 3 had primary acquisition problems but good delayed recall. Haut & Shutty (1992) concluded that the results support the existence of distinct types of memory dysfunction associated with head injury. The profiles of memory performance obtained from the 3 clusters appeared to be related to consolidation, encoding, and retrieval deficits.

Rationale For The Present Study

In reviewing the cognitive changes associated with PD, the existence of cognitive deficits has been well established. More specifically, verbal memory deficits commonly occur in PD. These include deficits in recall and recognition memory, inconsistent recall across trials and lower levels of recall overall, deficient use of semantic memory, high intrusion rates and difficulty in discriminating items previously learned from those that were not. Impaired recall is thought to be associated with retrieval deficits, while impaired recognition appears related to encoding deficits.

On the basis of the results obtained with head-injured patients, the present study attempted to determine whether sub-groups of verbal memory deficit could also be identified in PD. The study sought initially to establish the existence of verbal memory deficits in the PD group and then determine whether sub-groups of memory deficits existed within this group. The memory processes occurring within the PD group were examined to determine the nature of the deficit and whether different memory processes were present in any distinct sub-groups identified.

Reid, Broe et al. (1989) provide evidence that distinct sub-types of PD exist in relation to early or late onset, with late-onset patients appearing to have more serious cognitive decline. In addition, the predominately tremulous form of PD appears more benign in terms of cognitive and motor functioning than the dyskinetic and rigidity-prone form of PD. Thus, there are some precedents for an investigation into the existence of sub-types of memory deficit in PD.

The study was confined to an examination of encoding and retrieval processes in PD. Consolidation processes were not examined, as these impairments in memory processing normally result from deep temporal damage consistent with severe head injury (Squire, 1987). PD patients do not have such extensive brain damage.

In assessing the retrieval processes in PD, it was expected that PD participants with retrieval deficits would show consistently lower recall overall while recognition memory remained intact. PD participants with encoding difficulties were expected to show a reduced ability to organise information into semantic categories, impaired recognition memory and more susceptibility to interference.

The present study followed a format similar to Haut and Shutty (1992) and also used the same questionnaire (CVLT) to assess memory deficits. Use of the CVLT allowed an examination of the separate processes of memory, namely, recall compared to recognition, semantic organisation, delayed memory recall and ability to discriminate correct responses. These processes cannot be readily identified from an examination of the global scores which characterise many measures of memory functioning.

The CVLT has been used in a number of studies which have examined verbal memory performance in PD (Delis, Levin et al., 1987; Kramer et al., 1989; Massman et al., 1990; Buytenhuijs et al., 1994). Thus, the test was considered appropriate for the present examination of verbal memory deficits in PD.

Method

Participants

Twenty-two people with Parkinson's Disease (PD), members of the Kapiti-Horowhenua Parkinsonism Society, and 22 age and gender-matched Controls were recruited for the study. The PD group and the Control group, each consisted of 17 males and 5 females. Where possible, PD participants and Control participants were also matched on socio-economic factors, such as education and occupation. Ages ranged from 48-83, with a mean age of 72 and a *SD* of 7.64.

As an initial step in the recruitment of volunteers, the Kapiti-Horowhenua Parkinsonism Society was approached through the local field officer. The purpose of the study was explained and the assistance of the Society in recruiting volunteers was sought. A brief presentation was then presented to the Parkinsonism Society at the monthly meeting. Information sheets were distributed and a question time and discussion followed. Those interested in participating in the study were asked to supply a contact address and telephone number. A small number wished to take the information sheet away and consider further whether they wished to be involved. This latter group were subsequently contacted by telephone to ascertain if they were interested in participating.

All PD participants, with the exception of 1 younger member, were taking a commercial form of levodopa at the time of the evaluation and were currently stable on their medication. All had either received an initial diagnosis of PD from a neurologist or had the initial diagnosis confirmed by a neurologist. In addition, all PD participants had received a diagnosis of PD at least 1 year prior to evaluation in this study, including the participant who was unmedicated.

Within the age group most affected by PD (65-85 years) it is rare to find a participant who does not have some other health condition. In the present study, 7 PD participants self-reported mild depression. Of these 7, 2 were taking anti-depressant medication. Sleep disturbance was reported by 5 PD participants, 2 in concurrence with depression. Depression and sleep disturbance have been widely reported in conjunction with PD (Dooneief, Mirabello, & Bell, 1992: Koller et al., 1994)

In the PD group, 3 participants had sustained minor head injury involving loss of consciousness. Two of these injuries were related to horse riding incidents which occurred more than 50 years ago, and the other injury related to wartime service in 1940. A further 3 had sustained mild head injury with no loss of consciousness which occurred between 12 and 50 years previously. An additional 2 members of the PD group had sustained a mild stroke 5 and 11 years ago respectively, with no significant loss of functioning. One member had contracted childhood polio without significant ongoing cognitive effects.

These mild neurological conditions were thought to be marginal in terms of their likely contribution to overall memory scores. Thus, it was decided to use the Mini Mental State Examination (MMSE: Folstein, Folstein, & McHugh, 1975) as the basis for cognitive competence and ability to successfully complete the CVLT.

No PD group members nor Control group members were classified as cognitively impaired on the basis of the MMSE screening test. Furthermore, a comparison of Control group and PD group MMSE scores revealed no significant differences. In addition, scores on the CVLT for those people with mild neurological conditions were consistent with performance for their particular age group. Taken together, these results strongly suggest that the mild neurological impairments, which these participants may have suffered from in the past, did not significantly impact on memory performance in the present study.

Control participants were volunteers from the Kapiti Coast area, recruited from a variety of sources. Firstly, the PD volunteers were instrumental in providing matched Controls from their own peer group. The local bridge club, which had many members in a similar age group and whose membership included people with PD, also provided several Control participants. In addition, the Kapiti branch of Age Concern advertised the study and called for volunteers in their newsletter. Friends and neighbors of the researcher also assisted in providing volunteers.

As with the PD participants, Control group participants were screened for alcoholism, psychiatric illness, severe cerebral or cardio-vascular disease, head injury, epilepsy or any other significant neurological disorder. Two Control group members were excluded from the study. One of these had a form of clinical depression and the other had sustained a moderate head injury involving a coma of 12 hours and Post Traumatic Amnesia (PTA) of 3-4 days. The latter participant demonstrated marked vulnerability to proactive interference, recalling only 2 items from List B on the CVLT.

The remaining Control group members were in good general health at the time of assessment and none were taking psychotrophic medication. The PD group was recruited first and Control group members were then matched with the PD group for age and gender. This proved to be a time-consuming task and one which required tactful handling. Many of the Controls who volunteered could not be matched with participants in the PD group. They then had to be advised that participation was not possible.

All participation was on a voluntary basis. No pressure was exerted on any potential participant to become involved in the study. Participants were advised of their rights through the initial information sheet (see appendix A) where they were advised that they may withdraw from the study at any time. Several participants rang the researcher to volunteer their time and others actively sought friends who were possibly willing to participate. Most participants reported that they enjoyed the task and the opportunity for discussion and were happy to recommend the study to friends. The study was conducted in a community setting. All interviews were held at the participant's place of residence.

Table 1 summarises the gender, age, education and occupations of the sample. Each participant in the PD group was age-matched to within one year of the corresponding Control group participant. Where differences existed, they were in favor of the PD group, that is, the Control participant was the older of the matched pair. Note that means for age were identical for both groups. Table 1 also shows that years of education were also well matched across the two groups. Years of secondary and tertiary education were closely aligned between the two groups and approximately half of each of the PD and Control participants had completed some form of tertiary education.

Former occupations were similarly well matched, with few participants in lower socio-economic groups and a large proportion in professional occupations. Males markedly outnumbered females, which is consistent with incidence figures showing a prevalence of PD among men (Lezak, 1995).

Table 1 *Summary Characteristics of Participants*

	PD Group	Control Group
Gender		
Male	17	17
Female	5	5
Age		
Range	48-82	50-83
Mean	72.18	72.18
SD	7.93	7.53
***Education**		
Primary	1	3
Secondary	10	9
Tertiary	11	10
****Occupation**		
Homemaker	1	1
Clerical	1	1
Tradesperson	2	4
Technical	2	5
Professional	12	10
Farmer	1	0
Small Business Owner	2	1
Artist	1	0

* Figures indicate numbers of participants who completed each level.

* *Occupation before retirement - figures indicate numbers in each occupational category.

Ethical Requirements And Confidentiality

The present study required ethical approval from the Massey Human Ethics Committee and the Wellington Hospital Ethics Committee, the latter being affiliated with the health authority responsible for patient care in the PD participants' district. Approval from the Massey Human Ethics Committee was sought first, which facilitated the application to Wellington Hospital Ethics Committee. All names and individual performance scores remained confidential.

Informed Consent

All participants were given an information sheet prior to agreeing to take part in the study (see Appendix A). Separate information sheets were provided for the PD and Control groups, as requested by the ethics committee. The sheet contained a brief outline of what the study involved along with a description of the task which participants would be asked to do if they decided to participate in the study. Participants were also advised of their rights during the study and that they may withdraw from the study at any time. Ample time was given for questions following the participants' examination of the information sheet and at intervals during the study itself.

Once participants indicated that they were interested in participating in the study and had a clear idea of what was involved, they were asked to sign a consent form (see Appendix B). Where possible a witness was also present, this being indicated in Ethics Committee recommendations. This requirement was considered especially pertinent to the PD group. Once informed consent had been obtained, participation in the study began.

Materials

All participants completed the MMSE and the CVLT as part of the evaluation (see appendix C & D)

MMSE

The MMSE is a broad, one-page assessment of cognitive function designed to measure changes in cognitive status (Tombaugh, McDowell, Kristjansson, & Hubley, 1996). The test takes approximately 8-10 mins to administer and is used extensively in clinical settings as a screening measure. The test is broken into sub-sections as follows:

Orientation: Specifically, the participants' ability to orientate themselves in time and place, with questions such as, "What town are we in?" and "What day of the week is it today?".

Registration: Three objects are given (in this case: cup, shoe, chair) and immediate recall is requested. Trials may need to continue until the participant is able to recall all 3 objects accurately.

Attention and Calculation: The participant is requested to count backwards, from 100, in intervals of 7, for example: 93, 86, 79, 72, etc.

Recall: Delayed recall of the 3 objects given above is requested.

Language: A sequence of language tasks is required, including recognition, ability to follow a 3 stage command, carry out a written instruction, write a simple sentence and copy an embedded arrows design.

Level of Consciousness This is assessed along a continuum of "alert", "drowsy", "stupor" or "coma".

The test has a total score of 30, with norms indicating that a score of 24 may be regarded as a benchmark score. Scores below that level are considered to indicate cognitive impairment and scores above 24 to indicate the individual is cognitively intact (Tombaugh et al., 1996).

In the present study, the MMSE was administered as a screening measure, to rule out any possibility of widespread cortical degeneration such as might be expected to occur with dementia of the Alzheimer's type in participants. In addition, the test was employed as a criterion measure to establish cognitive competency to complete the CVLT. Some modification of the MMSE (USA normed) was made to render it more relevant to New Zealand participants. In the orientation section, the questions pertaining to state and hospital were omitted on the basis that they were not relevant to the participants involved in the study. A score of 2.5 points for each correct answer regarding town and country was allotted, giving a total score of 5 for the section, in keeping with the original scoring.

Many of the participants were high-functioning adults and it was evident from the first few minutes of conversation that global impairment was not indicated. It was considered necessary, therefore, to include a brief explanation on the basic nature of the MMSE in the test instructions (see procedure, page 11). Even so, many participants raised a questioning eyebrow when asked, "What day is it?".

Hall (1957) has made the following observation in respect of test questions of a basic nature:

> *"Individuals who, in a hospital setting, are asked to complete items which any child would know, are apt to be either frightened to death or insulted by the implications of the request. Neither reaction is conducive to securing data which are representative of the individual's behavior under more usual circumstances".*

Hall (1957) further argues that a common reaction to this type of questioning is for the participant to conclude that the psychologist thinks they are quite mad. It is important, therefore, to reduce the possibility of this mindset occurring in order to ensure that the tasks which follow fully reflect the participant's ability.

CVLT

The CVLT presents a list of 16 shopping items (list A) which is divided into 4 semantic categories: fruits, tools, herbs and spices (one category) and clothing (see Appendix C). The list is read aloud at the rate of approximately 1 item per s and immediate recall requested over each of 5 learning trials. Following these trials, an interference list (list B) with 4 semantic categories, 2 of which overlap with list A, is presented. Immediate recall of list B is then requested. Free recall of List A is then requested, followed by recall with the semantic categories provided as a prompt.

A 20 min delay follows, after which free recall of List A is again requested, then recall of List A with semantic category prompts. Finally, a recognition test requests participants to identify the original list A from a list of 44 shopping items. Sixteen of these are from list A, 8 are from list B and 20 are distractor and/or prototypical items. For example, "hammer" is presented as prototypical of the tools category.

From the procedure outlined above, a number of parameters of memory can be quantified. The test not only measures levels of total recall and recognition over all trials, but also semantic and serial learning strategies, serial position effects, learning rate across trials and consistency of item recall across trials. The degree of vulnerability to proactive and retroactive interference can also be assessed, as can comparisons of retention over short and long delay conditions. Recognition performance can be compared to recall and, in addition, other factors affecting recognition, namely discrimination and response bias, can be isolated. Patterns of errors made can be examined by numbers of perseverations and intrusions in recall and false positive responses in recognition.

Procedure

MMSE Administration

The procedure began with a brief introduction, during which participants were asked if they had any further questions arising from the information sheet and were asked to sign a consent form (see Appendix B). Following this introduction, the MMSE was introduced as follows:

> *"The first thing we will begin with is this short test. We do this test to rule out any widespread neurological degeneration such as might be present with a condition such as Alzheimer's disease. Now, it's obvious to me in talking with you that you don't have Alzheimer's, but we need to be able to rule it out for our research, otherwise someone could challenge us and say 'how do you know the effects you found are due to Parkinson's disease and not some other cognitive impairment?'. The test is very brief and takes about eight minutes to run through. Some of the questions are very basic, so please don't be offended when I ask you them. Shall we have a go at it?".*

The researcher then administered the test.

CVLT Administration

Before administering the CVLT, the participant was asked a number of questions relating to age, occupation, education level, years since onset of symptoms and medical history. The following instructions were then read out aloud:

> *"Let's suppose you are going shopping on Monday. I'm going to read a list of items for you to buy. Listen carefully, and when I'm through, I want you to say back as many of the items as you can. It doesn't matter what order you say them in, just tell me as many as you can. Are you ready?".*

The researcher then presented the 16 item shopping list, the Monday list. Words were read aloud at the rate of approximately 1 word per s. After each presentation of the list, immediate recall was requested, with these instructions:

> *"Now, tell me as many as you can".*

All responses were recorded verbatim. No information or feedback was given to participants while they were recalling the list. When participants had recalled as many items as they could remember, the researcher continued,

> *"I'm going to repeat Monday's shopping list again. I want you to say back as many items as you can in any order. Be sure to also say the items on the list that you told me the first time."*

Some participants were confused by the last sentence, in which case it was necessary for the researcher to elucidate:

> *"Don't omit the items you told me the first time".*

For trials 3 through 5 a similar instruction was given.

> *"I'm going to repeat Monday's shopping list. Again, I want you to say back as many items as you can, in any order, including items you may have already told me".*

Following these 5 learning trials of List A, list B was introduced, as follows:

> *"Now let's suppose you planned to go shopping again on Tuesday. I'm going to read a **new** list of items for you to buy. When I'm through, I want you to say back as many items as you can in any order".*

In the present study, many participants rolled their eyes in mock horror when informed of the new list, in which case a variant was added:

> *"I'm going to read a **new** list of items for you to buy and you **are** allowed to complain to the ethics committee".*

This generally produced a chuckle and appeared to relieve any tedium the participant may have been experiencing.

When recall of list B was completed the examiner asked:

> *"Now, can you tell me all of the shopping items you can from the Monday list?".*

When the short delay free recall trial was completed the cued trial followed, with the examiner asking:

> *"Can you tell me all of the shopping items from the Monday list that are spices and herbs?".*

Responses were recorded and then the examiner continued:

> *"Now, can you tell me all the items that are tools?".*

The procedure was repeated for the remaining categories of "fruits" and "clothing".

With the conclusion of the cued trial, a 20 min delay was imposed. In the present study, this offered an opportunity to discuss aspects of PD, or memory related to aging. This was generally conducted in a relaxed and informal manner over a cup of tea.

Following this break, the following instructions were given:

"I read some shopping items to you earlier. I'd like you to tell me all the items you can from the Monday list - that was the first list, the one I read to you five times. Go ahead".

At the completion of the long delay free recall trial, the final cued trials were carried out following the same procedure as in the earlier cued trial, thus:

"Tell me all the items from the Monday list that are items of clothing".

This procedure was then repeated for the other 3 categories.

Finally the recognition trial was administered, with the following instructions:

"I'm going to read a list of shopping items. After each item, say 'Yes' if the item was from the Monday list, and say 'No' if it was not".

A 44-word list was then read aloud. The recognition test concluded the administration of the CVLT.

At the conclusion of the interview and testing, participants were given an opportunity to discuss the task. For participants who were discouraged by their performance, some encouragement was provided as well as an attempt to relate performance to a normal age-related decline in memory. The CVLT is well normed and this proved to be beneficial to debriefing. Some participants who felt their performance was poor were surprised to find the norms for their age group compared favorably.

Paper and pencil procedures were used for scoring the MMSE and the CVLT (a computerised version of the CVLT is available). In the planning stage of the study, it was thought that the age group involved, particularly those aged 80-83 years, would be more at ease with procedures which were not highly technical. An attempt was made to provide an uncomplicated and relaxed setting for the study, in keeping with the participant's daily living environment.

No completion time was set for the task. The only time constraints which were operating were the presentation rates for each shopping list item (approximately 1 item per s) and the 20 min delay between short and long delay recall trials. Other trials followed consecutively with no delays imposed.

Hoehn and Yahr's (1967) motor impairment severity stages were not used in this study. The link between severity of cognitive impairment and severity of motor impairment is somewhat tenuous. In fact, some studies have suggested that cognitive impairment is independent of the degree of motor disability present in PD (Brown & Marsden, 1984; Portin & Rinne, 1984; Boller et al., 1984, all cited in Dubois et al., 1991). In addition, it was not considered optimal to extend the testing time of the neurological sample to include a third assessment measure.

Number of years since perceived onset of PD were calculated to provide an estimate of disease severity. It is acknowledged, however, that the relationship between onset and disease severity is a complex one, with late-onset PD patients often showing more severe cognitive deficit Reid, Broe et al., (1989). In the present study, the majority of PD participants were early-onset patients. Of the 22 participants assessed, only 4 showed initial PD symptoms at age 75 or beyond. Of these 4 late-onset participants, 3 were female with mild cognitive effects. The remaining late-onset male participant evidenced severe mobility problems, despite showing only mild cognitive deficits.

Statistical Analysis

All statistical analyses were carried out using the procedures in the Statistical Package for the Social Sciences (SPSS/PC+, SPSS Inc., 1986).

MMSE

Analysis of MMSE scores was relatively straightforward, with a simple *t* test to determine whether the means of the PD and Control groups were equal. In addition, individual scores for both groups were examined against the criterion score of 24, to determine cognitive competence.

CVLT

Initial examination of differences on the CVLT between the PD and Control groups involved the use of *t* tests which evaluated mean differences between the 2 groups on the 20 variables generated by the CVLT sub-tests. The study was interested in ascertaining whether the performance of the PD group would be lower than that of the Control group, reflecting impaired memory performance. To this end, differences in each variable of the CVLT were examined using *t* tests and a 5% significance level. It was not thought appropriate to employ multiple analysis of variance (MANOVA) procedures in the present study, due to the relatively small participant numbers. MANOVA, used with a small sample, lacks sufficient statistical power, increasing the likelihood of type II errors (Hager & Moller, 1986; Huberty & Morris, 1989).

Following the comparison between groups, other factors which may have contributed to CVLT performance were examined. Age and gender as contributing factors in performance were examined using a series of ANOVAS with age as covariate and main effects (factors) for group and gender (SPSS. INC., 1986).

In this section of the analysis and for subsequent sections, the large number of variables generated by the CVLT data were reduced to a set of key variables, which accounted for an estimated 90 percent of performance generated. Differences in serial position effects, intrusion rates and perseverations were very small and they were therefore omitted. The key variables used were those which had demonstrated an acceptable level of reliability and validity when subjected to psychometric analysis (Delis et al., 1987). These variables were significant in Phase I of analysis and also proved to be the most meaningful when subjected to further analysis in Phase II of the present study.

Using the same procedure as used to examine age, differences between male and female performance on the CVLT were also analysed. Sample size was a likely factor in the results achieved with this procedure. The groups consisted of 17 males and only 5 females, thus, the power of the statistical test to reveal any differences was very low.

Cluster Analysis

A hierarchical cluster analysis using Ward's method (SPSS Professional Statistics Manual, 1992) was performed to examine the characteristics of the scores for the PD group. The cluster analysis technique groups scores according to similarity and, in this study, identified homogenous clusters of scores with respect to recall and recognition. Each case in the analysis is described by the scores on the variables generated from the sub-tests of the CVLT. Cases which have scores of similar value are clustered together (Romesburg, 1984; Everitt, 1974).

The clustering process begins by assuming each case is a separate cluster. A new cluster is formed by combining 2 cases according to similarity of scores. The process continues with additional clusters being formed, either by joining a single case to an existing cluster, joining 2 cases into a single cluster or combining multiple clusters, based on similarity of scores. The steps are summarised in an agglomeration schedule which identifies the cases being combined at each stage. This process can theoretically continue until all cases are merged into a single cluster. However, a decision rule is usually employed which stops the process before the increase between 2 adjacent steps becomes too large. At each step, the squared Euclidean distance between the 2 cases being combined is provided. This coefficient is used as a criterion to determine when the increase between steps becomes too large. Large coefficients indicate that clusters with similar membership are being combined (SPSS Professional Statistics Manual, 1992).

The procedure maximises between-cluster variance and minimises within-cluster variance. The optimum number of clusters is generally regarded as the point beyond which within cluster variance exhibits the greatest relative increase as the number of clusters decrease, as indicated by the large increase in the coefficient.

Ward's method refers to the method used to determine the increase between 2 adjacent steps of the clustering procedure. This distance measure determines the number of clusters needed to represent the data. Using Ward's method of cluster analysis, the means for all variables are calculated. The squared Euclidean distance to the cluster mean is then calculated for each case.

Other studies of verbal memory involving clinical populations have also utilised Ward's method of hierarchical cluster analysis. Haut and Shutty (1992) utilised cluster analysis on verbal memory data generated by the CVLT (see Rationale section for further discussion on this study). Tweedy et al. (1982) also used a clustering procedure to examine organisation of free recall by semantic category in PD patients. It was considered appropriate, therefore, to implement the same procedure in the present study, so as to preserve comparability with the results of other similar investigations.

The clustering procedure adopted by Haut and Shutty (1992) clustered only the 8 recall trials of the CVLT (List A trials 1-5, List B, short delay free recall, and long delay free recall) and omitted all cued recall trials, recognition performance variables, semantic clustering scores, intrusions, perseverations and primacy/recency effects. These authors then validated the cluster analysis results with a series of concurrent measures of cognitive functioning, such as the immediate and delayed recall Logical Memory sub-test of the WMS, and digits forward and back, from the WAIS (Wechsler, 1939).

The second phase of the analysis, in which the cluster analysis was run, was regarded as essentially exploratory in nature. Within this context, initial attempts to run the cluster analysis over a wide range of variables ran into difficulty. Recognition and recall variables may be independent, and it appeared that the clustering procedure could not group these dissimilar items according to similarity without dismantling the patterns of performance in recall scores. This problem may also have been encountered in the cluster analysis carried out by Haut and Shutty (1992), which formed part of the rationale for the present study, as only the 8 recall trials of the CVLT were used in their study. In order to group cases which had some consistency in performance, therefore, the main recall and recognition trials of the CVLT only were used in the cluster analysis (see Results section, Phase II). Other aspects of recognition performance, such as false positive responses and discrimination scores were examined separately for each cluster grouping, as was number of years since onset of PD.

Cluster analysis makes no assumptions regarding the nature of the similarities found within clusters. For this study, the cluster analysis merely established that 3 specific groups of participants were similar with respect to scores, but provided no information as to the magnitude of the differences across clusters. Once the 3-cluster solution was obtained, therefore, a series of one-way analyses of variance (ANOVA) were run to confirm which differences between variables were statistically significant. By examining the differences, a profile of verbal memory performance for each cluster emerged.

Psychometric Evaluation

The purpose of this section is to examine the reliability and validity of the measures used in the present study, namely, the CVLT and the MMSE. Statistical information provided with the test manuals is evaluated, and supporting research conducted by the tests authors, as well as independent studies using the measures, are examined.

MMSE

The MMSE was developed in response to the need for a simple test of cognitive functioning which could be administered quickly. It has widespread use in clinical and research applications and is frequently used as a screening test for dementia. The test was not designed to constitute a definitive diagnoses of dementia, however, and should not be used for this purpose. The test authors caution that an accurate diagnosis depends on evidence developed from the psychiatric history of the patient, a full mental status examination, physical status and relevant laboratory data. A low score on the MMSE should not be taken as an indication of dementia, but rather that further investigation is required (Folstein et al., 1975; Tombaugh et al., 1996).

The test has a straightforward application. Administration and scoring are standardised with respect to allocation of points. The test is brief in consideration of the elderly population it generally encompasses. Initially developed 22 years ago by Folstein et al. (1975), the test is somewhat meagre in its original norms. Sixty three control participants with a mean age of 73.9 comprised the standardised population.

Anthony, LeResche, Niaz, Von Korff, and Folstein (1982) conducted a follow-up study for the purpose of studying a larger sample, examining the scores of a further 97 patients. Using a cut-off score of 23/24, the MMSE had a sensitivity of 87% and a specificity of 82% in detecting delirium or dementia when judged against a research psychiatrist's standardised clinical diagnosis of these disorders. Sensitivity refers to the ability of the test to detect these disorders, while specificity refers to the ability of the test to classify as non-cases, individuals with no delirium or dementia. The MMSE showed a test-retest reliability of .85 for the 58 participants judged to be without dementia, .90 for 12 participants judged to have dementia and .56 for 7 participants with delirium. Intuitively, one would expect participants with delirium to show a wide variance in scores, thus, test-retest reliability for this group would not be high.

Tombaugh and McIntyre (1992), in a recent review, concluded that the reliability coefficients of the MMSE were moderate to high, that the test was sensitive to cognitive deficits in moderate to severe cases of Alzheimer's disease and showed the cognitive decline typically seen in dementia patients. Criticisms of the test included its failure to distinguish between mild cases of dementia and no dementia, and limited ability to detect focal lesions, particularly in the right hemisphere.

Tombaugh and McIntyre (1992), in a meta-analysis of approximately 30 studies which used the MMSE, concluded that reliability and construct validity were satisfactory. They noted, however, that the MMSE was highly verbal and not all items were equally sensitive to cognitive impairment. In conclusion, it appears reliability of the MMSE is generally high in clinical populations of people with psychiatric and neurological disorders. Folstein et al. (1975) and Folstein and McHugh (1979) report that test-retest reliability has not fallen below .89 and inter-rater reliability has not fallen below .82 (both cited in Anthony et al., 1982).

Caution is needed, however, when interpreting results for participants with a (US) grade 8 education or less (Anthony et al., 1982). Specificity of the test for these participants was a low 63.3% compared to 100% for those with more years of education. These limitations need to be born in mind when administering the test. The need to match participants for age, gender and education is highlighted by these results. It is possible that the effects of low levels of education can either mask or duplicate symptoms of dementia.

The MMSE was considered a suitable screening measure for the present study, despite its limitations. As the sample was carefully matched for age and gender and closely matched for education, many of the shortcomings of the MMSE were circumvented. There were no borderline cases within the 2 groups, with all participants scoring well above the 23/24 cutoff point generally regarded to indicate cognitive impairment. In addition, the majority of participants were from professional or technical occupations, with tertiary qualifications. Thus, the problems with low-education level groups identified in other studies were not apparent here. In addition, as the main assessment tool in the study was a test of verbal memory (CVLT), the verbal bias of the MMSE was not considered to be a confounding factor.

The California Verbal Learning Test

With reference to memory functioning, it has long been thought that verbal memory is a complex cognitive function consisting of multiple component processes and parameters (Underwood, 1948; Tulving, 1966; Atkinson & Shiffron, 1971, all cited in Delis et al., 1987). However, many current tests of memory provide only total recall scores which offer no contrast between recall and recognition and little insight into what strategies may have been used to achieve such scores. Thus, the infrastructure of the memory processes which occur when testing verbal memory has been largely ignored.

A major impetus in the development of the CVLT has been research evidence suggesting that anterograde amnesia is not a unitary disorder, as previously thought, but one which can differ across patients depending on which part of the brain's component functions have been damaged (Butters & Miliotis, 1984; Mayes, 1986; Squire, 1987). Kaplan (1983, cited in Delis, Freeland, Kramer, & Kaplan, 1988), has demonstrated that brain damaged patients with different cognitive deficits may achieve the same total scores on a range of neuropsychological tests. The scores, however, tell us very little about the nature of the deficit or which processes may have been spared.

Thus, the CVLT was developed in response to calls for clinical tests that provide information about how tasks are solved, as well as providing overall achievement levels. It allows measurement of a number of memory processes, including semantic clustering in recall and recognition, primacy-recency effects, learning rate across trials, recall consistency, susceptibility to interference, retention of information over short and long delays, patterns of errors made and discrimination in recognition. In examining CVLT scores, impairments in the underlying processes which contribute to memory functioning may be identified.

Norms

The CVLT normative data are based on the scores of a reference group of 273 neurologically intact adults. This group had a mean age of 58.93 with a SD of 15.35 and a mean educational level of 13.83 years with a SD of 2.70. Normative participants were screened for any history of neurological or psychiatric disorder or other health problems known to affect memory functioning. Raw data were smoothed using multiple regression as the normative group consisted of several independently collected samples.

Age and gender effects were examined by means of a hierarchical multiple regression analysis. This procedure highlighted the need to control for age and gender differences when applying the CVLT to clinical populations. Gender was a significant predictor of 13 of the 22 variables (including all recall but not recognition trials). Age significantly predicted only 2 of the variables, list A total, trials 1-5, and recall consistency. SDs increased significantly with age on 10 of the variables, indicating a wider variance of scores among older participants. This should be taken into account when evaluating normal performance in older participants.

The CVLT, however, is one of the few memory tests which provide normative data for older age groups, presumably because the problems of increased variance with age are endemic across a broad spectrum of tests. The WMS (Wechsler, 1987) and the Memory Assessment Scale (Williams, 1991) are not normed beyond age 75, while the CVLT incorporates norms up to and including 80 years. In short, it appears that the authors consider norms with increasing variance are preferable to no norms at all.

Mapou (1995), in a review of memory tests, concludes that the normative database of the CVLT is better than other tests of verbal learning, such as the Rey Auditory Verbal Learning Test (Rey, 1964) or the Selective Reminding Test (Buschke, 1973). Similarly, Lezak (1995) concludes that the norms provided for the CVLT are detailed, and that studies undertaken to examine the reliability and validity of the CVLT support the conceptual framework of the test.

Reliability

Internal reliability was assessed using 133 participants from the normal reference group used to establish normative data. The study used 51 males and 82 females with a mean age of 60.89 (SD of 7.80). This group reflected a relatively narrow age range and provided a homogenous group, which was considered to be a stringent test of internal consistency. Homogenous groups tend to produce underestimations of the internal consistency of a test (Delis et al., 1987). The internal consistency reliability coefficients are summarised in Table 2.

Table 2 *Internal Consistency Reliability Coefficients and Standard Errors of Measurement (SEm) for List A Total Score, Trials 1-5*

Variables	Method	Reliability	SEm
Five trial scores	Odd-even	.92	2.61
Semantic categories	Split-half	.77	4.42
	Alpha	.74	4.72
Item totals across 5 trials (a)	Odd-even	.70	5.07
	Alpha	.69	5.21
Item totals across 5 trials (b)	Odd-even	.85	5.62
	Alpha	.86	5.43

(a) Nonclinical sample (N=133)
(b) Mixed clinical and nonclinical sample (N=78)
Note: From "The California Verbal Learning Test Manual" (p. 39) by Delis, Kramer, Kaplan, and Ober, 1987, New York, The Psychological Corporation.

An analysis of total scores for trials 1-5, using a split half correlation with a Spearman-Brown formula applied to the average of the correlations, yielded a reliability coefficient of .92. The split half technique compared trials 1-3 with trials 2-4 and trials 2-4 with trials 3-5. A second reliability coefficient was obtained by examining total learning trial scores on 2 independent halves of the test. Each half-test contained 8 words, representing 4 words each from 2 categories. These categories were paired in order to balance average scores, with "fruits" and "tools" making 1 pair and "clothing" and "spices and herbs" making up the other.

In this way a measure of consistency was obtained across different sets which were semantically unrelated. This procedure gave an estimated total test reliability of .77. Coefficient alpha based on the 4 category scores was .74. The correlation between total scores on odd-numbered and even-numbered words was obtained For the relatively homogenous normal sample a coefficient alpha of .69 was achieved, with an estimated test-retest reliability of .70. This procedure was also carried out on a mixed clinical and non-clinical sample yielding a coefficient of .86 and an estimated test-retest reliability of .85. The authors note that the internal consistency is higher for this more heterogeneous mixed group. In conclusion, taken together, the coefficient alphas shown in Table 2 appear reasonably high and establish the internal consistency reliability of the CVLT.

However, the test authors note that there are special problems when estimating the internal consistency of recall items in memory tests. Because memory capacity is limited, recalling any one word on a given trial decreases the likelihood of any other word being recalled on the same trial. Thus, there is item-interdependence within trials. Secondly, the fact that a given item is recalled on one trial increases the likelihood of it being recalled on another. Therefore, there is item interdependence across trials. For this reason, total trial scores were used in the reliability calculations to minimise, but not totally eliminate, the effects of item interdependence. In this context, it is also not meaningful to examine covariance of memory test items, since they are, by definition, highly interdependent.

Test-Retest Reliability

In neuropsychological testing, repeated assessments over time are often necessary to determine changes in cognitive functioning. Accordingly, an alternate form of the CVLT, known as form II, was developed, providing a further measure of test-retest reliability (Delis, Massman, Kaplan, McKee, Kramer, & Gettman, 1991). Forms I and II were administered in counterbalanced order to 41 normal volunteers with no history of neurological injury or disease. The test-retest interval of an average 8 days was somewhat narrow. While alternate forms were used, thus decreasing practice effects, the short time interval could still have resulted in the retention of learning strategies.

Reliability coefficients were somewhat borderline for 13 of the 19 CVLT variables. All recall and recognition trials, with the exception of list A trial 1, showed coefficient alphas ranging between .62 and .84. Primacy and recency recall, learning slope, list B recall, recall consistency, intrusions and perseverations all produced a coefficient alpha below .50. However, list A total trials 1-5, an important indicator variable (Delis et al., 1987), produced a coefficient alpha of .84. Five other variables also yielded coefficients between .70 and .85, including list A trial 5, short delay free recall, long delay free recall, long delay cued recall, and semantic cluster ratio. These latter 6 variables were statistically adequate. The recognition hits coefficient at .67 was also low. It is possible that some practice effects may have been operating in the test-retest conditions. Difficulties with practice effects are encountered frequently in memory testing validation. Wechsler (1987) concedes that there are practice effects operating in the data put forward to support the test-retest reliability of the WMS. Overall, however, reliability of the CVLT has been well established by the supporting research examined in this section.

Validity

The construct validity of the CVLT has been examined by 3 separate methods. Firstly, the CVLT variables were subjected to factor analysis and, secondly, the variables were correlated with the WMS (Wechsler, 1987). Thirdly, the CVLT was administered to a series of well-defined clinical samples to provide information on the profiles obtained for each clinical group.

Delis, Freeland et al. (1988) conducted a factor analysis to determine whether the CVLT variables clustered into linearly independent domains or whether they were simply redundant measures of a single learning factor. The study examined the scores of 399 individuals, 286 neurologically intact participants and 113 neurological patients. This latter group was made up of 55 patients with multiple sclerosis, 8 with Huntington's disease, 24 with chronic alcoholism and 26 with Parkinson's disease.

A 6 factor solution emerged from the analysis. The first factor was interpreted as a general verbal learning factor consistent with other studies of memory tests. Secondly, a response discrimination factor was isolated, which reflected an ability to determine relevant from irrelevant responses and was considered to be a primary mechanism related to memory failure. Learning strategy was the dimension considered to underlie factor 3, with loadings on semantic and serial clustering which were inversely related. Semantic clustering also loaded significantly on the general verbal learning factor, while serial recall did not, suggesting that semantic clustering is a more effective learning strategy than serial recall.

The fourth factor was interpreted as Proactive Effect. List B recall and list B versus list A trial 1 recall were the only 2 variables which loaded on this factor. This finding is consistent with research showing that proactive interference is a fundamental aspect of normal learning (Underwood, 1974). The fifth factor was termed Serial Position Effect. Percent primary recall and percent recency recall loaded inversely on this factor. This inverse loading was interpreted as providing further weight to the theory that greater primary recall reflects a more active learning strategy whereas greater recency recall reflects a more passive learning approach (Posner, 1964; Detterman & Ellis, 1970; both cited in Delis, Freeland et al., 1988). The sixth factor was defined as Acquisition Rate.

The learning slope variable loaded on this factor, reflecting individual differences in learning patterns. The test authors concluded that a number of theoretically meaningful factors underlie verbal memory performance as measured by the CVLT, which substantiates the construct validity of the test.

Criterion-Related Validity

In an examination of criterion-related validity, Delis, Cullum, Butters, & Cairns (1988) administered the CVLT and WMS to 105 participants. Of these, 55 were brain damaged patients, 25 were substance abuse patients and 25 were heterogeneous psychiatric patients. The 2 tests were administered on the same or successive days and the order was varied. The WMS was expanded to include a 20 min delay condition for the logical memory stories, visual reproduction designs and verbal associate learning in order that scores could be directly compared with the CVLT. Table 3 presents a summary table of key correlations of CVLT variables with WMS scores.

Table 3 *Correlations of Key CVLT Variables with WMS Scores*

CVLT Variables	Mental Control	Memory Quotient	Logical Memory Immediate	Logic Memory Delayed
List A Total Recall	.66	.66	.56	.46
List B	.49	.52	.37	.35
Short Delay Free	.47	.43	.45	.40
Short Delay Cued	.50	.47	.46	.41
Long Delay Free	.57	.53	.55	.41
Long Delay Cued	.54	.47	.52	.43
Semantic Cluster	.38	.49	.47	.20
Recognition Hits	.56	.51	.50	.56

Note: From Wechsler Memory Scale-Revised and the California Verbal Learning Test: Convergence and divergence, by D. C. Delis, C. M . Cullum, N. Butters and P. Cairns. (1988). *The Clinical Neuropsychologist*, 2, 188-196.

List A correlations with mental control and logical memory immediate are modest, but appear to substantiate the CVLT general verbal learning factor identified in the factor analysis. The semantic clustering score of the CVLT also correlates positively with the WMS while serial clustering does not. This adds further support to the interpretation made earlier, also in relation to factor analysis data, that semantic clustering is a more effective strategy than serial clustering. Overall, however, the correlations generated by the analysis were too low to be conclusive.

This does not necessarily call into question the validity of the CVLT *per se*. While the WMS has a demonstrated verbal bias (Zielinski, 1993), it also incorporates many aspects of visual memory. Thus, the WMS is not directly comparable to the CVLT, which is a specific test of verbal memory. In addition, the reliability of the WMS itself does not bear up well under close scrutiny, and reliability is a necessary condition for validity. A sample of the WMS reliability coefficients, for the age-group 55-64, is presented in Table 4. The numbers are illustrative of the above points.

Table 4 *Reliability Coefficients for the WMS-R*

Variable	r
Mental Control	.50
Figural Memory	.27
Logical Memory I	.67
Logical Memory II	.55
Verbal Paired Assoc. II	.46
Verbal Memory	.69
Digit Span	.86
Visual Memory Span	.83

Note: From "Wechsler Memory Scale-Revised Manual, by D. Wechsler, (1987). San Antonio, TX: Psychological Corporation.

While digit span and visual memory span coefficients are within an acceptable range, many of the coefficients relating to verbal memory performance are poor. Thus, while the 2 tests may be comparable on general memory items, comparisons between specific sub-tests cannot be made with confidence.

In a third examination of construct validity, profiles for selected clinical groups have been provided (Delis et al., 1987). Distinctive profiles emerged for Alcoholism, and Alzheimer's disease, and these were, in turn, distinct from Parkinson's Disease and Huntington's Disease which together showed a similar profile. In addition, Delis et al., (1991) examined the CVLT profiles of Alzheimer, Huntington and Korsakoff patients. The study was specifically interested in examining whether the CVLT profiles of these clinical groups were consistent with findings of other research which described the characteristics of these disorders.

The Alzheimer and Korsakoff patients showed no significant differences on the 20 CVLT variables analysed. Both groups showed severely impaired immediate recall, flat learning rates and inconsistent recall across trials, ineffective use of semantic clustering, a tendency to use a passive recall strategy, poor retention over delay intervals, high rates of intrusions and false positives, poor recognition discriminability, a tendency toward yes responses and no improvement on recognition compared to free recall (suggesting difficulties predominantly with encoding and storage processes).

These findings are consistent with studies which have investigated the explicit memory functioning of these groups (Butters, 1985; Kopelman, 1987; Salmon & Butters, 1987; all cited in Delis et al., 1991). The Huntington group also showed patterns of impaired recall consistent with that found in other studies (Caine, Ebert, & Weingartner, 1977; Butters, 1985, 1986; all cited in Delis et al., 1991). However, this group also displayed better retention on delayed recall, lower intrusions and false positives, less vulnerability to interference and more intact recognition.

In addition, analysis of the CVLT provides normative data on error types. The pattern of high rates of intrusions in recall and false positives in recognition is characteristic of patients with mesial temporal-diencephalic damage, such as occurs with Alzheimer's disease and Korsakoff's syndrome. Perseverative errors are also important indicators of deficits in frontal lobe functioning (Luria, 1981, cited in Delis et al., 1991).

The test authors conclude that the CVLT is useful in distinguishing memory disorders associated with a prototypical subcortical dementia, such as Huntington's disease, from a prototypical cortical dementia such as Alzheimer's disease. In summary, they further state that by assessing recall only, the severity of the amnesia in patients with primarily subcortical disorders such as Huntington's and Parkinson's diseases is likely to be exaggerated. Conversely, assessing recognition only is likely to minimise the deficit in subcortical patients. Both recall and recognition should be assessed concurrently. In doing this, the authors maintain that the retrieval deficits prominent in subcortical patients and the encoding and the storage impairment of mesial temporal-diencephalic patients can be identified.

Results

Results for the PD and Control groups were analysed in 2 phases. In the first phase, group differences between the PD and the Control groups were examined on all measures of the CVLT. The possible contribution made by age and gender factors was also examined. In the second phase, the PD group was analysed to explore the processes underlying performance in this group.

MMSE

Individual scores on the MMSE were examined to determine whether each participant was cognitively competent to participate in the study. All participants scored better than the minimum cut-off score (>24). Accordingly, the results of all participants were used in the subsequent analyses. In addition, differences in MMSE means for the PD group and the Control group were examined to determine whether the 2 groups were equal with respect to scores on the test. Scores were closely aligned, with only a 0.5 point difference between the means of the 2 groups (see Table 5). This difference was not significant, $t(42) = 1.60$, $p > .05$.

Table 5 *MMSE Total Scores: Group Means and Standard Deviations*

	M	*SD*	*t*
PD Group	28.59	1.22	1.60
Control Group	29.09	0.81	

CVLT: Phase I

Results from the CVLT were firstly analysed by examining between-group differences, using ***t*** tests. The results are presented in Table 6.

Table 6 *Group Means, Standard Deviations and t Values for CVLT Scores*

CVLT Trials	PD Group		Control Group		*t*
	MEAN	*SD*	*MEAN*	*SD*	*t*
List A Total	36.81	8.12	50.00	11.41	4.42 **
List A Trial 1	4.45	1.47	6.41	1.99	3.70 **
List A Trial 5	9.31	2.06	12.54	2.46	4.72 **
List B	4.04	1.53	5.91	1.79	3.71 **
Short Delay Free Recall	6.68	2.46	10.22	3.23	4.09 **
Short Delay Cued Recall	9.00	2.72	11.27	2.97	2.64 **
Long Delay Free Recall	7.59	3.03	10.50	3.23	3.08 **
Long Delay Cued Recall	9.00	2.64	11.41	3.03	2.81 **
Semantic Category Score	7.36	4.45	16.36	11.03	3.55 **
Serial Recall Score	3.00	1.85	4.31	2.33	2.07 *
Percent Correct Primacy Region	10.90	3.26	14.68	2.59	4.25 **
Percent Correct Middle Region	15.27	6.29	21.82	7.59	3.11 **
Percent Correct Recency Region	10.59	4.01	13.50	4.10	2.38 *
Perseverations	2.64	2.85	2.77	2.27	0.18
Free Recall Intrusions	2.68	3.03	0.86	1.55	2.51 **
Cued Recall Intrusions	2.41	2.15	1.23	3.04	1.49
Recognition Hits	13.86	1.28	14.59	1.47	1.75
False Positives	3.55	2.97	1.45	1.99	2.74 **
Discrimination	87.08	7.47	93.48	6.70	2.99 **
Response Bias	0.06	0.59	-0.05	0.50	0.72

* $p = < .05$
** $p = < .01$

Table 6 shows the means and standard deviations for both PD and Control groups. It is notable that means for all recall trials, list A total (a composite score of trials 1-5), List A trial 1, List A trial 5, List B, short delay free recall, short delay cued recall, long delay free recall and long delay cued recall, are lower in the PD Group. This result indicated a general impairment in recall overall. (All differences described below were statistically significant, unless otherwise indicated.) List A total score, regarded as an important indicator variable (Delis et al., 1987), was markedly higher for the controls. Similarly, the list A trial 5 score was considerably higher in the Control group. List A short delay free recall scores were also strikingly divergent. Likewise, the mean long delay free recall trials were higher for the Control group, although these differences were not as marked as those obtained with the short delay condition.

Both PD and Control groups showed a wide range of scores for list A total, with scores ranging from 21 to 52 in the PD group (SD 8.12) and 30 to 72 (SD 11.41) in the Control group. In the PD group, a comparison between younger and older participants accounts for much of this variance. Younger PD participants, aged between 48 and 74, showed a mean of 37.60 with a SD of 11.14 for list A total scores, which ranged from 21 to 52 in this younger group. Such heterogeneity of scores is not atypical of performance within this clinical group. Of these younger PD participants, those aged 62-68 tended to produce scores comparable to the norms for this age group (Delis et al., 1987), while those aged 69-74 showed a marked decline in performance. Hence, the wide variation in scores. Older PD participants (75-83), in contrast, showed little variance in scores on list A total. Scores were consistently low, ranging between 30-43, with a mean of 36.16 and SD of 4.80.

In the Control group, however, both young and older members showed a large SD on list A total scores and this variance did not appear to be explained by age factors. A correspondingly high variance was obtained on semantic category scores, with both younger and older Control group members showing SD's of 12.06 and 10.02 respectively.

Thus, the results showed a wide variation in this group's ability to utilise semantic categorisation with a corresponding wide range in total scores achieved on the learning trials. The relationship between scores on recall trials and semantic category scores is examined further below.

Both groups appeared to benefit from the provision of external cues in the long delay cued condition, where categories were provided as prompts. The PD group moved from a mean of 7.59 in the long delay free recall trials to a mean of 9.00 in the long delay cued recall trials. The Control group also showed a modest increase from a mean of 10.50 in the long delay free recall trials to a mean of 11.41 in the cued recall condition. Overall, the results obtained from all recall trials of the CVLT indicated that the performance of PD participants showed clear deficits in verbal memory recall when compared to that of the Control group

Differences in position effects were examined by calculating the number of items correctly recalled from the beginning, middle and end of list A, referred to in the CVLT as the primacy, middle and recency regions. While the level of recall was markedly lower in the PD group when compared to Controls, the PD group did not show a tendency to favor items from either the beginning or end of the list. Means for both regions were comparable (M = 10.90 and 10.59, respectively). The PD group, however, recalled fewer items from the middle of the list than the Control group.

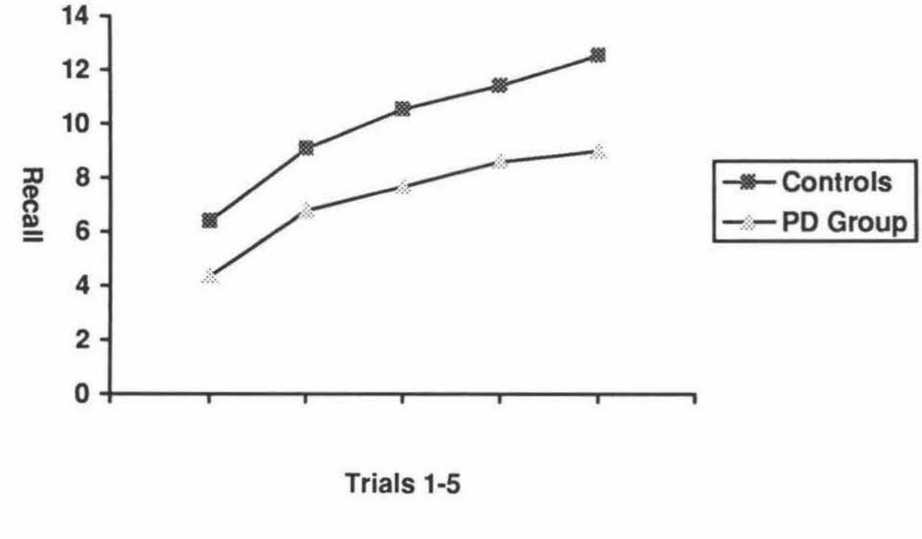
The difference in semantic category scores between the 2 groups was highly significant. This score measured ability to group items on the list into the 4 semantic categories. A direct correlation was observed in both groups between the number of semantic clusters used during recall and scores obtained on list A, trials 1-5. Greater use of semantic clusters as a strategy resulted in higher total scores (Pearson's r = .82). Use of semantic categories was significantly higher in the Control group, which contributed to the higher recall overall for this group.

The difference between serial recall scores was also significant, though not as marked as that of semantic category scores. Serial recall scores were low in absolute terms for both groups, as indicated by the means of 3.00 and 4.31 serial clusters formed over the 5 learning trials for the PD and Control groups, respectively.

Recognition hit scores showed only minor variation between the 2 groups, with a mean of 13.86 items for the PD group compared with 14.59 for the Control group. However, it is notable that the direction of the difference is in line with the overall profile for recall measures, with lower recognition hits for the PD group. Response bias, which measures a tendency to respond "yes" or "no" on recognition test items, varied marginally either side of a neutral response tendency (see Table 6). The difference was not significant. However, there were distinct differences in discrimination and false positive responses between the 2 groups. Discrimination, which measures ability to distinguish target items from distractor items, is expressed as an index score between 1-100. False positives are responses of "yes" on distractor items, that is, responding that an item was on the list when it was not. These results indicate that, while the differences between recognition hit scores for the 2 groups was not significant, the group's ability to distinguish correct responses was affected. This is reflected in the higher number of false positive responses of the PD group, which is also a component of the discrimination index score. In other words, the significant difference in recognition (discrimination) scores between the 2 groups was principally due to a difference in false positive rates, with PD participants showing the higher rate.

The means for list A trials 1-5 were also calculated to examine differences in learning patterns. These are presented in Figure 2, and show the poorer performance overall of the PD group.

Figure 2 *Mean Recall of List A, Trials 1-5, for PD and Control Groups*



Visual inspection of the 2 plots shows that the PD group may have been slower to learn. However, while the learning curve appears marginally flatter for this group, differences in learning rate were not marked.

In summary, scores for the PD group overall were consistently lower than those of the Control group. Taken together, these results show that a general impairment in verbal memory recall was present for the PD group. Participants who engaged in a semantic recall strategy tended to produce higher overall scores and this effect was notably higher in the Control group. Participants in the PD group showed a distinct decrease in the number of items recalled from the middle of the list when compared with the Control group. In addition, some aspects of recognition were affected, in that the discrimination measure was affected by an increased false positive rate in the PD group. The 2 groups did not differ in regard to hit rate.

Age Factors

As outlined in the Method section, participants in the present study were matched for age and gender in an attempt to rule out these factors as confounding variables in the analysis. However, this matching did not exclude age and gender from having a possible influence on the performance of participants within the PD and Control groups. As PD is a progressive, neurodegenerative condition, intuitively, we would expect the process of aging to assist in the progression of the disease. In addition, this progression may vary qualitatively between males and females. Therefore, it was considered necessary to examine these factors in the present study.

In order to establish whether age was a factor in the results obtained for the PD group, the group was split into 2 groups. A younger group comprised PD participants aged 63-74 and the older group were aged between 75-82. A series of one-way ANOVAS were calculated for the 2 groups. The results of these tests on age for the main recall and recognition trials of the CVLT, are presented in Table 7. The main recall and recognition trials of the CVLT were used in the age-group comparisons instead of the full number of variables, as these trials have proved to be the most consistent and reliable predictors of performance over time (Delis et al., 1987).

Table 7 *Anovas for CVLT Variables: Young and Older PD Participants*

CVLT Trials	**Young Mean**	**Older Mean**	*SS*	*Df*	*F*	*P*
List A Total	37.60	36.16	156.40	1	2.92	.10
List A Trial 1	4.70	4.25	2.69	1	1.44	.24
List A Trial 5	9.30	9.33	7.65	1	1.90	.18
List B	4.00	4.08	4.57	1	2.06	.17
Short Delay Free Recall	7.10	6.33	11.81	1	2.53	.13
Short Delay Cued Recall	9.20	8.83	20.74	1	3.52	.07
Long Delay Free Recall	7.60	7.58	20.34	1	2.43	.14
Long Delay Cued Recall	9.30	8.75	13.74	1	2.37	.14
Semantic Category Score	7.40	7.33	28.89	1	1.50	.23
Recognition Hits	14.40	13.41	.84	1	.56	.46
False Positives	4.00	3.16	25.09	1	3.01	.09
Discrimination	87.26	86.35	90.24	1	1.75	.20

No significant effect for age was found on any of the recall and recognition trials of the CVLT. A large SS for list A total scores, at 156.40 was noted. As list A total is a composite score, incorporating recall from trials 1-5, some variance is to be expected. Group numbers were small, with 10 participants in the younger PD group and 12 participants in the older PD group. Statistical power in examining these groups was therefore low.

Gender Differences

A further comparison was made of differences in scores between males and females in the PD group. The sample contained a majority of males, with 17 males and 5 females with PD taking part in the study, consistent with prevalence rates in the general population. Estimates vary from 1.36:1 to 3.7:1, in favor of males. (Diamond et al., 1990). These male and female PD participants were matched for gender in the Control group, giving a total of 34 males and 10 females who took part in the study. Using the same procedure which was applied to the age-group comparisons, gender differences were examined using ANOVA. The results are presented in Table 8.

Table 8 *ANOVAS for CVLT Variables by Gender in the PD Group*

CVLT Trials	Male Mean	Female Mean	*SS*	Df	*F*	*P*
List A Total	36.52	37.80	70.10	1	1.21	.28
List A Trial 1	4.41	4.60	2.11	1	1.11	.30
List A Trial 5	9.17	9.80	4.67	1	1.12	.30
List B	4.11	3.80	.01	1	.00	.96
Short Delay Free Recall	6.52	7.20	11.70	1	2.50	.13
Short Delay Cued Recall	8.94	9.20	5.93	1	.89	.35
Long Delay Free Recall	7.47	8.00	6.65	1	.73	.40
Long Delay Cued Recall	8.84	9.60	12.07	1	2.05	.17
Semantic Category Score	6.82	9.20	46.43	1	2.54	.13
Recognition Hits	13.94	13.60	.02	1	.01	.90
False Positives	3.88	2.40	14.01	1	1.58	.22
Discrimination	86.49	88.06	38.22	1	.70	.41

As Table 8 illustrates, no significant differences based on gender were found in any of the recall and recognition trials of the CVLT. As the sample size was small, with only 5 females participating in the study, statistical power was also low in this analysis.

While the ANOVA results were not generally significant for differences in gender, scores when taken as a whole, do identify an interaction. Females, on average, were 7 years older than males in the present study, and despite the older age, females scored modestly higher on the majority of list A recall trials and had fewer false positives in the recognition test.

In summary, no significant effects were found for age or gender in the PD group, although some strong trends were evident, particularly for gender.

Phase II

The analysis carried out in Phase I showed that the PD group's scores overall on the CVLT were markedly lower than those in the Control group. However, while age and gender have been examined for possible contributions to the memory deficits observed in the PD group, the memory processes responsible for the lower performance of this group have not been identified. Therefore, a further analysis was carried out to explore the underlying memory processes in the PD group's performance. This exploration was centered on the memory mechanisms involved in encoding and retrieval processes.

A hierarchical cluster analysis (Ward's method: SPSS, Inc.1986) was performed using score similarity as the basis for clustering. From this analysis, described in the Method section, 3 distinct clusters emerged which appeared to best represent the data. Using the decision rules discussed in the Method section, these clusters appeared to reveal homogenous groups of participants with respect to recall across trials. Cluster 1 contained 10 cases, and cluster 2 and 3 comprised 6 cases each. Only the scores on the main recall and recognition trials of the CVLT were examined for each cluster, as discussed in the Method section. A profile of cluster membership emerged from the examination of means and standard deviations, as Table 9 illustrates.

Table 9 *Means of PD Group Performance In Clusters 1, 2 and 3*

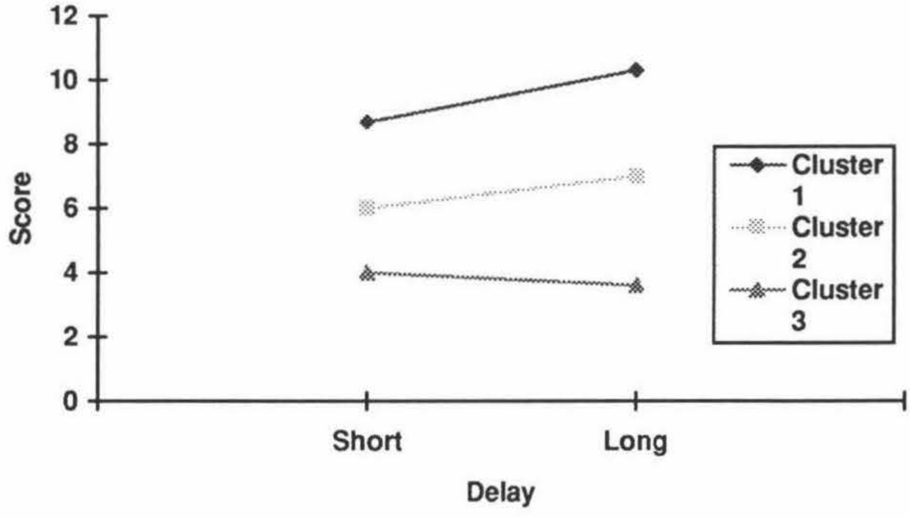
CVLT TRIALS	CLUSTER 1		CLUSTER 2		CLUSTER 3	
	MEAN	*SD*	*MEAN*	*SD*	*MEAN*	*SD*
Age	70.00	10.46	72.83	5.52	75.16	3.97
List A Total	43.70	5.12	34.83	3.06	27.33	3.55
List A Trial 1	5.30	1.25	4.33	1.50	3.16	0.75
List A Trial 5	10.80	1.62	9.00	1.41	7.16	0.98
List B	4.00	1.49	4.33	1.86	3.83	1.47
Short Delay Free Recall	8.70	1.88	6.00	1.09	4.00	0.63
Short Delay Cued Recall	11.00	1.56	8.83	1.94	5.83	1.72
Long Delay Free Recall	10.30	1.41	7.00	0.63	3.66	1.03
Long Delay Cued Recall	11.10	1.28	8.83	1.17	5.67	1.63
Semantic Category Score	10.80	3.91	6.00	1.79	3.00	1.90
Recognition Hits	14.40	0.52	14.33	1.36	12.50	1.23

Age did not appear to be uniform across cluster membership. Participants in cluster 1 (mean age of 70.00 years), were generally younger than those in cluster 2. Cluster 2 participants (mean age of 72.83), were younger than those in cluster 3 (mean age of 75.16). There were exceptions to the general trend, however, with 2 participants aged 82 in cluster 1. These exceptions also accounted for the relatively large SD of 10.46 for this group. These older members of cluster 1 were high-functioning individuals whose scores were markedly higher than the mean for the PD group as a whole. Participants in clusters 2 and 3, however, showed more uniformity with respect to age as can be seen from the standard deviations of 5.52 for cluster 2 and 3.97 for cluster 3.

Performance scores on all recall and recognition trials were highest for cluster 1. Cluster 2, in turn, was higher than cluster 3. The performance on list A total, which represents the total of the 5 learning trials, highlights the superior recall of participants in cluster 1. These participants scored a mean of 43.70 compared with 34.83 for cluster 2 and 27.33 for cluster 3.

The long delay free recall trials were also illustrative of this trend toward higher performance in cluster 1, with scores for this group showing a mean of 10.30. Cluster 2 showed a mean of 7.00 and cluster 3, a mean of just 3.66. These results for the long delay trials are evident in the divergent pattern of performance on the short and long delay trials shown in Figure 3.

Figure 3 *Comparison of short and long delay performance on the CVLT over Clusters 1, 2 and 3*



Cluster 1 participants showed improved recall from the short to the long delay condition. Cluster 2 showed only a modest improvement, while cluster 3 showed a divergent trend, with a decline in number of items correctly recalled.

The use of semantic categories also declined markedly across clusters. Cluster 1 employed a mean of 10.80 semantic categories throughout the 5 learning trials of list A. Cluster 2 scored a mean of 6 semantic categories and cluster 3 scored just 3 semantic categories. Recognition scores were also notably different in clusters 1 and 3. Recognition hits showed little difference between clusters 1 and 2, with a mean of 14.40 and 14.33 respectively, while the decline in performance for cluster 3, with a mean of 12.5 items recognised, is conspicuous.

The data relating to the false positive and discrimination variables were not able to be included in the cluster analysis without substantially distorting the relationships between recall scores (as discussed in the Method section). These variables were examined separately, therefore, within the cluster membership defined by the recall and recognition hits scores as set out in Table 9. Cluster 1 scored a mean of 2.7 false positive responses, cluster 2 a mean of 4.0 and cluster 3 a mean of 5.5 false positive responses. The Discrimination index followed a similar trend, with cluster 1 showing 90.22 percent, cluster 2, 89.38 percent and cluster 3 showing 79.54 percent. Thus, while these variables produced marked distortions in the cluster analysis procedure when included in the analysis, when excluded and examined in relation to existing cluster membership, the overall profile of declining performance across clusters was preserved. It is also notable that the results for the discrimination index are similar to those obtained for long delay free recall and recognition hits. The discrimination scores for cluster 3 are also consistent with the recognition hits achieved by this group.

The number of years since perceived onset of PD was also examined separately to determine if this was a factor in cluster membership. Cluster 1 showed a mean of 8.80 years since onset of symptoms, cluster 2 showed a mean of 9.33 years and cluster 3 a mean of 10.83 years. Thus, years since onset showed a direct relationship with cluster membership and memory performance. In addition, 1 outlier in cluster 1, who was diagnosed with PD at an early age, lengthened the mean years since onset for this group considerably. If this outlier is excluded, the mean years since onset for cluster 1 drops to 7.77, which further highlights the relatively short duration of PD in participants in cluster 1.

Differences were also noted in susceptibility to interference within clusters. Retroactive interference was assessed by the difference between recall on trial 5 and short delay free recall. Proactive interference was assessed by the number of items recalled on list B compared to list A, trial 1. Age-related norms for list B were also taken into account, with 5-6 items recalled being the norm on list B over the majority of age-groups. A decrease of 1-2 items on list B compared to list A trial 1, therefore, was not unexpected. Clusters 1 and 2 showed either no interference effects or mild, usually retroactive, interference. Cluster 3 showed more entrenched interference effects which were predominately proactive in nature.

Overall, there appeared to be a general pattern of higher scores and intact performance for cluster 1. Cluster 2 showed intermediate effects, with scores highlighting a similar trend but at a lower level. Cluster 3, however, showed a pattern of performance which was divergent from that of clusters 1 and 2, encompassing lower scores on the long delay trials, and more severe proactive interference in contrast to the milder retroactive interference of clusters 1 and 2. A marked decline in ability to utilise semantic clusters was also apparent for cluster 3, as was impaired recognition.

In order to determine which variables were significant with respect to cluster membership and to clarify the differences between clusters, a series of one-way ANOVAS, with *post hoc* Scheffe tests (SPSS, Inc., 1986) was conducted (see Appendix E). The results are presented in Table 10. The asterisks in the table indicate that the associated variables were significantly different across the 3 clusters. Thus, for example, with the variable list A total, all comparisons were significantly different. Comparisons showing no difference are left blank. The ANOVA results confirm the pattern of cluster membership seen in Table 9. Differences between the clusters were most striking on list A total, reflecting a progressive impairment in recall overall. The differences between long delay recall trials for each cluster were also significant, adding weight to the differences found in short to long delay performance on Table 9.

Table 10 *Statistically Significant Differences In CVLT Variables Across Clusters*

	Cluster 1 vs 2	Cluster 2 vs 3	Cluster 1 vs 3
Age			
List A Total	*	*	*
List A Trial 1			*
List A Trial 5			*
List B			
List A Short Delay Free Recall	*		*
List A Short Delay Cued Recall		*	*
List A Long Delay Free Recall	*	*	*
List A Long Delay Cued Recall	*	*	*
Semantic Cluster Ratio	*		*
Recognition		*	*

* = $p < .05$ (Scheffe)
Note: Cells left blank indicate a nonsignificant difference

The ability to utilise semantic clusters also proved to be significantly different between clusters 1 and 3 and between clusters 1 and 2. Differences between clusters 2 and 3 on semantic use, despite a mean difference which represented a 50 percent decline for cluster 3, were not significant.

The recognition trials also showed significant differences across the 3 clusters. Differences were significant between clusters 1 and 3, and also between 2 and 3. However, there was no difference between clusters 1 and 2. This result highlights a distinct difference in performance on the recognition task for participants in cluster 3. Cluster 3 showed a marked divergence in performance overall when compared with cluster 1, with 9 out of the 10 variables measured showing a significant difference. The only variable which did not reach significance with this comparison was list B free recall which was not significant on any comparison. In contrast, clusters 1 and 2 when compared, showed significant differences on only 5 out of the 10 variables measured, as did comparisons between clusters 2 and 3. Of the 5 variables significant in the cluster 1 and 2 comparison, only 2 were significant for cluster 1 and 2 alone.

In summary, the results shown in Table 9 together with the ANOVAS highlight a steady decline in overall recall performance across the 3 clusters. Long delay recall was differentially affected, however, being relatively preserved in clusters 1 and 2, but declining in cluster 3. Differences in ability to utilise semantic categories were also apparent. Cluster 1 participants demonstrated a moderate ability to utilise semantic categorisation while clusters 2 and 3 did not. Performance on the recognition task was also affected, with clusters 1 and 2 showing intact recognition in contrast to cluster 3 participants who evidenced a marked decline.

The implications of these results will discussed in full in the following chapter, with the aim of providing an interpretation of these clusters within a theoretical framework which attempts to account for the memory profiles observed.

The purpose of this study was firstly, to establish the existence of verbal memory deficits in PD and, secondly, to examine the underlying processes which contribute to this deficit, with specific emphasis on encoding and retrieval processes. The results showed that impaired verbal memory was present in the PD group. This was evidenced by lower recall overall, less utilisation of semantic categories, fewer items recalled from the middle of the list, more susceptibility to interference, and difficulty in discriminating previously learned items, when compared to the Control group.

MMSE

Results of the MMSE showed that all participants who were involved in the study were cognitively competent to do so. Thus, the results obtained are unlikely to be attributable to unidentified dementia, Alzheimer's disease, or other cognitive impairment of a global nature. The differences observed between the PD group and the Control group are most likely to be due to the influence of PD.

CVLT

Phase I

The study produced conclusive evidence of verbal memory deficits in PD. In phase I of the analysis, results showed a general impairment in recall with significant differences between the PD and Control groups on all recall trials of the CVLT. PD group performance was characterised by lower levels of recall overall, less efficient use of semantic categories and more susceptibility to interference relative to the Control group. The PD group did not show evidence of primacy or recency effects, but recalled fewer items from the middle of the list relative to the Control group. This pattern of response suggested that maintaining items from the middle of the list in working memory was difficult, which is consistent with a generalised impairment in short term memory (Lezak, 1995).

Intrusion rates on recall trials were also significantly higher in the PD group, which highlighted difficulties in discriminating between "list" and "non-list" items. Intrusions usually belonged to the same categories as the list items, and many were prototypical, for example, "hammer" as an item in the tools category. Many PD participants persisted in using these non-list intrusions, despite the feedback provided by the following learning trial, which indicated they were not on the list. These results and observations are consistent with other studies which have established that PD patients have difficulty discriminating between competing responses (Dubois et al., 1991; Flowers & Robertson, 1985; Channon, Jones, & Stephenson, 1993; Raskin, Borod, & Tweedy, 1992; Richards, Cote, & Stern, 1993). This difficulty in discrimination also appears to incorporate a failure to inhibit wrong responses even when the PD participant is aware of the strategy required (Flowers & Robertson, 1985).

Results achieved for both PD and Control groups showed that recall was considerably enhanced by the use of a semantic clustering strategy. The relative inability of the PD group to organise recall semantically, however, suggests inefficient use of planning and organisational aspects of memory. This organisation of the list occurs during the encoding stage of the task. The encoding processes involved in efficiently organising the list into semantic categories appear moderately deficient in the PD group overall. This, in turn, may be related to difficulties with the planning and organisational aspects of memory tasks which characterise PD performance on a number of cognitive tasks, such as the Tower of London (Robbins, James, Owen, Lange, Lees, Leigh, Marsden, Quinn, & Summers, 1994), Odd Man Out (Flowers & Robertson, 1985) and the Trail Making Test (Sullivan et al., 1989) and appear to be linked to damage in the dorsolateral prefrontal cortex (Owen et al., 1995; Gotham et al., 1988; Taylor et al., 1986; Petrides, 1985).

Thus, planning and organisational deficits result in a reduced ability to effectively organise material at the encoding stage of the learning process. Verbal material is encoded in a relatively random or haphazard fashion which, in turn, leads to difficulty in efficiently retrieving the material previously encoded.

To effectively organise the list into semantic categories, it is probable that the participant needs to encode the information twice. The list is firstly learnt either in a serial or random order, then held in working memory for several minutes while it is rearranged into the 4 categories. Thus, an elaborative encoding process occurs as meaning is attached to the learned material. This "search for meaning" is thought to involve the prefrontal cortex (Gabrieli, Desmond et al., 1996). The mean results for each of the learning trials achieved by the Control group (Figure 2) are consistent with this argument In trial 1, recall was largely confined to the "7- plus or minus 2" rule of short term memory span (Ebbinghaus, 1885, cited in Klatzky, 1975). In the process of carrying out trials 2 and 3, however, the participant appeared to uncover the semantic structure of the list as a possible learning strategy. During trial 4, performance tended to reach a plateau (trial 3, $M = 10.55$, trial 4, $M = 11.41$), while the participant reordered the list. This was then followed by an increase in mean number of items recalled on trial 5 ($M = 12.55$). This process was more noticeable for the higher scoring Control participants, 6 of whom correctly recalled 15-16 of the items on the 16 word list during trial 5.

The results showed that the PD group had a tendency to utilise serial recall, but serial clusters were low in absolute terms (a mean of 3.00 serial clusters in total for the 5 learning trials). The overall profile which emerged for the PD group highlighted a failure to adopt a consistent strategy in recall.

The high standard deviation for the Control group on the semantic categories variable indicates a wide variance in scores (range 6-49), with younger Control group members tending to obtain the higher scores. It was noted, however, that several of the Control group members whose scores were in the lower end of the semantic cluster range, also produced total recall scores for the 5 learning trials which were close to the mean for their group. These participants appeared able to cope with the demands of the task moderately well by use of a serial recall strategy, often recalling between 12 and 14 of the items on the list by trial 5.

In contrast, the PD group appeared unable to adopt a consistent recall strategy of any type, with recall which was haphazard, disorganised and significantly below that of the Control group. Thus, it would appear that *any* learning strategy which imposes some structure on the task will enhance recall. The semantic categorisation strategy, however, appeared the most efficient and, when employed consistently, produced higher recall overall. This result is consistent with the results from factor analysis of the CVLT, as presented in the Psychometric Analysis section (Delis, Freeland et al., 1988)

Gender studies have noted that men generally do not organise information into semantic categories as well as women and show a tendency toward more serial organisation on the CVLT (Kramer et al., 1988). This was predominately a male group. However, as the groups were also matched for gender, the male performance of the PD group was matched with normal male performance. Despite this matching, significant differences between the 2 groups in semantic utilisation were obtained.

Participants in the PD group appeared to have great difficulty with the mechanics of the semantic clustering process. The "search for meaning" normally undertaken by the prefrontal cortex (Gabrieli, Desmond et al., 1996) did not seem to occur. In a neural network context, it is possible that activation could not be maintained over a sufficient period of time to ensure reorganisation of the list. It is also likely that the corticocortical connections from the prefrontal cortex to semantic memory networks could not be made, with activation unable to spread to outlying cortical regions. The list, therefore, remained at the first stage of the encoding processes discussed above, usually in a random order and no further meaning was attached through the process of semantic categorisation.

Many PD participants also fell back on a phonetic strategy to facilitate recall of parts of the list. Thus, some responded "Plums, Parsley, Paprika, Pliers, Pants" (instead of slacks), or "chives/chisel", or "sweater/slacks". Within the PD group, 10 participants used phonetic clusters in this manner and semantic cluster scores for these 10 were predictably low, ranging between 2-12 clusters utilised over the 5 learning trials. Phonetic memory, according to Fuster's (1995) model, is lower down the neural hierarchy, and thus has less complex processing requirements. A list-learning task such as this, therefore, where semantic memory is affected, appears to have produced a tendency to rely on the phonetic properties of the list as an aid to recall.

Retrieval of information can also be affected by the inability to organise the list semantically at encoding. Conway and Engle (1994) have presented evidence which supports the interpretation that, once material is encoded in a semantic format, access to the information is in the form of an "address". Thus, in the context of the present study, the address "fruits" would contain "plums, apricots, grapes, tangerines". Once the address is accessed, retrieval of the individual items on the list is automatic. Retrieval is therefore not influenced by list length (Conway & Engle, 1994). If information is not encoded in this format, however, the PD participant may have to carry out a controlled and effortful search to recall each item on the list. This may result in inefficient and incomplete retrieval of list items.

A neural network explanation also offers some insight into the lower recall of PD participants relative to the Control group. Activation failure results in the appropriate neural networks not being activated and maintained while the retrieval process is carried out. This also results in difficulty discriminating list items from previously learned items of a similar category. Hence, a higher rate of intrusions for the PD group. In addition, because the information was not well organised at encoding, it may require access to a large number of neural networks to effect retrieval.

This extensive, time-consuming and effortful search may be further complicated by the inability to maintain a neural set due to activation failure. Participants in the PD group had difficulty recalling items from the middle of the list, and this was possibly due to an inability to maintain a neural set. This activation failure is thought to be a direct result of the loss of dopamine in the prefrontal cortex, as activation does not occur and a memory task cannot be maintained when dopamine uptake in the prefrontal cortex is blocked (Sawaguchi & Goldman-Rakic, 1991).

The number of false positive responses in the PD group suggested that recognition memory was not as robust as many previous studies have suggested (Tweedy et al., 1982; Sullivan et al., 1989; Buytenhuijs, Berger et al., 1994). Results for the group as a whole showed a mean of 3.55 false positives, but it was noted that 6 of the PD group participants had scores ranging from 5-11 false positives on the recognition task. The false positive scores for the PD group were not influenced by a tendency to respond with "yes" responses, as the response bias calculations indicated a neutral response tendency. The discrimination index, however, which is calculated using both false positive scores and recognition hits was significantly different between the 2 groups and suggested that the PD group had difficulty in discriminating between list A, list B and distractor items. These discrimination difficulties are thought to be the result of impaired encoding processes (Delis et al., 1987; Massman et al., 1990;).

Overall, the results obtained in Phase I of the analysis are consistent with other studies of verbal memory performance in PD. Delis et al. (1987), Kramer et al. (1989) and Massman et al. (1990) have also found evidence of impaired recall and difficulty with discrimination on the recognition task, along with low utilisation of semantic categories in PD patients tested on the CVLT. Other studies have also noted that verbal memory deficits are more marked when material is not semantically organised (Taylor et al., 1986; Caltagirone et al., 1989). The present study showed a direct relationship between the use of semantic categories and number of items recalled. In addition, the failure of PD participants in this study to organise material semantically or to adopt a consistent recall strategy of any type can be related to other studies which show disorders of planning and strategy (Flowers & Robertson, 1985; Owen, et al., 1995).

The finding of increased susceptibility to interference in PD participants is consistent with results obtained by Delis et al. (1987). Similar results, using the CVLT, have also been reported with head injured participants (Haut & Shutty, 1992), and it would appear that increased susceptibility to interference is a feature of many neurological disorders (Lezak, 1995).

Age

The effect of age on verbal memory performance was not significant for the PD group as a whole. This may have been an artifact of the small sample size, however, rather than an outright lack of effect. As the PD group was split in two for this section of the analysis, group numbers were small and this resulted in low statistical power. The non-significant result is somewhat counter-intuitive, given the neurodegenerative nature of PD. As the disease progresses with age, it is more probable that memory processes would be more severely affected in older PD participants. However, it appears that in this and other studies (Sullivan et al., 1989; Tweedy et al., 1982), age and disease severity are confounded.

Despite the fact that the age analysis did not detect an effect, the older PD group participants were largely responsible for the wide variation in List A total scores identified in Phase I of the results section. Younger PD participants tended to show milder memory deficits, and the differences between the younger and older participants of the PD group lead to a wide range in scores. Sullivan et al. (1989) also found that the "low" PD group, classed on the basis of BDS scores and comprised of younger members, showed memory impairments which were mild or non-existent. Reid, Broe et al. (1989) found early-onset, and therefore younger, patients performed better than the late-onset group and only moderately below controls on a range of cognitive tasks. This younger group then, appeared to have mild memory deficits. Thus, the results of these studies lend more support to the interpretation that the lack of effect for age in the present PD group was a result of small sample size

It should be acknowledged, however, that in this and other studies, there are problems in attempting to separate age from disease severity. The 2 factors show a high degree of interdependence in this neurodegenerative condition which is age-related, increasing in incidence beyond age 65. While late-onset PD may produce more severe cognitive decline, this could also be due, in part, to the neurodegenerative processes of aging. Younger PD patients may show only mild memory deficits, but that may be less to do with age and more to do with the levels of dopamine depletion found in the early stages of the disease. Thus, age is a function of disease severity and disease severity is a function of age.

Gender

As with the age comparisons, no significant effects were found for gender within the PD group. Once again, sample size may have been a factor in the results obtained. Numerous studies have established the superiority of female performance on verbal tasks (Kolb & Whishaw, 1990; Kramer et al., 1988; Maccoby & Jacklin, 1974), and it is unlikely that the results obtained in the present study, based on a sample of only 5 matched females, would refute the solid evidence presented in the literature. It is therefore difficult to justify the non-significant results for gender differences which were obtained in this study, when only 5 matched females participated.

While gender differences for the group as a whole were not significant, it was noted that the female participants were generally older (M = 77.30 compared to M = 70.67 for males), and, despite the older age group, performed moderately better than their younger male counterparts. Thus, a general picture emerged of different cognitive profiles for women participants with PD. The deficits occurring in these women with PD appeared milder, with memory functioning generally more preserved. Of the 5 females in the study, 3 had late-onset PD, but with mild cognitive deficits, which is contrary to general results achieved in other studies (Reid, Broe et al., 1989) Late-onset PD generally results in greater cognitive impairment than that of early-onset PD.

It was also noted that the women in this group remained active, carrying out most daily living activities and pursuing a wide range of interests despite their advancing years. In contrast, male PD participants who were of a similar age group, were largely housebound and had given up many of their former leisure pursuits. Thus, there appeared to be distinct psychosocial differences between men and women in PD.

Of the 3 clusters identified in the cluster analysis as representing mild, moderate and more severe deficits respectively, no females were in cluster 3, despite cluster 3 being composed of males of a similar age. Three women were in cluster 2 and the remaining 2 in cluster 1. The inclusion of these females in cluster 1 also increased the mean age of this group. Thus, the analysis of age as a factor in PD was somewhat distorted by the milder deficits observed in these older females. Further research with larger samples is necessary to more clearly delineate these gender differences.

Phase II

In phase II of the analysis, the cluster analysis revealed distinct subgroups of memory performance within the PD group. Three clusters emerged which appeared to best represent the data. Cluster 1 was characterised by higher scores overall, compared to clusters 2 and 3. Less retroactive interference and either mild or no proactive interference was noted. Furthermore, cluster 1 showed improved recall from the short to long delay condition, and greater ability to utilise semantic categories. Discrimination and false positive scores for this group, while examined separately from the cluster analysis, also presented a profile of intact ability to discriminate between list and distractor items which suggested that encoding processes were not affected. Recognition memory thus appeared well preserved. Three members of cluster 1 had scores comparable to the Control group and showed no evidence of memory deficits. Cluster 1, overall, appeared to represent mild retrieval deficits with little evidence of impaired encoding processes.

Cluster 2 appeared to represent intermediate effects, showing mild proactive interference and moderate retroactive interference. Little or no improvement was seen from short to long delay trials. As Figure 3 indicates, the trend was relatively flat. Recognition appeared well preserved for this group, with false positive and discrimination scores, examined separately, which did not show the characteristic steady decline of the recall trials. Use of semantic categories in recall was significantly lower than cluster 1, however, and it appears that semantic organisation at the encoding stage is disrupted, even in the early stages of the disease progression. Retrieval difficulties were also more pronounced in this group, compared to cluster 1. This was evidenced by lower scores on all recall trials, more susceptibility to interference and more intrusions.

Cluster 3 showed a pattern of memory performance which was distinctive and divergent from that of clusters 1 and 2. Scores were lower on all recall and recognition trials and recall declined from the short to the long delay trials. Performance on the long delay trials is considered an important indicator of overall memory functioning (Delis et al., 1987), reflecting an ability to hold information in memory over time. Ability to utilise semantic categories was severely affected, with a group mean of only 3 categorisations over the 5 learning trials. This result is meagre when compared to a possible 60 categories which may be formed. Recognition memory was also impaired in contrast to clusters 1 and 2.

These results for cluster 3, when taken together, suggest a profile of encoding and retrieval deficits which are more encompassing than those in cluster 2. The encoding deficits in cluster 2 members seemed largely related to organisational deficits which occurred in conjunction with the encoding stage of the learning process. Cluster 2 participants appeared able to recall a number of items from a simple list, provided that there was no elaborative process involved in encoding the material to be learned. Recognition memory in cluster 2, therefore, remained largely intact. However, in cluster 3, recognition memory was affected, suggesting that core encoding processes were also impaired, as well as those encoding processes which relate to the organisational requirements of the task.

A number of factors influenced the clustering of the PD group into these 3 distinct subgroups. The first of these is simply the progressive nature of PD itself. As the disease progresses, memory functioning becomes more impaired, with memory processes differentially affected as severity increases. The severity of PD, as indicated by number of years since onset of symptoms, appears to be a factor in cluster membership. Mean years since onset showed a steady progression across clusters (cluster 1, $M = 8.80$ years, cluster 2, $M = 9.33$ years and cluster 3, $M = 10.83$ years). The mean for cluster 1 drops considerably with the removal of 1 outlier ($M = 7.77$). This isolated case was diagnosed at an early age and, in many respects, was not typical of idiopathic PD.

The profile of cluster membership obtained suggests that, in the initial stages of the disease, memory functioning appears unaffected, and any deficits which do exist are mild in functional terms. As the disease progresses, however, an intermediate stage is characterised by significant retrieval difficulties and a markedly reduced ability to organise semantic information during encoding operations. In the final stages of the disease, retrieval and encoding processes are more severely affected as evidenced by substantially lowered recall, minimal semantic utilisation and impaired recognition memory.

Secondly, age was a factor in the cluster representations. While it was not established in Phase I of the analysis that age was a factor in overall memory performance, age did increase across the 3 clusters. Age in cluster 1 was not uniform, while clusters 2 and 3 showed a systematic increase in age, along with a decline in performance. Cluster 1 contained 2 outliers who produced a wide variation in age for this group. Both these participants were very high functioning individuals prior to diagnosis and still retained a considerable percentage of former functioning in spite of advancing years.

These results show that the relationship between age and memory function in PD does not always hold. There are some exceptions to the general trend, consistent with the heterogeneity found with other symptoms. The majority of cluster 1 participants, however, were aged between 48-76, with a mean age of 67, when the 2 older outliers are excluded from the calculation.

Thus, both age and disease severity are contributing factors to cluster membership, with number of years since onset holding a more consistent relationship to cluster membership than age. As discussed in the Introduction, however, the relationship between age and PD is a complex one, with many other factors which may also contribute to cognitive decline. These include early or late-onset of symptoms (Ried, Broe et al., 1989), tremulous versus akinetic forms of the disease (Koller et al.,1994) and varying levels of dopamine depletion in the cortical and subcortical structures affected (Gotham et al., 1988).

It should be noted, that in the present study, the majority of PD participants had early-onset of symptoms. Those participants with late-onset of symptoms were predominately female with mild memory deficits. Late-onset PD tends to produce more cognitive impairment (Reid, Broe et al., 1989; Biggins, Boyd et al., 1992) and, in these cases, the relationship between disease severity and number of years since onset of symptoms does not hold. Thus, the factors affecting cluster membership in this study are specifically applicable to PD with early-onset.

General Discussion

An interpretation, based on an examination of neural network organisation, suggests that encoding and retrieval deficits in PD are due to an increasing failure in activation as the disease progresses. In the verbal memory deficits occurring in PD, this activation failure is thought principally to occur in the dorsolateral prefrontal cortex. While, as discussed earlier, the mechanisms which cause neural networks to activate remain unclear, it is likely that the chemicals involved in neural transmission play a major role in activation. One of these chemicals is dopamine, and it has been established that when dopamine is blocked in the prefrontal cortex, activation does not occur (Sawaguchi & Goldman-Rakic, 1990). While the results obtained by Sawaguchi and Goldman-Rakic do not tend to support an alternative explanation that activation failure is due to overactivity in other neurotransmitters, it may be simplistic to ascribe activation failure wholly to dopamine loss alone. Other neurotransmitters may also contribute to this failure. It is suggested that dopamine, however, plays a major role in activating neural memory networks, and as dopamine loss increases, with possible flow-on effects to the equilibrium of neural transmission, there is a corresponding increase in activation failure. In the initial stages of the disease, there may be sufficient dopamine present in the neural networks of the dorsolateral prefrontal cortex to ensure that activation occurs most of the time. During the intermediate stage of PD, the amplitude of initial activation is possibly lowered (Fuster, 1995; Iansek, 1997) and, as a direct consequence, maintenance of activation throughout a given memory task is thought to be insufficient to meet the demands of the task.

This includes organisation of material at encoding and the efficient retrieval of information. Access to memory networks outside of the dorsolateral prefrontal cortex via corticocortical links may also be restricted as activation cannot be maintained at a sufficient level to ensure effective connections are made. Thus, discrimination between old and new items is affected and links to semantic memory networks may be disrupted.

In the latter stages of the disease, dopamine loss is substantial. Postmortem studies have shown dopamine is reduced to 40% in the frontal cortex (Agid et al., 1987). This, in turn, may produce a correspondingly high rate of activation failure. Initial activation may be absent or below the threshold necessary for neural networks to become active. With initial activation levels low, subsequent maintenance of activation would be minimised. Thus, the ability to maintain a mental set is severely compromised and information which is necessary for the successful completion of the task cannot be held in memory. It is likely that memory span would be therefore limited.

In this scenario, encoding processes would be disrupted, with participants unable to recognise material previously presented. This may account for the impaired recognition of cluster 3 participants. Organisation of semantic information would be impaired, as activation cannot be sufficiently maintained and links to semantic memory would not be established. Meaning would not be attached to learned material which, if it is encoded at all, would be entered in memory in a disorganised, haphazard manner. This deficiency in organising material at encoding was reflected in the inability of cluster 3 participants to utilise semantic categories, their limited recall overall and the random order in which list items were recalled. Similarly, PD participants in cluster 3 were unable to effectively retrieve previously learned information, as evidenced by the poorer performance of cluster 3 participants on the long delay trials, compared to recall during the short delay condition.

Fuster (1995) has presented a description of cortical memory organisation that links perceptual and motor memory systems in the dorsolateral prefrontal cortex. Gabrieli, Desmond et al. (1996) have shown that the prefrontal cortex mediates semantic memory and Awh et al., (1996) have demonstrated that frontal mechanisms are involved in verbal memory. In addition, Middleton and Strick (1994) have presented evidence that the dorsolateral prefrontal cortex is the cortical target of a pallido-thalamocortical pathway which is involved in non-motor aspects of cognition, such as working memory, rule based learning and planning for the future. Thus, the links to basal ganglia structures also appear to involve the dorsolateral prefrontal cortex. Finally, Sawaguchi & Goldman Rakic (1991) have demonstrated that when dopamine uptake is blocked in the dorsolateral prefrontal cortex, activation failure occurs. These results, therefore, provide strong support to an interpretation based on neural network organisation, which suggests that verbal memory deficits in PD occur when activation in the dorsolateral prefrontal cortex fails. The results obtained in this study, are consistent with this interpretation.

Further research is needed to identify the mechanisms which cause neural networks to activate. From this understanding it should be possible to identify why activation fails. Dopamine appears crucial to the activation process, however, (Sawaguchi & Goldman-Rakic, 1991) and it is suggested in this interpretation that dopamine loss is a major component of activation failure.

Sub-Group or Continuum?

Among PD participants, a pattern of gradual decline in memory functioning was noted in this study. Memory processes, however, were differentially affected as the disease progressed, with retrieval deficits which became apparent in the intermediate stages of the disease along with organisational difficulties at encoding. Significant encoding and retrieval deficits tended to occur in the latter stages of the disease process.

How these findings are classified is largely open to debate. Sullivan et al. (1989) in observing what appeared to be two distinct sub-types of memory deficit in PD, concluded that the mild memory deficits of younger PD participants and the dementia of the older PD group represented a continual decline consistent with disease severity.

Crosson et al. (1989) and Haut and Shutty (1992) considered the clusters they observed in head injured participants to be distinct sub-groups of memory deficit. The clusters identified in the head injury studies resulted in distinct groups, with either encoding or retrieval deficits occurring respectively for each cluster. Encoding and retrieval deficits were thus not identified as coexisting in any single cluster grouping.

The clusters identified in PD, however, present a more mixed picture. Cluster 1 showed only mild retrieval deficits, while cluster 2 showed moderate retrieval deficits along with difficulty in semantic organisation at the encoding stage of the learning process. In cluster 3, which was interpreted as representing an advanced stage of PD, both encoding and retrieval deficits were observed.

However, the 3 clusters were distinct from each other in the profiles of memory performance obtained. While a consistent decline in scores across clusters was noted for the majority of recall trials, cluster 2 appeared to have a specific difficulty with the semantic organisation of material at encoding. Cluster 3 showed distinct divergence on performance over the long delay trials. Recognition performance was also divergent for this group. The present study suggests, then, that there is a continuous progression in verbal memory deficit associated with disease severity in PD, but that this progression is characterised by identifiable stages with respect to the memory processes affected.

A neural network interpretation suggests a possible reason for this seemingly paradoxical finding. It is possible that memory processes which are further up the neural heirarchy and, therefore, have more complex processing requirements, are the first to be affected in PD. Thus, recall and semantic organisation appeared to be impaired at a relatively early stage of the disease progression, as noted in cluster 2 participants. Gurd and Ward (1989) noted that early-onset cases of PD showed impaired semantic categorisation and suggested that identifying this impairment may be an aid to early diagnosis. In the latter stages of the disease progression, the relatively less effortful processes of recognition appear affected, as evidenced by the poorer performance of cluster 3 participants. Thus, the processing demands of the task may be a determinant of which specific memory processes are affected.

Implications for Management of PD

Crosson et al. (1989) and Haut and Shutty (1992) identified learning conditions which would allow head injured participants with encoding and retrieval deficits to compensate for the difficulties encountered in verbal learning tasks. They recommended that patients with encoding deficits would benefit from brief breaks and short delays while learning, and that this learning should also consist of short sessions. The provision of an external structure, such as cues to recall, is also helpful in allowing more organisation of material to be carried out. Retrieval deficits could be partially alleviated by long delays between learning sessions to take advantage of relatively more preserved consolidation processes. Semantic categories provided as prompts would also take advantage of preserved ability to organise material semantically at the encoding stage. Enhancement of memory through repetition was also recommended, taking advantage of intact encoding and preserved delayed recall.

If one considers that encoding or retrieval deficits are functionally similar, regardless of the underlying pathology, then PD patients with these difficulties should also receive some benefit from these learning conditions. For those PD patients with retrieval deficits who are still reading, a 20 min break between chapters of a book may facilitate retention. In general, reading material which is able to be easily put down and picked up again is preferable to a novel with a long and convoluted plot. Newspapers and short stories are more likely to retained than a weighty volume of fiction. PD patients with retrieval deficits may also need time to retrieve information. Because retrieval processes are not thought to be as efficient as they were previously, more time may be required. As outlined earlier in the discussion, PD patients may have to carry out a controlled and effortful search which involves access to a large number of neural networks to effect retrieval. They should not be rushed, have sentences finished for them or be put under pressure to respond.

The results of the present study suggest that there are 2 components to encoding deficits in PD, one relating to organisational difficulties such as those experienced by participants in cluster 2. The other component relates to information which is missing, which appeared to be a feature of encoding deficits in cluster 3. PD patients with encoding deficits are likely to benefit from the provision of an external structure, such as timetabling, weekly activities calendar or other organisational programs which allow the person with PD access to information in a well structured format. Before beginning on a task, the person with encoding deficits may benefit from a prompt to use an attenuation strategy. That is, attenuating the demands of the task, breaking it down into smaller components, giving selective attention to such aspects as, what needs to be done, how they are going to achieve this, and the steps necessary along the way. These steps could be recorded in some way, perhaps with a personal voice recorder. Thus, in the absence of an internal and largely implicit "goal path", the person consciously constructs an external "goal path" of their own which then can then refer back to.

Similar strategies are employed in head injury rehabilitation, with tasks required in daily living broken into the component steps needed to complete the task. The head injured patient may also be encouraged to use a memory diary to timetable activities, or a personal recording device, such as a "memory pen" or compact cassette recorder to remember items which need to be purchased, or details of daily living activities which the person may need to refer back to (Prigatano, 1983, 1988; Wilson, 1987,1991; Wilson & Moffat, 1992; Wesolowski & Zencius, 1994).

Both encoding and retrieval deficits may be partially alleviated through having information presented in video form. For encoding deficits, this represents a less effortful processing task than the reading of information. A video format also tends to present the information in a highly structured format, thus providing some external organisation to facilitate efficient encoding. In addition, this format is easily repeated, enabling people with retrieval deficits to benefit from the repetition of information. The information contained in the video is able to be viewed with family members, with specific aspects able to be repeated and clarified, if necessary. These considerations may be particularly applicable to such areas as financial and retirement planning, where important decisions affecting the PD patient and their family are often made.

Thus, it is possible that PD patients with encoding and retrieval deficits will benefit from the same learning conditions as those proposed for head injured patients with respect to brief breaks while learning, the provision of an external structure and the use of repetition (Crosson et al., 1989; Haut & Shutty, 1992).

Future Research and the Limitations of the Present Study

The measure used in the present study, the CVLT, introduced a number of limitations which future research should take account of. The CVLT has a strong clinical application which does not necessarily translate to optimal suitability for research purposes. The number of trials used and the large number of variables measured result in the generation of a substantial amount of data. It should be noted, however, that this problem is endemic to all clinical memory tests and not simply an isolated limitation of the CVLT. Commonly used memory assessment measures such as the WMS, RAVLT, and others all have large numbers of variables which are measured over repeated trials. To accurately assess memory, it is necessary that material be first learnt, and then retained over time. It is the nature of memory that such repetitions are necessary.

The solution to these limitations presents a challenge, given the state of our present knowledge regarding memory functioning. However, more cross-pollination between test authors and researchers with respect to research objectives, may lead to the construction of memory tests more suited for research purposes. In general, it is more appropriate in memory research to focus on 1 or 2 specific aspects of memory rather than spread investigations over a wide range of memory functions. This is because, in accounting for the results obtained, a detailed analysis is required, rather than an overall clinical profile of the client. Thus, memory tests are needed which allow this depth of analysis to be carried out.

The revision of the WMS (Wechsler, 1987) has been similarly criticised for a lack of depth and a failure to incorporate the advances in understanding of memory processes arising from research (Sewell, Downey, & Sinnett, 1988; Loring, 1989). The CVLT, however, does attempt an examination of memory processes which is based on findings from current research (as discussed in the Psychometric Evaluation section). In general, there appears to be a need for more dialogue between those involved in research and clinical methods.

As the above points suggest, there are complex psychometric issues surrounding the use of memory tests. A major factor in this complexity concerns the interdependence of memory items. As discussed in the section on Psychometric Evaluation, memory capacity is limited. As a result, recalling an item on any given trial decreases the likelihood of any other item being recalled on the same trial. Therefore, there is item interdependence within trials. In addition, recalling an item on any given trial increases the likelihood of the item being recalled on a subsequent trial. Thus, there is item interdependence across trials. This interdependence should be taken into account in the analysis used.

Where an investigation intends to divide a sample into sub-groups, the estimated statistical power needs to be evaluated. This is particularly pertinent to studies such as this one and others (Sullivan et al., 1989; Tweedy, et al., 1982) where the clinical population is separated into sub-groups during the analysis. In studies such as these, sub-groups are small, consisting of 6-10 participants. There is an inherent danger in the analysis of these groups that an effect will go undetected because the statistical power of the tests used to examine any differences is too low. However, it should be noted that, in the present study, the ANOVA used to clarify the differences between sub-groups (clusters) showed significant differences between the 3 groups.

There appears to be a general consensus within the literature that the cognitive deficits found in PD are often subtle (Dubois et al., 1991). Small effects are therefore more common. It is important in this context, therefore, to employ an experimental design which will maximise experimental sensitivity and preserve statistical power.

As discussed earlier, the recognition test of the CVLT may benefit from expansion. A possible criticism which as been leveled at the CVLT, is that the recognition test constitutes too few trials to satisfy its theoretical underpinnings. The recognition test is based on a signal detection theory approach. Howevei, in signal detection applications, large numbers of trials are normally needed to reliably establish the data. Only 44 trials are used in the CVLT. Crosson et al. (1989) note that, for each participant, tests based on signal detection theory require large numbers of trials.

While the recognition test could, on theoretical grounds, be extended to include more trials, there are practical constraints in doing so. In any neuropsychological assessment procedure there must be a trade-off between theoretical rigor and the capacity of neurological participants to contribute to lengthy assessment procedures. As Kline (1993) argues;

> *"The shorter the test, the more useful it is. A psychological test which requires ten hours to complete, no matter its reliability and validity, is essentially useless. Participants would not complete it and no testers would be given time to use it. Thus, in the applied setting there is a trade-off between brevity and reliability."*
>
> (Kline, 1993: "The Handbook of Psychological Testing")

This comment is particularly pertinent to neurological conditions which are also age related. Thus, the propensity of participants with neurological disabilities to become easily fatigued must be taken into account in the experimental design and choice of assessment measures used. A participant who requires several hours of bedrest following assessment is not well served by the research process. Folstein et al. (1975), in developing the MMSE, noted that;

> *"Elderly patients, particularly those with delirium or dementia syndromes cooperate well only for short periods".*
>
> (Folstein, Folstein, & McHugh, 1975)

These considerations were important factors in the choice of assessment measures used.

While the clinical focus of the CVLT presented some challenges to the data management necessary for research, its clinical strength was apparent in the applied setting in which it was used. The test proved easy to administer, with participants having little difficulty understanding task requirements.

Most participants reported that they enjoyed the task, which may have added to the tests effectiveness in assessing normal memory performance. In addition, the test proved to be sensitive in identifying other underlying pathology in the participants tested. It quickly identified a prior head injury in a control participant which was not reported and was also sensitive to a case of meningitis which had occurred 77 years previously.

In the process of carrying out assessments, the CVLT quickly highlighted the presence of a short term memory deficit within the first 2 learning trials. Participants with memory difficulties would invariably first report the last 3 items on the list, often in reverse order. These items would be given in great haste, as if the participant was anxious to respond before all trace of the item in memory faded. The participant would then return to the beginning of the list and recall several items. They would have considerable difficulty, however, recalling anything from the middle section of the list. It was immediately apparent with this pattern of response on the CVLT, that a deficit existed. These observations were subsequently confirmed by the analysis of scores.

The timeframe available for subject recruitment in the present study meant that participant numbers were small (*N* = 22 / gp). However, overall differences between PD and Control participants were clearly established, suggesting that the number of participants was statistically adequate. Many PD patients approached had concurrent neurological problems other than PD, such as stroke, brain tumor, or epilepsy and, thus, had to be excluded. Others had mild dementia or a history of neurological injury which was relatively severe. The age-matching process proved time consuming, but ultimately rewarding in terms of the quality of data obtained. Careful matching resulted in an identical mean age for each group. However, this matching process, coupled with the time spent finding suitable participants and the travel to carry out interviews, resulted in a lengthy data collection process. Within the time constraints and scope defined by the academic requirements of the study, therefore, a larger sample size was not practicable. While statistical power was considered adequate, future research could consider using a larger sample size to ensure that statistical power is maximised.

Implications for Further Research

The interpretation given in the General Discussion section, in an attempt to account for verbal memory deficits in PD, needs to be substantiated with empirical data. Future research which is able to identify the mechanisms responsible for activation in the prefrontal cortex is needed in order to fully understand why activation fails. Further animal studies which establish the role of neurotransmitters other than dopamine in facilitating or inhibiting activation are also needed. In addition, the interpretation of the current study that dopamine loss affecting receptors in the dorsolateral prefrontal cortex results in activation failure and consequent verbal memory deficits needs to be supported by studies which examine the extent of activation in the prefrontal cortex of patients with PD. These studies need to examine both amplitude and duration along with spread of activation in mild and severe cases of PD. The existence of verbal memory deficits in participants involved in these studies should be established prior to any further investigations and a comparison with neurologically normal Controls made. It may be possible, for example, that a low level of activation in neurologically normal controls does not result in memory deficit. In addition, there may be other compensatory mechanisms involved. It is envisaged that, in the near future, technological advances will enable us to isolate activation processes in a memory system which is dynamic. These advances may enable studies, such as those suggested above, to be carried out.

Gender differences in PD are worthy of further examination. Clinical observations carried out during this study noted that, in comparison to males, the women in the present study appeared to have a milder form of PD overall, with less cognitive impairment and a lessening of the psychosocial consequences of the disease. However, it is difficult to confidently substantiate these observations with only 5 females with PD involved in the study. Diamond et al. (1990) examined male and female differences in the progression of PD and concluded that the disease followed the same progression in men and women. The study was largely confined to an examination of motor symptoms as measured by the University of California: LA (UCLA) scale. Cognitive symptoms were confined to 7 activities of daily living as reported by the participant. While incidence of dementia was recorded, an independent assessment of memory functioning was not made. Psychosocial differences were not examined. Thus, there is a need for future research which examines the cognitive profiles and psychosocial deficits of men and women with PD. If distinct differences do exist in memory and psychosocial deficits, then this has implications for the management of PD in men and women.

Future studies should examine these differences with a larger sample of women. There are practical constraints to achieving this, however, as the incidence of PD in women is generally low. As discussed in the introduction, male:female ratios have been reported which range from 1.36:1 to 3.7:1 (Diamond et al., 1990). The authors conclude that most studies find PD to be more common in men than women. Therefore, any prospective study would need to be conducted in a setting where time allocated for subject recruitment is not limited.

A future study could also consider a separate analysis of the performance of male and female participants with PD. The females in this study significantly extended the age group means for cluster 1, and, despite their advancing years, showed little evidence of severe memory impairment. In contrast, males of a similar age in the study were all concentrated in cluster 3, which was considered to represent those with advanced memory deficits. Thus, the general trend of advancing memory difficulty with increased age was somewhat distorted by the performance of the females in this study. All these factors are deserving of closer scrutiny.

The participant characteristics obtained by several studies (Tweedy et al., 1982; Sullivan et al., 1989; Massman et al., 1990) showed that PD patients, overall, had more years of education than is found in the population generally. In clinical observation of the PD group, the majority of these participants were high-functioning individuals who, despite their PD, still retained a degree of former functioning capacity. They appeared, in short, to be coming off a very high base of pre-morbid functioning.

Future research could incorporate an estimate of premorbid intelligence such as The North American Adult Reading Test (NAART: Nelson & O'Connell, 1978; Nelson, 1982; Blair & Spreen, 1989), which would give a clearer indication of the degree of impairment present as a result of PD. The NAART presents a list of irregularly pronounced words, such as "gauche" or "abstemious". Given the difficulties which some PD patients have with the mechanics of the reading process, an adaptation to the NAART may be advisable for use with PD groups. Words could be presented individually in large print on a computer screen to avoid any difficulties associated with having to read a list.

This study highlights the need to match participants for age and gender in examination of cognitive deficits in PD. They should also, where possible, be closely matched on education and occupational grounds, to control for general verbal ability.

The present study found that both age and disease severity were contributing factors to cluster membership, with number of years since onset holding a more consistent relationship to cluster membership than age. However, as discussed earlier in this section, the 2 factors appear interdependent and raise a question which future research must address. There are few examples in the literature of studies which separate age and disease severity as factors affecting verbal memory deficits. While both items are usually assessed, they also both influence the severity of any deficits found. Future research needs to address ways in which these 2 factors can be disentangled when examining memory deficits in PD.

It is also apparent from the present study that disease severity needs to be examined in relation to early or late-onset of PD, particularly in regard to severity of cognitive symptoms. In addition, other studies have highlighted the need to ensure that any examination of deficient processes in PD examines both mild and advanced cases of the disease before stating that any specific function is preserved.

Finally, in considering the approach which future research into memory deficits in PD might take, the views expressed by Welford (1980) seem appropriate. In discussing the work of the late George Talland, "Disorders of Memory and Learning", Wellford described him as:

> " ...a *man who could rise above minutiae of methodology and petty theoretical squabbles to look his problems straight in the face, applying logic and common sense to the results of acute observation, and stating his conclusions with simplicity, penetrating insight, and a refreshing touch of humanity. This is the stuff of which great scientists in any discipline are made - qualities not always endearing to those who like their ideas to remain well ordered, but which are the means of driving a subject forward, freeing it from an endless round of ever more trivial details."*

In the past, the discourse relating to memory research has suffered from some of the criticisms implied in Wellford's (1980) statement. There is much work to be done in the field of memory disorders. Adopting the approach of Talland would add considerably to our ability to advance the body of knowledge in this field.

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Appendix A

Information Sheets

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FACULTY OF SOCIAL SCIENCES
DEPARTMENT OF PSYCHOLOGY

INFORMATION SHEET

COGNITIVE DEFICITS IN PARKINSON'S DISEASE

Researchers:

Marie O'Sullivan
Postgraduate Researcher
Ph: [redacted]

Dr John Podd
Principal Researcher
Massey University
Ph: W: (06) 356-9099
Ph: H: [redacted]

The study will look at the way in which people with Parkinson's Disease remember information and how they recall new material. Some people may have difficulty as the information goes into memory and others may have difficulty as the information is taken out of memory. These difficulties give rise to different learning patterns and these can be identified by the different patterns of errors which are made. From our results we hope to write recommendations for rehabilitative procedures with Parkinson's Disease patients.

Should you decide to participate in the study, the researcher will travel to your residence to gather the information needed. Alternatively, a meeting place can be arranged which is convenient to you.

Two measures will be used in the study. The first is a simple screening measure and takes between 5-10 minutes to complete.

The second measure is the California Verbal Learning Test which examines the way people learn new material. You will be given a list of shopping items to remember and then asked to recall them. The way in which these items are presented is varied along with the length of time between presentation and recall. There is no time limit on the test, however, and you will be under no time pressure to give answers. The test should not cause you any undue stress or fatigue and contains items which are encountered on a daily basis. This test takes about 1 hour to complete. The total time of the study will be about one and a half hours.

continued over ...

Should you decide to participate in the study, you will have the following rights:

A. To refuse to answer any particular question, and to withdraw from the study at any time.

B. To ask any further questions about the study that occur to you during your participation.

C. You will provide information on the understanding that it is completely confidential to the researchers. All information is collected anonymously, and it will not be possible to identify you in any reports that are prepared from the study.

D. To be given access to a summary of the findings from the study when it is concluded.

Should you wish to have an interpreter present for this study, please indicate below :

English:	I wish to have an interpreter	Yes	No
Maori:	E hiahia an koe ki tetahi tangata hei kai whakamohio	Yes	No

COGNITIVE DEFICITS IN PARKINSON'S DISEASE

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INFORMATION SHEET FOR PARTICIPANTS IN THE PARKINSON'S DISEASE COMPARISON GROUP

Researchers:

Marie O'Sullivan
Postgraduate Researcher
Ph: [redacted]

Dr John Podd
Principal Researcher
Massey University
Ph: W: (06) 356-9099
Ph: H: [redacted]

The main focus of our study is the investigation of how people with Parkinson's disease remember new information and recall new material. Some people may have difficulty as the information goes into memory and others may have difficulty as the information is taken out of memory. These difficulties produce learning and memory patterns that are different from those we find in comparison, or control, individuals. When establishing a control group, such as the one you are being asked to join, we try to "match" the sex and approximate age of each Parkinsonian participant with that of a comparison person who does not have Parkinson's disease. This matching allows us to rule out sex and age as factors that might affect learning and memory. This means that we can be more confident that Parkinson's disease itself is the cause of any differences we find between the two groups. Therefore, a control group of "normal" people is essential to the running of our study, and others like it.

Should you decide to participate in the study, the researcher will travel to your residence to gather the information needed. Alternatively, a meeting place can be arranged which is convenient to you.

Two measures will be used in the study. The first is a simple screening measure and takes between 5-10 minutes to complete.

continued over ...

The second measure is the California Verbal Learning Test which examines the way people learn new material. You will be given a list of shopping items to remember and then asked to recall them. The way in which these items are presented is varied along with the length of time between presentation and recall. There is no time limit on the test, however, and you will be under no time pressure to give answers. The test should not cause you any undue stress or fatigue and contains items which are encountered on a daily basis. This test takes about 1 hour to complete. The total time of the study will be about one and a half hours.

Should you decide to participate in the study, you will have the following rights:

A. To refuse to answer any particular question, and to withdraw from the study at any time.

B. To ask any further questions about the study that occur to you during your participation.

C. You will provide information on the understanding that it is completely confidential to the researchers. All information is collected anonymously, and it will not be possible to identify you in any reports that are prepared from the study.

D. To be given access to a summary of the findings from the study when it is concluded.

Should you wish to have an interpreter present for this study, please indicate below :

English:	I wish to have an interpreter	Yes	No
Maori:	E hiahia an koe ki tetahi tangata hei kai whakamohio	Yes	No

Appendix B

Consent Forms

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CONSENT FORM

COGNITIVE DEFICITS IN PARKINSON'S DISEASE

The following is a statement to be signed in the presence of the researcher and, where possible, a witness.

a) I have read the Information Sheet and have had the opportunity for discussions with Marie O'Sullivan.

b) I know that I may withdraw from the study at any time, and I understand that this withdrawal will not adversely affect my further health care.

c) I understand that this study has been approved by the Central Regional Health Authority Wellington Ethics Committee and if I have any concerns about the study, I may contact the Ethics Committee, Wellington Hospital. Telephone : 385-5999 Ext : 5185.

I agree to take part in this study.

Signature .... (participant) / / (date)

.... (witness) / / (date)

Witness Name .... (Print Name)

Statement by Investigator

I have discussed with ....

(participant's name) the aims of and procedures involved in this study.

Signature .... (investigator) / / (date)

COGNITIVE DEFICITS IN PARKINSON'S DISEASE

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DEPARTMENT OF PSYCHOLOGY

CONSENT FORM

PARKINSON'S DISEASE COMPARISON GROUP

The following is a statement to be signed in the presence of the researcher and, where possible, a witness.

a) I have read the Information Sheet and have had the opportunity for discussions with Marie O'Sullivan.

b) I know that I may withdraw from the study at any time.

c) I understand that this study has been approved by the Central Regional Health Authority Wellington Ethics Committee and if I have any concerns about the study, I may contact the Ethics Committee, Wellington Hospital. Telephone : 385-5999 Ext : 5185.

I agree to take part in this study.

Signature .... (participant) / / (date)

.... (witness) / / (date)

Witness Name .... (Print Name)

Statement by Investigator

I have discussed with ....

(participant's name) the aims of and procedures involved in this study.

Signature .... (investigator) / / (date)

Appendix C

CVLT

CVLT CALIFORNIA VERBAL LEARNING TEST

RESEARCH EDITION

ADULT VERSION

Dean C. Delis, Joel H. Kramer, Edith Kaplan, and Beth A. Ober

Examinee Information:

Name ______________________________ ID No. ______________

Sex ______ Age ______ Race ______ Education ______________________

Date of Birth ______________ Occupation ______________________

Handedness ______________ Familial Left-handedness? ______________________

Current Medications ______________________________________

Diagnoses* Date of Onset

1. ______________________________________

2. ______________________________________

3. ______________________________________

4. ______________________________________

5. ______________________________________

6. ______________________________________

*Diagnoses should include history of: 1) neurological injury or disease, 2) medical illness, 3) psychiatric disorder, 4) loss of consciousness and duration of episode, 5) substance abuse, and/or 6) developmental learning disability.

Examiner ______________________________ Date of Administration ______________

THE PSYCHOLOGICAL CORPORATION
HARCOURT BRACE JOVANOVICH, INC.

9-030503

Summary of Scores:

	Raw Score	Standard Score[c]
Recall Measures (Number Correct)		
List A Total Trials 1-5[a]	______	______
List A Trial 1	______	______
List A Trial 5	______	______
List B[a]	______	______
List A Short-Delay Free Recall[a]	______	______
List A Short-Delay Cued Recall[a]	______	______
List A Long-Delay Free Recall[a]	______	______
List A Long-Delay Cued Recall[a]	______	______
Learning Characteristics, List A Trials 1-5		
Semantic Cluster Ratio (Observed/Expected)[b]	______	______
Serial Cluster Ratio (Observed/Expected)[b]	______	______
Percent of Correct Recall from: Primacy Region	______	______
: Middle Region	______	______
: Recency Region	______	______
Learning Slope (Increment in Words Recalled/Trial)[b]	______	______
Percent Recall Consistency Across Trials 1-5[b]	______	______
Recall Errors (Lists A & B)		
Perseverations (Free and Cued Recall Total)	______	______
Free Recall Intrusions (Total)	______	______
Cued Recall Intrusions (Total)	______	______
Recognition Measures		
Recognition Hits[a]	______	______
Discriminability[a]	______	______
False Positives	______	______
Response Bias	______	______
	Percentage Change	Difference Score
Contrast Measures		
List B compared with List A Trial 1	______	______
Short-Delay Free Recall compared with List A Trial 5	______	______
Long-Delay Free Recall compared with Short-Delay Free Recall	______	______
Recognition Hits compared with Long-Delay Free Recall	______	______
Discriminability compared with Long-Delay Free Recall		______

[a] Primary Score.

[b] Index not easily computed by hand. See CVLT Research Edition Administration and Scoring Software.

[c] For List A Total Trials 1-5, Mean = 50 and SD = 10; for all other indices, Mean = 0 and SD = 1.

LIST A (Monday List)
[redacted]

LIST A: Immediate Free Recall, Trials 1-3

Instructions to Examinee:

Trial 1:

Let's suppose you were going shopping on Monday. I'm going to read a list of items for you to buy. Listen carefully, and when I'm through, I want you to say back as many of the items as you can. It doesn't matter what order you say them in — just tell me as many as you can. Are you ready?

Trial 2:

I'm going to repeat Monday's shopping list. Again, I want you to say back as many items as you can, in any order. Be sure to also say the items on the list that you told me the first time.

Trials 3-5:

I'm going to repeat Monday's shopping list. Again, I want you to say back as many items as you can, in any order, including items you may have already told me.

KEY FOR CODING RESPONSE TYPE
C = Correct
P = Perseveration
I = Intrusion

	Trial 1 Responses	Type	Semantic Cluster
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14			
15			
16			
17			
18			
19			
20			

	Trial 2 Responses	Type	Semantic Cluster
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14			
15			
16			
17			
18			
19			
20			

	Trial 3 Responses	Type	
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14			
15			
16			
17			
18			
19			
20			

LIST A: Immediate Free Recall, Trials 4 & 5

	Trial 4 Responses	Type	Semantic Cluster
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14			
15			
16			
17			
18			
19			
20			

	Trial 5 Responses	Type	Semantic Cluster
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14			
15			
16			
17			
18			
19			
20			

Table of Free-Recall Word Order

LIST A (Monday List)	Immediate 1	2	3	4	5	Short Delay	
[redacted]							
Serial cluster score:							

Summary Table: LIST A (Monday List) Immediate Free Recall

TRIAL	Correct (C)	Persever-ations (P)	Intrusions (I)	Semantic Cluster
1				
2				
3				
4				
5				
TOTAL				

Record these totals in the Summary Table of Recall Errors (p. 9)

Table of Serial Position Recall: LIST A Immediate Free Recall, Trials 1-5

Region	Number Correct	Percent of Total
Primacy		
Middle		
Recency		
TOTAL		100%

LIST B
(Tuesday List)

Instructions: *Now let's suppose that you planned to go shopping again on Tuesday. I'm going to read a* **new** *list of items for you to buy. When I'm through, I want you to say back as many as you can, in any order.*

LIST B (Tuesday List)	Recall Order
[redacted]	

	Responses	Type	Semantic Cluster
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14			
15			
16			
17			
18			
19			
20			

Correct (C):	[redacted]
Perseverations (P):	______
Intrusions (I):	______
Semantic cluster score:	______
Serial cluster score:	______

LIST A:
Short-Delay Free Recal

Instructions: *Now I'd like you to tell n all of the shopping items you can from Monday list.*

	Responses	Type	Semantic Cluster
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14			
15			
16			
17			
18			
19			
20			

Correct (C):	[redacted]
Perseverations (P):	______
Intrusions (I):	______
Semantic cluster score:	______

LIST A:
Short-Delay Cued Recall

Instructions: *Tell me all of the shopping items from the Monday list that are:* (category)

	Responses	Type
Spices & Herbs:		
Tools:		

	Responses	Type
Fruits:		
Clothing:		

Correct (C):	
Perseverations (P):	______
Intrusions (I):	______

Time of day Short-Delay Cued Recall completed: ______

LIST A:
Long-Delay Free Recall

Time of day Long-Delay Free Recall begun: ________

Time of day Short-Delay Cued Recall completed: ________

Total delay: ________

(Note: The total delay should be about 20 minutes.)

Instructions: *I read some shopping items to you earlier. I'd like you to tell me all the items you can from the Monday list—that was the first list, the one that I read to you five times. Go ahead.*

	Responses	Type	Semantic Cluster
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14			
15			
16			
17			
18			
19			
20			

Correct (C):	________
Perseverations (P):	________
Intrusions (I):	________
Semantic cluster score:	________

LIST A:
Long-Delay Cued Recall

Instructions: *Tell me all of the shopping items from the Monday list that are:* (category)

	Responses	Type
Clothing:		
Fruits:		

	Responses	
Tools:		
Spices & Herbs:		

Correct (C):	
Perseverations (P):	
Intrusions (I):	

Summary Table of Recall Errors

Error Type	List A, Tr. 1-5 Total	List B	Short-Delay Recall Free	Short-Delay Recall Cued	Long-Delay Recall Free	Long-Delay Recall Cued	Total
Perseverations							
Intrusions: Free Recall							
Cued Recall							

LIST A:
Long-Delay Recognition

Instructions: *I'm going to read a list of shopping items. After I read each item, say "Yes" if the item was from the Monday list, and say "No" if it was not.*

(Note: For each "Yes" response, place a (✓) in the one blank space to the right of the word; for each "No" response, leave the space blank.)

A	=	List A-Correct (Hits)
BS	=	List B-Shared
BN	=	List B-Nonshared
NP	=	Neither List-Prototypical
PS	=	Neither List-Phonemically Similar
UN	=	Neither List-Unrelated

ITEM	A	FALSE POSITIVES BS	BN	NP	PS	UN
[redacted]						
TOTAL						
POSSIBLE	16	4	4	4	8	8
	A	BS	BN	NP	PS	UN

Total number of hits: ______

Total number of false positives: ______

Misses = 16 − Hits = ______

Discriminability:

$$\left(1 - \frac{\text{\_\_\_\_ (False Positives)} + \text{\_\_\_\_ (Misses)}}{44}\right) \times 100$$

$$= (1 - \text{\_\_\_\_}) \times 100 = \text{\_\_\_\_}$$

Response Bias:

$$\frac{\text{\_\_\_\_ (False Positives)} - \text{\_\_\_\_ (Misses)}}{\text{\_\_\_\_ (False Positives)} + \text{\_\_\_\_ (Misses)}} = \text{\_\_\_\_}$$

Note: If False Positives = 0 or Misses = 0, substitute a value of 1 when computing Response Bias.

CVLT
General Questions

1. Is memory for List B notably worse than for Trial 1 of List A (which may reflect a clinically significant vulnerability t proactive interference)? Are more List B nonshared-category items recalled than shared-category items?

2. Is Short-Delay Free Recall of List A notably worse than Trial 5 of List A (which may reflect both the effects of the sho delay interval and a clinically significant vulnerability to retroactive interference)?

3. Does cued recall enhance memory performance over free recall (which may have implications for the relative integrit of encoding versus retrieval processes)?

4. What types of perseverations occur (e.g., proximal versus distal)? Are perseverations semantically clustered? What types of intrusions occur (e.g., novel intrusions; intrusions from the other CVLT list; semantically-related versus unr lated to CVLT items)? Do more intrusions occur on cued recall than on free recall? Error analysis can contribute to th characterization of different memory disorders.

5. Is recognition performance notably better than free recall and cued recall (which may have implications for the relati integrity of encoding versus retrieval processes)?

6. Do false positives occur on recognition? If so, what types (e.g., semantically related; phonemically related; from List B; etc.)? The nature of recognition errors may be related to problems in discriminability and/or response bias.

7. Other observations which reflect the examinee's learning strategy (e.g., semantic clustering versus serial-order clus tering; primacy-recency effect; consistency of item recall and recall strategy across trials, as shown in the Free-Reca Word Order table)?

Appendix D

MMSE

Patient................................
Examiner............................
Date..................................

"MINI-MENTAL STATE"

Maximum Score	Score	
		ORIENTATION
5	( )	What is the (year) (season) (date) (day) (month)?
5	( )	Where are we: (state) (country) (town) (hospital) (floor).
		REGISTRATION
3	( )	Name 3 objects: 1 second to say each. Then ask the patient all three after you have said them. Give 1 point for each correct answer. Then repeat them until he learns all 3. Count trials and record.
		ATTENTION AND CALCULATION
5	( )	Serial 7's. 1 point for each correct. Stop after 5 answers. Alternatively spell "world" backwards.
		RECALL
3	( )	Ask for the 3 objects repeated above. Give 1 point for each correct.
		LANGUAGE
9	( )	Name a pencil, and watch (2 points) Repeat the following "No ifs, ands or buts" (1 point) Follow a 3-stage command: "Take a paper in your right hand, fold it in half, and put it on the floor". (3 points) Read and obey the following: CLOSE YOUR EYES (1 point) Write a sentence (1 point) Copy design (1 point)
	______	Total Score

ASSESS level of consciousness along a continuum________________________________

Alert Drowsy Stupor Coma

Appendix E

Hierarchical Cluster Analysis

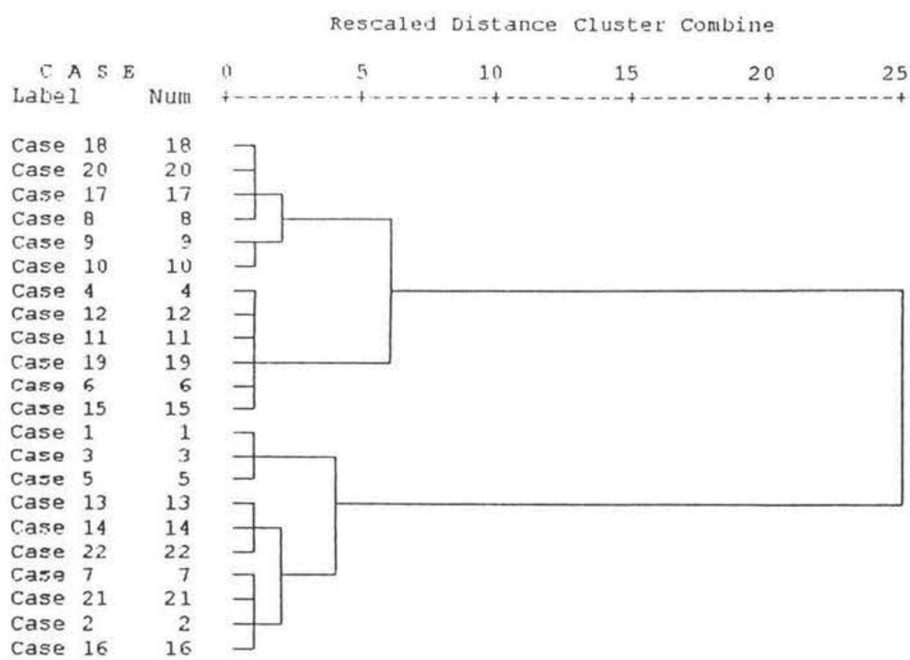
* * * * * * H I E R A R C H I C A L   C L U S T E R   A N A L Y S I S * * * * * *

Agglomeration Schedule using Ward Method

Stage	Clusters Combined Cluster 1	Cluster 2	Coefficient	Stage Cluster 1st Appears Cluster 1	Cluster 2	Next Stage
1	18	20	5.000000	0	0	3
2	4	12	13.000000	0	0	7
3	17	18	21.333332	0	1	5
4	1	3	32.833332	0	0	10
5	8	17	45.750000	0	3	17
6	7	21	60.250000	0	0	8
7	4	11	74.916664	2	0	9
8	2	7	91.750000	0	6	15
9	4	19	111.333336	7	0	16
10	1	5	131.166672	4	0	19
11	13	14	153.666672	0	0	14
12	9	10	177.166672	0	0	17
13	6	15	210.166672	0	0	16
14	13	22	247.666672	11	0	18
15	2	16	290.333344	8	0	18
16	4	6	350.250000	9	13	20
17	8	9	435.833344	5	12	20
18	2	13	558.690491	15	14	19
19	1	2	789.000000	10	18	21
20	4	8	1102.083374	16	17	21
21	1	4	2603.636230	19	20	0

* * * * * * H I E R A R C H I C A L   C L U S T E R   A N A L Y S I S * * * * * *

Dendrogram using Ward Method



Variables (Cluster Membership) Saved into Working File

CLU3_2 to CLU2_2 for Ward Method

Appendix F

ANOVA Tables

Table F.1 *Cluster ANOVA's with Scheffe Tests*

```
     Variable  LATOT        LIST A TOTAL
  By Variable  CLU3_1       Ward Method

                                Analysis of Variance

                                   Sum of          Mean           F       F
      Source            D.F.       Squares         Squares        Ratio   Prob.

Between Groups            2       1037.0061       518.5030      28.4508   .0000
Within Groups            19        346.2667        18.2246
Total                    21       1383.2727

     Variable  LATOT        LIST A TOTAL
  By Variable  CLU3_1       Ward Method

Multiple Range Tests:  Scheffe test with significance level .05

The difference between two means is significant if
  MEAN(J)-MEAN(I)  >= 3.0187 * RANGE * SQRT(1/N(I) + 1/N(J))
  with the following value(s) for RANGE: 3.75

   (*) Indicates significant differences which are shown in the lower triangle

                        G G G
                        r r r
                        p p p

                        3 2 1
   Mean       CLU3_1

   27.3333    Grp 3
   34.8333    Grp 2     *
   43.7000    Grp 1     * *


                     - - - - -  O N E W A Y  - - - - -

     Variable  LAONE        LIST A TRIAL ONE
  By Variable  CLU3_1       Ward Method

                                Analysis of Variance

                                   Sum of          Mean           F       F
      Source            D.F.       Squares         Squares        Ratio   Prob.

Between Groups            2         17.1879         8.5939       5.7766   .0110
Within Groups            19         28.2667         1.4877
Total                    21         45.4545

                     - - - - -  O N E W A Y  - - - - -

     Variable  LAONE        LIST A TRIAL ONE
  By Variable  CLU3_1       Ward Method

Multiple Range Tests:  Scheffe test with significance level .05

The difference between two means is significant if
  MEAN(J)-MEAN(I)  >= .8625 * RANGE * SQRT(1/N(I) + 1/N(J))
  with the following value(s) for RANGE: 3.75

   (*) Indicates significant differences which are shown in the lower triangle

                        G G G
                        r r r
                        p p p

                        3 2 1
   Mean       CLU3_1

   3.1667     Grp 3
   4.3333     Grp 2
   5.3000     Grp 1     *
```

Table F.1 *Cluster ANOVA's with Scheffe Tests*

```
      Variable  LAFIVE       LIST A TRIAL FIVE
   By Variable  CLU3_1       Ward Method

                                    Analysis of Variance

                                     Sum of          Mean              F       F
         Source              D.F.    Squares         Squares         Ratio   Prob.

Between Groups                 2      50.3394        25.1697       12.4430   .0004
Within Groups                 19      38.4333         2.0228
Total                         21      88.7727

                           - - - - -  O N E W A Y  - - - - -

      Variable  LAFIVE       LIST A TRIAL FIVE
   By Variable  CLU3_1       Ward Method

Multiple Range Tests:  Scheffe test with significance level .05

The difference between two means is significant if
  MEAN(J)-MEAN(I)  >= 1.0057 * RANGE * SQRT(1/N(I) + 1/N(J))
  with the following value(s) for RANGE: 3.75

   (*) Indicates significant differences which are shown in the lower triangle

                             G G G
                             r r r
                             p p p

                             3 2 1
    Mean         CLU3_1

    7.1667       Grp 3
    9.0000       Grp 2
   10.8000       Grp 1       *


                           - - - - -  O N E W A Y  - - - - -

      Variable  LBFREE       LIST B FREE RECALL
   By Variable  CLU3_1       Ward Method

                                    Analysis of Variance

                                     Sum of          Mean              F       F
         Source              D.F.    Squares         Squares         Ratio   Prob.

Between Groups                 2        .7879          .3939         .1554   .8572
Within Groups                 19      48.1667         2.5351
Total                         21      48.9545


                           - - - - -  O N E W A Y  - - - - -

      Variable  LBFREE       LIST B FREE RECALL
   By Variable  CLU3_1       Ward Method

Multiple Range Tests:  Scheffe test with significance level .05

The difference between two means is significant if
  MEAN(J)-MEAN(I)  >= 1.1259 * RANGE * SQRT(1/N(I) + 1/N(J))
  with the following value(s) for RANGE: 3.75

- No two groups are significantly different at the  .050 level
```

Table F. 1 *Cluster ANOVA's with Scheffe Tests*

```
      Variable  LASFREE      LIST A SHORT DELAY FREE RECALL
   By Variable  CLU3_1       Ward Method

                                    Analysis of Variance

                                   Sum of         Mean             F       F
     Source               D.F.     Squares        Squares          Ratio   Prob.

Between Groups              2       86.6727        43.3364       20.5334   .0000
Within Groups              19       40.1000         2.1105
Total                      21      126.7727


      Variable  LASFREE      LIST A SHORT DELAY FREE RECALL
   By Variable  CLU3_1       Ward Method

Multiple Range Tests:  Scheffe test with significance level .05

The difference between two means is significant if
  MEAN(J)-MEAN(I)  >= 1.0273 * RANGE * SQRT(1/N(I) + 1/N(J))
  with the following value(s) for RANGE: 3.75

   (*) Indicates significant differences which are shown in the lower triangle

                          G G G
                          r r r
                          p p p

                          3 2 1
     Mean       CLU3_1

     4.0000     Grp 3
     6.0000     Grp 2
     8.7000     Grp 1     * *


                    - - - - -  O N E W A Y  - - - - -


      Variable  LASCUED      LIST A SHORT DELAY CUED RECALL
   By Variable  CLU3_1       Ward Method

                                    Analysis of Variance

                                   Sum of         Mean             F       F
     Source               D.F.     Squares        Squares          Ratio   Prob.

Between Groups              2      100.3333        50.1667       17.1228   .0001
Within Groups              19       55.6667         2.9298
Total                      21      156.0000


      Variable  LASCUED      LIST A SHORT DELAY CUED RECALL
   By Variable  CLU3_1       Ward Method

Multiple Range Tests:  Scheffe test with significance level .05

The difference between two means is significant if
  MEAN(J)-MEAN(I)  >= 1.2103 * RANGE * SQRT(1/N(I) + 1/N(J))
  with the following value(s) for RANGE: 3.75

   (*) Indicates significant differences which are shown in the lower triangle

                          G G G
                          r r r
                          p p p

                          3 2 1
     Mean       CLU3_1

     5.8333     Grp 3
     8.8333     Grp 2     *
    11.0000     Grp 1     *
```

Table F.1 *Cluster ANOVA's with Scheffe Tests*

```
       Variable  LALFREE      LIST A LONG DELAY FREE RECALL
    By Variable  CLU3_1       Ward Method

                                   Analysis of Variance

                                   Sum of           Mean              F        F
        Source              D.F.   Squares          Squares           Ratio    Prob.

Between Groups                2    167.8848         83.9424           62.7093  .0000
Within Groups                19     25.4333          1.3386
Total                        21    193.3182

       Variable  LALFREE      LIST A LONG DELAY FREE RECALL
    By Variable  CLU3_1       Ward Method

Multiple Range Tests:  Scheffe test with significance level .05

The difference between two means is significant if
  MEAN(J)-MEAN(I)  >= .8181 * RANGE * SQRT(1/N(I) + 1/N(J))
  with the following value(s) for RANGE: 3.75

   (*) Indicates significant differences which are shown in the lower triangle

                          G G G
                          r r r
                          p p p

                          3 2 1
     Mean      CLU3_1

     3.6667    Grp 3
     7.0000    Grp 2      *
    10.3000    Grp 1      * *

                        - - - - -  O N E W A Y  - - - - -

       Variable  LALCUED      LIST A LONG DELAY CUED RECALL
    By Variable  CLU3_1       Ward Method

                                   Analysis of Variance

                                   Sum of           Mean              F        F
        Source              D.F.   Squares          Squares           Ratio    Prob.

Between Groups                2    110.9333         55.4667           30.0532  .0000
Within Groups                19     35.0667          1.8456
Total                        21    146.0000

       Variable  LALCUED      LIST A LONG DELAY CUED RECALL
    By Variable  CLU3_1       Ward Method

Multiple Range Tests:  Scheffe test with significance level .05

The difference between two means is significant if
  MEAN(J)-MEAN(I)  >= .9606 * RANGE * SQRT(1/N(I) + 1/N(J))
  with the following value(s) for RANGE: 3.75

   (*) Indicates significant differences which are shown in the lower triangle

                          G G G
                          r r r
                          p p p

                          3 2 1
     Mean      CLU3_1

     5.6667    Grp 3
     8.8333    Grp 2      *
    11.1000    Grp 1      * *
```

Table F.1 *Cluster ANOVA's with Scheffe Tests*

```
   Variable  SCRATIO     SEMANTIC CATEGORY RATIO
By Variable  CLU3_1      Ward Method

                                   Analysis of Variance
```

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	2	243.4909	121.7455	13.4800	.0002
Within Groups	19	171.6000	9.0316		
Total	21	415.0909			

```
                         - - - - -  O N E W A Y  - - - - -

   Variable  SCRATIO     SEMANTIC CATEGORY RATIO
By Variable  CLU3_1      Ward Method

Multiple Range Tests:  Scheffe test with significance level .05

The difference between two means is significant if
  MEAN(J)-MEAN(I)  >= 2.1250 * RANGE * SQRT(1/N(I) + 1/N(J))
  with the following value(s) for RANGE: 3.75

   (*) Indicates significant differences which are shown in the lower triangle

                         G G G
                         r r r
                         p p p

                         3 2 1
    Mean      CLU3_1

    3.0000    Grp 3
    6.0000    Grp 2
   10.8000    Grp 1      * *

                         - - - - -  O N E W A Y  - - - - -

   Variable  RECOGH      RECOGNITION HITS
By Variable  CLU3_1      Ward Method

                                   Analysis of Variance
```

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	2	15.3576	7.6788	7.5856	.0038
Within Groups	19	19.2333	1.0123		
Total	21	34.5909			

```
   Variable  RECOGH      RECOGNITION HITS
By Variable  CLU3_1      Ward Method

Multiple Range Tests:  Scheffe test with significance level .05

The difference between two means is significant if
  MEAN(J)-MEAN(I)  >= .7114 * RANGE * SQRT(1/N(I) + 1/N(J))
  with the following value(s) for RANGE: 3.75

   (*) Indicates significant differences which are shown in the lower triangle

                         G G G
                         r r r
                         p p p

                         3 2 1
    Mean      CLU3_1

   12.5000    Grp 3
   14.3333    Grp 2      *
   14.4000    Grp 1      *
```

Table F. 2 *Age comparisons (young/old) in the PD group*

```
* * * * * * A n a l y s i s   o f   V a r i a n c e -- design  1 * * * * * *

 Tests of Significance for LATOT using UNIQUE sums of squares
 Source of Variation          SS       DF        MS         F  Sig of F

 WITHIN+RESIDUAL         1016.45       19     53.50
 REGRESSION               355.61        1    355.61      6.65      .018
 YOUNGOLD                 156.40        1    156.40      2.92      .104

 (Model)                  366.82        2    183.41      3.43      .054
 (Total)                 1383.27       21     65.87

 R-Squared =           .265
 Adjusted R-Squared =  .188

 - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - -
 Effect Size Measures and Observed Power at the .0500 Level
                       Partial Noncen-
 Source of Variation   ETA Sqd trality     Power

 Regression               .259   6.647      .685
 YOUNGOLD                 .133   2.924      .369

* * * * * * A n a l y s i s   o f   V a r i a n c e -- design  1 * * * * * *

 Tests of Significance for LAONE using UNIQUE sums of squares
 Source of Variation          SS       DF        MS         F  Sig of F

 WITHIN+RESIDUAL           35.53       19      1.87
 REGRESSION                 8.82        1      8.82      4.72      .043
 YOUNGOLD                   2.69        1      2.69      1.44      .245

 (Model)                    9.93        2      4.96      2.66      .096
 (Total)                   45.45       21      2.16

 R-Squared =           .218
 Adjusted R-Squared =  .136

 - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - -
 Effect Size Measures and Observed Power at the .0500 Level
                       Partial Noncen-
 Source of Variation   ETA Sqd trality     Power

 Regression               .199   4.720      .539
 YOUNGOLD                 .070   1.437      .205

* * * * * * A n a l y s i s   o f   V a r i a n c e -- design  1 * * * * * *

 Tests of Significance for LAFIVE using UNIQUE sums of squares
 Source of Variation          SS       DF        MS         F  Sig of F

 WITHIN+RESIDUAL           76.48       19      4.03
 REGRESSION                12.28        1     12.28      3.05      .097
 YOUNGOLD                   7.65        1      7.65      1.90      .184

 (Model)                   12.29        2      6.15      1.53      .243
 (Total)                   88.77       21      4.23

 R-Squared =           .138
 Adjusted R-Squared =  .048

 - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - - -
 Effect Size Measures and Observed Power at the .0500 Level
                       Partial Noncen-
 Source of Variation   ETA Sqd trality     Power

 Regression               .138   3.052      .382
 YOUNGOLD                 .091   1.901      .258
```

Table F.2 *Age comparisons (young/old) in the PD group*

Tests of Significance for LBFREE using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	42.16	19	2.22		
REGRESSION	6.75	1	6.75	3.04	.097
YOUNGOLD	4.57	1	4.57	2.06	.167
(Model)	6.79	2	3.40	1.53	.242
(Total)	48.95	21	2.33		

R-Squared = .139
Adjusted R-Squared = .048

Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncen- trality	Power
Regression	.138	3.044	.381
YOUNGOLD	.098	2.060	.275

* * * * * * A n a l y s i s o f V a r i a n c e -- design 1 * * * * * *

Tests of Significance for LASFREE using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	88.85	19	4.68		
REGRESSION	34.71	1	34.71	7.42	.013
YOUNGOLD	11.81	1	11.81	2.53	.128
(Model)	37.92	2	18.96	4.05	.034
(Total)	126.77	21	6.04		

R-Squared = .299
Adjusted R-Squared = .225

Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncen- trality	Power
Regression	.281	7.423	.732
YOUNGOLD	.117	2.526	.327

* * * * * * A n a l y s i s o f V a r i a n c e -- design 1 * * * * * *

Tests of Significance for LASCUED using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	112.08	19	5.90		
REGRESSION	43.18	1	43.18	7.32	.014
YOUNGOLD	20.74	1	20.74	3.52	.076
(Model)	43.92	2	21.96	3.72	.043
(Total)	156.00	21	7.43		

R-Squared = .282
Adjusted R-Squared = .206

Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncen- trality	Power
Regression	.278	7.320	.726
YOUNGOLD	.156	3.516	.428

Table F.2 *Age comparisons (young/old) in the PD group*

Tests of Significance for LALFREE using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	159.11	19	8.37		
REGRESSION	34.20	1	34.20	4.08	.058
YOUNGOLD	20.34	1	20.34	2.43	.136
(Model)	34.21	2	17.10	2.04	.157
(Total)	193.32	21	9.21		

R-Squared = .177
Adjusted R-Squared = .090

- -

Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncen-trality	Power
Regression	.177	4.084	.482
YOUNGOLD	.113	2.428	.316

* * * * * * A n a l y s i s o f V a r i a n c e -- design 1 * * * * * *

Tests of Significance for LALCUED using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	110.38	19	5.81		
REGRESSION	33.97	1	33.97	5.85	.026
YOUNGOLD	13.74	1	13.74	2.37	.141
(Model)	35.62	2	17.81	3.07	.070
(Total)	146.00	21	6.95		

R-Squared = .244
Adjusted R-Squared = .164

- -

Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncen-trality	Power
Regression	.235	5.846	.629
YOUNGOLD	.111	2.366	.309

* * * * * * A n a l y s i s o f V a r i a n c e -- design 1 * * * * * *

Tests of Significance for SCRATIO using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	365.23	19	19.22		
REGRESSION	49.83	1	49.83	2.59	.124
YOUNGOLD	28.89	1	28.89	1.50	.235
(Model)	49.86	2	24.93	1.30	.297
(Total)	415.09	21	19.77		

R-Squared = .120
Adjusted R-Squared = .027

- -

Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncen-trality	Power
Regression	.120	2.592	.334
YOUNGOLD	.073	1.503	.212

Table F.2 *Age comparisons (young/old) in the PD group*

Tests of Significance for RECOGH using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	28.85	19	1.52		
REGRESSION	.47	1	.47	.31	.585
YOUNGOLD	.84	1	.84	.56	.465
(Model)	5.74	2	2.87	1.89	.178
(Total)	34.59	21	1.65		

R-Squared = .166
Adjusted R-Squared = .078

- -

Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncentrality	Power
Regression	.016	.309	.066
YOUNGOLD	.028	.556	.132

* * * * * * A n a l y s i s o f V a r i a n c e -- design 1 * * * * * *

Tests of Significance for FPOS using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	158.01	19	8.32		
REGRESSION	23.66	1	23.66	2.84	.108
YOUNGOLD	25.00	1	25.00	3.01	.099
(Model)	27.45	2	13.72	1.65	.218
(Total)	185.45	21	8.83		

R-Squared = .148
Adjusted R-Squared = .058

- -

Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncentrality	Power
Regression	.130	2.845	.360
YOUNGOLD	.137	3.006	.377

* * * * * * A n a l y s i s o f V a r i a n c e -- design 1 * * * * * *

Tests of Significance for DISCRIM using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	926.62	18	51.48		
REGRESSION	200.91	1	200.91	3.90	.064
YOUNGOLD	90.24	1	90.24	1.75	.202
(Model)	205.24	2	102.62	1.99	.165
(Total)	1131.86	20	56.59		

R-Squared = .181
Adjusted R-Squared = .090

- -

Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncentrality	Power
Regression	.178	3.903	.463
YOUNGOLD	.089	1.753	.240

Table F.3 *Gender comparisons in the PD group*

```
* * * * * * A n a l y s i s   o f   V a r i a n c e -- design  1 * * * * * *

 Tests of Significance for LATOT using UNIQUE sums of squares
 Source of Variation          SS      DF       MS        F  Sig of F

 WITHIN+RESIDUAL         1102.75      19    58.04
 REGRESSION               274.28       1   274.28     4.73      .043
 GENDER                    70.10       1    70.10     1.21      .285

 (Model)                  280.52       2   140.26     2.42      .116
 (Total)                 1383.27      21    65.87

 R-Squared =           .203
 Adjusted R-Squared =  .119

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 Effect Size Measures and Observed Power at the .0500 Level
                        Partial Noncen-
 Source of Variation    ETA Sqd trality     Power

 Regression                .199   4.726      .540
 GENDER                    .060   1.208      .181

* * * * * * A n a l y s i s   o f   V a r i a n c e -- design  1 * * * * * *

 Tests of Significance for LAONE using UNIQUE sums of squares
 Source of Variation          SS      DF       MS        F  Sig of F

 WITHIN+RESIDUAL           36.10      19     1.90
 REGRESSION                 9.22       1     9.22     4.85      .040
 GENDER                     2.11       1     2.11     1.11      .305

 (Model)                    9.35       2     4.68     2.46      .112
 (Total)                   45.45      21     2.16

 R-Squared =           .206
 Adjusted R-Squared =  .122

* * * * * * A n a l y s i s   o f   V a r i a n c e -- design  1 * * * * * *

 Tests of Significance for LAFIVE using UNIQUE sums of squares
 Source of Variation          SS      DF       MS        F  Sig of F

 WITHIN+RESIDUAL           79.47      19     4.18
 REGRESSION                 7.81       1     7.81     1.87      .188
 GENDER                     4.67       1     4.67     1.12      .304

 (Model)                    9.31       2     4.65     1.11      .349
 (Total)                   88.77      21     4.23

 R-Squared =           .105
 Adjusted R-Squared =  .011

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 Effect Size Measures and Observed Power at the .0500 Level
                        Partial Noncen-
 Source of Variation    ETA Sqd trality     Power

 Regression                .089   1.866      .254
 GENDER                    .056   1.117      .172
```

Table F.3 *Gender comparisons in the PD group*

```
Tests of Significance for LBFREE using UNIQUE sums of squares
Source of Variation          SS      DF       MS         F  Sig of F

WITHIN+RESIDUAL           46.73      19     2.46
REGRESSION                 1.84       1     1.84       .75      .398
GENDER                      .01       1      .01       .00      .957

(Model)                    2.23       2     1.11       .45      .642
(Total)                   48.95      21     2.33

R-Squared =           .046
Adjusted R-Squared =  .000

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Effect Size Measures and Observed Power at the .0500 Level
                      Partial Noncer-
Source of Variation   ETA Sqd trality     Power

Regression               .038    .748      .154
GENDER                   .000    .003      .042


* * * * * * A n a l y s i s   o f   V a r i a n c e -- design   1 * * * * * *

Tests of Significance for LASFREE using UNIQUE sums of squares
Source of Variation          SS      DF       MS         F  Sig of F

WITHIN+RESIDUAL           88.97      19     4.68
REGRESSION                36.07       1    36.07      7.70      .012
GENDER                    11.70       1    11.70      2.50      .130

(Model)                   37.80       2    18.90      4.04      .035
(Total)                  126.77      21     6.04

R-Squared =           .298
Adjusted R-Squared =  .224

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Effect Size Measures and Observed Power at the .0500 Level
                      Partial Noncen-
Source of Variation   ETA Sqd trality     Power

Regression               .288   7.702      .748
GENDER                   .116   2.498      .324

* * * * * * A n a l y s i s   o f   V a r i a n c e -- design   1 * * * * * *

Tests of Significance for LASCUED using UNIQUE sums of squares
Source of Variation          SS      DF       MS         F  Sig of F

WITHIN+RESIDUAL          126.89      19     6.68
REGRESSION                28.85       1    28.85      4.32      .051
GENDER                     5.93       1     5.93       .89      .358

(Model)                   29.11       2    14.55      2.18      .141
(Total)                  156.00      21     7.43

R-Squared =           .187
Adjusted R-Squared =  .101

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Effect Size Measures and Observed Power at the .0500 Level
                      Partial Noncen-
Source of Variation   ETA Sqd trality     Power

Regression               .185   4.320      .504
GENDER                   .045    .888      .158
```

Table F.3 *Gender comparisons in the PD group*

Tests of Significance for LALFREE using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	172.80	19	9.09		
REGRESSION	19.43	1	19.43	2.14	.160
GENDER	6.65	1	6.65	.73	.403
(Model)	20.52	2	10.26	1.13	.344
(Total)	193.32	21	9.21		

R-Squared = .106
Adjusted R-Squared = .012

Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncen- trality	Power
Regression	.101	2.137	.284
GENDER	.037	.731	.153

* * * * * * A n a l y s i s   o f   V a r i a n c e -- design 1 * * * * * *

Tests of Significance for LALCUED using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	112.06	19	5.90		
REGRESSION	31.61	1	31.61	5.36	.032
GENDER	12.07	1	12.07	2.05	.169
(Model)	33.94	2	16.97	2.88	.081
(Total)	146.00	21	6.95		

R-Squared = .232
Adjusted R-Squared = .152

Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncen- trality	Power
Regression	.220	5.360	.592
GENDER	.097	2.046	.274

* * * * * * A n a l y s i s   o f   V a r i a n c e -- design 1 * * * * * *

Tests of Significance for SCRATIO using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	347.69	19	18.30		
REGRESSION	45.58	1	45.58	2.49	.131
GENDER	46.43	1	46.43	2.54	.128
(Model)	67.40	2	33.70	1.84	.186
(Total)	415.09	21	19.77		

R-Squared = .162
Adjusted R-Squared = .074

Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncen- trality	Power
Regression	.116	2.491	.323
GENDER	.118	2.537	.328

Table F.3 *Gender comparisons in the PD group*

Tests of Significance for RECOGH using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	29.67	19	1.56		
REGRESSION	4.47	1	4.47	2.86	.107
GENDER	.02	1	.02	.01	.907
(Model)	4.92	2	2.46	1.58	.233
(Total)	34.59	21	1.65		

R-Squared = .142
Adjusted R-Squared = .052

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Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncen-trality	Power
Regression	.131	2.863	.362
GENDER	.001	.014	.043

* * * * * * A n a l y s i s o f V a r i a n c e -- design 1 * * * * * *

Tests of Significance for FPOS using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	169.00	19	8.89		
REGRESSION	7.96	1	7.96	.90	.356
GENDER	14.01	1	14.01	1.58	.225
(Model)	16.45	2	8.23	.92	.414
(Total)	185.45	21	8.83		

R-Squared = .089
Adjusted R-Squared = .000

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Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncen-trality	Power
Regression	.045	.895	.158
GENDER	.077	1.575	.220

* * * * * * A n a l y s i s o f V a r i a n c e -- design 1 * * * * * *

Tests of Significance for DISCRIM using UNIQUE sums of squares

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN+RESIDUAL	978.64	18	54.37		
REGRESSION	145.24	1	145.24	2.67	.120
GENDER	38.22	1	38.22	.70	.413
(Model)	153.22	2	76.61	1.41	.270
(Total)	1131.86	20	56.59		

R-Squared = .135
Adjusted R-Squared = .039

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Effect Size Measures and Observed Power at the .0500 Level

Source of Variation	Partial ETA Sqd	Noncen-trality	Power
Regression	.129	2.671	.341
GENDER	.038	.703	.151