

The Graphemic Buffer and Attentional Mechanisms

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Two patients with acquired dysgraphia were reported. The patients' performance in various written and oral spelling tasks converge in support of the hypothesis that they have selective damage, within the spelling system, to the Graphemic Buffer. Although the patients present with comparable patterns of error types, they differ in the distribution of errors as a function of letter position in words. The patients' patterns of errors are compared to previously reported patterns of spelling errors in dysgraphic patients and are discussed in terms of hypothesized mechanisms that operate on the representations that are stored in the Graphemic Buffer. © 1989 Academic Press, Inc.

Recent studies of dysgraphic patients have provided important information concerning the cognitive and linguistic mechanisms that underlie spelling (e.g., Beauvois & Derouesne, 1981; Caramazza, Miceli, & Villa, 1986; Goodman & Caramazza, 1986; Newcombe & Marshall, 1980; Shallice, 1981). This research has helped reveal the principal components of the spelling process and their structure and has led to the formulation of a widely accepted, if not uncontested, functional architecture of that process.

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On this account of the spelling process a major distinction is drawn between those processes that compute the graphemic structure of familiar words—lexical processes—and those processes that compute the graphemic structure of unfamiliar words (or nonwords)—nonlexical processes (e.g., Caramazza et al., 1986). A competing account of the spelling process does not distinguish between lexical and nonlexical processes in the spelling system (e.g., Campbell, 1983). Although alternative models of spelling vary with respect to the hypothesized procedures for computing graphemic representations of familiar and unfamiliar words, all models may need to postulate a processing stage—the Graphemic Buffer—in which the graphemic representation of a word is held during the execution of appropriate output processes (e.g., Caramazza, Miceli, Villa, & Romani, 1987; Ellis, 1982; Miceli, Silveri, & Caramazza, 1985; Newcombe & Marshall, 1980). Evidence for such a working memory system is provided by the dysgraphic performance of two patients, F.V. (Miceli et al., 1985) and L.B. (Caramazza et al., 1987), who presented with a pattern of performance compatible with hypothesized damage to the Graphemic Buffer.

Caramazza et al. (1987) have proposed a set of criteria for identifying selective damage to the Graphemic Buffer. The proposed criteria are the following: (1) A similar pattern of spelling errors should obtain for novel (nonwords) and for familiar words, across all output modalities (oral and written spelling and typing) and all input modalities (spontaneous writing, written naming, writing to dictation, and delayed copying), since the Graphemic Buffer is equally implicated in each of these spelling tasks; (2) spelling accuracy should not be affected by lexical factors such as word frequency, grammatical word class or concreteness since, by hypothesis, lexical components of the system are intact; (3) nor should spelling accuracy be affected by orthophonological factors such as phonology-to-orthography probability mappings since phoneme-to-grapheme mapping processes are hypothesized to be intact; (4) semantically, morphologically, or phonologically similar word errors should not occur for the same reason as in (2); and (5) spelling errors should occur with increased frequency as a function of word length, since each grapheme to be stored introduces a potential error.

Additional criteria proposed by Caramazza et al. (1987) for identifying selective damage to the Graphemic Buffer include features of spelling errors. Because errors resulting from damage to the Graphemic Buffer must arise in the storage of an accurate series of graphemes, the errors should reflect the degradation of the spatially encoded, graphemic representation of the target word.¹ The expected types of spelling errors

¹ We assume that graphemes are abstract units, and as such, are "spatially encoded" in the sense that the serial position of each graphemic unit is specified in an internal

are deletions, substitutions, insertions, and transposition of letters, which lead to the production of phonemically implausible nonwords (i.e., responses that do not respect phoneme-grapheme correspondence; e.g., *blame* → *blome*, *lbame*, and so on).

The rationale for each of the above predictions has been discussed in detail elsewhere (Caramazza et al., 1987) and will not be repeated in this paper. Rather, we will present case studies of two dysgraphic patients who present with the pattern of errors described above—a pattern that is explicable by assuming selective impairment (within the spelling system) to the Graphemic Buffer. Our primary objective in this paper was to record the replication of selective damage to the Graphemic Buffer in two English-speaking patients (previous studies involved Italian-speaking patients). We also compare these cases to each other and to previous cases, focusing in particular on the distribution of errors across relative letter positions in words, in order to constrain claims about the processing structure of the Graphemic Buffer. A contrast in the distribution of errors, skewed in opposite directions for the two patients with opposite sides of brain damage, is discussed in terms of the possibility that attentional deficits contribute to the overall clinical picture presented by the two cases.

CASE 1: D.H.

Case History

D.H., a 49-year-old, right-handed male with a high school education, is employed as a Quality Control Supervisor at a chemical plant. He suffered a thromboembolic stroke in May 1986. His primary complaints at the time of hospital admission were acute memory loss and difficulty reading, writing, formulating sentences, and word-finding. A neurological examination identified slight weakness of the right wrist flexors and extensors and impairments of recent memory, proverb interpretation, calculation, and writing. There was no evidence of cranial neuropathy, sensory or proprioceptive deficits, or cerebellar or extrapyramidal signs. Visual fields were normal. A CT scan 2 months post onset revealed a left frontoparietal infarct.

Clinical, speech-language pathology evaluation indicated aphasia characterized by dyslexia, dysgraphia, and mild/moderate anomia. By 3 months post onset, when this study was initiated, D.H.'s earlier paraphasias in spontaneous production had largely resolved. His speech was grammatical,

representation with spatial coordinates. This assumption entails a distinction between this level of abstract representation and a representation with letters or visual features as primitives and retinal coordinates (see Seymour, 1979, for review of numerous studies on normal subjects that have validated this proposal).

and predominantly fluent, with occasional hesitations for word retrieval. His score of 55/60 on the Revised Boston Naming Test (Goodglass, Kaplan, & Weintraub, 1983) was within the normal range for his age and education. He received full scores on sentence repetition and auditory-verbal comprehension subtests of the Boston Diagnostic Aphasia Examination (BDAE) (Goodglass & Kaplan, 1972) and a score of 35/36 on the Modified Token Test (DeRenzi & Faglioni, 1978). D.H. was able to recall six digits forward and four digits backward. His reading comprehension was only very mildly impaired; performance on the BDAE and The Reading Comprehension Battery for Aphasia (RCBA) (LaPointe & Horner, 1979) was 100% accurate for words and 90% accurate for sentences and paragraphs. However, his oral reading of single words exhibited visual errors primarily at the ends of words, including morphological errors. On The Johns Hopkins University Dyslexia Battery, D.H. produced 14 errors in reading aloud words. The frequency of visual errors was not affected by word class, concreteness, length, or orthographic regularity. He correctly read 95.5% of high-frequency words and 91.1% of low-frequency words. D.H. self-corrected all errors in oral reading of narratives, except when the errors were semantically compatible with the context.

D.H.'s total score on the Weschler Memory Scale (Weschler, 1972) was within normal limits (56.6; mean for age = 58.8). However, his performance on the visual reproduction subtest of this measure was significantly impaired (score 3/15; mean = 8.35; $SD = 3.17$). Although he made errors in drawing from memory (immediate recall) on both sides of abstract figures, he made more right-sided errors (e.g., he failed to draw the right sides of two small squares in the second figure), as shown in Appendix A. Similarly, his copy of the Rey Figure (Rey, 1941) revealed obvious errors, such as his omission of two vertical lines, only on the right side. In contrast, his direct copying of figured objects (e.g., flower, clock, etc.) was normal. However, D.H. performed below normal on several other standardized visual-spatial tests. He received a score of 24/30 on the Hooper Visual Organization Test (Hooper, 1957), corresponding to the "mild" range of deficit. His age-corrected score of 25/36 on Raven's Colored Progressive Matrices (RCPM) (Raven, 1956) is one standard deviation below the mean of 29. He showed a significant position preference on this test: 14 responses on the left, 7 on the right; five errors on the left, none on the right. This response pattern has been positively associated with the presence of visual-spatial neglect (Costa, Vaugh, Horwitz, & Ritter, 1969; Gainotti, 1968). Although a reduced score on this test has also been interpreted as impaired intellectual functioning or defective visual analog reasoning, D.H. demonstrated adequate analog reasoning in verbal tests: He scored in the normal range on the Woodcock Language Proficiency Battery (Woodcock, 1984) verbal anal-

ogies section. D.H. did not err in line cancellation or line bisection tests of neglect.

Experimental Study

The Johns Hopkins University Dysgraphia Battery was administered to D.H. He wrote 326 words and 44 nonwords to dictation, spelled aloud 120 words and 20 nonwords, and wrote the names of 51 pictured objects. Word lists were controlled for a variety of lexical parameters (word length in letters and syllables, frequency, concreteness, grammatical word class, and orthographic regularity). The frequency and pattern of his errors were essentially the same for words and nonwords, for written and oral spelling, and for all input modalities. Thus, for example, in oral and written spelling of words and nonwords to dictation, single letter errors predominated. Of these, the most common were letter substitutions (39.3% in writing; 39.1% in oral spelling) and deletions (42.4% in writing and 41.0% in oral spelling). Mixed errors (e.g., substitution + transposition) accounted for 16.3% (24/147) of the written spelling errors and 16.6% (6/36) of oral spelling errors. In both modalities, less than 10% of the responses contained multiple errors of the same type (e.g., two insertions). This pattern of predominantly single substitution and deletion errors, 15–20% mixed errors, and few multiple errors also obtained for written naming and delayed transcoding (Table 1). The homogeneity of his error pattern for all types of stimuli, across all input and output modalities, suggests that D.H.'s deficit must be in a process common to all spelling tasks. Additional evidence for this hypothesis was obtained by ruling out damage to other components of the spelling process.

D.H. spelled correctly 50.0% (60/120) of the words and 48.1% (26/54) of the nonwords in a list controlled for word length in letters. Also, he spelled correctly 70% (21/30) of orthographically regular words and 67.5% (54/80) of irregular words matched for frequency and word length in letters and phonemes. These results contraindicate selective disruption of phoneme-to-grapheme conversion processes. The similarity between D.H.'s written and oral spelling performance—which involve different motor output systems—rules out the possibility of selective disruption of motor processes as the basis for the observed pattern of spelling difficulties; writing to dictation was 51.7% (61/118) accurate and oral spelling to dictation was 53.6% (30/56) accurate. Performance on copy transcoding and naming tasks ruled out the possibility of a phonological input deficit as the basis for his errors. Writing to dictation and delayed copy transcoding were performed with the same level of accuracy—both 62.9% correct. Accuracy in written naming was somewhat higher (74.5%), but this finding can be explained by the shorter word length in this task (mean word length of 4.8 letters in written naming, compared to mean word length of 5.34 and 5.62 for dictation and transcoding, respectively).

TABLE 1
DISTRIBUTION OF ERRORS IN FOUR SPELLING TASKS: D.H.

	Writing to dictation		Oral spelling to dictation		Written naming	Delayed transcoding
	Words	Nonwords	Words	Nonwords		
Number of stimuli:	326	44	120	20	51	124
Spelling errors						
Single letter	64 (52.9)	12 (52.2)	34 (68.0)	6 (60.0)	9 (69.2)	31 (67.4)
Substitutions	25 (39.1)	5 (41.7)	15 (44.1)	3 (50.0)	4 (44.4)	10 (32.2)
Insertions	3 (4.7)	1 (8.3)	2 (5.8)	0 (0)	0 (0)	0 (0)
Deletions	27 (42.2)	5 (41.7)	13 (38.2)	3 (50.0)	5 (55.5)	15 (48.4)
Transpositions	9 (14.1)	1 (8.3)	4 (11.8)	0 (0)	0 (0)	6 (19.4)
Multiple errors (of 1 type)	6 (5.0)	2 (8.7)	4 (8.0)	1 (10.0)	1 (7.7)	0 (0)
Mixed errors	21 (17.4)	4 (17.4)	8 (16.0)	2 (20.0)	2 (15.4)	9 (19.6)
Unclassifiable						
(partial responses & visually similar words)	30 (24.8)	7 (30.4)	4 (8.0)	1 (10.0)	1 (7.7)	6 (13.0)
Total errors	121(100.0)	23(100.0)	50(100.0)	10(100.0)	13(100.0)	46(100.0)
% of total responses	37.1	52.3	41.7	50.0	25.5	37.1
× word length (No. letters)	5.34		5.68		4.80	5.62

Furthermore, D.H. produced similar types of spelling errors (including substitution, deletion, insertion, and transposition of single or multiple letters) in oral and written spelling to dictation, in delayed copy transcoding of words and nonwords from upper- to lower case or vice versa, and in written naming, as noted above (see Appendix B for examples).

Damage to lexical processes was ruled out by comparing lexical parameters of stimuli and by analyzing error responses. If misspellings were to arise at the level of the graphemic output lexicon, we would anticipate the production of phonemically plausible errors (with intact phoneme-grapheme correspondence; e.g., *phone* → *foan*) through non-lexical spelling processes, or production of related word errors through activation of a more available unit in the output lexicon when the target unit cannot be accessed. In contrast, our hypothesis that misspellings result from degradation of an accurate lexical entry in the Graphemic Buffer predicts the types of grapheme errors made by D.H. (described above), which need not maintain phonological or orthographic plausibility. Also consistent with the latter prediction, D.H. produced no semantic errors and only two morphological errors. The majority (59.7%) of his spelling errors were phonemically implausible nonwords (e.g., *blome*), of which a nonnegligible proportion were orthographically illegal (e.g., *length* → *lentgh*; *brisk* → *brsst*; *scrubbed* → *sbuced*). The few visually similar word responses (VSWs; e.g., *speak* → *speck*; *starve* → *stave*) and phonemically plausible errors (PPEs; e.g., *thread* → *thred*; *rinse* → *rince*) also consisted of deletions, substitutions, insertions, and transpositions of one or two letters, and therefore could also be attributed to degradation of an accurate lexical-graphemic representation.

Further evidence counter to a lexical basis as the source of the spelling deficit in D.H. was obtained by ruling out significant effects of grammatical word class, concreteness, and orthographic regularity. There were no word class effects on spelling accuracy. D.H.'s written spelling was correct for 50.0% (14/28) of the nouns, and 57.1% (16/28) of both the verbs and the adjectives, on a list counterbalanced for grammatical word class, frequency, letter length, and syllabicity. He also spelled correctly 41.7% (5/12) of the words in each open class in oral spelling of a similarly controlled list. Nor was concreteness a significant factor in spelling accuracy; D.H. spelled correctly 57.1% (12/21) of concrete words and 52.4% (11/21) of abstract words matched for frequency and length in letters. Also, he spelled correctly 70% (21/30) of the orthographically regular and 67.5% (54/80) of the orthographically irregular words on a list controlled for frequency and word length in letters and phonemes.

The only significant lexical factor was an effect of word frequency in written spelling. D.H. spelled correctly 74.7% (109/146) of the high frequency words and 49.3% (72/146) of the low frequency words in writing to dictation. This discrepancy appeared to be due to his tendency to

self-correct high frequency words in writing, when his initial attempt clearly did not "look right." He frequently self-corrected written, but not oral, spelling; and the frequency effect was not obtained in oral spelling.

Word length was the most striking determinant of spelling accuracy. D.H. exhibited steady decrements in spelling accuracy from 100% (14/14) for four-letter words, to 14.3% (2/14) for eight-letter words (Table 2). The proportion of error responses consistently increased as a function of word length, in oral and written spelling and delayed copy transcoding of words and nonwords (Table 3). Grapheme deletions and mixed errors also increased as a function of word length (Table 4). Furthermore, the mean length of correctly spelled words (5.42 letters) was significantly shorter than the mean length of misspelled words (6.38 letters; $t = 6.9$; $p < .0001$). This robust effect of stimulus length is consistent with the hypothesized impairment of a working memory system. Normal performance on immediate and delayed repetition of words and sentences confined the deficit to the Graphemic Buffer in the spelling system.

The possibility that the damage responsible for D.H.'s performance could be located at the level of the allographic conversion process is ruled out by the fact that all of D.H.'s errors were graphemic, rather than allographic. That is, he omitted, substituted, or inserted well-formed letters in words. There was no detectable pattern of visual similarity in letter substitution errors; many substituted letters were not visually similar to the target letters (e.g., $m \rightarrow e$; $r \rightarrow g$; $e \rightarrow t$). Furthermore, although the patient made many spelling errors in the delayed copy task, no case errors were produced (see examples in Appendix B), consistent with the hypothesis that the locus of damage is at the level of the Graphemic Buffer.

TABLE 2
SPELLING PERFORMANCE AS A FUNCTION OF WORD
LENGTH: D.H.

Word length	No. errors	% Errors
4 letter	0/14	0
5 letter	2/14	14.3
6 letter	4/14	28.6
7 letter	8/14	57.1
8 letter	12/14	85.7

Note. Bisyllabic; $\frac{1}{2}$ high frequency and $\frac{1}{2}$ low frequency; counterbalanced for frequency, number of phonemes per word, and word length.

TABLE 3
 SPELLING ERRORS AS A FUNCTION OF LENGTH IN THREE WRITING TASKS: D.H.^a

Number of letters	Written naming	Writing to dictation	Delayed copy transcoding
3	0/2 (0%)	—	—
4	1/15 (6.7%)	16/75 (21.3%)	1/4 (25.0%)
5	9/21 (42.9%)	30/110 (27.3%)	11/30 (36.7%)
6	6/11 (54.5%)	33/84 (39.3%)	15/42 (35.7%)
7-8	—	27/40 (67.5%)	3/6 (50.0%)

^a Misspelled words/total words (% errors).

CASE 2: M.L.

Case History

M.L. is a 55-year-old, left-handed woman with a 12th-grade education. She previously held a clerical position with the Census Bureau. She suffered a small right hemisphere stroke in June 1986. Neurological evaluation indicated mild residual paresis of both left extremities and mild dysarthria, but without aphasia. A CT scan at that time was normal. One month later she suffered a new thromboembolic stroke, resulting in dense left hemiplegia, left neglect, and aphasia. A CT scan in August revealed a large infarct in the right parietal and frontal regions and right basal ganglia. Neurologic examination indicated left facial palsy, a slight decrease in the left visual field, and poor movement and reduced tone in the left extremities, with intact tactile sensation and proprioception.

Speech-language pathology evaluation in October 1986 indicated aphasic symptoms of mild auditory comprehension deficits and "agrammatic" speech. Unilateral upper motor neuron involvement of cranial nerves VII, IX, and X was manifested in harsh phonation and slightly reduced range of volitional (but not reflexive) movements of the face and velum on the left side. Diadochokinesis and single word repetition showed normal articulation. She omitted function words in spontaneous speech, sentence repetition, and oral reading of sentences, although she read isolated functors without error. On The Johns Hopkins University Dyslexia Battery, M.L. made six errors in reading aloud 326 words. All incorrect responses were visual errors at the beginning of words (e.g., *germ* → *term*; *glove* → *love*), consistent with "neglect dyslexia" (Kinsbourne & Warrington, 1962). In reading aloud narratives, she self-corrected all such errors that were semantically incompatible with the context. However, she frequently omitted reading words on the left side of the page, resulting in mildly impaired reading comprehension. Reading performance on the BDAE was 100% accurate for words and sentences and 80% accurate for paragraphs. Due to left hemiplegia, M.L. wrote with her right, non-dominant hand. Her written output was characterized by spatially and

TABLE 4
DISTRIBUTION OF ERRORS AS A FUNCTION OF WORD LENGTH: D.H.^a

Letter length:	4	5	6	7-8	Total
Single errors	8 (50.0)	16 (53.3)	19 (57.6)	13 (48.1)	56 (52.8)
Substitutions	6 (75.0)	8 (50.0)	6 (31.6)	3 (23.1)	21 (38.5)
Insertions	0	1 (6.3)	1 (5.3)	0	2 (3.6)
Deletions	0	4 (25.0)	10 (52.6)	9 (69.2)	23 (41.1)
Transpositions	2 (25.0)	3 (18.8)	2 (10.5)	1 (7.7)	8 (14.3)
Multiple errors	1 (6.3)	2 (6.7)	1 (3.0)	1 (3.7)	5 (4.7)
Mixed errors	0 (0)	4 (13.3)	6 (18.1)	8 (29.6)	18 (17.0)
Unclassifiable	7 (43.8)	8 (26.7)	7 (21.2)	5 (18.5)	27 (25.5)
Total	16	30	33	27	106

^a Percentages are in parentheses.

syntactically disorganized sentences, as well as frequent misspellings. In spontaneous writing, she neglected the left side of the page (i.e., began writing in the middle of the page).

M.L.'s "agrammatic" speech, dyslexia and dysgraphia persisted for several months, although her auditory comprehension and memory had improved substantially. By the time the current study was initiated, M.L. responded with 100% accuracy on auditory comprehension subtests of the BDAE and received a score of 35.5/36 on the Modified Token Test. She received a score of 59/60 on the Revised Boston Naming Test. M.L.'s total score on the Weschler Memory Scale was within normal limits. All subtest scores fell in the normal range, with the exception of visual reproduction. Her reproduction of abstract designs exhibited persisting left neglect (see Appendix C). Neglect was also found to contribute to her performance in drawing objects from memory; for instance, she included only numbers 1 through 6 (on the right) in drawing a clock. Gross left-side errors were evidenced in copying the Rey Figure (Appendix C), although direct copying of object pictures and line cancellation tasks were performed normally. M.L.'s bisection of a 10-in. line was correct on two of three trials, and displaced $\frac{1}{2}$ in. to the right on the third trial. She presented with extinction of left-sided tactile and visual stimuli with bilateral stimulation. M.L. also performed poorly in other tasks involving spatial processing: Her performance on the Hooper Visual Organization Test (score = 21.5/30; normal = 25–30) and on the RCPM (score = 15/36, with a right position preference in responding—6:11) was impaired, presumably as a result of an attentional deficit.

Experimental Study

The Johns Hopkins University Dysgraphia Battery was also administered to M.L. Like D.H., she produced a pattern of errors that was nearly identical for words and nonwords and for written and oral spelling (Table 5). She correctly wrote 35.6% (36/104) of the words and 35.3% (12/34) of the nonwords matched for length in letters and phonemes. Written spelling of open class words was 34.6% (29/84) accurate, and oral spelling was 35.7% (15/42) accurate. Performance was very similar across all spelling tasks (dictation, delayed copying, and naming) and was uniformly characterized by a substantial length effect (Table 6) as well as frequent substitution, deletion, insertion, and transposition of single or multiple letters (see examples in Appendix D). The mean length of correctly spelled words was 4.62 letters, and the mean length of misspelled words was 5.52 letters ($t = 8.12$; $p < .0001$). M.L.'s performance on a list of words matched for frequency and number of phonemes per word revealed a strong effect of word length: She correctly spelled 50.0% of four- to five-letter words, but no seven- or eight-letter word was spelled correctly (Table 7). The distribution of error types was similar to that of D.H.

TABLE 5
DISTRIBUTION OF ERRORS IN FOUR SPELLING TASKS: M.L.^a

	Writing to dictation		Oral spelling		Written naming	Delayed copying
	Words	Nonwords	Words	Nonwords		
Number of stimuli	326	34	42	20	51	124
Spelling errors						
Single letter	135 (55.1)	14 (66.7)	17 (63.0)	7 (63.6)	23 (76.7)	60 (63.2)
Substitutions	29 (21.5)	6 (42.3)	6 (35.3)	5 (71.4)	5 (21.7)	14 (23.3)
Insertions	20 (14.8)	2 (14.3)	3 (17.6)	1 (14.3)	5 (21.7)	9 (15.0)
Deletions	51 (37.8)	3 (21.4)	7 (41.2)	0 (0)	6 (26.1)	26 (43.3)
Transpositions	35 (25.9)	3 (21.4)	1 (5.9)	1 (14.3)	7 (30.4)	11 (18.3)
Multiple errors (of 1 type)	20 (8.2)	0 (0)	3 (11.1)	1 (9.1)	1 (3.3)	6 (6.3)
Mixed errors	72 (29.4)	6 (28.6)	6 (22.2)	2 (18.2)	6 (20.0)	25 (26.3)
Unclassifiable (partial responses & visually similar words)	18 (7.3)	1 (4.8)	1 (3.7)	1 (9.1)	0 (0)	4 (4.2)
Total errors	245(100.0)	21(100.0)	27(100.0)	11(100.0)	30(100.0)	95(100.0)
% of total responses	75.2	61.8	64.3	55.0	58.8	76.6
× word length (No. letters)	5.34	5.68	5.68	5.68	4.80	5.62

^a Percentages are in parentheses.

TABLE 6
SPELLING ERRORS AS A FUNCTION OF LENGTH IN FIVE TASKS: M.L.

Letter length	Written naming	Writing to dictation		Delayed copy	Oral spelling
		Words	Nonwords		
3-4	4/17 (23.5)	34/75 (45.3)	7/12 (58.3)	8/15 (53.3)	1/7 (14.3)
5	12/22 (54.5)	95/123 (77.2)	4/7 (57.1)	26/41 (63.4)	7/19 (36.8)
6	11/12 (91.7)	69/86 (80.2)	6/9 (66.7)	50/59 (84.7)	18/28 (64.3)
7-8	—	40/42 (95.2)	4/6 (66.7)	11/11 (100)	8/8 (100)

and afore-cited cases. Single letter errors decreased, and mixed errors (e.g., substitution + deletion) increased as a function of word length (Table 8). Furthermore, letter transpositions and insertions decreased in longer words, while deletions increased.

Unlike D.H., M.L. did not exhibit a significant word frequency effect. She spelled correctly 28.1% (41/146) of the high frequency words and 26.0% (38/146) of the low frequency words on the battery. Her accuracy rates on lists controlled for word length and frequency were also roughly identical across grammatical word classes (21.4 to 28.6%, or 6-8/28 for each form class), concrete and abstract words (both 14.3%; 3/21), and regular and irregular words (36.7% and 30.0%, respectively). Nearly all (92.5%; 369/399) of her errors were phonemically implausible nonwords, and all other errors involved similar grapheme errors (e.g., PPE: *since* → *sinse*, and VSW: *palace* → *place*). A substantial proportion (41.7%; 153/369) of her errors were unpronounceable violations of English orthography (e.g., *priest* → *rpiest*). Many errors exhibited preservation of irregular spellings and silent letters (e.g., *dumb* → *dub*; *fight* → *fght*; *lamb* → *llamb*), indicating accurate lexical retrieval, with subsequent degradation of the retrieved lexical-graphemic representation. There were no instances of semantic errors and only one morphological error among her responses. There was also no evidence of visual similarity in letter substitutions or case errors in transcoding tasks (Appendix D), consistent

TABLE 7
SPELLING PERFORMANCE AS A FUNCTION OF WORD LENGTH: M.L.

Word length	No. errors	% Errors
4 letter	6	42.9
5 letter	8	57.1
6 letter	13	92.9
7 letter	14	100
8 letter	14	100

TABLE 8
DISTRIBUTION OF ERRORS AS A FUNCTION OF WORD LENGTH: M.L.^a

Letter length:	4	5	6	7-8	Total
Single errors	38 (77.6)	85 (68.0)	66 (54.5)	24 (39.3)	213 (58.4)
Substitutions	9 (23.7)	22 (25.9)	15 (22.7)	7 (29.2)	53 (24.9)
Insertions	4 (10.5)	19 (22.4)	8 (12.1)	1 (4.2)	32 (15.0)
Deletions	13 (34.2)	25 (29.4)	30 (45.5)	11 (45.8)	79 (37.1)
Transpositions	12 (31.6)	19 (22.4)	13 (19.7)	4 (16.7)	48 (22.5)
Multiple errors	0 (0)	4 (3.2)	15 (12.4)	7 (11.4)	26 (7.1)
Mixed errors	6 (12.2)	27 (21.6)	40 (32.0)	30 (49.2)	103 (28.2)
Unclassifiable	5 (10.2)	9 (7.2)	9 (7.2)	0 (0)	23 (6.3)
Total	49	125	121	61	365

^a All spelling tasks; percentages are in parentheses.

with spared allographic conversion mechanisms. Letter formation was unimpaired, with allowance for writing with her nondominant (right) hand.

Comparison of the Spelling Performance of D.H. and M.L.

The pattern of spelling performance reported for D.H. and M.L. is compatible with the hypothesis that these patients' dysgraphia results from damage to the Graphemic Buffer. For each patient the hypothesized locus of damage was narrowed down to a single mechanism within the spelling system, first by demonstrating a homogeneous pattern of errors in all spelling tasks and for all classes of stimuli, and second, by ruling out other possible input or output deficits.

Although the spelling performance of patients M.L. and D.H. may be characterized in each case as reflecting a deficit to the Graphemic Buffer, the two patients differ between themselves and from other reported cases of damage to the Graphemic Buffer in their distribution of errors as a function of letter position in words. Figure 1 displays the contrasting distribution of M.L.'s and D.H.'s errors across relative letter positions in spelling words and nonwords, using the formula in Fig. 1 (from Wing & Baddeley, 1980) to equate letter positions of words of varying length. M.L.'s errors occurred primarily toward the beginnings of words in all spelling tasks—the first two relative positions accounted for 65% (115/177) of her single-letter spelling errors. In marked contrast, D.H.'s errors increased toward the ends of words—the last two relative positions accounted for 60% (85/142) of his single-letter errors.

The distributions of single spelling errors as a function of letter position in words for M.L. and D.H. not only differed from each other, but also from those previously reported for L.B. For this latter patient, errors occurred primarily in the middle positions of words, reflecting the bow-shaped pattern of errors in normal writers' "slips of the pen." This

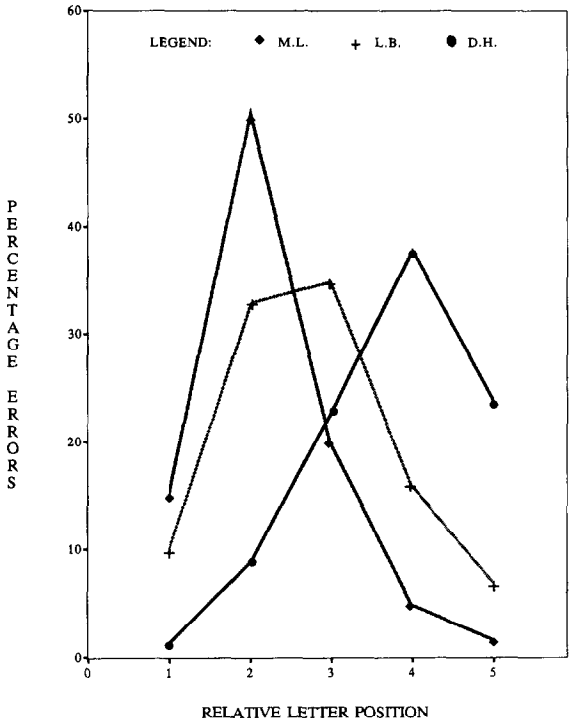


FIG. 1. Distribution of M.L.'s, L.B.'s, and D.H.'s errors as a function of letter "position" in strings. The formula used for normalizing letter positions of words of unequal lengths is taken from Wing and Baddeley (1980, p. 260):

Number of Letters Assigned to Each Position

k = integer values 0, 1, 2, 3 . . .

Total No. elements	Relative position				
	A	B	C	D	E
$k \times 5 + 1$	k	k	$k + 1$	k	k
$k \times 5 + 2$	$k + 1$	k	k	k	$k + 1$
$k \times 5 + 3$	$k + 1$	k	$k + 1$	k	$k + 1$
$k \times 5 + 4$	$k + 1$	$k + 1$	k	$k + 1$	$k + 1$
$(k + 1) \times 5$	$k + 1$	$k + 1$	$k + 1$	$k + 1$	$k + 1$

distribution of errors has been taken as empirical evidence for a "read-out" process in the Graphemic Buffer—the "read-out" process is sensitive to interference between immediately adjacent positions resulting in poorer performance for letters in word-medial positions (Wing & Baddeley, 1980). D.H. and M.L. both showed a variation on this normal pattern: in each case the distribution was bow-shaped, but skewed in opposite directions.

DISCUSSION

How can we account for the divergent patterns of errors among patients for whom it is hypothesized that they have damage to the same component of the spelling process? The opposing directions of the asymmetrical error distributions obtained for M.L. and D.H. could reflect random, individual variation in dealing with an impoverished database in the Graphemic Buffer. Alternatively, it could be the case that the observed asymmetries in the distribution of errors are "true" effects reflecting either different forms of impairment within the Graphemic Buffer or different additional impairments in each patient. Although at this time it is not possible to adjudicate among these possibilities with any certainty, a plausible case can be made for the hypothesis that contralesional attentional impairments may contribute importantly to the observed variation in the distribution of spelling errors. Our discussion will focus on this latter possibility.

Baxter and Warrington (1983) have reported a patient with left neglect, O.R.F., who presents with a pattern of spelling errors similar to that of M.L. These authors interpreted their results as evidence that spelling requires reading from an "inner screen," and that such "reading" is susceptible to similar impairments as those that may occur in ordinary reading from print. On this interpretation, O.R.F.'s hemispatial neglect errors in (oral) spelling performance has the same underlying cause as his neglect errors in ordinary reading performance. M.L.'s oral (and written) spelling errors also matched her word-reading errors which occurred primarily at the beginnings of words. She produced substitutions, deletions, transpositions, or insertions of single or multiple letters (graphemes or phonemes), at the beginnings of words both in oral reading and in writing. The reading performance reported for both O.R.F. and M.L. corresponds to a pattern that Kinsbourne and Warrington (1962) have reported for patients with left neglect: the production of "backward completion errors" (e.g., later → better). M.L.'s preponderance of spelling errors at the beginnings of words co-occurring with neglect in other, nonspelling tasks suggests the hypothesis that left neglect² contributes to her lateralized errors in "reading" letters from the *damaged* Graphemic Buffer. On this hypothesis, neglect is interpreted to be a clinical manifestation of a unilateral breakdown in the distribution of attention over

² In keeping with the bulk of the literature, we use the term, "unilateral spatial neglect," or simply "neglect," in reference to all varieties of phenomena evidencing impaired cognitive processing of internal representations or external stimuli, on the side contralateral to brain damage. We hasten to add that accumulating evidence (including results cited herein) indicates that the various behavioral and perceptual disorders reported under the clinical term of neglect are not symptoms of a unitary deficit, but dissociable symptoms of impaired attentional mechanisms which may operate at several levels of cognitive processing.

the internal spatially coded graphemic representation of the word (to be spelled or read).

In contrast with M.L.'s left-sided errors (secondary to right hemisphere damage), D.H. (with left hemisphere damage) exhibited the opposite pattern of performance in reading and spelling. D.H.'s unilateral errors included frequent suffix (but never prefix) substitutions, deletions, or insertions in both reading and writing of morphologically complex words (see Table 9), although he consistently self-corrected such errors in narrative reading. More than 60% of D.H.'s errors in reading lists of affixed and unaffixed words were visual confusions at the ends of words—errors that could be described as "forward completion" errors (e.g., happen → happy), of which 25% were morphological errors (e.g., suspend → suspended). D.H. produced a nearly identical distribution of errors in spelling the same words: 54% of errors at the ends of words, of which 31% were morphological paraphasias. Kinsbourne and Warrington noted the existence of forward-completion, reading errors associated with right neglect, although no cases were reported in detail. The preponderance of D.H.'s right-sided "completion" errors in reading, along with a right-sided skew of errors in spelling, could reflect the influence of a mild, right neglect, comparable to the hypothesized influence of left neglect in M.L.'s and O.R.F.'s performance.

If an attentional deficit were to be a contributing factor to M.L.'s and D.H.'s reading errors at the beginnings or ends of words, it could also be the basis for the contrasting distribution of their spelling errors as a function of letter position within words. The hypothesis considered here is not that an attentional deficit is the cause of these patients' spelling

TABLE 9
POSITION OF ERRORS IN D.H.'S READING AND WRITING PERFORMANCE^a

Position of error:	First 3rd of word	Middle 3rd of word	Last 3rd of word
Reading			
Letter substitutions	4	2	13
Insertions	3	4	14
Deletions	1	2	6
Transpositions	0	4	3
Total	8 (14.3)	12 (21.4)	36 (64.2)
Writing			
Letter substitutions	6	3	18
Insertions	1	1	1
Deletions	5	12	20
Transpositions	2	6	4
Total	14 (7.7)	22 (27.8)	43 (54.4)

^a Errors include visually similar words.

impairment, but that the presence of a mild hemispatial attentional deficit could modulate the outcome of an impaired computational process at the level of the Graphemic Buffer. In other words, we propose that the predominance of spelling errors at the beginnings or ends of words, for M.L. and D.H., respectively, reflects the contribution of a lateralized disruption of attention (neglect) in impaired "reading" from an inner screen. By contrast, the predominance of errors in the medial positions of words reported in earlier cases (e.g., L.B.) may reflect damage to the Graphemic Buffer without the influence of attentional impairment.

A possible obstacle to this explanation of the contrasting patterns of spelling errors in our two patients is the absence of clear evidence of neglect in traditional clinical tests of spatial neglect: line cancellation, line bisection, or drawing familiar objects. However, we do have some evidence of (subclinical) neglect in the reading process and in perception of nonlexical grapheme strings in our two patients. In a series of tachistoscopic experiments described elsewhere (Hillis & Caramazza, in preparation), D.H. and M.L. made significantly fewer errors in identifying a pair of identical letters in briefly presented random letter strings, when both members of the pair occurred on the side ipsilateral to brain damage, than when at least one of the pair occurred contralateral to brain damage, *regardless* of location along the visual field. Also, in a tachistoscopic lexical decision task, they made a disproportionate number of errors in rejecting nonwords with unpronounceable segments on the right or the left, when the segment occurred contralateral to the side of lesion: 48.0% left-sided errors vs. 11.3% right-sided errors for M.L. ($\chi^2 = 47.17$; $p \ll .0001$) and 7.3% left-sided errors vs. 29.3% right-sided errors for D.H. ($\chi^2 = 22.55$; $p \ll .0001$). These results cannot be attributed to homonymous hemianopia, since both patients had full visual fields at the time of the study. Hence, the comparable pattern of unilateral errors opposite to the side of lesion, across oral and written spelling, oral reading, and tachistoscopic perceptual tasks, along with mild indications of neglect in standardized tests (contralesional errors in drawing abstract figures and ipsilateral position preference on the RCPM), for both patients provides some support for the hypothesis that these patients present with mild attentional deficits.

Another potential obstacle for the conjectured role of attentional deficits in the spelling process is the absence of a unilateral distribution of spelling errors in some previously reported dysgraphic patients with left neglect. Ellis, Flude, and Young (1987a) have shown that neglect may affect writing performance at a more peripheral level, without causing lateralized grapheme errors. They reported the spelling performance of V.B., a nonaphasic woman with left neglect, who made spatial and spelling errors in writing. These authors attributed their patient's performance to impaired use of visual and kinesthetic feedback. One of her symptoms, a failure

to dot i's and cross t's, was largely lateralized to the left, although her letter and stroke errors were distributed fairly evenly across positions within the word. Unlike D.H. and M.L., this patient could spell aloud remarkably well. In a separate paper (Ellis et al., 1987b), these authors reported that V.B. made "backward completion" errors in reading, although these errors were eliminated when words were spelled aloud to her, or when print was rotated vertically. Her performance is in striking contrast to that of three other patients with neglect whom we have studied (R.B., N.G., and H.H.). These latter patients not only presented with contralesional symptoms comparable to V.B.'s in ordinary reading and written spelling but also showed impairments in tasks of vertical reading, recognition of orally spelled words, and oral spelling (Hillis & Caramazza, in preparation). We have argued that attentional deficits may affect various stages in the reading and spelling processes, such that unilateral errors in the computation of an internal representation (e.g., in oral spelling) and in directed attention to external stimuli (e.g., manifested in omission of words on one side in reading narratives and failure to dot i's on one side in writing) are dissociable. V.B.'s unilateral reading and spelling errors confined to horizontal processing of external, visual stimuli indicates that neglect can operate solely at the latter level. We found the opposite dissociation in one patient, H.H.: neglect in computing the internal representation of words in reading (vertical or horizontal print), in the presence of normal recognition of the letters that constitute the words, and in oral and written spelling, without symptoms of impaired scanning of external stimuli. By the same token, M.L.'s and D.H.'s unilateral spelling and reading errors, without obvious scanning impairment, may represent a similar dissociation.

Further evidence that neglect can operate in the domain of internal representation has been supplied in numerous studies by Bisiach and his colleagues (e.g., Bisiach, Luzzatti, & Perani, 1979; Bisiach & Berti, 1987), as well as in the cited study by Baxter and Warrington. It is in this vein that we have raised the possibility that D.H. and M.L.'s opposing distributions of spelling errors, both in oral and in written spelling, may reflect the influence of an attentional deficit at a level of internal "spatial" representation of the to-be-spelled word in the Graphemic Buffer.

Several alternative explanations of unilaterally skewed distribution of spelling errors have been put forth in the literature. For instance, Chedru and Geschwind (1972) reported, for a group of 21 hospitalized patients in acute confusional states and 20 hospitalized controls, that spelling errors were more frequent on the last two letters than the first two letters of words. This pattern, congruent with D.H.'s distribution of errors, could be assumed to reflect serial retrieval from abnormally rapid decay of representations stored in the Graphemic Buffer (Wing & Baddeley, 1980). On the other hand, Wing and Baddeley reported the opposite

pattern of reversal and substitution errors among normal writers' slips of the pen—these errors were more frequent on initial letters of words, resembling the pattern of errors reported for M.L. Thus, M.L.'s pattern could be interpreted as an exaggeration of the "normal" error distribution, although it must be noted that her most common errors were letter deletions (while in Wing & Baddeley's study of normal spellers' slips of the pen deletion errors were concentrated in the medial position of words). It is possible, then, that the different distributions of spelling errors in our two patients reflect different forms of damage to the Graphemic Buffer: M.L. could have an aspecific reduction of "processing capacity" in the Buffer resulting in an elevated but "normal" distribution of spelling errors; D.H. could have a deficit affecting the rate of decay of information in the Buffer with the consequence that the ends of words are adversely affected more than the beginnings of words.

Although we cannot reject this latter account as the basis for the reported asymmetries in spelling errors in our two patients, this hypothesis is less than parsimonious—it fails to account for the observed co-occurrences of deficits in M.L. and D.H. That is, the hypothesis of different forms of damage to the Graphemic Buffer for M.L. and D.H. is silent with respect to the overall patterns of reading and spelling impairments found in the two patients. For this reason we feel encouraged to interpret the coincident pattern of asymmetrical reading and spelling errors in D.H. and M.L., in each case with disproportionate errors on the side contralateral to brain damage, as suggesting the hypothesis that attentional deficits interact with a deficit to the Graphemic Buffer to produce the observed constellation of symptoms in our patients.

The hypothesis we have advanced as the basis for the asymmetrical distribution of errors in D.H. and M.L. forces us to consider an important theoretical issue concerning the nature of possible interactions between representations stored in the Graphemic Buffer and attentional mechanisms. By hypothesis, the representations stored in the Graphemic Buffer are "abstract" graphemic representations; that is, these representations do not correspond to particular letter shapes but merely specify the set of possible letter shapes for each of the graphemes that constitute a word. Although graphemic representations are "abstract" in the sense just given, they must specify "spatial" information pertaining to the order of the graphemes in a word. Thus, the representation stored in the Graphemic Buffer may be thought of as an ordered set of graphemes. Although the order information in a graphemic representation need not be represented spatially, there is no reason why it might not be. Indeed, if the account we have given as the basis for our two patients' spelling performance were to be correct, then we would have to be committed to the position that order information in the Graphemic Buffer is represented spatially. In other words, our hypothesis entails that graphemic repre-

sentations in the buffer are coded spatially and hence subject to spatial attentional deficits. It goes without saying, of course, that this hypothesis is in need of independent empirical evaluation.

To conclude, the results we have reported favor an interpretation of the Graphemic Buffer as a temporary storage system for the internal representation of a word that is "read off" as one spells. The two cases we have presented supplement the accumulating support for the functional architecture of the spelling process schematically represented in fig. 2, and, more specifically, they provide support for the Graphemic Buffer as a crucial component of the spelling process. These two cases also provide suggestive evidence in support of the hypothesized processing structure of the Graphemic Buffer and the potential interaction of the latter system with attentional mechanisms.

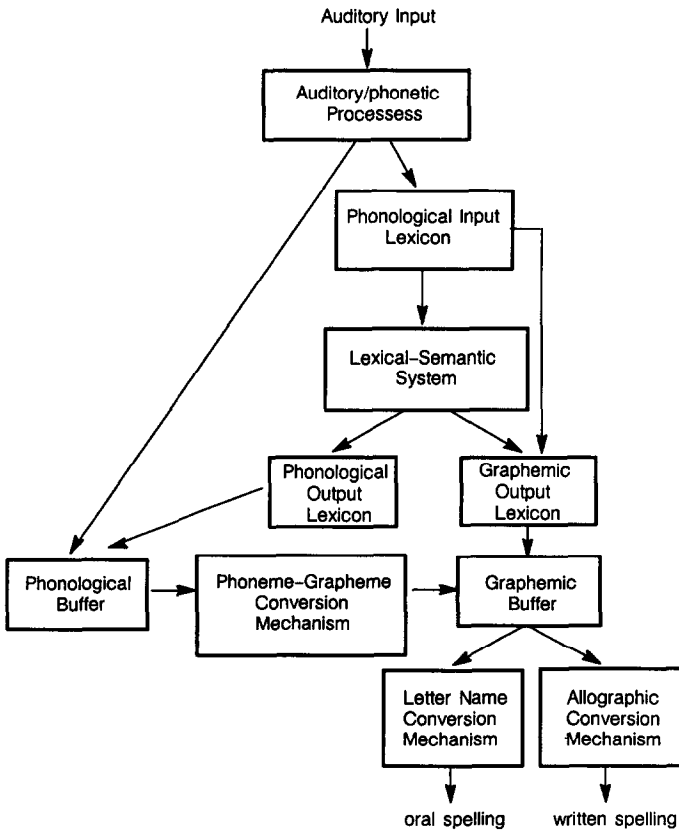
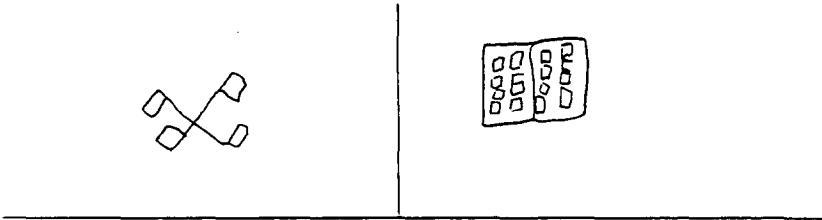


FIG. 2. Schematic diagram of the spelling process.

APPENDIX A

Examples of D.H.'s Drawings with Spatial Errors

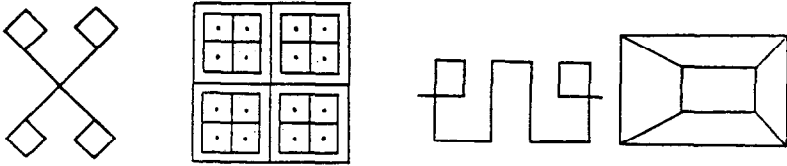
Delayed reproduction of the Weschler Memory Scale figures:



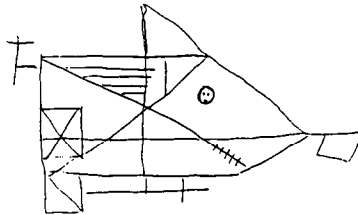
VI C-1. C-2



Stimulus figures from the WMS (Weschler, 1945):



Direct copy of the Rey Figure :



APPENDIX B

Examples of D.H.'s Spelling Errors:
Various Types Phonemically Implausible Nonword (PIN) Errors

	Words	Nonwords
1. Single errors		
Substitution	bump → buep	herm → hegm
Insertion	oyster → osyster	feen → feent
Deletion	faith → faih	reesh → reeh
Transposition	church → chucrth	ghurb → grub
2. Multiple errors		
Substitutions	urban → egban	kittul → keeful
Insertions	—	feen → frient
Deletions	since → sic	—
Transpositions	—	—
3. Mixed errors		
Substitutions and deletions	kitchen → kicken	—
Substitutions and transpositions	pursuit → presuit	reesh → richs
Insertions and deletions	afraid → affrid	—
Deletions and transpositions	pierce → peire	—
Deletions and transpositions and substitutions	fierce → feist	—
Multiple transpositions and deletions	vulgar → vagul	—
Multiple substitutions and deletions	—	—
Multiple deletions and substitutions	offense → offec	—
Multiple insertions and deletions	—	faunch → frouf
4. Unclassifiable		
Fragments	yawn → y	pytes → pi
Miscellaneous	courage → gorous	poys → fesh

Examples of D.H.'s Handwriting and Spelling Errors in Delayed Copy transcoding

afraid AFRID AFR

carry CARRY

length LENY LENGH

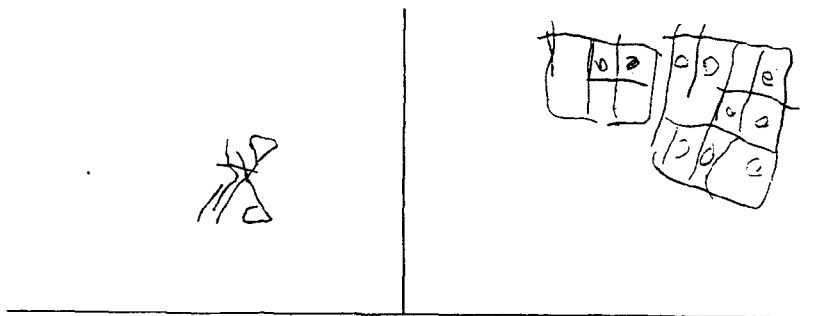
under UNDEA

talent TALTE TALENT

APPENDIX C

Examples of M.L.'s Drawings with Spatial Errors

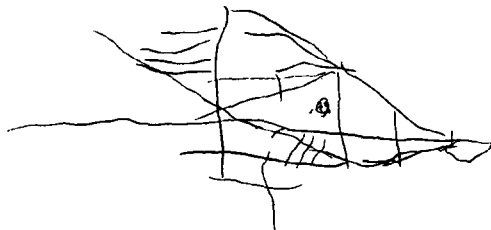
Delayed reproduction of the WMS figures:



VI C-1. C-2

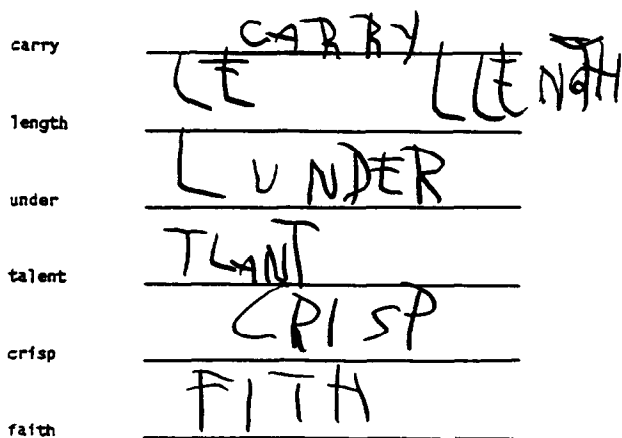


Direct copy of the Rey Figure:



Insertions and deletions	guitar → gita	pressure → pesure	skart → sarat
Deletions and transpositions	—	college → clegdc	—
Insertions and transpositions	—	sister → stser	murnee → rnmee
Deletions and transpositions and substitutions	carrot → caorrtto	threat → rheat	—
Substitutions and multiple deletions	razor → rzasor	fence → fcnece	kantree → katny
Deletions and multiple substitutions	—	status → satatsu	—
Substitutions and insertions	—	success → cssess	haygrid → hrgein
Substitutions and multiple insertions	—	talent → tanat	—
Substitutions and transpositions and insertions	—	—	merber → mmmr
Insertions and multiple deletions	—	offense → fencn	—
Insertions and multiple deletions	—	cable → cpble	—
Deletions and multiple transpositions	—	mend → ndend	mushrame → mukhrname
Insertions and multiple deletions	—	trout → trtorat	—
Deletions and multiple transpositions	—	crawl → healwl	—
Multiple deletions and transpositions	—	question → qstnion	—
	—	future → frhrure	—
	—	starve → svrte	—
	—	—	donsept → dspest

Examples of M.L.'s Handwriting and Spelling errors in Delayed Copy
Transcoding (Written with Her Right, Nondominant Hand, Due to
Persisting Hemiplegia)



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