Task Decomposition Analysis of Intertrial Free Recall Performance on the Rey Auditory Verbal Learning Test in Normal Aging and Alzheimer's Disease*

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ABSTRACT

Task decomposition provides supplementary data that complement traditionally computed performance indexes of multi-trial list learning. Both traditional and decomposition approaches can be combined to permit a thorough assessment of multiple aspects of learning and memory in patients with memory impairment. We applied task decomposition to investigate the relative roles of acquisition and consolidation in mediating the multi-trial learning deficit in patients with Alzheimer's disease. This goal was accomplished by decomposing recall performance across the five study-and-test trials of the Rey Auditory Verbal Learning Tests into measures that presumably tap intertrial acquisition and intertrial consolidation. As compared to matched controls, patients diagnosed with mild Alzheimer's disease showed lower gained access across trials, indicating that Alzheimer's disease impairs the ability to produce a stable memory representation of new material in long-term memory. Additionally, patients with Alzheimer's disease manifested higher lost access, which suggests that deficient consolidation leading to rapid intertrial forgetting also contributes to their poor learning. We argue that analytically decomposing learning curves will help both in uncovering the cognitive processes that underlie disease-related learning deficits in persons with memory disorders and can help to characterize potential areas for remediation.

Word list learning tasks are commonly used in clinical neuropsychology for the assessment of memory disorders and can provide a rich set of data to characterize memory functioning. Traditional analysis of performance across trials on these tasks yields considerable information, including acquisition, learning rate, susceptibility to proactive and retroactive interference, and retention/forgetting. These traditional measures typically rely on global comparisons between overall performance across specific trials. However, more fine-grained analysis of learning performance within trials can be of potential utility to identify specific cognitive deficits that could underlie impaired memory functioning. Characterizing learning performance along these dimensions can potentially facilitate diagnostic determinations as well as identify potential areas for remediation.

The Rey Auditory Verbal Learning Test (AVLT; Rey, 1964) was perhaps one of the first standard tests of multitrial list learning that achieved widespread clinical use. This test utilizes five critical study-test trials of a list of 15 unrelated words. On a given trial, each word is individually read aloud by the examiner. After

the final word of the list has been presented, the participant tries to recall as many list words as possible. Learning is operationalized by changes in the number of words recalled across the five trials. Aspects of retention can also be examined, because the fifth trial is followed by a study-test trial of a distractor list, which in turn is followed by immediate and delayed recall trials of the critical list. Thus, susceptibility to proactive and retroactive interference and retention/forgetting may be assessed with the AVLT. The California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan, & Ober, 1986) adopts the same basic format of the AVLT, but uses a total of 16 words (4 items from 4 semantic groups). The task is also presented in the context of learning a grocery list, and the CVLT also makes use of immediate and delayed cued recall. The Selective Reminding Task (SRT; Buschke, 1973; Buschke & Fuld, 1974) is another frequently used measure of list learning. Unlike the AVLT and CVLT, the SRT utilizes selective reminding of only those words that were not recalled on the immediately preceding trial after the first presentation of the list. In addition, constructs involving aspects of storage and retrieval are operationalized and scored for the SRT. Word list learning tasks are also implemented in the Consortium to Establish a Registry for Alzheimer's Disease (CERAD; Morris, Mohs, Rogers, Fillenbaum, & Heyman, 1988; Welsh, Butters, Hughes, Mohs, & Heyman, 1991; Welsh, Butters, Hughes, Mohs, & Heyman, 1992) neuropsychological battery and the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS; Randolph, 1998; Randolph, Tierney, Mohr, & Chase, 1998).

The AVLT has been reported to be the most frequently used measure of list learning in clinical neuropsychology, with a reported usage of 46% among International Neuropsychological Society members responding to a survey of usage of neuropsychological test instruments (Butler, Retzlaff, & Vanderploeg, 1991), compared to usages of 36% and 28% for the CVLT and SRT, respectively. Traditional summary measures of list acquisition and recall are routinely calculated. Acquisition has typically been evaluated by tallying the total number of words re-

called across the first five recall trials, and learning rate can be examined by comparing the number of words recalled on the first trial with the number of words recalled on the fifth recall trial, illustrating whether repeated presentations of the word list contribute to gains in recall performance. Retention/forgetting has also been commonly assessed by comparing the number of words recalled on the fifth recall trial with the number of words recalled after a 20-minute delay period. Although these relatively simple comparisons *across* trials are routinely calculated by clinicians and have diagnostic utility, they neglect more specific trial-to-trial components of the learning curve that can potentially highlight discrete aspects of a patient's learning strategy. Performance *within* individual trials may involve both deficits in encoding as well as in forgetting, but these processes are not easily distinguishable in the global across-trial measures (e.g., total number of words recalled) that are typically computed for multi-trial list learning tasks.

One purpose of this study was to illustrate the use of a trial-to-trial decomposition method with the AVLT, which is designed to yield measures of both gained items and lost items across adjacent study-test trials. Because relatively little is known about the manner by which encoding, consolidation, or rapid forgetting deficits may combine to limit performance of patients with dementia of the Alzheimer's type (DAT) across the individual study-test trials, this decomposition method was applied to a group of patients with DAT. These individuals are presumed to exhibit deficits in encoding and consolidation, as well as accelerated forgetting (Corkin et al., 1984; Delis et al., 1991; Freed, Corkin, Growdon, & Nissen, 1989; Hart, Kwentus, Harkins, & Taylor, 1988; Kopelman, 1985; Martin, Brouwers, Cox, & Fedio, 1985; Moss, Albert, Butters, & Payne, 1986; Ober, Koss, Friedland, & Delis, 1985; Shimamura, Salmon, Squire, & Butters, 1987; Tierney et al., 1994; Weingartner et al., 1981). Performance of patients with DAT was contrasted with a group of healthy, age– and education-matched control participants.

When this task decomposition approach is used, the AVLT (and similar multi-trial list

learning tasks, such as the CVLT) can be used to evaluate whether these factors jointly mediate performance, because trial-by-trial performance can be decomposed into measures that presumably tap intertrial recall gains and intertrial recall failures. Decomposing AVLT performance into these measures highlights the fact that overall recall performance should not be treated as a process-pure indicator of encoding effectiveness.

Trial-by-trial performance on the AVLT can be decomposed into a measure of gains in access to items from one trial to the next ('gained access') and a measure of losses in access to items from one trial to the next ('lost access'). These measures are derived as follows: Gained access is the proportion of items correctly recalled on trial $n + 1$ that had not been recalled on trial n, whereas lost access is the proportion of items not recalled on trial $n + 1$ that had been correctly recalled on trial n (Blachstein, Vakil, & Hoofien, 1993; Dunlosky & Salthouse, 1996; Salthouse & Dunlosky, 1995); cf. gained access and lost access to the measures of intra-trial retention and intertrial forgetting (Tulving, 1964). Gained access reflects intertrial acquisition and hence can be presumed to be a function of the degree to which a representation of an item in memory is strengthened during a particular study trial. Lost access reflects intertrial consolidation deficits that lead to rapid intertrial forgetting and may be conceptualized as the proportion of items that do not possess sufficient strength to be recalled consistently.

METHOD

Participants

The patient group was composed of 6 patients who met National Institute of Neurological and Communicative Disorders and Stroke-Alzheimer's Disease and Related Disorders Association criteria for probable Alzheimer's disease (McKhann et al., 1984). Diagnostic consensus was obtained between a licensed psychologist and a board-certified neurologist. Each patient had been referred for neuro-

psychological evaluation, and each patient completed the AVLT as part of a larger neuropsychological battery. The mean Dementia Rating Scale score (Mattis, 1973) was 111.5, with SD of 11.7 and range from 91 to 126.

For each DAT patient, 3 adults were chosen from a pool of individuals from a larger study (Salthouse, Fristoe, & Rhee, 1996). The participants in this larger study were community-dwelling individuals between the ages of 18 and 94 recruited via newspaper advertisement for a study of age-related performance on a larger neuropsychological battery. The average self-reported health rating for the entire sample was in the very good range, and 93% of the participants in the overall sample rated their health as good, very good, or excellent. The 3 control participants were chosen based on their match to a given patient with respect to age, education, and sex. Because age-related effects (as compared to education and sex) would most likely be greater on the various measures of performance from AVLT, age was matched as closely as possible before matching on the other characteristics. If exact matches were not possible, we attempted to produce differences between groups that slightly favored the patients with DAT (e.g., the control participant was older or had less education). Table 1 includes demographic characteristics of the patients with DAT and the matched controls.

Materials and Procedure

The AVLT was administered using standardized materials and procedures described by Spreen and Strauss (1991). The technique involves study and test trials of two 15-word lists (one critical list and one distractor list) that consist of concrete nouns. Stimulus words were read aloud at a rate of approximately one word per second. Participants were instructed to remember as many words as possible and to recall them in any order. All responses (but not the order of response output) were recorded.

Words on the critical list were presented in the same order for five study-test trials. After the fifth test trial of the critical list, the distractor list was presented for one study-test trial. Immediately following this study-test trial of the distractor list, participants were asked to recall all of the words from the critical list. Finally, participants completed 20-min of non-memory-related activities, after which they were again asked to recall the words from the critical list.

Table 1. Demographic Characteristics of Patients with Alzheimer's Disease with Corresponding Mean Values for Each Patient's Three Matched Controls.

| | Subject characteristics | | | | | | |
| | Alzheimer patients | | | | Matched controls | | |
Subject number	Age (years)	Education[a]	Gender		Age (years)	Education[a]	Gender
1	78	3	F		78.7	2.7	2 F
2	69	3	F		69.0	3.7	3 F
3	75	5	F		75.0	2.3	1 F
4	72	3	M		72.0	3.3	2 F
5	64	5	M		64.3	3.7	2 F
6	69	5	M		69.0	4.3	1 F
Overall	71.3	3.3	3 F		71.2	4.0	11 F

Note. F = female; M = male; other entries indicate the number of females (F) of the three matched controls or the total number of females for the group.
[a] Education ranges from 1 to 5, where 1 = less than 12 years, 2 = High School graduate, 3 = 13-15 years, 4 = College graduate, and 5 = more than 16 years.

RESULTS

The proportion of words correctly recalled is plotted as a function of trials separately for individuals diagnosed with DAT and for the matched controls in Figure 1. Before reporting the central analyses of gained access versus lost access, we first summarize the analyses of traditional AVLT performance measures, including (a) acquisition (performance across the five study-test trials and total number of words recalled across trials 1 to 5), (b) retention (recall at trial 5 compared to recall at trial 7 and recall at trial 6 compared to recall at trial 7), (c) susceptibility to proactive interference (recall at trial 1 compared to recall for the distractor list), (d) susceptibility to retroactive interference (recall at trial 5 compared to trial 6), and (e) serial-position effects across the five critical study trials. Although some of these analyses have been reported by other investigators (e.g., Tierney, Snow, Reid, Zorzitto, & Fisher, 1987), we focus on them first to establish any DAT-related deficits in recall as well as to connect with previous research. All comparisons declared as significant had *p*-values that were less than .05.

Acquisition
The analysis on trials 1 through 5 revealed main effects of group, $F(1,22) = 42.0$, $MSe = .07$, and trial, $F(4,88) = 26.9$, $MSe = .006$, and a significant Group x Trial interaction, $F(4,88) = 6.62$. As evident from inspection of Figure 1, this interaction is due to less of an increase in performance across trials for individuals with DAT as compared to the matched controls. Note, however, that the curves shown in Figure 1 do not necessarily represent differential rates of learning for the two groups (e.g., scale-dependent interaction, Loftus, 1978) because (a) initial performance on trial 1 is different for the two groups, $t(22) = 5.24$, $p < .001$, and (b) performance does significantly increase from trial 1 to trial 5 for individuals with DAT, $t(5) = 5.48$, $p = .003$. Nevertheless, as in previous research, the differences in trial-by-trial performance were substantial, with performance on trial 5 being greater for 17 (out of 18) of the matched controls than for the patients with DAT. The sum of words recalled across trials 1 through 5, another traditional measure of acquisition, was significantly different between the two groups ($t(22) = 6.48$, $p < .001$) with patients with DAT performing significantly below controls (DAT $M = 18.3$, $SD = 8.4$; Control $M = 44.5$, $SD = 8.6$).
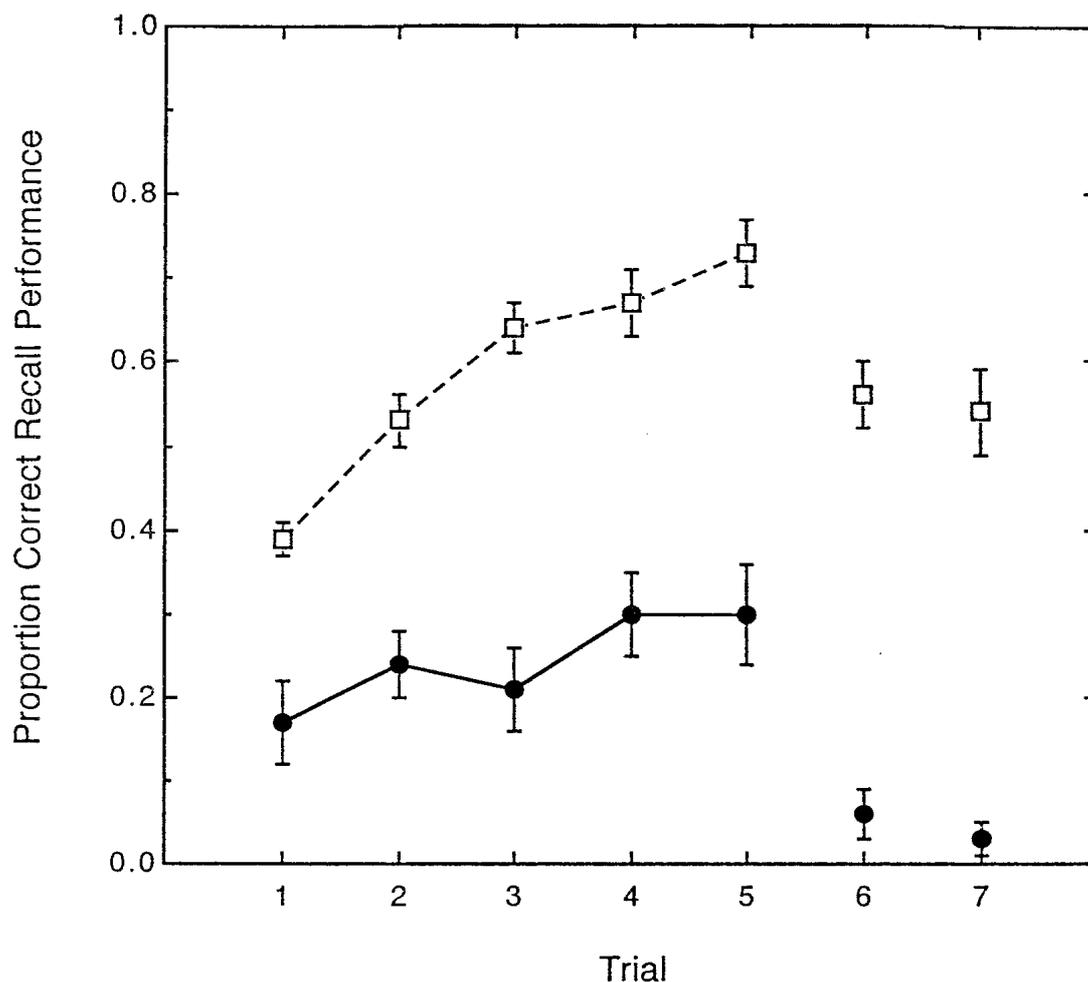
Fig. 1.   For the patients with Alzheimer's disease (circles) and for the matched controls (squares), the mean (across individuals) proportion of correctly recalled items on the critical list is plotted as a function of the five study-test trials and for test trial 6 (which occurred after a study-test trial of a distractor list) and for test trial 7 (which occurred about 20 min after trial 6). Trials 6 and 7 did not include a study trial. Bars represent standard errors of the corresponding mean.

Retention

The number of words recalled at trial 7 was compared against two different reference points. First, the trial 5 versus trial 7 comparison examines the retention of list words after 20 min relative to the most likely 'best' performance after five consecutive learning trials. The main effects for group and for trial were both significant in the ANOVA contrasting trial 5 versus trial 7, $Fs > 31.0$, $MSes < .05$. The Group x Trial interaction was not significant, $F(1,22) = 0.91$, but this may be attributable to the near-floor level of performance on trial 7 for patients with DAT. Second, the trial 6 versus trial 7 comparison compares the number of words recalled after 20 min with the number of words recalled after having been presented with a distractor list. The contrast between trials 6 and 7 revealed a main effect of group, $F(1,22) = 39.8$, $MSe = .06$, but the main effect of trial and the interaction were

not significant, Fs < 0.55, MSe = 0.009, which again might be related to the floor effects. When the traditional 'savings' score (percent of words recalled after 20 min relative to the number of words recalled at trial 5) was compared directly for the two groups, a significant difference was again observed ($t(22)$ = 5.9, p < .001; DAT M = .12, SD = .20; Control M = .74, SD = .23).

Susceptibility to proactive interference
The extent to which prior learning interferes with the recall of a new word list can be evaluated by comparing the number of words recalled after the first presentation of the first word list to the number of words recalled from the distractor list (trial 1 recall vs. List B recall). Although the main effect for group was significant ($F(1,22)$ = 17.6, p < .001), neither the main effect for trial nor the Group × Trial interaction were significant Fs(1,22) < 3.6, ps > .05. This absence of a significant interaction effect again is likely to be attributable to the near floor level of performance for List B recall.

Susceptibility to retroactive interference
The extent to which learning a new list interferes with recall of a previously learned list may be examined by comparing recall at trial 5 (prior to presentation of the distractor list) to recall at trial 6 (following presentation of the distractor list). The main effects for group and for trial were both significant in the ANOVA contrasting trials 5 versus trial 6, Fs > 37.0, MSes < .05. The Group × Trial interaction was not significant, $F(1,22)$ = 1.35, but this may be attributable to the near-floor level of performance on trial 6 for individuals with DAT.

In sum, recall performance was substantially less for individuals with DAT than for age-matched controls across all trials, with patients with DAT showing less improvement in performance across the five study-test trials. The near-floor performance of patients with DAT on trials 6 and 7, as well as during recall of List B, reduced our ability to detect a significant Group × Trial interaction on traditional measures of AVLT performance due to diminished variability in the DAT group.

Recall performance as a function of serial positions
To provide a more complete characterization of multi-trial performance, we examined performance on the AVLT across various serial positions of the presentation of items. As described previously (Dunlosky & Salthouse, 1996), performance across the serial positions was collapsed into three segments: 'Primacy' is performance collapsed across serial positions 1-3, 'asymptote' is performance collapsed across positions 5-11, and 'recency' is performance collapsed across serial positions 13-15. Means across individual subject's values for each of the three segments and for the five critical study-test trials are reported in Table 2.

A 2 (patients with DAT vs. control) × 5 (critical study-test trials) × 3 (primacy, recency, asymptote) ANOVA was conducted. As evident from inspection of Table 2, main effects occurred for group, $F(1,22)$ = 38.47, MSe = .21, for trial, $F(4,88)$ = 13.51, MSe = .03, and for serial position, $F(2,44)$ = 30.76, MSe = .12. Two interactions were also significant: (a) the Group × Trial interaction, $F(4,88)$ = 3.80, indicating that performance generally increased across trials for the matched controls but showed little increase for the patients with DAT, and (b) the Group × Serial position interaction, $F(2,44)$ = 3.77, indicating a somewhat smaller difference in scores between asymptote and primacy positions for the patients with DAT than for the matched controls. The Trial × Serial position interaction and the three-way interaction were not significant, Fs < 1.40, MSes = .04.

Although the Group × Serial position interaction is compromised by floor effects of the patients with DAT, this interaction does not compromise the significant main effects. Namely, where interpretation is not limited by floor effects, patients with DAT and the matched controls show both primacy and recency effects across multiple trials, which is consistent with previous findings based on a single study-test trial (Pepin & Eslinger, 1989).

Gained Access and Lost Access
Finer-grained analyses of trial-by-trial performance involved examining gained access versus

Table 2. Proportion Correct Recall Performance as a Function of Serial Position

Trial	Segment of Serial-position Curve					
	Primacy		Asymptote		Recency	
	M	(SD)	M	(SD)	M	(SD)
Alzheimer patients						
1	.06	(.06)	.05	(.03)	.61	(.16)
2	.33	(.15)	.07	(.05)	.61	(.16)
3	.22	(.17)	.12	(.04)	.50	(.14)
4	.39	(.16)	.10	(.05)	.61	(.13)
5	.22	(.11)	.17	(.09)	.67	(.17)
Matched controls						
1	.52	(.06)	.24	(.04)	.67	(.05)
2	.65	(.06)	.38	(.05)	.80	(.06)
3	.78	(.07)	.50	(.05)	.85	(.05)
4	.82	(.05)	.56	(.05)	.83	(.06)
5	.83	(.04)	.66	(.06)	.80	(.06)

Note. Entries in parentheses are standard errors of the corresponding mean. The apparent group differences in standard errors are at least partially attributable to the differences in sample size.

lost access between trials, which were calculated as follows: Gained access was the proportion of items recalled on a given trial that were not recalled on the previous trial, and lost access was the proportion of items not recalled on a given trial that were recalled on the previous trial. The means (across individuals) of the measure of gained access and of the measure of lost access are shown in Figure 2. A Group × Trial × Kind of fluctuation (gained vs. lost access) ANOVA was conducted separately for performance on the five critical study-test trials and for performance across the retention trials.

The ANOVA for the five study-test trials revealed a marginal effect for group, $F(1,21) = 4.29$, MSe = .07, p = .051, and a main effect of trial, $F(3,63) = 3.76$, $MSe = .03$. The effect of kind of fluctuation, $F(1,21) = 2.91$, $MSe = .08$, was not significant. Most important, the Group × Kind of fluctuation interaction was significant, $F(1,21) = 15.58$, $MSe = .08$ (all other interactions were not significant, $Fs < 2.70$, $MSes < .04$). This interaction is due to the crossover interaction evident in Figure 2: Gained access was *less* for patients with DAT than for matched controls, whereas lost access was *greater* for patients with DAT than for matched controls.

The ANOVA for the retention trials (5 and 6) revealed no main effects for group or for trial, $F(1,18)s < 3.25$, $MSes < .07$. The main effect for kind of fluctuation, $F(1,18) = 8.39$, $MSe = .08$, and the Group × Kind of fluctuation interaction, $F(1,18) = 7.88$, $MSe = .08$, were significant. The latter is due to a crossover interaction across the retention trial in which gained access was again less for patients with DAT than for matched controls, whereas lost access was greater for patients with DAT than for matched controls. Another interesting aspect concerning gained access across the retention trial is that patients with DAT had no upward fluctuations of recall across the two retention trials. All other interactions were not significant, $Fs < 3.0$, $MSes < .06$.

Relationships between traditional measures and gained and lost access

The trial-to-trial measures of gained access were aggregated to reflect the *total* gained access across the five study-test trials. In like manner, the lost access measures across trials were aggregated to reflect the *total* lost access across the five study-test trials. Pearson product-moment correlations were performed separately for the controls and patients with DAT between tra-
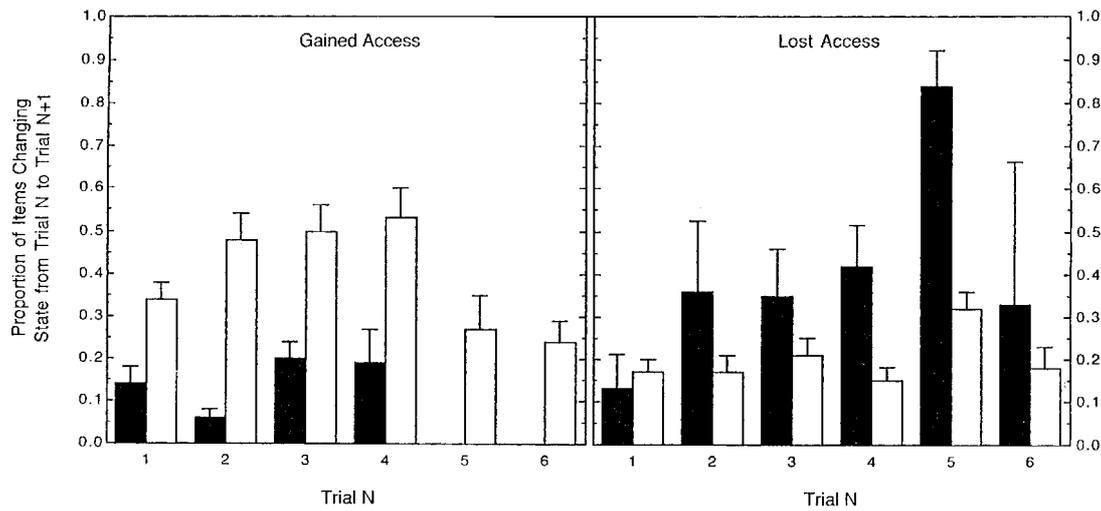
Fig. 2.   Gained access and lost access plotted as a function of group, where gained access is the mean proportion of recalled items on trial n+1 of those that were not recalled on trial n, and lost access is the mean proportion of items not recalled on trial n+1 of those that were recalled on trial n. Accordingly, note that values labeled 1 through 4 represent fluctuations in recall across the 5 critical study-test trials, whereas values labeled 5 and 6 represent fluctuations in recall across the retention trials. Black bars = Alzheimer's patients; white bars = matched controls.)

ditional measures of acquisition (sum of words recalled across trials 1 through 5), learning rate (difference between words recalled at trial 5 and words recalled at trial 1), and retention ('savings' score) on the one hand and gained and lost access on the other hand. Although our small sample size (particularly in the DAT group) limits our power to detect significant correlations, this analysis was performed to determine if general trends may be evident to support the construct validity of gained and lost access.

First, measures of gained and lost access were uncorrelated in either group, suggesting their relative independence. For the control group, gained access showed a significant relationship with both acquisition ($r = +0.88$, $p < .001$) and learning rate ($r = +0.52$, $p = .03$) but not with retention ($r = +0.25$, $p = .31$). For the DAT group, gained access also showed a significant relationship with acquisition ($r = +0.91$, $p < .02$) but not with learning rate ($r = +0.18$, $p = .74$) or retention ($r = –0.13$, $p = .80$). The diminished gained access and learning rate in the DAT group may have resulted in a restriction of range

relative to control participants. Nevertheless, the construct of gained access demonstrated a substantial overlap with a traditional AVLT measure of acquisition. The relationship between lost access and acquisition showed a nonsignificant trend ($r = –0.43$, $p < .08$). A nonsignificant trend was also seen between lost access and learning rate ($r = –0.40$, $p = .10$), but lost access was uncorrelated with retention ($r = +0.11$, $p = .67$) in the control group. Again, because controls showed less lost access than the DAT group, a restriction of range may have decreased the correlation between measures relative to the DAT group. For the DAT group, lost access was significantly correlated with learning rate ($r = –0.90$, $p < .04$) but was not correlated with acquisition ($r = –0.21$, $p = .73$) or retention ($r = +0.09$, $p = .89$).

In summary, traditional measures of AVLT performance, including number of words recalled across trials, savings (percent of words recalled after 20 min relative to the number of words recalled at trial 5), and susceptibility to proactive and retroactive interference all demon-

strated significant consolidation and 20-min retention deficits in the patients with DAT relative to matched controls. With respect to measures of gained access and lost access, patients with DAT demonstrated greater lost access and less gained access relative to the control group. Correlational analyses suggested independence between gained access and lost access. A substantial overlap was observed between gained access and a traditional measure of acquisition in both groups. Traditional measures of retention were uncorrelated with either gained or lost access. However, for patients with DAT (who showed greater levels of lost access than controls), lost access showed a significant negative relationship with learning rate.

GENERAL DISCUSSION

The present study illustrates the potential utility of decomposing learning curves in order to understand the manner by which learning might be impaired in various memory disorders. This approach allows the differentiation between deficits in intertrial acquisition versus deficits in intertrial consolidation. This decomposition technique can be used to analyze learning curves for any of the numerous multi-trial learning tasks that are commonly used in neuropsychological research. Such an approach has previously been applied to understanding the nature of the impaired learning curve in patients with closed-head injury (Blachstein et al., 1993), as well as age-related differences in verbal learning in non-demented adults (Dunlosky & Salthouse, 1996).

The results of this study suggest that both impaired encoding and impaired consolidation of items *between adjacent trials* underlie impaired multi-trial performance of patients with DAT. Namely, relative to matched controls, patients with DAT showed substantial decline in gained access to words across trials, and they recalled fewer words from those that they had recalled on the preceding trial (lost access), which reflects relatively incomplete storage even with intervening maintenance trials.

Besides having difficulties encoding and retaining items across the five critical trials, patients with DAT showed poor retention across the distractor trial as has been noted previously (Tierney et al., 1994). They had no upward fluctuations (Figure 2) across the retention interval and also showed considerable lost access across the same interval. Such deficits combined to produce nearly complete failure of overall recall across the retention interval. Furthermore, as has been observed in previous studies, all of the patients with DAT were below all of the control participants on performance during the retention interval.

In a study examining age-related differences in gained access versus lost access in non-demented adults (Dunlosky & Salthouse, 1996), substantial age-related differences occurred in the measure of gained access, whereas little age-related difference occurred in the measure of lost access. These findings are informative here because they demonstrate that one factor (normal aging) can substantially affect multi-trial free recall through specific reductions in intertrial acquisition while leaving the measure of intertrial consolidation relatively spared. Previous studies using the AVLT (Mitrushina, Satz, Chervinsky, & D'Elia, 1991; Petersen, Smith, Ivnik, Kokmen, & Tangalos, 1994) have shown that retention is generally stable across age, whereas acquisition typically shows an age-related decline. In preclinical patients with DAT, acquisition was shown to be diminished relative to age-matched controls, whereas retention did not differ when a memory testing procedure that maximized learning by inducing deep semantic processing and by controlling study and test conditions was used (Grober & Kawas, 1997). However, in addition to impaired acquisition, a retention deficit did become evident in these same patients three years later using the same free and cued selective reminding procedure (Buschke, 1984; Grober & Buschke, 1987).

The increase in lost access seen in patients with DAT appeared to have a substantial adverse impact on learning rate across the five critical study-test trials. Because lost access was uncorrelated with the traditional measure of forgetting ('savings') in either group, it is likely to

reflect something other than retention. The correlation between lost access and learning rate seen in the patient group lends support to its conceptualization as reflecting the proportion of items lost due to a consolidation deficit. This deficit may be manifested by either retrieval failure associated with inconsistent recall from long-term memory (analogous to the random long-term retrieval [RLTR] measure from the Buschke SRT) or due to a reliance on recalling information from short-term memory rather than long-term memory (analogous to the short-term retrieval [STR] measure from the Buschke SRT. In addition to impaired acquisition and rapid forgetting, patients with DAT have been shown to exhibit both inconsistent recall from long-term memory as well as an over-reliance on re-calling information from short-term memory (cf., Bondi, Salmon, & Butters, 1994).

Although the generalizability of the results of the present study are potentially limited by the small patient sample size, the overall performance of the participants was consistent with prior studies of multi-trial learning performance in patients with DAT (e.g., Tierney et al., 1994). Furthermore, the statistical contrasts were often statistically significant, suggesting that the effect sizes were generally large.

In conclusion, decomposition of learning curves can complement traditional performance indexes by highlighting the trial-to-trial learning processes that cannot be determined directly by global measures. As such, they can serve to distinguish between deficient acquisition skills and inefficient consolidation that may underlie deficient memory functioning. In doing so, this approach can potentially highlight areas that might be targeted for remediation. Future research along these lines might also include subjects from different diagnostic groups (e.g., Parkinson's disease, Huntington's disease) to help determine whether the relatively high lost access and relatively low gained access pattern might be specific to patients with DAT, or whether it may be seen with patients from other diagnostic categories.

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