

Mechanisms of verbal memory impairment in four neurodevelopmental disorders

Sharon Nichols,^{a,*} Wendy Jones,^b Mary J. Roman,^c Beverly Wulfeck,^d Dean C. Delis,^e
Judy Reilly,^f and Ursula Bellugi^b

^a Department of Neurosciences, University of California, 9500 Gilman Drive, MC-0935, San Diego, CA 92093-0935, USA

^b Salk Institute for Biological Studies, La Jolla, CA, USA

^c Open Gates Dyslexia Program, University of Texas Medical Branch, Galveston, TX, USA

^d School of Speech, Language, and Hearing Sciences, San Diego State University and Center for Research in Language, University of California, San Diego, CA, USA

^e Department of Veterans Affairs Medical Center, La Jolla, CA, USA

^f San Diego State University, San Diego, CA, USA

Accepted 2 April 2003

Abstract

Profiles of verbal learning and memory performance were compared for typically developing children and for four developmental disorders characterized by different patterns of language functioning: specific language impairment, early focal brain damage, Williams Syndrome, and Down Syndrome. A list-learning task was used that allowed a detailed examination of the process of verbal learning, recall, and recognition (California Verbal Learning Test—Children's Version). Distinct patterns of performance characterized the four disorders. These patterns were consistent with the language deficits typically seen in the disorders, with the exception of a dissociation seen in Williams Syndrome.

© 2003 Elsevier Science (USA). All rights reserved.

Keywords: Verbal learning; Verbal memory; Developmental disorders; Brain lesions; Specific language impairment; Williams Syndrome; Down Syndrome

1. Introduction

In recent years, the study of children with developmental disorders or early brain injury has contributed enormously to our understanding of brain–behavior relationships underlying language acquisition (cf. Bates, 1997). This line of investigation has underscored the differences between the developing and adult brain, as well as the potential for, and limitations of, neural plasticity. In particular, it has shown us that the long-term consequences for language of frank structural damage such as that occurring in early stroke can be far less devastating than the consequences of a disorder such as specific language impairment, which is not associated with obvious brain lesions. It has also demon-

strated that language and other cognitive abilities can be dissociated by disorders of brain development.

The development of verbal learning and memory, both in typical children and in those with brain injury, has also seen a recent surge of scientific interest, made possible partly by innovations in assessment tools. Like language, verbal learning is an area with both theoretical and practical implications. The latter is particularly true for school-age children, who spend a huge proportion of their time engaged in tasks that are dependent on verbal learning and memory. For this reason, the assessment of verbal learning is an important part of any evaluation done following childhood traumatic brain injury or for the purpose of educational planning for children with learning or other developmental disabilities. Although a literature addressing these issues has begun to accumulate, the brain–behavior relationships underlying verbal learning and memory in children are

* Corresponding author. Fax: 858-587-8050.

E-mail address: slnichols@ucsd.edu (S. Nichols).

still poorly understood, as is the relationship of verbal learning to language abilities. The purpose of this study was to compare verbal learning and memory in early, focal brain injury and three developmental disorders: specific language impairment (SLI), Williams Syndrome (WMS), and Down Syndrome (DS). Because these groups of children have different patterns of language impairment that are well characterized, we are able to examine the similarity of verbal learning ability to language characteristics in general. Furthermore, we are able to evaluate its plasticity, or potential for reorganization, following focal damage and compare it to brain dysfunction that is possibly, in the case of SLI, or certainly, in the case of WMS or DS, associated with more diffuse abnormalities in brain development.

In adults, brain–behavior relationships underlying verbal learning and memory appear to parallel other language functions in that deficits are most likely following damage to the left hemisphere. Evidence for this asymmetry exists both for the primary memory functions of the medial temporal lobes (but see Dobbins, Kroll, Tulving, Knight, & Gazzaniga, 1998, for a dissenting view) and for the organizational and other memory-related functions of the frontal lobes (Stuss et al., 1994). Damage to the right hemisphere can result in qualitative changes in verbal learning, such as decreased recency effects (Cappa, Papagno, & Vallar, 1990), but a preponderance of evidence supports an association between the left hemisphere and many aspects of verbal memory performance in adults. In the case of damage outside the medial temporal lobes, some investigators have suggested that deficits in verbal memory can be attributed to language dysfunction (Hermann, Seidenberg, Haltiner, & Wyler, 1992; Ween, Verfaellie, & Alexander, 1996), although it should be noted that some studies have found dissociations between aphasia and verbal memory deficits (Beeson, Bayles, Rubens, & Kaszniak, 1993). Given the complexity of memory processes in the brain (see Squire, 1987), it is reasonable to conclude that memory deficits can have multiple causes, aphasia being among them; however, the association between left hemisphere damage and verbal memory deficits remains fairly strong in adults.

Focal lesions occurring early in development produce a markedly different relationship between lesion localization and language functioning than that seen in adults. Although delays in early language acquisition are common, only subtle language problems typically remain by the time the children reach school age, unlike the frank aphasia that can occur in adults with focal left hemisphere damage (Reilly, Bates, & Marchman, 1998). Furthermore, early left and right hemisphere lesions produce comparable effects on language by school age (Bates et al., 2001). Less is known about learning and memory following early, focal lesions. Aram and Ekelman (1988) assessed children with unilateral focal lesions using the

Woodcock–Johnson Psycho-Educational Battery and found that both left- and right-hemisphere lesions were associated with lowered performance on the memory cluster. Studies of verbal learning and memory in children with hippocampal pathology (Vargha-Khadem et al., 1997), closed head injury (Levin et al., 1996), and sickle cell disease (Watkins et al., 1998) have also begun to address the effects of lesion location; however, the latter two disorders are typically associated with widespread damage that complicates the interpretation of the findings.

The subtle language problems of children with early strokes provide an interesting contrast to the relatively severe language dysfunction seen in children with specific language impairment (SLI). Children with SLI have deficits in expressive and/or receptive language that are disproportionately greater than other cognitive problems. Although it could be assumed that their brains are in some manner different from those of children without SLI, obvious brain lesions have not been detected by neuroimaging procedures. However, it is possible that more subtle abnormalities exist that are able to exert a significant impact on brain systems associated with language, without allowing for reorganization, by virtue of their diffuse distribution or subcortical location (Bates, 1997). Differences between children with SLI and typically developing children have been described for a number of aspects of verbal memory, including free and cued retrieval (Kail, Hale, Leonard, & Nippold, 1984), memory scanning speed (Sininger, Klatzky, & Kirchner, 1989), phonological working memory (Montgomery, 1995, 2000), and verbal capacity (Kirchner & Klatzky, 1985; Weismer, Evans, & Hesketh, 1999). Studies comparing auditory short-term memory of children with SLI and language-matched controls, using such measures as digit span or word list tasks, have produced conflicting results (see Gathercole & Baddeley, 1995; van der Lely & Howard, 1993, 1995). However, tasks that look at the learning of supraspan lists over several trials and that assess delayed recall have found that children with SLI perform more poorly than chronological age controls. In a study of 12 children aged 8–9 with SLI, Shear, Tallal, and Delis (1992) found that, although the children had normal immediate memory span, they recalled fewer correct words over the learning trials of the California Verbal Learning Test—Children's Version (Delis, Kramer, Kaplan, & Ober, 1994), a list-learning task, and made more perseverative, but not intrusion, errors than control children. Although their free recall following a short delay was intact, they had difficulty recalling the list after a longer delay and failed to benefit from semantic cueing to the degree that controls did. Shear and colleagues suggested that the verbal learning and memory deficits of children with SLI might result from a limitation in information processing. The number of children in their study was relatively small, however, which may have precluded detecting some group differences.

Children with Williams Syndrome (WMS) represent another group whose language is incongruent with their other abilities. However, in contrast to specific language impairment, language represents a relative strength for children with WMS rather than a weakness. WMS is a rare genetic disorder caused by a deletion of one copy of the elastin gene and other surrounding genes on chromosome 7. The neuropsychological profile of WMS is highly uneven and includes a series of intriguing dissociations both across and within domains of cognitive functioning. Although individuals with WMS have mild to moderate levels of mental retardation, language abilities are often relatively preserved in the face of prominent impairments in spatial cognition. One of the general goals in research with people with WMS is to better understand the interrelationship of components of cognition by examining the pattern of spared and impaired abilities in the disorder. As part of this goal, researchers are currently investigating memory as one possible component of the unusual profile seen in WMS.

Past studies on memory in WMS have focused primarily on short-term memory and its importance as a phenotypic characteristic of the disorder. Memory abilities of people with WMS appear to be characterized by deficits in visuo-spatial short-term memory, but relative strengths in verbal short-term memory (Jarrold, Baddeley, & Hewes, 1999; Mervis, Morris, Bertrand, & Robinson, 1999; Vicari, Brizzolara, Carlesimo, Pezzini, & Volterra, 1996; Wang & Bellugi, 1994). In contrast, individuals with Down Syndrome show the opposite pattern, with strengths in visual short-term memory relative to verbal short-term memory (Carlesimo, Marotta, & Vicari, 1997; Wang & Bellugi, 1994). Down Syndrome (DS) is a genetic disorder, caused by abnormalities of chromosome 21, which is often included in studies of WMS as a basis of comparison for the effects of mild to moderate mental retardation on aspects of cognition. This difference in verbal versus spatial short-term memory in WMS and DS is consistent with the overall patterns of cognitive strengths and weaknesses in these disorders, and has been interpreted by some researchers (Jarrold et al., 1999; Wang & Bellugi, 1994) as evidence for dissociations in short-term or working memory. Because of the association between relatively good verbal working memory and strengths in the more general domain of language in people with WMS, some researchers (Vicari et al., 1996) hypothesize that normal verbal working memory underlies the relatively strong language abilities in this disorder.

In contrast to the findings related to short-term memory, long-term memory appears to be affected equally in children with WMS and DS. In addition, long-term memory also appears to be affected independent of cognitive domain, with deficits found both in verbal as well as spatial long-term memory in both WMS and DS (Vicari et al., 1996). However, only one

study has examined this component of the profile in WMS versus DS, and additional studies are needed to corroborate this finding.

Assessing verbal learning and memory in children has been facilitated in the past decade by the development of sophisticated memory measures influenced by findings from the burgeoning field of cognitive science. One of the most widely used measures has been the California Verbal Learning Test. The children's version of this test (CVLT-C; Delis et al., 1994) has been used to examine characteristics of verbal learning and memory in children with acquired head injury (Roman et al., 1998; Yeates, Blumenstein, Patterson, & Delis, 1995a) and meningomyelocele (Yeates, Enrile, Loss, Blumenstein, & Delis, 1995b), among other disorders. An advantage of the CVLT-C is that it allows the assessment of qualitative aspects of learning and memory such as proactive and retroactive interference, encoding and retrieval processes, efficiency strategies, retention of information over a delay, and facilitation of memory by semantic cues.

The purpose of the present study was to compare verbal learning and memory, using the CVLT-C, in five groups of children: typically developing children and children with SLI, early acquired focal lesions, WMS, and DS. The performance of these groups was predicted to parallel their patterns of language development. In addition to the comparisons of each disordered group with controls, we were interested in whether children with focal brain injury would show less long-term impact of their disorder on verbal learning than would children with more widespread damage (WMS and DS) or dysfunction in the system presumed to underlie language development (SLI). Based on previous studies and our preliminary findings, we hypothesized the following. (1) Children with early acquired brain injury would perform better than the other study groups but show mild deficits in verbal memory compared to controls. (2) Children with specific language impairment would show a generalized deficit affecting both learning and delayed recall and would possibly make more perseverations and intrusion errors than controls. (3) Children with Williams Syndrome would do worse than control children overall but show relative strengths on variables reflecting immediate auditory memory. (4) Children with Down Syndrome would do poorly on all aspects of the task as well, and show a high rate of intrusion errors and difficulty discriminating relevant words from distractors during recognition memory testing.

2. Materials and methods

2.1. Participants

Five groups of children from the Center participated in the current study. All participants included in the

study were from monolingual English backgrounds, and had hearing and corrected vision within normal limits.

Twenty-eight children with specific language impairment (SLI) were included in the study. Participants ranged in age from 6 to 14 (mean age = 8.96 years). There were 20 males and 8 females. The SLI children had been recruited from area speech-language pathologists, psychologists and physicians. These children had documented language impairment and no evidence of frank neurologic abnormalities or developmental disorders such as mental retardation or autism. To participate in the present study, each SLI participant was required to have expressive and/or receptive language scores, assessed by the Clinical Evaluation of Language Fundamentals—Revised (CELF-R; Semel, Wiig, & Secord, 1987), of at least 1.5 *SD* below the mean for his or her age. In addition, each SLI participant was also required to have a nonverbal IQ, assessed by the Wechsler Intelligence Scale for Children—Revised (WISC-R; Wechsler, 1974) Performance I.Q. or the Leiter International Performance Scale (Leiter, 1948), within 1 *SD* of the mean for his or her age.

Twenty-three adolescents and young adults with Williams Syndrome (WMS) were included and were matched to the other experimental groups using the mental age as estimated by the Full Scale I.Q. on the WISC-R or Wechsler Adult Intelligence Scale—Revised (WAIS-R; Wechsler, 1981). These participants ranged in age from 9 to 25 years (mean age = 15.2 years); there were 11 males and 12 females. The mean mental age of the WMS group was 8.0 years (*SD* = 2.8). All WMS participants were required to have been clinically diagnosed with the disorder using criteria developed by the Williams Syndrome Association Medical Advisory Board. Any participant who had a history of medical or neurological abnormality more severe than what is typically found in the syndrome was excluded from the present study (for instance, participants with histories of seizures, stroke, or cancers were not included).

Fourteen adolescents and young adults with Down Syndrome (DS) were included as well. The 4 male and 10 female DS participants ranged in age from 9 to 21 years (mean age = 15.0 years) and had a mean mental age of 7.9 (*SD* = 2.9). The WMS and DS groups did not differ significantly in mental age. All DS participants were diagnosed with Trisomy 21 by a clinical geneticist prior to inclusion in the current study. As with the WMS participants, any participant who had a history of medical or neurological abnormality more severe than what is typically found in the syndrome was excluded from the present study.

Fourteen children with prenatal or perinatal focal brain lesions (FL) were also included. The FL children ranged in age from 6 to 15 years (mean age = 7.8 years), and there were 10 males and 4 females. Children in this group were required to have evidence of a single, acute

onset, unilateral brain lesion documented by CT or MRI scan. The onset of the lesion must have been prior to 6 months of age. Additional exclusionary criteria included the presence of multiple or bilateral lesions; history of a condition that might have caused more global brain damage, such as bacterial meningitis, encephalitis, or head trauma, or evidence of an evolving lesion such as a brain tumor.

Finally, 29 typically developing children (TD), ranging in age from 6 to 15 years (mean age = 9.5 years), were included as a control group. There were 14 males and 15 females. Control children were screened for intellectual and language abilities within the normal range using a battery of standardized tests administered in separate sessions. Control participants were also screened for abnormal neurological soft signs as tested by a neurologist, and for developmental or medical abnormalities as described by their parents during a comprehensive interview.

Participants in the SLI, FL, and TD groups were matched on age and nonverbal intelligence as estimated by the Block Design subtest of the WISC-R. Children with Block Design scaled scores greater than 2 *SD* from the mean (i.e., below 4 or above 16) were excluded from the study. This resulted in the exclusion from the initial sample of 7 (all high) TD, 2 (1 high, 1 low) SLI, and 3 (1 high, 2 low) FL children, resulting in the group sizes given above. Characteristics of the groups are shown in Table 1. Mean standard scores for the Peabody Picture Vocabulary Test—Revised (PPVT-R; Dunn & Dunn, 1981), a test of receptive vocabulary, are provided as an estimate of language functioning free of word retrieval demands. Because participants with WMS and DS typically have mild to moderate mental retardation, these groups were matched to the other experimental groups on the basis of mental age, as assessed with the WISC-R or WAIS-R. One-way Analysis of Variance with age (or mental age for WMS and DS) by experimental group showed that our groups were well matched on this factor ($F(6, 110) = 1.32, p = .26$).

Table 1
Group means (*SD*) for age, PPVT-R, and Block Design

Group	<i>N</i>	Chron. age (years)	Block Design	
			Standard score ^a	Scaled score ^b
SLI	28	8.9 (2.4)	84.96 (10.79)	10.4 (2.4)
WMS	23	15.2 (4.2)	63.69 (14.74)	2.5 (1.4)
DS	14	15.0 (3.7)	42.33 (11.75)	3.0 (2.3)
FL	14	7.8 (2.5)	104.38 (18.36)	9.9 (2.7)
TD	29	9.5 (2.9)	111.93 (15.31)	11.1 (2.7)

^a PPVT-R scores were unavailable for 1 FL, 2 SLI, and 2 DS participants.

^b Block Design scores were unavailable for four participants in each of the WMS and DS groups. These children underwent I.Q. testing through their local schools, and specific subtest scores were not recorded in the reports resulting from these assessments.

In addition, one-way ANOVA with group (SLI, FL, and TD) by Block Design score showed that the SLI, FL, and TD groups were well matched on nonverbal intelligence as well ($F(2, 70) = 1.08, p = .34$).

2.2. Procedure

Participants were administered the California Verbal Learning Test—Children’s Version (CVLT-C) as part of a larger battery of tests. The CVLT-C is a learning and memory test in which the participant is read a “shopping list” of 15 words (List A) from three semantic categories (fruit, clothing, and toys). The words are presented in a fixed random order at the rate of approximately one word per second. The list is presented five times, and the participant is asked to recall as many of the words as possible on each of the five trials. Following the fifth trial of List A, an interference list of 15 words is read (List B), and the participant is asked to recall as many words as possible. The words on List B are also from three semantic categories (fruit, sweets, and furniture). After recalling List B, the participant is then asked to recall the words from List A, first freely (Short-Delay Free Recall) and then cued by semantic category (Short-Delay Cued Recall). After a 20-min delay during which the participant performs nonverbal tasks, free and cued recall of List A are again elicited (Long-Delay Free, Long-Delay Cued Recall). Following these trials, recognition memory is tested with a yes/no recognition test. The participant is read a list of 45 words (15 List A words and 12 List B words interspersed with non-list distracters from the same semantic categories, phonemically similar words, and unrelated words) and asked to say “yes” if each word was from List A, and “no” if it was not. The number of target words identified correctly and the number of false positives endorsed are used to compute the Discriminability Index. The number of non-list words given during the learning trials and free and cued recall (Intrusion Errors) and repetitions of words (Perseverations) were also computed.

Table 3
Mean z scores by group

Variable	TD	SLI	WMS ^a	DS ^a	FL	Group comparisons ^b
List A Trial 1	.25	-.39	-.19	-1.60	.28	TD, SLI, WMS, and FL > DS
List A Trial 5	.46	-.83	-1.25	-1.36	-.83	TD > SLI, WMS, DS, and FL
List B Recall	.30	-.69	-.15	-1.42	-.49	TD > SLI, DS; WMS > DS
SD Free Recall	.37	-.78	-.97	-1.46	-.55	TD > SLI, WMS, and DS
LD Free Recall	.43	-1.08	-1.06	-1.55	-.82	TD > SLI, WMS, DS, and FL
Total intrusions ^c	.39	-1.93	-1.69	-4.38	-.11	TD > SLI, WMS, and DS; FL > DS, SLI
Discriminability	.33	-1.47	-2.31	-2.51	-.61	TD > SLI, WMS, and DS

^a z scores for the WMS and DS groups were computed relative to mental age rather than chronological age.

^b The comparisons listed were significant at $p < .05$ using Bonferroni correction.

^c The means for Intrusions have been reversed in sign for clarity in presentation. Thus, higher values represent better performance.

Table 2
Variables included in the analyses

Factor	CVLT-C variables used in factor
1. Attention/Immediate Memory Span	List A Trial 1; List B
2. Learning efficiency	List A Trial 5
3. Delayed Free Recall	Short Delay Free Recall, Long Delay Free Recall
4. Intrusions	Total Intrusion Errors
5. Recognition memory	Discriminability Ratio

Responses for each participant were scored using the CVLT-C normative database (Fridlund & Delis, 1994). Raw scores were converted into z scores using age- and gender-stratified norms from the 920 children ages 5–16 included in the normative sample. Mental age (rather than chronological age) was used for z score computations for the WMS and DS participants to provide a measure of the degree to which verbal learning is consistent with intellectual level in these groups. In order to limit the number of analyses performed, we selected for analysis several key CVLT-C variables that have been shown to be reliable and clinically important measures of learning and memory (cf. Donders, 1999). These variables, with the areas of functioning they are theorized to represent, are given in Table 2. Follow-up analyses using related CVLT-C variables are described in Section 3.

3. Results

The data were analyzed using either ANOVA or MANOVA with group as the independent variable. Post hoc comparisons were performed using Bonferroni α correction. The number of intrusion errors was non-normally distributed and, as a result, a logarithmic transformation was performed on this variable for inclusion in parametric analyses. Mean z scores and SD for each group are shown in Table 3, with the means for Intrusions reversed in sign for clarity in presentation. Thus, higher values represent better performance for all variables.

3.1. Attention/Immediate Memory Span

Group differences on Attention/Immediate Memory Span were significant (Pillai's $F(8, 206) = 6.15$, $p < .001$). Univariate F tests revealed significant effects of group on List A Trial 1 performance ($F(4, 103) = 8.05$, $p < .001$) and on List B performance ($F(4, 103) = 8.94$, $p < .001$). Post hoc follow-up comparisons using Bonferroni correction indicated that participants with DS had statistically significantly lower z scores on List A Trial 1 than SLI (mean difference = -1.21 , $p = .009$), WMS (mean difference = -1.42 , $p = .002$), FL (mean difference = -1.88 , $p < .001$) or TD participants (mean difference = -1.85 , $p < .001$), but there were no significant differences in performance between participants with SLI, WMS, FL, or TD (all comparisons ns at $p = .05$). Follow-up comparisons also revealed that participants with DS had statistically significantly lower z scores on List B than WMS (mean difference = -1.27 , $p = .002$) or TD participants (mean difference = -1.72 , $p < .001$), while SLI participants performed significantly worse than TD participants (mean difference = $-.99$, $p = .002$). All other comparisons were not significant.

3.2. Learning efficiency

A significant group effect was seen for performance on Trial 5 ($F(4, 107) = 7.08$, $p < .001$). Post hoc comparisons revealed that FL, SLI, WMS, and DS groups all differed significantly from the TD group (mean difference from TD group: SLI = -1.30 , $p = .005$; FL = -1.30 , $p = .043$; WMS = -1.71 , $p < .001$; DS = -1.82 , $p = .001$) but did not differ from one another. A follow-up analysis was performed to examine group differences on Slope, a measure of new learning per trial (computed as the slope of the least-squares regression line fitted across the five immediate recall trials of List A). The groups differed significantly on Slope, ($F(4, 107) = 4.79$, $p = .001$). Post hoc comparisons revealed that FL and WMS groups differed significantly from the TD group (mean difference from TD group: FL = 1.16 , $p = .01$; WMS = 1.00 , $p = .009$).

3.3. Delayed free recall

MANOVA showed a significant effect of group on free recall (Pillai's $F(8, 206) = 4.37$, $p < .001$). Univariate F tests revealed significant group effects on SD Free Recall performance ($F(4, 107) = 6.93$, $p < .001$) and LD Free Recall performance ($F(4, 107) = 9.51$, $p < .001$). Post hoc comparisons indicated that SLI, WMS and DS participants performed significantly worse than the TD participants on SD Free Recall trials (mean difference SLI = -1.16 , $p = .006$; WMS = -1.34 , $p = .002$; DS = 1.83 , $p < .001$), while FL participants performed comparably to typically developing controls. Although

participants with WMS, DS and SLI performed worse than the TD group, participants within these clinical groups performed similarly to each other (all comparisons ns). In addition, participants in all clinical groups had significantly lower z scores than TD participants on LD Free Recall trials (mean difference SLI = -1.52 , $p < .001$; WMS = -1.49 , $p = .001$; DS = -1.98 , $p < .001$; FL = -1.26 , $p = .017$), but they did not perform significantly differently from each other on this variable (all comparisons ns , $p > .05$).

3.4. Recognition memory

ANOVA with group by Discriminability performance revealed a significant difference between the groups ($F(4, 107) = 5.75$, $p < .001$). Post hoc Bonferroni corrected comparisons revealed that the SLI, WMS, and DS participants obtained lower recognition scores than the TD participants (mean difference TD – SLI = -1.80 , $p = .48$; TD – WMS = -2.65 , $p = .001$; TD – DS = -2.83 , $p < .003$), while the FL participants performed comparably to the TD participants. Participants with SLI, WMS, and DS did not differ significantly from each other. A follow-up ANOVA compared the groups on false positive errors (using a log transform) on the recognition trial. It showed a significant group difference ($F(4, 107) = 7.561$, $p < .001$). Again, post hoc comparisons showed significant differences between TD children and SLI, WMS, and DS participants, as well as between FL and WMS participants. Mean raw scores for number of false positive errors (out of a possible 30) are shown in Table 4.

3.5. Intrusions

Analysis of errors made during the learning trials revealed that extra-list intrusion errors differed as a function of clinical group ($F(4, 107) = 14.20$, $p < .001$). Post hoc comparisons showed that participants with SLI, WMS, and DS gave significantly more intrusion errors than TD participants (mean difference TD – SLI = $-.33$, $p < .001$; TD – WMS = $-.29$, $p < .001$; TD – DS = $-.49$, $p < .001$), while FL participants did not differ significantly from TD participants on this measure. Participants with SLI also

Table 4
Mean (SD) raw scores for Recognition False Positive (FP) Errors and Free and Cued Recall Intrusion Errors

Group	Total FP errors	Free Recall intrusions	Cued Recall intrusions
SLI	5.57 (6.54)	16.21 (13.77)	8.75 (8.21)
WMS	11.39 (10.04)	14.91 (20.11)	13.61 (14.59)
DS	10.50 (11.81)	28.21 (31.96)	20.86 (16.82)
FL	3.71 (4.32)	7.64 (8.59)	5.43 (7.87)
TD	2.27 (2.27)	2.90 (3.38)	0.83 (1.20)

made more intrusion errors than FL participants (mean difference FL – SLI = $-.22$, $p = .037$), as did participants with DS (mean difference FL – DS = $-.39$, $p < .001$). Follow-up analyses were performed to examine intrusion errors on free and cued recall trials separately. A MANOVA comparing the groups was significant (Pillai's $F(8, 206) = 6.801$, $p < .001$), and univariate analyses showed that the groups differed significantly on both free ($F(4, 107) = 10.356$, $p < .001$) and cued ($F(4, 107) = 12.426$, $p < .001$) intrusions. Post hoc Bonferroni corrected comparisons of both intrusion types demonstrated that participants in the SLI, WMS, and DS groups, compared to TD children, made significantly more free and cued intrusion errors. The FL group made significantly fewer errors of both types than the DS group, and significantly fewer free recall intrusions than the SLI group. Mean raw scores for free and cued recall intrusion errors are given in Table 4 to illustrate the rates of intrusion errors given by the five groups.

4. Discussion

The results of this study provide support for the hypothesis that differential patterns of language functioning predict different patterns of verbal learning and memory deficits in children. For the purposes of discussion, the findings for each clinical group compared to typically developing children are first summarized. Differences between the clinical groups and general patterns are then discussed.

4.1. Clinical groups

4.1.1. Specific language impairment

Children with SLI performed more poorly than typically developing children on all variables except for the first learning trial. This pattern is consistent with their pervasive problems with language and supports the prevailing view that these children have problems with verbal learning and memory. The finding of poor performance on learning and both short and long delay recall trials, and the large number of intrusion errors, differs somewhat from the results of Shear et al. (1992). Our results suggest that children with SLI have difficulty initially encoding the word list in a manner that allows efficient learning and differentiation of relevant words from extra-list information. The SLI group's comparable performance on both short and long delay recall indicates that they are able to retain verbal material once it has been learned, however. Furthermore, their failure to improve with recognition testing, compared to recall, argues against a retrieval deficit. In a previous study, we found that SLI and TD children were similar in their use of clustering strategies and in serial position effects, suggesting that their approaches to the task did

not differ and further supporting the contention that the SLI children's impairment is in initial encoding (Nichols, Roman, Wulfeck, & Delis, 1996). Our failure to find a significant difference on the first learning trial offers some support to previous suggestions that a problem with short-term auditory memory does not play a primary role in memory impairment for children with SLI. However, the SLI group did not differ from controls in learning slope, suggesting that their performance across the learning trials is lower overall than that of controls.

4.1.2. Early acquired focal lesions

Our results support the hypothesis that, by school age, children with early focal damage show problems in verbal learning and memory that are, like their language deficits, relatively mild. Although the performance of our FL group was somewhat lower than that of controls on all variables except for the first learning trial, these differences reached significance only for the fifth learning trial and long delay recall. In other words, the differences between them and children with (presumably) intact brains are subtle, only reaching a statistically meaningful level on variables that represent the "end product" of the learning process. For example, the FL children perform adequately on the first learning trial of the task; however, by the fifth trial, they are no longer quite able to keep up with their age mates, possibly because their learning is not as efficient. This is supported by the difference in learning slope between FL and TD children. Similarly, their difficulty with retrieval becomes statistically apparent only after a relatively long delay, when greater consolidation would be expected. Thus, although verbal learning and memory show considerable plasticity when damage is early, it is incomplete. Furthermore, preliminary comparisons of children with left and right hemisphere lesions from our sample have suggested that side of lesion is not related to the degree of verbal learning and memory impairment (Nichols, Jones, Delis, Wulfeck, & Trauner, 2000), providing further evidence that verbal learning parallels language functioning in this population. One noteworthy practical aspect of these findings is that, although these children are doing fairly well overall, the problems that they have on this task are those that might particularly impact their ability to perform in challenging and memory-intensive environments such as school.

4.1.3. Williams syndrome

Their relatively good language might cause one to predict that verbal memory would represent an area of strength for the WMS group. However, the study by Vicari et al. (1996) has raised the issue that language and auditory short-term memory might be dissociated from longer-term verbal memory in this population. Our findings support this hypothesis. Participants with WMS

differed significantly from controls on all variables except for the first learning trial and list B. Thus, despite relative strength in some areas of language functioning, their verbal learning and memory abilities are poor compared to controls even when their performance is scored according to mental rather than chronological age. Their isolated advantage on recall of List A Trial 1 and List B is consistent with other findings of anomalously good short-term auditory recall. However, this advantage does not result in preserved verbal learning and memory overall. Participants with WMS also discriminate relevant from irrelevant information poorly, as demonstrated by their elevated false positive error rates on recognition testing and very high number of intrusion errors. Preliminary findings also suggest that participants with WMS, as well as DS, use poor organizational learning strategies on the CVLT-C (Jones, Nichols, Delis, & Bellugi, 2000).

4.1.4. Down syndrome

In this study, the children with DS differed from controls on every CVLT-C variable, again despite the use of *z* scores based on mental rather than chronological age. This is consistent with the literature describing these children as having language deficits that are worse than their global delays and supports our prediction that this group would perform poorly on all aspects of the task. The children with DS were the only group in our study that differed significantly from controls even on the first learning trial of the task. They were particularly susceptible to interference from extra-list information, as shown by their extremely high number of intrusion errors and their false positive errors on the recognition trial.

5. Conclusions

The groups of children included in our study are all, with the exception of the controls, at risk for abnormalities in language development. Our findings support the hypothesis that these children also have problems in verbal learning and memory, but with distinct patterns of deficits associated with each disorder.

The results of this study highlight the necessity of looking beyond immediate auditory memory in assessing verbal learning. The only children who had significant difficulty on the first learning trial of the CVLT-C compared to controls were those with the most global developmental disorder, DS. By the fifth learning trial, however, the failure of the other groups to learn at the same rate as the controls had emerged. A paradigm with multiple learning trials challenged the ability of the clinical groups to keep up with their age (or mental age) peers and revealed significant problems in learning verbal material.

It is important to note that all four groups were able to maintain the information that they had learned over a delay; their impairment was in the initial learning of the list. The FL children appeared to do more poorly on long delay free recall than on short delay; however, they rebounded on the recognition measure, suggesting that the information was in fact learned and retained but may have been difficult for them to retrieve efficiently after a delay. This may imply that these children would especially benefit from testing that uses a recognition format in educational settings.

The participants with SLI, WMS, and DS had significant difficulty in discriminating relevant from irrelevant information compared to the typically developing group. This was true both when they were asked to select target words from a list containing distractors (false positive errors) and when they were asked to recall the lists (intrusion errors). All three groups made significantly more errors of both types than did controls. Intrusion errors occurred during free recall as well as cued recall and are therefore probably not disinhibited associations to the semantic prompt. A detailed characterization of the intrusion errors made by these groups is beyond the scope of this paper. However, a generalization can be made that these three disorders are characterized more by interference and poor discrimination than is the group with early acquired focal brain damage.

To what degree do our findings support the hypothesis that verbal learning and memory can be predicted from language functioning? Our data agree with the hypothesis in the most general sense; all four clinical groups demonstrate both some degree of language impairment and problems on at least some aspects of the CVLT-C compared to controls. With regard to specific clinical groups, the patterns of performance seen on the CVLT-C are consistent with overall language abilities for the SLI, FL, and DS groups as shown by their PPVT-R scores (Table 2). However, WMS, a disorder already known to have interesting dissociations between different cognitive domains, appears to be characterized by yet another dissociation, this time between their relatively strong language and their poor verbal learning and memory. It is possible that their preserved language cannot compensate for their difficulty with the other cognitive demands of the task, and that relationships between language abilities and verbal learning are weakened in the context of significant global impairment or deficits in specific non-language cognitive functions. It is interesting to note that the CVLT-C profiles are similar, in many respects, for participants with SLI and WMS even though language represents a relative weakness for one group and a strength for the other.

In summary, it appears that distinct patterns of verbal learning and memory are associated with early focal damage, SLI, WMS, and DS. The FL group, despite having the clearest evidence of structural brain damage,

showed the least impairment across most of the CVLT-C variables, providing further support for the idea that language-related functions have greater plasticity following focal structural damage than with presumably more diffuse dysfunction. The profiles of performance associated with each disorder may also have treatment and educational implications and suggest that clinical evaluations of these individuals should include a detailed assessment of verbal learning processes and verbal memory.

Acknowledgments

We thank the following colleagues for their assistance in scheduling participants and compiling data: Tracy Powell, Kathy Scarvie, Gayle Simon, and the testing staff at the San Diego Project in Cognitive and Neural Development/Language Research Center (University of California, San Diego). We also express our gratitude to the children and their parents for donating their time to the study. This research was supported in part by funding from NIH-NINDS Grant P50 NS22343.

References

- Aram, D. M., & Ekelman, B. L. (1988). Scholastic aptitude and achievement among children with unilateral brain lesions. *Neuropsychologia*, *26*, 903–916.
- Bates, E. (1997). Origins of language disorders: A comparative approach. *Developmental Neuropsychology*, *13*, 447–476.
- Bates, E., Reilly, J., Wulfeck, B., Dronkers, N., Opie, M., Fenson, J., Kriz, S., Jeffries, R., Miller, L., & Herbst, K. (2001). Differential effects of unilateral lesions on language production in children and adults. *Brain and Language*, *79*, 223–265.
- Beeson, P. M., Bayles, K. A., Rubens, A. B., & Kaszniak, A. W. (1993). Memory impairment and executive control in individuals with stroke-induced aphasia. *Brain and Language*, *45*, 253–275.
- Cappa, S. F., Papagno, C., & Vallar, G. (1990). Language and verbal memory after right hemispheric stroke: A clinical-CT scan study. *Neuropsychologia*, *28*, 503–509.
- Carlesimo, G. A., Marotta, L., & Vicari, S. (1997). Long-term memory in mental retardation: Evidence for a specific impairment in subjects with Down's syndrome. *Neuropsychologia*, *35*, 71–79.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (1994). *The California Verbal Learning Test—Children's Version*. San Antonio, TX: Psychological Corporation.
- Dobbins, I. G., Kroll, N. E. A., Tulving, E., Knight, R. T., & Gazzaniga, M. S. (1998). Unilateral medial temporal lobe memory impairment: Type deficit, function deficit, or both? *Neuropsychologia*, *36*, 115–127.
- Donders, J. (1999). Structural equation analysis of the California Verbal Learning Test—Children's Version in the standardization sample. *Developmental Neuropsychology*, *15*, 395–406.
- Dunn, L. M., & Dunn, L. M. (1981). *Peabody Picture Vocabulary Test—Revised*. Circle Pines, MN: American Guidance Service.
- Fridlund, A., & Delis, D. (1994). *IBM user's guide for the children's version of the California Verbal Learning Test*. San Antonio, TX: Psychological Corporation.
- Gathercole, S. E., & Baddeley, A. D. (1995). Short-term memory may yet be deficient in children with language impairments: A comment on van der Lely & Howard (1993) [Letter]. *Journal of Speech and Hearing Research*, *38*, 463–466.
- Hermann, B. P., Seidenberg, M., Haltiner, A., & Wyler, A. R. (1992). Adequacy of language function and verbal memory performance in unilateral temporal lobe epilepsy. *Cortex*, *28*, 423–433.
- Jarrold, C., Baddeley, A. D., & Hewes, A. K. (1999). Genetically dissociated components of working memory: Evidence from Down's and Williams syndrome. *Neuropsychologia*, *37*, 637–651.
- Jones, W., Nichols, S., Delis, D. C., & Bellugi, U. (2000). Verbal list learning in Williams Syndrome and Down Syndrome. *Journal of the International Neuropsychological Society*, *2*, 162.
- Kail, R., Hale, C. A., Leonard, L. B., & Nippold, M. A. (1984). Lexical storage and retrieval in language-impaired children. *Applied Psycholinguistics*, *5*, 37–49.
- Kirchner, D. M., & Klatzky, R. A. (1985). Verbal rehearsal and memory in language-disordered children. *Journal of Speech and Hearing Research*, *28*, 556–565.
- Leiter, R. G. (1948). *Leiter International Performance Scale*. Chicago: Stoelting.
- Levin, H. S., Fletcher, J. M., Kusnerik, L., Kufera, J. A., Lilly, M. A., Duffy, F. F., Chapman, S., Mendelsohn, D., & Bruce, D. (1996). Semantic memory following pediatric head injury: Relationship to age, severity of injury, and MRI. *Cortex*, *32*, 461–478.
- Mervis, C. B., Morris, C. A., Bertrand, J., & Robinson, B. F. (1999). Williams Syndrome: Findings from an integrated program of research. In H. Tager-Flusberg (Ed.), *Neurodevelopmental disorders* (pp. 65–110). Cambridge, MA: The MIT Press.
- Montgomery, J. W. (1995). Sentence comprehension in children with specific language impairment: The role of phonological working memory. *Journal of Speech and Hearing Research*, *38*, 187–199.
- Montgomery, J. W. (2000). Verbal working memory and sentence comprehension in children with specific language impairment. *Journal of Speech, Language, and Hearing Research*, *43*, 293–308.
- Nichols, S., Jones, W., Delis, D., Wulfeck, B., & Trauner, D. (2000). The effects of early, focal brain injury on verbal learning and memory. *Journal of the International Neuropsychological Society*, *2*, 131.
- Nichols, S., Roman, M., Wulfeck, B., & Delis, D. (1996). Verbal learning in children with specific language impairment. Presented at the annual meeting of the American Psychological Society, San Francisco, CA.
- Reilly, J. S., Bates, E., & Marchman, V. (1998). Narrative discourse in children with early focal brain injury. *Brain and Language*, *61*, 335–375.
- Roman, M. J., Delis, D. C., Willerman, L., Magulac, M., Demadura, T. L., de la Pena, J. L., Loftis, C., Walsh, J., & Kracun, M. (1998). Impact of pediatric traumatic brain injury on components of verbal memory. *Journal of Clinical and Experimental Neuropsychology*, *20*, 245–258.
- Semel, E., Wiig, E. H., & Secord, W. (1987). *Clinical Evaluation of Language Fundamentals—Revised*. San Antonio, TX: The Psychological Corporation.
- Shear, P. K., Tallal, P., & Delis, D. C. (1992). Verbal learning and memory in language impaired children. *Neuropsychologia*, *30*, 451–458.
- Sininger, Y. S., Klatzky, R. L., & Kirchner, D. M. (1989). Memory scanning speed in language-disordered children. *Journal of Speech and Hearing Research*, *32*, 289–297.
- Squire, L. R. (1987). *Memory and brain*. New York: Oxford University Press.
- Stuss, D. T., Alexander, M. P., Palumbo, C. L., Buckle, L., Sayer, L., & Pogue, J. (1994). Organizational strategies of patients with unilateral or bilateral frontal lobe injury in word list learning tasks. *Neuropsychologia*, *8*, 355–373.
- van der Lely, H. K. J., & Howard, D. (1993). Children with specific language impairment: Linguistic impairment or short-term mem-

- ory deficit. *Journal of Speech and Hearing Research*, 36, 1193–1207.
- van der Lely, H. K. J., & Howard, D. (1995). Specific language impairment in children is not due to a short-term memory deficit: Response to Gathercole & Baddeley [letter]. *Journal of Speech and Hearing Research*, 38, 466–472.
- Vargha-Khadem, F., Gadian, D. G., Watkins, K. E., Connelly, A., Van Paesschen, W., & Mishkin, M. (1997). Differential effects of early hippocampal pathology on episodic and semantic memory. *Science*, 277, 376–380.
- Vicari, S., Brizzolara, D., Carlesimo, G. A., Pezzini, G., & Volterra, V. (1996). Memory abilities in children with Williams syndrome. *Cortex*, 32, 503–514.
- Wang, P. P., & Bellugi, U. (1994). Evidence from two genetic syndromes for a dissociation between verbal and visual-spatial short-term memory. *Journal of Clinical and Experimental Neuropsychology*, 16, 317–322.
- Watkins, K. E., Hewes, D. K. M., Connelly, A., Kendall, B. E., Kingsley, D. P. E., Evans, J. E. P., Gadian, D. G., Vargha-Khadem, F., & Kirkham, F. J. (1998). Cognitive deficits associated with frontal-lobe infarction in children with sickle cell disease. *Developmental Medicine & Child Neurology*, 40, 536–543.
- Wechsler, D. (1974). *Manual for the Wechsler Intelligence Scale for Children—Revised*. San Antonio, TX: The Psychological Corporation.
- Wechsler, D. (1981). *Manual for the Wechsler Adult Intelligence Scale—Revised*. San Antonio, TX: The Psychological Corporation.
- Ween, J. E., Verfaellie, M., & Alexander, M. P. (1996). Verbal memory function in mild aphasia. *Neurology*, 47, 795–801.
- Weismer, S. E., Evans, J., & Hesketh, L. J. (1999). An examination of verbal working memory capacity in children with specific language impairment. *Journal of Speech, Language, and Hearing Research*, 42, 1249–1260.
- Yeates, K. O., Blumenstein, E., Patterson, C. M., & Delis, D. C. (1995a). Verbal learning and memory following pediatric closed-head injury. *Journal of the International Neuropsychological Society*, 1, 78–87.
- Yeates, K. O., Enrile, B. G., Loss, N., Blumenstein, E., & Delis, D. C. (1995b). Verbal learning and memory in children with myelomeningocele. *Journal of Pediatric Psychology*, 20, 801–815.