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Verbal Learning and Memory in Patients with Dementia with Lewy Bodies or Parkinson's Disease with Dementia

J. Vincent Filoteo[1,2], **David P. Salmon**[3], **Dawn M. Schiehser**[1], **Amy E. Kane**[3], **Joanne M. Hamilton**[3], **Laurie M. Rilling**[4], **John A. Lucas**[5], **Vanessa Zizak**[2], and **Douglas R. Galasko**[2,3]

[1]Department of Psychiatry, UCSD

[2]VA San Diego Healthcare System

[3]Department of Neurosciences, UCSD

[4]Department of Psychiatry, UT Southwestern Medical Center

[5]Departments of Psychiatry and Psychology, Mayo Clinic, Jacksonville

Abstract

This study compared verbal learning and memory in patients with autopsy-confirmed dementia with Lewy Bodies (DLB) and patients with Parkinson's disease with dementia (PDD). Twenty-four DLB patients, 24 PDD patients, and 24 normal comparison participants were administered the California Verbal Learning Test. The three groups were matched on demographic variables and the two patient groups were matched on the Mattis Dementia Rating Scale. The results indicated that DLB patients recalled less information than PDD patients on all but one recall measure and displayed a more rapid rate of forgetting. In contrast, the PDD patients committed a greater percent of perseveration errors than the DLB patients. The two groups did not differ in the percentage of recall intrusion errors or any measures of recognition. A discriminant function analysis (DFA) using short delay cued recall, percent perseveration errors, and list b recall, differentiated the DLB and PDD groups with 81.3% accuracy. The application of the DFA algorithm to another sample of 42 PDD patients resulted in a 78.6% correct classification rate. The results suggest that, despite equivalent levels of general cognitive impairment, patients with DLB or PDD exhibit a different pattern of verbal learning and memory deficits.

Dementia with Lewy Bodies (DLB) and Parkinson's Disease with Dementia (PDD) are both characterized by significant cognitive impairment and movement abnormalities. The primary pathology associated with DLB and PDD are Lewy neurites and Lewy body inclusions that are primarily composed of α-synuclein aggregation. Both diseases are associated with profound cognitive deficits as well as motor impairment, and the primary clinical distinction is the relative timing of the onset of cognitive relative to motor symptoms, with the onset of cognitive symptoms occurring less than one year after the onset of motor symptoms in DLB and greater than one year in PDD (McKeith et al., 2005). The cognitive deficits associated with both PDD and DLB include impairment in executive functioning, memory, attention, and visual cognition. Other features include hallucinations, fluctuating cognition, sleep abnormalities, and autonomic symptoms (Lippa et al., 2007).

Although there is potentially greater nigral pathology in PDD as compared to DLB (Tsuboi & Dickson, 2005), these diseases are difficult to distinguish neuropathologically and

Correspondence concerning this article can be addressed to J. Vincent Filoteo, Psychology Service 116B, 3350 La Jolla Village Dr., San Diego, CA, 92161 or to vfiloteo@ucsd.edu .

neurochemically (Aarsland, Ballard, & Halliday, 2004; Burton, McKeith, Burn, Williams, & O'Brien, 2004; Harding & Halliday, 2001; Tiraboschi et al., 2000), calling into question whether they are nosologically distinct. Past studies contrasting the cognitive abilities of these patient groups have also been equivocal in demonstrating different patterns of impairment, with some revealing a different level and/or profile of cognitive deficits in the two groups (Aarsland et al., 2003; Cormack, Gray, Ballard, & Tovee, 2004; Downes et al., 1998) and others showing no differences (Ballard et al., 2002; Cahn-Weiner et al., 2003; Cormack, Aarsland, Ballard, & Tovee, 2004; Horimoto et al., 2003; Noe et al., 2004). These discrepant findings could be due to a number of methodological issues, including (a) the failure to match DLB and PDD patients on global level of cognitive impairment, (b) the misdiagnosis of patients, and (c) the use of insensitive cognitive measures.

Past studies that have taken these important methodological issues into consideration have identified subtle differences in the pattern of cognitive impairment in these two groups. For example, Aarsland et al. (2003) found that mild to moderately demented DLB patients performed worse than PDD patients on the Conceptualization subscale of the Mattis Dementia Rating Scale (MDRS) (Mattis, 1988), but not on the Attention, Initiation/Perseveration, Construction, or Memory subscales. Importantly, this study took two critical methodological issues into account in that the diagnosis of the DLB patients was confirmed at autopsy and the DLB and PDD groups were matched for their overall level of cognitive impairment (based on total score of the MDRS). A methodological problem remains with this study, however, because the failure to identify differences between the two groups in cognitive abilities other than conceptualization might be related to the relative insensitivity of the MDRS subscales. To address this issue, Kane and colleagues (Kane et al., 2008) compared PDD patients and patients with autopsy-confirmed DLB on a more extensive neuropsychological test battery that measured memory, language, visuospatial processes, executive functions, and attention/ psychomotor speed. Consistent with a recent study by Noe and colleagues (Noe et al., 2004), results showed no significant differences between DLB and PDD patients in their cognitive profiles (although there was a trend). It should be noted, however, that the PDD and DLB cognitive deficit profiles in the Kane et al. study differed from those of patients with Alzheimer's disease (AD) in distinct ways. Specifically, patients with DLB, but not those with PDD, performed worse than AD patients on measures of visuospatial abilities and executive functions, whereas patients with PDD, but not those with DLB, performed worse than AD patients on measures of attention/psychomotor speed. In addition, patients with PDD, but not those with DLB, performed better than AD patients on measures of memory. These results suggest that, while the majority of studies to date have found few differences between carefully matched groups of DLB and PDD patients, subtle differences that can only be detected with rigorous cognitive testing methods may exist.

The purpose of the present study was to further examine potential differences in the ognitive deficits exhibited by patients with DLB or PDD. The study focused on memory processes because this cognitive domain tends to be one of the most sensitive in distinguishing among patients with various forms of dementia (Butters, Delis, & Lucas, 1995), and there is some evidence that DLB results in greater atrophy than PDD in brain regions that are believed to be involved in declarative memory processes, such as the medial temporal lobes (Tam, Burton, McKeith, Burn, & O'Brien, 2005). We included autopsy-confirmed patients with DLB who were matched to a group of PDD patients on global level of cognitive impairment (based on the MDRS total score) and important demographic factors such as age, education, and gender. We compared the groups' performances on several key measures from the California Verbal Learning Test (CVLT) (Delis, Kramer, Kaplan, & Ober, 1987), a rigorous test constructed to quantify specific aspects of verbal learning and memory that had not been considered in previous clinical measures. For example, the CVLT not only provides quantitative measures of learning and recall, but also evaluates the manner in which the subject approaches the task,

measures the number and type of learning and recall errors produced, and quantitatively and qualitatively measures recognition memory in addition to recall. These indices have proven invaluable in characterizing the memory deficits associated with various forms of dementia, including PDD (Filoteo et al., 1997; Zizak et al., 2005), DLB (Hamilton et al., 2004; Simard et al., 2002), Huntington's disease (HD) (Lundervold, Karlsen, & Reinvang, 1994; Massman, Delis, Butters, Levin, & Salmon, 1990; Zizak et al., 2005), and AD (Bayley et al., 2000; Pillon et al., 1994), and in identifying differences in the memory processes affected by these various neurodegenerative disorders. While memory profiles of PDD and DLB have been studied separately using the CVLT (Hamilton et al., 2004; Zizak et al., 2005), there has been no study to date which has directly compared the performance of these groups on this measure. However, those studies that have examined DLB patients on the CVLT have compared their performances to those of patients with AD, and have tended to demonstrate greater levels of memory impairment in patients with AD (Hamilton et al., 2004; Simard et al., 2002). In contrast, studies of patients with PDD on the CVLT have shown that these patients typically do not demonstrate improvement on recognition testing, such as patients with HD (Zizak et al., 2005). Based on our previous work suggesting that DLB patients may have greater memory impairment than PDD (Kane et al., 2008), we hypothesized that the DLB patients would perform worse than the PDD patients on the various indices of the CVLT.

METHOD

Subjects

Twenty-four patients with DLB, 24 patients with PDD, and 24 normal comparison (NC) participants were included in the study. The mean ages (with standard deviations and ranges) of the three groups were as follows: PDD = 72.50 (*SD* = 6.87; range 54 – 84), DLB = 74.49 (*SD*=5.80; range 63 – 86), NC = 73.04 (*SD* = 5.18; range 63 – 83), and did not differ statistically based on a one-way ANOVA, *F*=0.7. The mean levels of education (with standard deviations and ranges) in years were as follows: PDD = 14.21 (*SD* = 2.40; 11 – 20), DLB = 15.21 (*SD* = 2.52; 9 – 20), NC = 15.25 (*SD* = 2.35; range 9 – 19), and did not differ statistically based on a one-way ANOVA, *F*=1.4. The male to female gender ratios for the three groups were the following: PDD = 19:5, DLB = 17:7, NC = 17:7, and based on a χ^2 were not different statistically. The mean MDRS scores (with standard deviations and ranges) for the three groups were as follows: PDD = 115.83 (*SD* = 9.44; range 98 – 137), DLB = 114.3 (*SD* = 10.53; range 91 – 136), NC = 138.71 (SD = 2.77; 133 – 144), and as expected, there was a significant difference among the three groups in total score on the MDRS based on a one-way ANOVA, *F*=64.7, *p*<.001, with the NC group having greater MDRS total scores than both the PDD and DLB groups based on Tukey HSD post-hoc comparisons (*p*<.001 for both contrasts); however, the DLB and PDD groups did not differ on the MDRS. Thus, overall the three groups were well matched demographically, and the PDD and DLB groups were well matched in terms of global cognitive impairment.

All DLB patients had been participants at the University of California at San Diego (UCSD) Alzheimer's Disease Research Center (ADRC), through which they received annual medical, neurologic, and neuropsychological evaluations. Data on these patients were collected between 1986 and 1998. Autopsy was performed according to established UCSD ADRC protocols (Hansen & Samuel, 1997). In brief, the left hemibrain was fixed by immersion in 10% formalin for 5 to 7 days. The paraffin-embedded blocks from midfrontal, rostral superior temporal, and inferior parietal areas of neocortex, hippocampus, entorhinal cortex, basal ganglia/substantia innominata, mesencephalon, and pons were cut at 7 μm thickness for hematoxylin and eosin (H–E) and thioflavin-S staining. Neuritic plaques were assessed on thioflavin-S or Bielschowsky silver stains of cerebral neocortex. Total plaque, neuritic plaque, and neurofibrillary tangle counts were determined by the same examiner with the same criteria

used consistently. Lewy bodies were detected using H-E or antiubiquitin immunostaining as recommended by the Consortium on DLB criteria for a pathologic diagnosis of DLB (McKeith et al., 1996), and confirmed using alpha-synuclein immunostaining. Cases were neuropathologically diagnosed with DLB only if Lewy bodies were found in the locus coeruleus, substantia nigra, or nucleus basalis of Mynert, as well as in the neocortex. In addition, each brain was staged for the degree of neurofibrillary pathology by a modification (Hansen & Samuel, 1997) of the method of Braak and Braak (Braak & Braak, 1991). Five DLB patients were in modified Braak stage I, 4 patients were in modified Braak stage III, 6 patients were in modified Braak stage IV, 6 patients were in modified Braak stage V, and 3 patients were in modified Braak stage VI. At the time of testing, the DLB patients had a mean length of illness of 3.8 years (*SD*=1.7). One patient was taking perphenazine, 1 patient was taking verapamil and oxybutynin, 1 patient was taking clozapine, 1 patient was taking pemoline, 1 patient was taking donepezil, and 1 patient was taking donepezil and risperidone. The remaining 18 DLB patients were taking no psychotropic medication. There were no differences in CVLT performance on the indices presented below between patients who were on psychotropic medications compared to those who were not on psychotropic medications. All DLB patients were diagnosed with dementia at their clinical evaluation based on the presence of a memory impairment, impairment in one other cognitive domain, and a functional impact of such deficits. Of the 24 DLB patients, 4 were clinically diagnosed with DLB, and 19 with possible (n=4) or probable (n=15) Alzheimer's disease (AD). The diagnosis of probable AD was made when dementia was present in the absence of other neurologic, psychiatric, or systemic disorders sufficent or likely to cause dementia, but disease onset, presentation, or course was atypical for AD. Given that some of these patients were diagnosed clinically prior to the development of formal clinical DLB criteria, it would be expected that a large number of these patients would have been considered to have AD at the time of their initial evaluation. Importantly, however, all DLB patients presented with dementia without motor impairment for at least 12 months (McKeith *et al.*, 1996) and a diagnosis of idiopathic Parkinson's disease was never considered in any of these cases during their clinical evaluations. The CVLT performance of the majority of DLB patients was presented previously as part of another study (Hamilton et al., 2004).

Parkinson's disease was diagnosed in the PDD group by a board-certified neurologist with specialty training in movement disorders based on the presence of 2 of 4 cardinal features of the disease (tremor plus rigidity, bradykinesia, or postural instability) (Larsen, Dupont, & Tandberg, 1994). Patients were only given the diagnosis of Parkinson's disease if they were responsive to dopaminergic therapy and they did not present with atypical findings (e.g., bilateral motor symptoms at onset). The diagnosis of dementia in the PDD group was based on the presence of memory impairment, impairment in one other cognitive domain, and a functional impact of such deficits (i.e., DSM-IV criteria). For each of the PDD patients, motor symptoms were present for at least one year prior to the onset of their cognitive impairment (McKeith et al., 1996). The 24 PDD patients included in the main analysis were drawn from a total sample of 66 PDD patients tested between 1995 and 2005. Those PDD patients included were matched to the DLB patients based on their overall scores on the MDRS, age, level of education, and gender. An attempt was made to match each individual DLB patient with an individual PDD patient based on these variables, and when this could not be done, we matched based on the variables in this order of importance: MDRS, age, level of education, and gender. The PDD p0061tients were evaluated as part of either a research protocol or a clinical evaluation at one of three different sites: the University of Utah in Salt Lake City, Mayo Clinic in Jacksonville, Florida, or Brown University in Providence, Rhode Island. The PDD patients had a mean length of illness of 9.1 years (*SEM*=1.3) and a mean Hoehn and Yahr (1967) rating of 2.6 (*SD*=0.2). Twenty-two of the 24 PDD patients were taking some form of dopamine replacement medication (i.e., carbidopa-levodopa, levodopa) at the time of testing and 2 were also taking an anticholinergic medication (i.e., trihexyphenidyl).[1] In addition, 6 of the PDD

patients were taking l-depryl, 3 were taking pramipexole, 1 was taking ropinirole, 2 were taking entacapone, and 1 was taking amantadine at the time of testing. The diagnosis of Parkinson's disease (PD) was confirmed by autopsy in two out of two cases. However, previous studies (Aarsland, Perry, Brown, Larsen, & Ballard, 2005) with PDD patients using similar clinical criteria as those used in the present study have yielded high diagnostic accuracy and displayed the neuropathology consistent with the diagnosis of PD.

NC participants were recruited from the community and were included only if they had no history of neurological or psychiatric disease. The 24 NC participants were drawn from a larger sample of comparison participants (n=55) and matching was based first on age, level of education, and then gender. Table 1 shows the mean age, years of education, gender ratios, and total scores on the MDRS for the two patient groups and the NC subjects. All data in this study were obtained in compliance with the local human subjects review boards from the different institutions listed above.

Materials and Procedures

The CVLT was administered using the methods described in the standardization manual (Delis et al., 1987). Briefly, the CVLT is a word-list learning task that consists of two 16-item word lists, one of which (list A) is presented over 5 learning trials (e.g., auditory presentation and immediate recall) and a second (list B) presented on a single learning trial. Following the recall of list B, subjects are asked to recall list A spontaneously (short-delay free recall) and following semantic cues (short-delay cued recall), and after a 20-minute delay they are again asked to recall list A spontaneously (long-delay free recall) and following semantic cues (long-delay cued recall). Finally, subjects are administered a "yes/no" recognition test in which they are presented with the 16 target words embedded within a list of 28 non-target words and asked to say "yes" if the word was from list A and "no" if it was not.

The CVLT provides measures of recall based on the number of words produced by the subject on the various recall trials. Measures of learning characteristics are also provided, including a semantic clustering ratio (a measure of the degree to which the subject provides the words back to the examiner in a semantically organized manner) and a serial clustering ratio (a measure of the degree to which the subject provides the words back to the examiner in the order in which they were read to the subject). Greater scores on these indices indicate a greater use of that particular strategy. Other learning characteristic measures include the percentage of words recalled from the first part of the word list (primacy region), the middle part of the word list (middle region), or the last part of the word list (recency region). Larger scores on those indices indicate a greater percentage of words recalled from that region of the word list. The CVLT also provides several measures of recognition. As an overall measure of recognition performance, we computed the difference between the number of recognition hits (i.e., the number of times the participant correctly identified a word as being a member of the first list) minus the number of false positive errors (i.e., the number of times a subject says "yes" to a word that was not part of the target list). Greater scores are indicative of better recognition memory performance. A final recognition memory index that is included is the response bias measure, which indicates a subject's propensity to say "yes" (indicated by a positive response bias score) or "no" (indicated by a negative response bias score) during recognition testing.

Additional analyses were conducted excluding the 2 PDD patients on trihexyphenidyl because of its anticholinergic effects. Results were the same when these patients were excluded (see below) with the exception that significant differences among the three groups became nonsignificant on serial cluster ratio and free recall intrusions.

Statistical Analysis

The analyses of the various CVLT indices were designed to examine specific components of learning and memory. *Recall Level* indices included total words recalled on all learning trials (trial 1–5), trial 1, trial 5, list B, short-delay free recall, short-delay cued recall, long-delay free recall, and long-delay cued recall. We also examined the subjects' ability to retain information when taking into account level of acquisition. To do so, we computed a percentage savings score by dividing the number of words recalled on the long-delay free recall trial by the number of words recalled on trial 5 of the learning trials. *Learning Characteristics* indices included the semantic cluster ratio, the serial cluster ratio, and the percentage of words recalled from the primacy, middle, and recency regions of the word list during the learning trials (trials 1–5). *Recall Error* indices included the number of perseverations produced during short- and long-delay free and cued recall trials, the number of intrusions produced during short- and long-delay free recall trials, and the number of intrusions produced during short- and long-delay cued recall trials. Because there were significant group differences in the total number of words recalled across the trials (see below), we computed the percentage of errors for each error type (perseverations, free recall intrusions, and cued recall intrusions) by dividing the total number of each error type by the total number of words provided (each error type + correct words recalled). These indices allowed us to determine the percentage of each error type committed by the subjects while taking into account the total number of correct words they provided. *Recognition* indices included recognition discriminability, recognition hits, recognition false positives, and recognition response bias. Because groups were matched on important demographic variables known to impact learning and memory, CVLT raw scores were analyzed rather than derived standardized scores.

Comparisons of the three groups' CVLT index scores, along with demographic data and MDRS total scores, were conducted using one-way ANOVAs with Tukey's HSD pair-wise comparisons, the latter of which allowed for a more stringent test of the all pair-wise comparisons if the one-way ANOVA was found to be significant. Tables 1–4 list the *F*-values for the one-way ANOVAs, whether they were significant, and if so, the *p*-values for the pair-wise contrasts derived from Tukey's HSD tests, which strikes a good balance between the possibility of committing a Type I or Type II error. If any of the pair-wise comparisons were significant for the contrast between the PDD and DLB groups, effect sizes were calculated using Cohen's *d* based on the mean score and pooled standard deviation of these two groups. Gender ratios were contrasted using a 3×2 χ^2 test. A direct discriminant function analysis was used to determine whether DLB and PDD patients could be discriminated using key indices from the CVLT. Sensitivity, specificity, and positive predictive values for distinguishing between PDD and DLB patients were computed using standard methods. All tests were considered significant at $p<.05$.

RESULTS

Demographics and MDRS Total Score

CVLT Indices

Recall Level: Table 2 lists the mean number of words recalled for the various recall indices. Results indicated that the three groups differed significantly on all of the recall indices, with both the PDD and DLB groups performing significantly worse than the NC participants, and the DLB group performing significantly worse than the PDD group on all of the indices except for trial 5 (see Table 2). On those indices on which the PDD and DLB differed, the effect sizes for the differences between PDD and DLB groups were all large and ranged from 0.84 – 1.18. These results indicate that both PDD and DLB patients learned and recalled fewer words than the NC participants on all the recall trials, and that the DLB patients recalled fewer words than the PDD patients on all but one of the recall indices (trial 5).

Learning Characteristics and Recall Errors: Table 3 lists the mean values for the three groups on the learning characterstic and recall error indices. Note that 3 of the DLB patients were excluded from the analysis examining the semantic cluster ratio because the index could not be computed due to a low recall level. Results indicated that both the PDD and DLB groups had lower semantic ratio scores than the NC participants, but their scores did not differ from each other. The three groups also differed on the serial cluster ratios, but post-hoc tests indicated that this was mostly due to the DLB patients having greater ratios than the NC participants, whereas the PDD groups did not differ from the other two groups. Thus, both patient groups were less likely than NC participants to use a semantic cluster strategy when learning the word list, but only the DLB patients were more likely than NC subjects to use a serial cluster strategy. The mean percentage of words recalled by each group from the primacy, middle, and recency regions of the word list are also listed in Table 3. Results indicated that the three groups differed only in the percentage of words recalled from the recency region of the word list, but the post-hoc tests failed to reach signifiance for any of the pair-wise comparisons.

Recall Errors: The mean percent perseveration errors, intrusion errors during free recall trials, and intrusion errors during cued recall trials are listed in Table 3. Results indicated that the three groups differed in the percentages of perseveration errors, free recall intrusions, and cued recall intrusions. Post-hoc contrasts indicated that the PDD patients committed a greater percentage of perseveration errors than both the DLB patients and the NC subjects, whereas the DLB and NC subjects did not differ. The effect size for the difference between the PDD and DLB groups was 0.90. For both free and cued recall intrusions, post-hoc comparisons indicated that both the PDD and DLB patients committed a significantly greater percentage of intrusions than the NC subjects, but that the two patient groups did not differ from one another on either index. Thus, in terms of recall errors, the main finding was that PDD patients committed a greater percentage of perseveration errors than did the DLB patients, and the PDD and DLB patients committed a greater percentage of free and cued recall intrusions than NC subjects.

Recognition Measures: Scores for the various recognition indices are listed in Table 4. Results indicated that the three groups differed on discriminability, false positives, and response bias, and the post-hoc comparisons revealed that, relative to the NC group, both the PDD and DLB groups had poorer discriminability, more false positives, and more of a "yes" response bias. The PDD and DLB patients did not differ on any of the recognition measures. Taken together, these results suggest that PDD and DLB patients were similarly impaired in their recognition performance relative to NC subjects.

Discriminant Function Analysis

To determine whether select variables from the CVLT could adequately discriminate between the DLB and PDD groups, a linear discriminant function analysis was conducted using short delay cued recall, list b recall, and the percentage of perseveration errors. Short delayed recall was included in the analysis because this variable had the largest effect size for the difference between the PDD and DLB patients. List b recall was used because it had a large effect size and it was not redundant with the other variables included (e.g., using long delay cued recall would have been somewhat redundant with short delay cued recall). Percentage of perseveration errors was used because it was the only variable on which the PDD patients were more impaired than the DLB patients. The discriminant function was significant, $\chi^2(3)=23.8$, $p<.001$. Based on standardized canonical discriminant function coefficients, percent perseveration contributed most to the discriminant function (standardized coefficient=.627), followed by short delay cued recall (standardized coefficient=.625), and list b recall (standardized coefficient=.325). Classification function coefficients are displayed in Table 5. The overall classification accuracy for the two groups was 81.3% (18/24 correct classification

for the PDD patients and 21/24 for the DLB patients), and the sensitivity for the diagnosis of PDD was 75.0% (95% CI: 52.9–89.4%) and the specificity was 87.5% (95% CI: 66.5–96.7%). The positive predictive power, which is defined as the probability of an individual having a given diagnosis given that the discriminant function classified the individual with that diagnosis, was 85.7% (95% CI: 62.6–96.2%) for the PDD patients and 77.8% (95% CI: 57.3–90.6%) for the DLB patients. Of course, the actual positive and negative predictive values would depend on the base rates of PDD and DLB in any given clinical setting.

To provide further validation of the discriminant function, we applied the derived algorithm to the CVLT data of the group of 42 PDD patients who were not included in the original analyses. The mean demographic and MDRS data for this sample was the following: age= 69.6 (*SEM*=1.1), education=12.8 (*SEM* =0.4), male:female ratio=30:12, and total MDRS score=122.4 (*SEM* =1.1). The results indicated that 78.6% (33/42) of this new sample of PDD patients was correctly classified, suggesting a fairly high level of discrimination within this cross-sample. There were no differences between those PDD patients that were correctly and incorrectly classified in age, education, or MDRS total scores (all p's > .05).

DISCUSSION

The main finding from the present study was that patients with DLB and patients with PDD differed in certain aspects of the verbal learning and memory deficits they exhibited. In particular, DLB patients were more impaired than the PDD patients on all but one of the recall measures, a finding that was highlighted by the fact that DLB patients displayed a more rapid rate of forgetting than that of PDD patients. PDD patients, on the other hand, committed a greater percentage of perseveration errors when recalling the word list than did either the DLB patients or NC subjects. These differences were rather robust when one considers the effect sizes (see Tables 2 and 3). Importantly, these observed differences cannot be attributed to overall level of cognitive impairment in that the DLB and PDD patients did not differ in their total scores on the MDRS, nor can they be due to differences in demographic factors such as age, gender, or level of education, as the two patient groups were matched on these variables.

Despite the differences in recall level and recall errors between the DLB and PDD groups, the two groups showed similar patterns in their learning characteristics. The semantic cluster and serial cluster ratios for the DLB and PDD patients did not differ significantly, although DLB patients had higher serial clustering ratios than the NC group, which was not the case for the PDD patients. This may be due a greater frontal lobe pathology (Double et al., 1996; Harrington et al., 1994) and executive dysfunction (Aarsland et al., 2004; Aarsland et al., 2003; Downes et al., 1998) in DLB compared to PDD, as higher serial clustering ratios have been reported in demented individuals with greater frontal lobe pathology (Glosser, Gallo, Clark, & Grossman, 2002). There were no significant differences in terms of recalling words from the primacy, middle, or recency regions of the word list. When considered with the observed differences between DLB and PDD patients in recall and percent savings, these results suggest that the two groups differ less in terms of their approach to acquiring verbal information than in their ability to actually learn and retain such information.

Although mathematically the DLB patients had lower recognition discriminability scores than PDD patients, the two groups did not differ significantly on any of the recognition measures. Thus, DLB and PDD patients show fairly comparable levels of recognition memory despite the fact that DLB patients have worse learning and retention of verbal information. These findings suggest that patients with DLB might benefit more from recognition testing than do patients with PDD. Because recognition memory is less dependent upon effortful retrieval processes than free recall, the enhanced recognition benefit of the DLB patients compared to PDD patients may indicate that they have a greater retrieval deficit than the PDD patients.

These results are consistent with recent reports indicating that patients with PD do not always display improved performance on recognition testing compared to other patients with primarily subcortical pathology (Zizak et al., 2005).

The finding that DLB and PDD patients demonstrate differences in learning and memory is important given that neuropathological and structural imaging studies have not been able to consistently distinguish between these two disorders (Burton et al., 2004; Guo, Itaya, Takanashi, Mizuno, & Mori, 2005; Harding & Halliday, 2001; Tam et al., 2005; Tsuboi & Dickson, 2005), even when examining the medial temporal lobe brain regions believed to be critical for the type of declarative memory tested by the CVLT (although see Tam et al., 2005, for some evidence of greater medial temporal lobe atrophy in DLB than PDD). The failure to observe consistent structural medial temporal lobe differences stands in contrast to our results showing poorer learning, delayed recall and more rapid forgetting in DLB than in PDD patients. Impaired delayed recall and rapid forgetting are characteristic features of patients with medial temporal lobe damage, such as patients with AD or focal medial temporal lobe damage (Kohler et al., 1998; Squire, Stark, & Clark, 2004). It is possible that the memory differences we observed are due to greater concomitant AD pathology in the DLB patients as compared to the PDD patients. Indeed, the majority of DLB patients in the current study had significant AD pathology in medial temporal lobe regions as indicated by a Braak stage of IV or greater. However, neither the CVLT savings score (r=−.04, p=.87) nor cued intrusions (r=.02, p=.93) of the DLB patients correlated with Braak stage, so it does not appear that differences in medial temporal lobe involvement can entirely account for the differences in forgetting rates in the two groups.

The finding that PDD patients committed a greater percentage of perseveration errors than DLB patients was somewhat surprising given previous reports of worse executive dysfunction in DLB patients than in PDD patients (Aarsland et al., 2004; Aarsland et al., 2003; Downes et al., 1998), and greater frontal lobe pathology in DLB than PDD (Double et al., 1996;Harrington et al., 1994). However, a recent study found that PDD patients have a greater decrease in cholinergic activity in the mediodorsal nucleus of the thalamus as compared to patients with DLB (Ziabreva et al., 2006). This brain region has extensive connections to the anterior cingulate cortex, so it is possible that the greater magnitude of perseverations in the PDD patients is related to dysfunction in this region. In addition, it has become increasingly clear that various executive functions can be dissociated (Faw, 2003; Possin et al., 2005) and that different frontal regions might contribute to various executive processes (Faw, 2003; Fuster, 1999). As such, it is possible that the greater executive functioning deficits previously observed in DLB patients and the greater tendency towards perseverative responding in the PDD patients in the present study are due to the involvement of different regions of the frontal cortex.

The results of the discriminant function analysis using short delay cued recall, percent perseverations, and list b recall differentiated the DLB and PDD patients with 81.3% accuracy. Sensitivity for the diagnosis of PDD relative to DLB was fairly adequate (75%) as was the specificity (87.5.0%). The application of the discriminant function algorithm to the PDD patients not included in the original analyes resulted in a 78.6.% correct classification rate. These results are somewhat promising in that a fairly high accuracy was obtained with just a single memory test. It is very possible that the inclusion of other tests, such as measures of executive functions, would provide an even greater degree of diagnostic accuracy. To test this possibility, future studies will have to include additional measures as well as conduct a cross-validation of the discriminant function from this study in a new sample of DLB patients.

A majority of previous studies have failed to identify clear differences between DLB and PDD patients on various cognitive measures (Ballard et al., 2002; Cormack, Aarsland et al., 2004; Horimoto et al., 2003; Noe et al., 2004), leading some investigators to conclude that DLB and

PDD result in only subtle, if any, differences in cognition that will have little clinical utility in distinguishing between these diseases (Aarsland et al., 2004; Ballard et al., 2002). The conclusions that can be drawn from these studies are limited, however, because they usually employed relatively broad and insensitive cognitive measures. The results of the current study show that a more in-depth analysis of cognition, such as that provided by the multiple measures of verbal learning and memory of the CVLT, can identify potentially important differences between patients with DLB and PDD. This was also recently shown in the domain of visual cognition. Although a study examining basic visual perceptual and visual spatial processes in DLB and PDD failed to identify any differences between these groups (Mosimann et al., 2004), another study showed that DLB patients, but not those with PDD, were impaired on a visual search task that provided an intricate assessment of visual pre-attentive processes (Cormack, Gray et al., 2004).

There are several limitations of our study that should be noted. First, only verbal learning and memory were examined so it is impossible to determine the extent to which deficits in other cognitive abilities may have contributed to the observed differences in CVLT performance. As noted above, prior studies have shown that DLB patients perform worse than PDD patients on certain measures of executive functioning (Aarsland et al., 2003; Downes et al., 1998; Gnanalingham, Byrne, Thornton, Sambrook, & Bannister, 1997), and it is possible that these differences play an important role in the divergent CVLT performances. Second, the PDD patients were drawn from different geographic locations and tested during different years than the DLB patients, so there may be cohort differences that could account for our findings. It should be noted, however, that standardized criteria for the diagnosis of PDD were applied at each of the sites to minimize cohort differences, and that the DLB patients were all autopsy-confirmed to further reduce this potential source of bias. Third, we had autopsy-confirmation on only two of the PDD patients, so it impossible to determine if other pathological features, such as concomitant AD, could account for the pattern of memory deficits they exhibited. However, Braak and colleagues (Braak, Rub, Jansen Steur, Del Tredici, & de Vos, 2005) recently reported that cognitive status in PD could be adequately accounted for by the stage of Parkinson's pathology, and that although AD-type pathology can occur in PD patients with cognitive impairment, the degree of such pathology may not be sufficient to account for the significant cognitive deficits observed in PDD (see also) (Aarsland et al., 2005). Finally, we identified differences between mildly demented patients with DLB or PDD, but it is not known if such differences remain in patients with a more severe level of cognitive impairment. Future studies should address this issue.

In conclusion, the results of the current study indicate that patients with DLB and patients with PDD exhibit different patterns of verbal learning and memory deficits. These differences suggest that there are distinct pathological features in the two diseases, although these features cannot be identified at this point. Overall, these results provide some support for the validity and usefulness of separate clinical diagnoses of DLB and PDD, and offer a potential clinical feature that may help in making a differential diagnosis.

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Table 1

Mean (*Standard Deviation*) number of words recalled for the learning trials, immediate recall trials, delayed recall trials, and percent savings, and mean values for the various learning, recall, and recognition indices for the PDD patients, DLB patients, and **NC participants**. One-way ANOVA *F*-values are based on a comparison among the three groups and effect sizes for the F-values are based on η_p^2. Pair-wise comparison *p-values* are based on Tukey's HSD multiple-comparison tests. Effect sizes are based on a comparison between the PDD and DLB groups using Cohen's *d*. Confidency intervals (95% C.I.) are based on the difference of the pair-wise comparisons.

	PDD ***M*** ***(SD)***	**DLB** ***M*** ***(SD)***	**NC** ***M*** ***(SD)***	**One-Way ANOVA** ***F value*** **(η_p^2)**	**PDD vs DLB** ***p value*** ***(d value)*** ***(95% C.I.)***	**PDD vs NC** ***p value*** ***(d value)*** ***(95% C.I.)***	**DLB vs NC** ***p value*** ***(d value)*** ***(95% C.I.)***
Recall Measures							
Trials 1–5 Total	25.54 (10.49)	17.08 (6.51)	45.21 (11.33)	53.36*** (0.61)	.01 (0.97) (1.77 - 15.15)	<.001 (1.80) (−12.98 - −26.36)	<.001 (3.04) (−34.82 - −21.43)
Trial 1	3.25 (1.51)	2.00 (1.47)	5.75 (1.82)	33.73*** (0.49)	.02 (0.84) (0.14 - 2.36)	<.001 (1.50) (−3.61 - −1.39)	<.001 (2.23) (−4.86 - −2.64)
Trial 5	6.08 (3.31)	4.25 (1.42)	10.92 (3.01)	38.81*** (0.53)	.06 (0.72) (−0.04 - 3.71)	<.001 (1.53) (−6.71 - −2.96)	<.001 (2.83) (−8.54 - −4.79)
List B	3.00 (1.82)	1.42 (1.06)	5.63 (1.69)	44.67*** (0.56)	.002 (1.06) (0.51 - 2.66)	<.001 (1.50) (−3.70 - −1.55)	<.001 (2.98) (−5.29 - −3.13)
Short Delay Free Recall	3.83 (2.14)	1.71 (2.05)	8.75 (3.43)	45.69*** (0.57)	.02 (1.01) (0.31 - 3.94)	<.001 (1.72) (−6.73 - −3.11)	<.001 (2.49) (−8.85 - −5.23)
Short Delay Cued Recall	5.67 (2.60)	2.92 (2.02)	10.38 (2.95)	52.49*** (0.60)	.001 (1.18) (0.99 - 4.51);	<.001 (1.69) (−6.47 - −2.94)	<.001 (2.95) (−9.22 - −5.69)
Long Delay Free Recall	3.63 (2.65)	1.54 (1.93)	9.17 (3.89)	43.23*** (0.56)	.04 (0.90) (0.05 - 4.11)	<.001 (1.66) (−7.57 - −3.51)	<.001 (2.48) (−9.66 - −5.59)
Long Delay Cued Recall	5.46 (2.98)	2.54 (2.13)	9.58 (3.59)	34.34*** (0.50)	.003 (1.13) (0.87 - 4.96)	<.001 (1.25) (−6.17 - −2.08)	<.001 (2.39) (−9.09 - −5.00)
Percent Savings	55.95 (31.83)	30.22 (29.71)	81.76 (21.21)	20.38*** (0.37)	.006 (0.84) (6.39 - 45.06)	.006 (0.95) (−45.14 - −6.47)	<.001 (2.00) (−70.87 - −32.20)
Learning Indices							
Semantic Cluster Ratio	1.27 (0.70)	1.32 (0.87)	2.14 (0.92)	8.04** (0.20)	.98 (0.06) (−0.65 - 0.55)	.002 (1.06) (−1.45 - −0.29)	.005 (0.92) (−1.42 - −0.22)
Serial Cluster Ratio	3.03 (2.30)	3.58 (3.53)	1.73 (1.43)	3.28* (0.09)	.74 (0.18) (−2.33 - 1.22)	.20 (0.68) (−0.48 - 3.07)	.04 (0.69) 90.07 - 3.62)
Primacy Recall Percentage	26.08 (13.31)	29.46 (19.28)	29.88 (5.95)	0.53 (0.2)	.68 (0.20) (−13.02 - 6.27)	.62 (0.37) (−13.44 - 5.86)	.99 (0.03) (−10.07 - 9.23)
Middle Recall Percentage	33.04 (19.09)	29.75 (18.19)	40.79 (7.35)	3.08 (0.08)	.75 (0.17) (−7.64 - 14.22)	.21 (0.53) (−18.68 - 3.18)	.05 (0.80) (−21.97 - −0.11)
Recency Recall Prcentage	40.67 (19.44)	40.54 (21.61)	29.42 (8.99)	3.25* (0.09)	1.0 (0.01) (−12.02 - 12.27)	.08 (0.74) (−0.90 - 23.40)	.08 (0.67) (−1.02 - 23.27)

	PDD ***M*** ***(SD)***	**DLB** ***M*** ***(SD)***	**NC** ***M*** ***(SD)***	**One-Way ANOVA** ***F value*** **(η_p^2)**	**PDD vs DLB** ***p value*** ***(d value)*** ***(95% C.I.)***	**PDD vs NC** ***p value*** ***(d value)*** ***(95% C.I.)***	**DLB vs NC** ***p value*** ***(d value)*** ***(95% C.I.)***
Recall Error Indices							
Percent Perseverations	9.83 (9.21)	3.11 (5.25)	4.19 (3.14)	7.66** (0.18)	.001 (0.90) (2.30 - 11.13)	.009 (0.82) (1.22 - 10.05)	.83 (0.25) (−5.50 - 3.33)
Percent Free Recall Intrusions	15.85 (16.07)	19.32 (15.95)	4.23 (3.95)	8.51*** (.20)	.64 (0.22) (−12.65 - 5.70)	.009 (0.99) (2.44 - 20.79)	.001 (1.29) (5.92 - 24.27)
Percent Cued Recall Intrusions	37.94 (26.02)	45.73 (24.67)	8.32 (7.76)	20.84*** (0.38)	.41 (0.31) (−22.44 - 6.86)	<.001 (1.54) (14.97 - 44.26)	<.001 (2.05) (22.76 - 2.05)
Recognition Indices							
Recognition Hits - False Positives	5.75 (5.97)	2.92 (6.12)	12.08 (3.32)	18.69*** (0.35)	.16 (0.47) (−0.84 - 6.51)	<.001 (1.31) (−10.01 - − 2.67)	<.001 (1.86) (−12.84 - −5.49)
Recognition Response Bias	0.30 (0.32)	0.29 (0.49)	−0.04 (0.43)	4.96*	.99 (0.02) (−0.28 - 0.30)	<.05 (0.89) (0.05 - 0.63)	<.05 (0.72) (0.03 - 0.61)

* $p<.05$,

** $p<.01$,

*** $p<.001$

Table 2

Classification function coefficients derived from discriminant function analysis.

	Predicted Group	
	PDD	**DLB**
Short Delay Cued Recall	0.93	0.49
Percent Perseverations	0.21	0.08
List B Recall	0.60	0.24
Constant	−5.28	−1.69