Pergamon

Archives of Clinical Neuropsychology
16 (2001) 547–560

Archives of CLINICAL NEUROPSYCHOLOGY

Different underlying mechanisms for deficits in concept formation in dementia

Tania Giovannetti[a,b], Melissa Lamar[a,b], Blaine S. Cloud[c], Rod Swenson[d], Deborah Fein[c], Edith Kaplan[f,g], David J. Libon[a,b,h,*]

[a]*Neuropsychology Program, Drexel University, Philadelphia, PA, USA*
[b]*Neuropsychology Service, Crozer Chester Medical Center, Upland, PA, USA*
[c]*Department of Neurology, Thomas Jefferson University Hospital, Philadelphia, PA, USA*
[d]*Merit Care Neuroscience Center, Fargo, ND, USA*
[e]*Department of Psychology, University of Connecticut, Storr, CT, USA*
[f]*Departments of Neurology and Psychiatry, Boston University Medical Center, Boston, MA, USA*
[g]*Department of Psychology, Suffolk University, Boston, MA, USA*
[h]*Department of Psychiatry, Medical College of Pennsylvania/Hahnemann University Hospital, Philadelphia, PA, USA*

Accepted 9 May 2000

Abstract

We investigated the different mechanisms that may underlie deficits in verbal concept formation among patients with Alzheimer's disease (AD) and ischaemic vascular dementia (IVD) associated with periventricular and deep white matter alterations. Concept formation was assessed with the WAIS-R Similarities subtest (SIM). Two types of errors were re-coded from the 0-point responses as scored by the WAIS-R manual. *In set* errors (e.g., *dog–lion* "they're alive") were coded when patients reported a very vague superordinate concept for the word pair. *Out of set* responses (e.g., *dog - lion* "the lion roars and the dog barks") were coded when a response was clearly out of mental set, i.e., when participants were unable to provide a superordinate concept for the word pair. Between-group comparisons demonstrated no difference in SIM test performance according to the scoring system described in the WAIS-R manual. Nonetheless, AD patients produced a greater proportion of *in set* errors, while IVD patients produced a greater proportion of *out of set* errors. *Out of set* errors were highly associated with measures of executive function, while *in set* errors were associated with measures related to delayed recognition memory and semantic intrusion errors. We conclude that the underlying deficits that contribute to poor concept formation differ between AD and IVD patients. In IVD impaired concept formation is related to deficits in the executive systems necessary to monitor responses and sustain

* Corresponding author. Neuropsychology Service, Crozer Chester Medical Center, Upland, PA 19013, USA. Tel.: +1-610-447-6014.

0887-6177/01/$ see front matter
PII: S0887-6177(00)00066-4

mental set. In AD, by contrast, the deficit appears to be secondary to impaired verbal response selection. © 2001 National Academy of Neuropsychology. Published by Elsevier Science Ltd.

Keywords: Alzheimer's disease; Vascular dementia; Concept formation

1. Introduction

Concept formation refers to the ability to identify and extract the common and pertinent features of objects or concepts (Cronin-Golomb, Corkin, & Rosen, 1993; Lezak, 1995). The WAIS-R Similarities subtest (SIM), a measure that requires one to identify a superordinate category or features shared by word pairs (Kaplan, Fein, Morris, & Delis, 1991), has been used extensively to assess concept formation among normal older adults and neurological patients with diverse conditions (Cronin-Golomb, Rho, Corkin, & Growdon, 1987; Lezak, 1995; Whelihan & Lesher, 1985). The goal of the present study is to test the hypothesis that the difficulties in concept formation among participants with distinct dementia syndromes are associated with differing underlying mechanisms.

For the most part, research has shown that individuals suffering from Alzheimer's disease (AD) obtain lower scores on the SIM, as well as other tests of concept formation, compared to age-matched control subjects (Cronin-Golomb et al., 1987; Lafleche, & Albert, 1995; Martin & Fedio, 1983; Pillon, Dubois, Lhermitte, & Agid, 1986). Similar results have been reported among patients with a wide variety of dementia syndromes, such as progressive supranuclear palsy (PSP; Albert, Feldman, & Willis, 1974), Parkinson's disease (PD; Cronin-Golomb, Corkin, & Growdon, 1988; Ledesma, Nicdao, Dantes, & Perez, 1986; Maher, Smith, & Lees, 1985; Pillon et al., 1986; Reitan & Boll, 1971), and multi-infarct dementia (Perez et al., 1975; Villardita, 1993).

Although the current literature suggests that concept formation is deficient among a wide range of dementia syndromes, the underlying mechanisms for poor performance on tests of concept formation such as the SIM have not been explored. In the present study, we assume that concept formation is a multi-dimensional construct. Moreover, we suggest that accurate SIM performance requires an adequate fund of knowledge of objects/concepts (semantic knowledge), as well as the ability to hold and simultaneously process multiple units of information (executive systems functioning).

Recent investigations have documented distinct patterns of neuropsychological deficits between patients with different dementing illnesses (Libon et al., 1997). For example, neuropsychological comparisons between patients with AD and patients suffering from PD, Huntington's disease (HD), and ischaemic vascular dementia (IVD) show that AD patients typically demonstrate greater problems on tests of semantic knowledge and declarative memory (Chan et al., 1993; Giovannetti-Carew, Lamar, Cloud, Grossman, & Libon, 1997; Rosser & Hodges, 1994); while patients with IVD, PD, and HD invariably show greater executive systems deficits (Almkvist, 1994; Butters, Wolfe, Granholm, & Martone, 1986; Kertesz & Clydesdale, 1994; Lamar et al., 1997; Libon et al., 2000; Sagar & Sullivan, 1988).

Given the evidence for semantic knowledge deficits among patients with AD, we believe that their poor concept formation, as assessed with the SIM may be related to a breakdown in semantic knowledge. Semantic knowledge deficits would result in the inability to extract the most salient or distinguishing features of concepts (Smith, 1995). Therefore, on the SIM, semantic deficits may cause participants to report only trivial similarities between word pairs or vague responses, such as "they're alive" (dog–lion). Because executive systems functions are less impaired earlier in the course of AD (Lafleche & Albert, 1995), AD patients should retain the ability to stay in set for the task and generate a superordinate relationship between word pairs.

With respect to participants with IVD, we believe their concept formation deficits may be related to executive function impairments (e.g., see Almkvist, 1994; Butters et al., 1986; Kertesz & Clydesdale, 1994; Lamar et al., 1997; Libon et al., 2000; Sagar & Sullivan, 1988). Thus, a response such as "one roars and one barks" (dog–lion) suggests an inability to operate within the parameters of the task as specified by task instructions, i.e., inability to sustain the necessary mental set. Although IVD participants may be capable of retrieving detailed semantic knowledge regarding the word pair, their deficits in such executive systems functions as mental control and working memory (Cloud et al., 1994; Lamar, Giovannetti, & Libon, 1999) may preclude the efficient comparing and contrasting of semantic information.

Despite reports demonstrating that performance on the SIM is impaired in dementia, previous studies have limited analyses to the total number of absolute correct responses as specified by the WAIS-R manual. Restricting analyses to the sum of correct responses, however, may mask subtle differences in task performance and limit a complete understanding of the underlying cognitive mechanisms responsible for deficient concept formation. In an effort to develop a more sensitive coding scheme for the SIM, we turned to Kaplan et al. (1991), who have commented upon a variety of distinctive qualitative errors made by neurologic patients, including instances when patients report how the word pair is different (*eye–ear*: "one is for seeing and one is for hearing"), when patients respond to only one of the stimulus words (*table–chair*: "you sit on them"), and instances when patient's responses are "stimulus-bound" (*fly–tree*: "a fly sits on a tree"; *egg–seed*: "a chicken eats a seed and then lays eggs"). Kaplan et al. reported that these errors likely reflect executive system dysfunction; however, these observations were never formalized into a concrete scoring system or tested experimentally.

We agree that the SIM errors described by Kaplan et al. (1991) reflect executive dysfunction, as the examples demonstrate a loss of mental set, or an inability to respond according to the task instructions, i.e., to compare two objects and determine a way in which the objects are similar. We will refer to this category of errors as *out of set*. By contrast, errors that reflect preservation of the basic task demands, but contain only vague or trivial similarities between word pairs (*dog lion*: "they're alive") may not necessarily suggest executive function deficits. Rather, such errors might be associated with deficits in semantic knowledge processing. We will use the term *in set* error to refer to this category of responses.

In the present study the SIM was administered to participants suffering from AD and IVD associated with subcortical periventricular and deep white matter alterations. We developed a scoring system to categorize 0-point responses as either *in set* or *out of set*. The goal of this study is to test the hypothesis that impairment in concept formation, as assessed with the SIM,

in AD and IVD is related to the semantic knowledge and executive function deficits that characterize these disorders, respectively. Therefore, our first prediction is that AD patients will make more *in set* errors, while IVD patients will make more *out of set* errors. Regarding the underlying mechanisms for *in set* and *out of set* errors, our second prediction is that when SIM errors are factor analyzed along with other neuropsychological measures, *in set* errors will load with variables that are related to semantic knowledge functioning. *Out of set errors*, on the other hand, will load with variables related to executive systems functioning.

2. Methods

2.1. Participants

All patients were outpatients recruited from the Crozer Chester Medical Center's Alexander Silberman Geriatric Assessment Program Center. All patients were examined by a social worker, geriatrician, neurologist, psychiatrist, and neuropsychologist. An MRI or CT study of the brain and appropriate laboratory studies were obtained for all participates. AD patients with verified MRI/CT cortical or subcortical infarction were excluded. A clinical diagnosis was established at an inter-disciplinary team conference. Forty-nine patients diagnosed with NINCDS-ADRDA probable AD (McKhann et al., 1984) were studied.

Forty-two patients were diagnosed with probable/possible IVD using the California Criteria of Chui et al. (1992). In addition to periventricular and deep white matter alterations, all patients diagnosed with probable IVD ($n=25$) had evidence of two or more ischaemic strokes on the basis of their history, neurological examination, and/or a T1 weighted MRI study of the brain. Patients diagnosed with possible IVD ($n=17$) presented with either evidence of a single stroke without a clear documented relationship to the onset of their dementia, and/or Binswanger's disease as defined by Chui et al. Patients with cortical CVAs on MRI/CT scans were excluded.

There were no differences between the two patient groups in age, education, level of dementia as assessed with the Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975), or level of depression (Geriatric Depression Scale, GDS; Yesavage, 1986; Table 1).

Patients had no record of head injury, substance abuse, epilepsy, B12, folate, thyroid deficiency, or major psychiatric disorders (including major depression). This information was

Table 1
Demographic data and neuropsychological test performance

	AD		IVD	
	M	(SD)	M	(SD)
Age	77.2	(5.6)	79.4	(6.0)
Education	12.3	(2.7)	11.9	(2.7)
MMSE	21.3	(3.8)	21.1	(4.0)
GDS	5.5	(4.4)	6.0	(3.8)

AD = Alzheimer's disease; IVD = Ischaemic Vascular Dementia; MMSE = Mini-Mental State Examination; GDS = Geriatric Depression Scale.

obtained through an extensive interview conducted by one of us (DJL) with a knowledgeable informant and a review of past medical records.

2.2. The SIM

The SIM was administered and scored according to standard instructions provided in the manual (Wechsler, 1987). In addition, several dependent variables not described in the standard scoring criteria were also coded.

(1) *Number of test items administered* — This refers to the total number of items administered before the discontinuation criterion was met.
(2) *Number of responses scored* — Very often patients offered more than one response to the examiner's query. This variable refers to the total number of responses obtained.
(3) *Two-point responses* — These are fully correct, abstract responses as defined by the WAIS-R manual.
(4) *One-point responses* — These are concrete responses as defined by the WAIS-R manual.

All responses that were given a score of zero according to criteria set forth in the WAIS-R manual were coded using the following criteria.

2.2.1. In set responses

(1) *Vague responses* — This was scored when the patient provided a superordinate, but superficial categorical response (e.g., dog–lion: "they eat", coat–suit: "you buy them").
(2) *Subordinate responses* — These responses relate to shared concrete attributes (coat–suit: "they both have sleeves"), or highly specific properties about the test items that may not be correct in all instances (e.g., boat–automobile: "they both have motors").

2.2.2. Out of set responses

(1) *One object responses* — This was scored when patients responded to only one member of the word pair (e.g., coat–suit: "one is minus a pair of pants").
(2) *Juxtaposition responses* — This was scored when patients provided a description of how one member of the word pair might interact with the other member. (e.g., dog–lion: "the lion can eat the dog"; fly–tree: "the fly has a place to land on").
(3) *Different responses* — This refers to instances when patients accurately described how the two items of the word pair are different (e.g., eye–ear: "you see with your eyes and hear with your ears").

2.2.3. Additional error types

Four additional types of errors were coded for 0-point responses that could not be classified as either *in set* or *out of set*.

(1) "They're not" — This was scored when patients refused to acknowledge that the word pairs could be related in any way and responded "they're not alike".

(2) *Unrelated/Incorrect* — This code was assigned when the response was either not germane to the word pair, or was clearly wrong (dog–lion: "they're enemies").
(3) "I Don't Know" — This response was given by patients who were unable to formulate an answer and responded "I don't know."

2.3. Perseverations

Any 0-point response that was re-stated from a previous item was given one of the codes listed above. In addition, the response was also flagged as a perseveration.

In an attempt to facilitate scoring of 0-point responses, a code book was constructed that listed instances representing each type of error. Using this code book, inter-rater reliability was quite high (0.84) between two coders (TG and DJL).

2.4. Neuropsychological assessment

In addition to the SIM, measures designed to assess executive functioning, semantic knowledge and language, and declarative memory were obtained for the IVD and AD participants. These domains of functioning were selected because such functions are associated with concept formation. The specific measures selected for each domain are described below:

(1) *Executive Systems Functioning* — was assessed with the Boston Revision of the Wechsler Memory Scale (WMS) Mental Control subtest (Cloud et al., 1994; Wechsler, 1945). In addition to the three tasks that comprise the standard WMS-Mental Control subtest, (i.e., counting from 20 to 1, reciting the alphabet, and adding serial 3's; Wechsler, 1945), the Boston Revision of the WMS-Mental Control subtest includes four additional tasks: reciting the months of the year forward and backward, an alphabet rhyming task that requires patients to identify letters that rhyme with the word "key", and an alphabet visualization task that requires patients to provide all block printed letter that contain curved lines. Participants were allowed to work as long as necessary on these tasks provided they were working meaningfully. The dependent variable derived from this test was an accuracy index (MC-AcI) composed from the three non-automatized tasks (i.e., months backward, alphabet rhyming, and alphabet visualization). This accuracy index was based on the following algorithm: WMS MC-AcI $=[1-(\text{false positive}+\text{misses}/\#\text{ possible correct})]\times 100$. This algorithm yielded a percentage score ranging from 0 to 100, such that participants obtaining a score of 100% correctly identified all targets and made no false positive responses or misses. The average WMS MC-AcI for each non-automated mental control task was calculated and then averaged for each participant.

Executive systems functioning was also assessed with the Goldberg Graphical Sequence Test-Dementia Version (GST-D; Goldberg, 1986; Goldberg & Tucker, 1979; Lamar et al., 1997). On the GST-D patients are asked to draw geometric shapes and letters. At various times throughout the test patients must switch their mode of output, i.e., instead of drawing shapes, patients are required to write the words "circle," "square," etc. The dependent variable was the total number of perseverations.

(2) *Language and Semantic Knowledge* — was assessed with the 60-item version of the Boston Naming Test (BNT; Kaplan, Goodglass, & Weintraub, 1983), and a test of semantic word list generation (WLG) for "animals" (Giovannetti-Carew et al., 1997; Monsch et al., 1994). The dependent variable derived from the BNT was the number of correct responses. The dependent variable derived from the "animal" WLG was the total association index (AI). The AI measures the degree to which successive responses are semantically related. A high AI is believed to reflect intact semantic memory stores. Complete details regarding how this measure was derived and scored can be found in Giovannetti-Carew et al.

(3) *Declarative Memory* — This was assessed with the 9-word dementia version of the California Verbal Learning Test (CVLT, Delis, Kramer, Kaplan, & Ober, 1987; Libon et al., 1996). Three dependent variables were used, total free recall of list A trials 1–5, the number of responses from the two cued recall test trials that were intrusion errors, and the recognition discriminability index.

3. Results

Between-group differences were primarily tested with a series of univariate analyses of variance (ANOVA). Follow-up analyses, with the Bonferroni correction, were also performed. ANOVAs were first conducted for the number of SIM test items that were administered, the number of responses that were coded, and number of 2- and 1-point responses that were produced. These analyses were not significant. There was also no between-group difference for the total SIM raw score (Table 2).

To test our first prediction that participants with AD would produce a greater proportion of *in set* errors, while IVD participants would produce a greater proportion of *out of set* errors, we used a 2 (AD vs. IVD) × 2 (*in set* vs. *out of set*) repeated measures ANOVA (Fig. 1). Diagnosis was the between-group factor, and SIM error type was the within-group factor. The decision to use repeated measures ANOVA was made because of our need to assess the significance of the two-way interaction. For this analysis all types of *in set* responses (vague and subordinate), and all types of *out of set* (different, one object, and juxtaposition) responses were summed and then divided by the total number of 0-point responses that were produced (Table 3). Using the proportion score allowed us to compare differences between *in set* and *out of set* errors while controlling for variation in the total number of 0-point responses produced. This ANOVA showed a significant main effect for

Table 2
Performance on the SIM (raw scores)

	AD		IVD		
	M	(SD)	M	(SD)	Significance
Total raw score	7.5	(4.8)	7.1	(4.6)	ns
Number of 0-point responses	8.4	(2.8)	8.7	(3.4)	ns
Number of 1-point responses	2.4	(1.5)	2.7	(1.8)	ns
Number of 2-point responses	2.9	(2.3)	2.4	(2.2)	ns

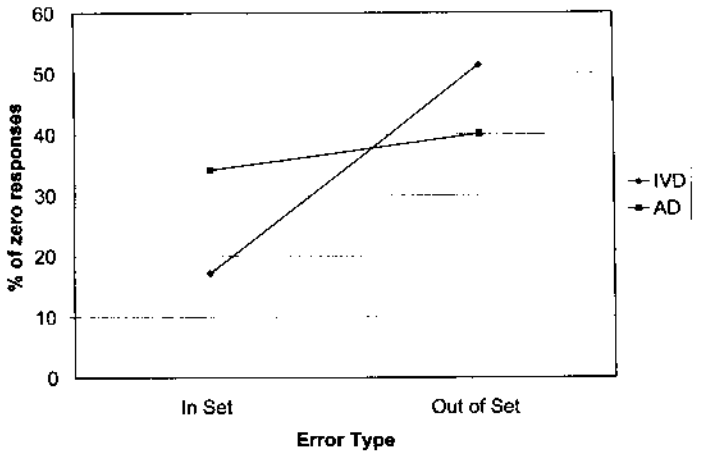

Fig. 1. The proportion of zero-point responses codes as in set and out of set for AD and IVD patients.

error type, (F[1,89]=30.0, p<0.001), and a significant diagnosis by error type interaction (F[1,89]=14.9, p<0.001).

Between-group follow-up analyses (Bonferroni correction, p<0.01) yielded results that supported our first prediction, that is, AD patients made more *in set* responses as compared to the IVD group (AD-M=34.2%, SD=20.3; IVD-M=17.2%, SD=14.2; t[89]=4.55, p<0.001). IVD patients made a greater number of *out of set* responses than AD patients (IVD-M=51.4%, SD=25.3; AD-M=40.2%, SD=18.8, t[89]=−2.42, p<0.017). The results of within-group analyses were, however, somewhat equivocal. As predicted, IVD patients made more *out of set* (M=51.4%, SD=25.3) as compared to *in set* errors (M=17.2%, SD=14.2; t[41]=−6.40, p<0.001); however, among AD patients there was no difference in the proportion of in set (M=34.2%, SD=20.3) versus out of set errors in AD, (M=40.2%, SD=18.8; t[48]=−1.19, ns).

Table 3
Between-group error analysis of the SIM (percent of total responses)

	AD		IVD		
	M	(SD)	M	(SD)	Significance
Vague	23.9%	(17.1)	11.7%	(11.4)	p<0.001
Subordinate	10.3%	(15.7)	5.5%	(9.0)	p<0.08
Different	24.2%	(19.4)	27.5%	(23.2)	ns
One object	11.9%	(14.3)	15.4%	(15.7)	ns
Juxtaposition	4.1%	(7.2)	8.5%	(12.0)	p<0.04
Other SIM errors					
Incorrect	8.6%	(11.2)	7.5%	(10.0)	ns
They're not	7.5%	(13.5)	8.5%	(10.3)	ns
Don't know	9.5%	(14.4)	15.4%	(17.4)	p<0.08

ANOVAs were performed to explore between-group differences for specific SIM error types (Table 3). Results showed AD patients produced more vague responses than IVD patients ($F[1,89]=15.30$, $p<0.001$), while IVD patients made more juxtaposition errors than AD patients ($F[1,89]=4.50$, $p<0.04$). Two trends were also noted which suggested that AD patients made more subordinate errors, $F(1,89)=3.10$, $p<0.08$, and IVD patients made more "Don't Know" responses, $F(1,89)=3.22$, $p<0.08$. No other analyses reached statistical significance. A Mann–Whitney *U*-test was used to explore a difference for the number of perseverations. A non-parametric test was used because very few perseverations were made (AD M=0.29, SD=0.54; IVD-M=0.26, SD=0.54). This analysis was not significant.

Our second prediction was that *in set* errors are related to deficits in semantic knowledge, whereas *out of set* errors are more closely associated with executive system impairment. To test this prediction both vague and superordinate; and one object, juxtaposition, and different errors were summed to create *in set* and *out of set* composite scores. These two variables, along with the seven neuropsychological measures described above, were then entered into a principal component analysis (PCA) with varimax rotation. This yielded a three-factor solution that accounted for 54.0% of the variance (Table 4).

The first factor (22.0% of variance) relates to immediate free recall, naming, and semantic knowledge. As immediate free recall from the CVLT-list A increased, so did the AI derived from the "animal" WLG, and total output from the BNT. The second factor (17.6% of variance) contained positive and negative factor loadings, and appears to reflect executive systems ability to establish and maintain mental set, and to monitor one's behavior. Thus, as patients made more perseverations on the GST-D, they also obtained lower scores on the WMS MC-AcI, and SIM *out of set* errors increased. The third factor (14.4% of variance) also contained positive and negative factor loadings, and may be related to verbal response selection or discrimination. As patients made more CVLT-cued recall intrusion errors, their

Table 4
Factor analysis of SIM in set and out of set scores and other neuropsychological tests

	Factor 1	Factor 2	Factor 3
CVLT: trials 1–5	0.83513	0.14078	0.14779
Boston Naming Test	0.73022	−0.26143	−0.09422
Total AI	0.50009	0.14397	−0.19360
Graphical Sequence	−0.16173	0.76480	−0.09885
WMS MC-AcI	−0.06611	−0.65691	0.19693
SIM-out of set	0.16298	0.64662	0.09314
CVLT-cued recall intrusions	0.07308	0.13643	0.82120
CVLT-Recog Discrim	0.39353	0.11241	−0.63018
SIM-in set	−0.02818	−0.24377	0.57370
Eigen values	1.9	1.5	1.2

CVLT = California Verbal Learning Test; Total AI = total association index; WMS MC-AcI = Wechsler Memory Scale Non-Automatized Index; Recog Discrim = CVLT-recognition discriminability; SIM = WAIS-R Similarities subtest.

CVLT recognition discriminability index declined, and greater numbers on of SIM *in set* errors were made.

4. Discussion

The SIM was used to explore the underlying deficits responsible for poor concept formation in AD and IVD. Our results showed that although participants with AD and IVD did not differ on measures derived from the WAIS-R scoring manual, further analysis of zero-point responses revealed significant between-group differences. That is, patients with AD produced a higher proportion of *in set* errors as compared to IVD patients, while patients with IVD generated a higher proportion of *out of set* errors compared to individuals with AD. Furthermore, when *in set* and *out of set* errors were factor analyzed with neuropsychological tests of executive function, language and semantic knowledge, and memory, the SIM error types loaded on two different factors. Out of set errors loaded with measures of executive function, while in set errors loaded with measures of recognition memory and semantic intrusion errors.

We hypothesized that on the SIM IVD patients, who suffer from greater executive function deficits than AD patients (Almkvist, 1994; Butters et al., 1986; Kertesz & Clydesdale, 1994; Lamar et al., 1997; Libon et al., 2000; Sagar & Sullivan, 1988), would report attributes pertaining to only a single object, or report how concepts are different (i.e., *out of set* errors). On the other hand, AD patients, who suffer from degradation of their semantic knowledge stores (Chan et al., 1993; Giovannetti-Carew et al., 1997; Rosser & Hodges, 1994), would make errors that reflect only very superficial or trivial generalizations between concepts (i.e., *in set* errors) more often than IVD patients. Consistent with our first prediction, when the number of *in set* and *out of set* SIM errors were compared between the two dementia groups, a significant group by error type interaction was observed. That is, individuals with AD produced a greater proportion of *in set* errors when compared to IVD patients, while individuals with IVD produced a greater proportion of *out of set* errors compared to AD patients. In addition, analyses of the specific error types made between groups showed that individuals with AD produced more vague responses, while there was a trend for and individuals with IVD to produce more juxtaposition responses.

Inconsistent with this prediction, however, were the results of within-group analyses, which demonstrated that although IVD participants were more likely to make more *out of set* than *in set* errors, there was no difference in the number of *in set* versus *out of set* errors made by AD participants. Although a double dissociation between the dementia groups was not obtained, these finding are consistent with the notion that the underlying deficits in impaired concept formation differ between AD and IVD patients; and, that the underlying cognitive mechanisms associated with impaired concept formation as assessed with the SIM may be related, at least in part, to the specific pattern of cognitive impairment that is associated with these two dementia syndromes.

Our second prediction was that when in set and out of set SIM errors were factor analyzed with neuropsychological tests, in set and out of set errors would load on separate factors. We predicted that *in set* errors would load with tests related to language and semantic knowledge

(e.g., Boston Naming Test, the "animal" WLG AI), while out of set errors would load with tests of executive functioning. Consistent with our prediction, the factor analysis reported above showed *in set* and *out of set* errors loaded on different factors, suggesting that different mechanisms might underlie the production of *these* SIM errors. Furthermore, *out of set* errors loaded with tests of executive functions that measure perseveration, capacity for inhibition, and behavior monitoring (i.e., the WMS Mental Control Non-Automatized Index and Graphical Sequence Test-Dementia Version; Lamar et al., 1999). Thus, the results of the factor analysis provide evidence to support that the underlying mechanism for poor concept formation among IVD patients may be related to an inability to monitor behavior, attain mental set, and inhibit impulsive answers.

Contrary to our expectation, the results of the factor analysis showed *in set* errors were not associated with measures of semantic knowledge. Rather, *in set* errors loaded with measures of declarative memory, such that as in set errors increased, CVLT-cued recall intrusion errors also increased, and CVLT recognition discriminability test scores declined. We interpret this finding as a possible link between *in set* errors and impaired response selection. That is, increased intrusion errors and poor recognition memory may be related to difficulties in selecting the most appropriate response from a set of competing alternatives. Likewise, *in set* errors may represent an inability to select the most appropriate response from a set of related and simultaneously activated semantic concepts. For example, when asked to provide a superordinate response regarding how a coat and a suit are alike, the information that they are *clothing*, and that they can be *bought in a store* may both become activated. Although clothing is a more optimal and salient superordinate characteristic, a deficit in response selection may cause any activated unit of information to be reported. A primary deficit in semantic knowledge may, indeed, underlie this behavior. However, we acknowledged that no direct evidence for this hypothesis was obtained in the present research. We also acknowledge that because of our modest sample size the results of the factor analysis must be interpreted with caution.

Our findings may be interpreted within the conceptual framework of frontal systems dysfunction in dementia as set forth by Lamar et al. (1997). In that study we proposed a hierarchical model of executive functions impairment among patients with AD and IVD. The model suggests that the executive systems dysfunction seen in AD is specific to the semantic/representational difficulties that are present in the disorder. By contrast, the executive systems dysfunction seen in individuals with IVD is associated with pervasive impairment of very rudimentary cognitive functions. The results of the present study parallel the findings of Lamar et al. AD patients were capable of responding within the general parameters of the task; however, response selection difficulties arose when they were required to retrieve and process semantic information. By contrast, the deficit observed among IVD subjects appeared to be at an earlier stage of processing, such that they were unable to sustain mental set to respond according to the task requirements. Thus, it appears that the production of *out of set* errors may be related to a more primary or pervasive impairment in the ability to monitor behavior, inhibit impulsive responses, and sustain mental set.

In sum, the present research suggests that impairment in concept formation as assessed with the SIM may be related to deficits in more elementary cognitive operations, such as

attaining mental set or response selection. We acknowledge, however, that our interpretations regarding the relationship between SIM variables and neuropsychological test performance are post-hoc and require further investigation. We believe that a better understanding of the cognitive components of concept formation may have implications for addressing issues regarding treatment strategies for cognitively impaired patients, as concept formation or the ability to make comparisons between two or more objects or events is crucial for carry over of rehabilitation gains (Cicerone & Tupper, 1991).

References

Albert, M. L., Feldman, R. G., & Willis, A. L. (1974). The 'subcortical dementia' of progressive supranuclear palsy. *Journal of Neurology, Neurosurgery and Psychiatry, 37*, 121–130.

Almkvist, O. (1994). Neuropsychological deficits in vascular dementia in relation to Alzheimer's disease: reviewing evidence for functional similarity or divergence. *Dementia, 5*, 203–209.

Butters, N., Wolfe, J., Granholm, E., & Martone, M. (1986). An assessment of verbal recall, recognition, and fluency abilities in patients with Huntington's disease. *Cortex, 22*, 11–32.

Chan, A. S., Butters, N., Paulsen, J. S., Salmon, D. P., Swenson, M. R., & Maloney, L. T. (1993). An assessment of semantic networks in patients with Alzheimer's disease. *Journal of Cognitive Neuroscience, 5*, 254–261.

Chui, H. C., Victoroff, J. I., Margolin, D., Jagust, W., Shankle, R., & Katzman, R. (1992). Criteria for the diagnosis of ischemic vascular dementia proposed by the State of California Alzheimer's Disease Diagnostic and Treatment Centers. *Neurology, 42*, 473–480.

Cicerone, K. D., & Tupper, D. E. (1991). Neuropsychological rehabilitation: treatment of errors in everyday functioning. In D. E. Tupper, & K. D. Cicerone (Eds.), *The neuropsychology of everyday life: issues in development and rehabilitation* (pp. 50–63). Boston: Kluwer Academic Publishers.

Cloud, B. S., Swenson, R., Malamut, B. L., Kaplan, E., Sands, L. P., Gitlin, H. L., & Libon, D. J. (1994). The Boston Revision of the Wechsler Memory Scale-Mental Control subtest. Poster presented at the 22nd annual meeting of the International Neuropsychological Society, Cincinnati, OH.

Cronin-Golomb, A., Corkin, S., & Growdon, J. H. (1988). Impaired problem solving in Parkinson's disease: influence of set shifting deficit. *Abstract Society of Neuroscience, 14*, 218.

Cronin-Golomb, A., Corkin, S., & Rosen, T. J. (1993). Approaches to the study of dementia. In P. J. Whitehouse (Ed.), *Dementia* (pp. 101–112). Philadelphia: F. A. Davis.

Cronin-Golomb, A., Rho, W. A., Corkin, S., & Growdon, J. H. (1987). Abstract reasoning in age-related neurological disease. *Journal of Neural Transmission, 24* (Suppl.), 79–83.

Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (1987). *California Verbal Learning Test manual*. San Antonio: Psychological Corporation.

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). Mini-mental state: a practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research, 12*, 189–198.

Giovannetti-Carew, T. G., Lamar, M., Cloud, B. S., Grossman, M., & Libon, D. J. (1997). Impairment in category fluency in ischaemic vascular dementia. *Neuropsychology, 11*, 400–412.

Goldberg, E. (1986). Varieties of perseveration: a comparison of two taxonomies. *Journal of Clinical and Experimental Neuropsychology, 8*, 710–726.

Goldberg, E., & Tucker, D. (1979). Motor perseveration: a comparison of two taxonomies. *Journal of Clinical and Experimental Neuropsychology, 1*, 273–288.

Kaplan, E., Fein, D., Morris, R., & Delis, D. (1991). *The Wechsler Adult Intelligence Scale-Revised as a neuropsychological instrument*. San Antonio: Psychology Corporation.

Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *The Boston Naming Test*. Philadelphia: Lea & Febiger.

Kertesz, A., & Clydesdale, S. (1994). Neuropsychological deficits in vascular dementia versus Alzheimer's disease. *Archives of Neurology, 51*, 1226–1231.

Lafleche, G., & Albert, M. S. (1995). Executive function deficits in mild Alzheimer's disease. *Neuropsychology, 9*, 313–320.

Lamar, M., Giovannetti, T., & Libon, D. J. (1999). Frontal systems deficits in cortical and subcortical dementia. *Journal of the International Neuropsychological Society, 5*, 151. Poster presented at the 27th annual meeting of the International Neuropsychological Society.

Lamar, M., Podell, K., Carew, T. G., Cloud, B. S., Resh, R., Kennedy, C., Goldberg, E., Kaplan, E., & Libon, D. J. (1997). Perseverative behavior in Alzheimer's disease and subcortical ischemic vascular dementia. *Neuropsychology, 11*, 523–534.

Ledesma, L. K., Nicdao, S. T., Dantes, M. B., & Perez, M. C. (1986). Neuropsychological profile of primary parkinsonism among Filipinos. In M. D. Yahr, & K. J. Bergmann (Eds.), *Advances in neurology: Parkinson's disease* (vol. 45. pp. 421–424). New York: Raven Press.

Lezak, M. D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.

Libon, D. J., Bogdanoff, B., Bonavita, J., Skalina, S., Cloud, B. S., Cass, P., & Ball, S. K. (1997). Neuropsychological deficits associated with ischaemic vascular dementia caused by periventricular and deep white matter alterations. *Archives of Clinical Neuropsychology, 12*, 239–250.

Libon, D. J., Bogdanoff, B., Swenson, R., Gitlin, H. L., Ball, S., Leopold, N., Hurka, R., Bonavita, J., & Skalina, S. (2000). *Neuropsychological profile associated with subcortical white matter alterations and Parkinson's disease — implications for the diagnosis of dementia* (in press).

Libon, D. J., Mattson, R. E., Glosser, G., Kaplan, E., Malamut, B., Sands, L. P., Swenson, R., & Cloud, B. S. (1996). A nine-word dementia version of the California Verbal Learning Test. *The Clinical Neuropsychologist, 10*, 237–244.

Maher, E., Smith, E. M., & Lees, A. J. (1985). Cognitive deficits in the Steele–Richardson–Olszewski syndrome (progressive supranuclear palsy). *Journal of Neurology, Neurosurgery, and Psychiatry, 48*, 1234–1239.

Martin, A., & Fedio, P. (1983). Word production and comprehension in Alzheimer's disease: the breakdown of semantic knowledge. *Brain and Language, 19*, 124–141.

McKhann, G., Drachman, D., Folstein, M., Katzman, R., Price, D., & Stadlan, E. M. (1984). Clinical diagnosis of Alzheimer's disease: report of the NINCDS-ADRDA work group under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. *Neurology, 34*, 939–943.

Monsch, A. U., Bondi, M. W., Butters, N., Paulsen, J. S., Salmon, D. P., Brugger, P., & Swenson, M. R. (1994). A comparison of category and letter in Alzheimer's and Huntington's disease. *Neuropsychology, 8*, 25–30.

Perez, F. I., Rivera, V. M., Meyer, J. S., Gay, J. R. A., Taylor, R. L., & Matthew, N. T. (1975). Analysis of intellectual and cognitive performance in patients with multi-infarct dementia, vertebrobasilar insufficiency with dementia, and Alzheimer's disease. *Journal of Neurology, Neurosurgery, and Psychiatry, 38*, 533–540.

Pillon, B., Dubois, B., Lhermitte, F., & Agid, Y. (1986). Heterogeneity of cognitive impairment in progressive supranuclear palsy, Parkinson's disease, and Alzheimer's disease. *Neurology, 36*, 1179–1185.

Reitan, R. M., & Boll, T. J. (1971). Intellectual and cognitive functions in Parkinson's disease. *Journal of Consulting and Clinical Psychology, 37*, 364–369.

Rosser, A., & Hodges, J. R. (1994). Initial letter and semantic category fluency in Alzheimer's disease, Huntington's disease, and progressive supranuclear palsy. *Journal of Neurology, Neurosurgery, and Psychiatry, 57*, 1389–1394.

Sagar, H. F., & Sullivan, E. V. (1988). Patterns of cognitive impairment in dementia. In C. Kanard (Ed.), *Recent advances in clinical neurology* (pp. 27–42). New York: Livingstone.

Smith, E. E. (1995). Concepts and categorization. In E. E. Smith, & D. N. Osherson (Eds.), *Thinking: an invitation to Cognitive Science* (2nd ed.; pp. 132–147). Cambridge: MIT Press.

Villardita, C. (1993). Alzheimer's disease compared with cerebrovascular dementia: neuropsychological similarities and differences. *Acta Neurologica Scandinavica, 87*, 299–308.

Wechsler, D. (1945). A standardized memory test for clinical use. *Journal of Psychology 19*, 87–95.

Wechsler, D. (1987). The Wechsler Adult Intelligence Scale-Revised. Psychology Corporation, San Antonio, TX. *Journal of Psychology, 19*, 87–95.

Whelihan, W. M., & Lesher, E. L. (1985). Neuropsychological changes in frontal functions with aging. *Developmental Neuropsychology, 1*, 371 380.

Yesavage, J. (1986). The use of self-rating depression scales in the elderly. In L. W. Poon (Ed.), *Handbook of clinical memory assessment of older adults* (pp. 213 217). Washington, DC: American Psychological Association.