Research Article

Phonological Dyslexia

A Test Case for Reading Models

Elise Caccappolo-van Vliet,[1,2] Michele Miozzo,[3] and Yaakov Stern[1,2,3]

[1]*Cognitive Neuroscience Division, Taub Institute for Research in Alzheimer's Disease and the Aging Brain;*
[2]*Gertrude H. Sergievsky Center; and* [3]*Columbia University*

ABSTRACT—***Following brain damage, skilled readers may encounter more severe problems in reading nonwords than familiar words, a type of deficit referred to as phonological dyslexia. We report on 2 individuals with Alzheimer's disease who show phonological dyslexia. Although highly accurate in reading familiar words aloud (even those with irregular spelling, such as*** sew***), they were quite impaired in nonword reading. Both patients performed well in phonological tasks involving the repetition, identification, and manipulation of phonemes of orally presented words and nonwords. These results challenge the idea, proposed in the context of connectionist and evolutionary theories, that phonological dyslexia originates from a phonological deficit. However, the results are consistent with reading models, such as the dual-route model, that attribute phonological dyslexia to a deficit that selectively affects the reading mechanisms responsible for deriving the sounds of nonwords. According to these models, such a deficit is not necessarily accompanied by a more general phonological impairment.***

The term phonological dyslexia is used by neuropsychologists to describe reading deficits that affect nonwords (*nep*, *cabe*) more severely than familiar words. This disturbance appears in skilled adult readers following cortical brain damage; a developmental form has also been reported in children who have no apparent cortical lesions (e.g., Temple & Marshall, 1983). The understanding of phonological dyslexia not only has obvious clinical consequences, but also has implications for the current debate on reading processing. Current models of reading offer different accounts of phonological dyslexia. Because a better knowledge of this deficit can severely constrain such models, it is of primary theoretical significance.

The dual-route model proposes that two types of mechanisms, which are in part neuroanatomically distinct, support reading aloud (see Coltheart, Rastle, Perry, Ziegler, & Langdon, 2001, for a recent instantiation of this account and a discussion of its variants). One series of mechanisms, the lexical route, is implicated in the retrieval of stored information about the orthography, semantics, and phonology of familiar words. An alternate route, the nonlexical route, allows readers to derive the sounds of written words by means of mechanisms that convert letters or letter clusters into their corresponding sounds. The nonlexical route is functionally limited in that it does not provide information about word meaning; nor, in a language like English or Italian, does it guarantee the correct pronunciation of a number of words. Nevertheless, the nonlexical route is responsible for deriving the sounds of nonwords; its selective damage would result in phonological dyslexia (Berndt, Haendiges, Mitchum, & Wayland, 1996; Coltheart, 1985; Derousné & Beauvois, 1985).

Another class of models, which we refer to as "triangle models," offers a different account of phonological dyslexia. According to these models, reading aloud depends on the joint processing of mechanisms that translate orthography into phonology and mechanisms that bind word meaning and phonology. This type of architecture has been proposed in several connectionist models (e.g., Plaut, McClelland, Seidenberg, & Patterson, 1996; Seidenberg & McClelland, 1989). Although differing on a number of details, the various triangle models all share the assumption that identical processes support the reading of words and nonwords. That is, in contrast to the dual-route model, they do not propose any mechanisms specifically involved in the processing of nonwords. Within the framework of triangle models, the cause of phonological dyslexia is assumed to be an impairment in the representation of phonological information (Friedman, 1996; Harm & Seidenberg, 2001; Patterson, 2000; Plaut et al., 1996). Various factors conspire to make nonwords more vulnerable to a phonological impairment than familiar words are. Because nonwords depend more on orthography-phonology mapping, have less stable phonological representations, and do not benefit from the collateral support of semantics, conditions that alter the representation of phonological information are expected to have sizable effects on nonword reading. These models further propose that when phonological impairment is mild, only nonword reading should be affected, thereby accounting for pure cases of phonological dyslexia in which reading of familiar words is spared (e.g., Beauvois & Derousné, 1979; Funnell, 1983; Shallice & Warrington, 1980). We refer to this explanation of phonological dyslexia as the *phonological-impairment hypothesis*.

Farah, Stowe, and Levinson (1996) also appealed to the phonological-impairment hypothesis, although from different theoretical premises. Essentially, their idea was that because reading is, from an evolutionary perspective, a recently acquired function, it cannot

---

Address correspondence to Michele Miozzo, Department of Psychology, Columbia University, 1190 Amsterdam Ave., New York, NY 10027; e-mail: michele@psych.columbia.edu.

depend on specialized brain regions. Consequently, brain damage cannot cause selective reading deficits. However, damage to cognitive functions with dedicated brain regions (e.g., vision or phonology) can affect reading. In particular, phonological deficits should have selective effects on nonword reading essentially for the reasons already explained: because nonwords require additional phonological processing for word sounds to be assembled.

Proponents of the dual-route model do not deny that phonological deficits could affect reading, particularly nonword reading. A point of divergence concerns whether such deficits should invariably accompany this form of dyslexia, as proposed by the phonological-impairment hypothesis, or whether other factors can also yield phonological dyslexia, as proposed by the dual-route model. It is striking that patients with acquired phonological dyslexia almost invariably fail in a range of phonological tests, including, for example, nonword repetition and phoneme-manipulation tasks (e.g., "repeat the word *sharp* without the initial phoneme"). This association of deficits lends support to the phonological-deficit hypothesis. However, there have also been individuals with acquired phonological dyslexia who have performed well on phonological tasks, although these cases have been reported much more rarely. In fact, only 2 or 3 such patients have been reported.

One such individual is LB (Derousné & Beauvois, 1985), a French-speaking patient who, for example, could assemble nonwords if provided with the individual phonemes (e.g., /g/, /r/, /a/ → /gra/) and could pronounce the last phoneme of a word spoken by the experimenter. The second individual, the Italian speaker RR (Bisiacchi, Cipolotti, & Denes, 1989), was able to pronounce the first phoneme of aurally presented words and performed within control subjects' range in the difficult spoonerism task, which requires swapping word onsets (as in *Niccolò Macchiavelli* → *Miccolò Nacchiavelli*). More recently, we (Caccappolo-van Vliet, Miozzo, & Stern, in press) documented phonological dyslexia in a subject (RG) diagnosed with Alzheimer's disease. Because of a general cognitive decline, RG could perform phonological tasks only if they required relatively simple instructions: word and nonword repetition, rhyme judgment ("Do the words *pair* and *bear* rhyme?"), and production of words that share onset phonemes (*shot* → *sugar*) or rhyme (*table* → *cable*). RG performed flawlessly on all these tasks. The dissociations between impaired nonword reading and preserved performance on phonological tasks documented in these patients are problematic for the phonological-deficit hypothesis. These dissociations, however, are in line with the dual-route model, which does not assume phonological deficits to be the principal cause of phonological dyslexia and thus does not anticipate a recurrent association between phonological deficit and nonword reading impairment.

Upon closer scrutiny, however, these dissociations might appear to be less compelling. Several questions have been raised about these patients' data. For example, Patterson (2000) questioned whether LB was a convincing case of phonological dyslexia, because his accuracy was not far better for words (range: 74–94%) than for nonwords (range: 48–85%). Patterson raised similar concerns about RR, as there were indications that this patient could read short nonwords but had some problems with low-frequency abstract familiar words. Harm and Seidenberg (2001) suspected that LB's relatively good performance in the phonological tasks resulted from rehabilitative training that emphasized phonological reading and improved LB's phonological awareness. Regarding RG, data were limited to phonological tasks with procedures simple enough to be understandable to this patient. In short, if previous neuropsychological data do not allow one to make conclusive claims about whether phonological dyslexia and phonological deficits dissociate, it also appears clear that this issue needs further investigation.

An opportunity to address this issue is offered by the 2 patients we report on here, M.O. and I.B. Both have progressive dementia and encountered relatively severe problems in reading nonwords. Their word reading was highly accurate, and they also performed remarkably well on a wide variety of phonological tasks. Their serious cognitive damage prevented us from administering a few tasks; still, our results provide significant constraints on current accounts of acquired phonological dyslexia.

CASE REPORTS

M.O. is a 48-year-old, right-handed Caucasian male with a master's degree in business who worked as an accountant. I.B. is a 77-year-old, right-handed African American female with 12 years of education who was previously employed as a secretary. Both patients were diagnosed with probable Alzheimer's disease according to strict neurological criteria based on full clinical evaluation and extensive neuropsychological testing. M.O. has a family history of autosomal dominant Alzheimer's disease; his father, paternal uncle, and three siblings were all diagnosed in their 40s. The 2 patients had no history of psychiatric disease, head trauma, alcohol abuse, or other medical diseases, and neither patient demonstrated behavioral or psychiatric symptoms. Neither patient showed developmental dyslexia, according to reports of their family members.

Neuropsychological testing of both patients revealed significant deficits in multiple cognitive domains, including verbal and visual learning and memory, attention, abstract reasoning, and confrontation naming (see Table 1). We analyzed whether oral picture-naming responses revealed phonological distortions (i.e., substitutions, additions, or omissions of one or more phonemes). Such errors were rarely observed with M.O. (5/770, 0.6%; e.g., *ruler* → "roller," *beanie* → "beeper") and never observed with I.B. (0/75). On a modified version of the Mini Mental Status Examination (mMMSE; Mayeux, Stern, Rosen, & Leventhal, 1981), I.B. obtained a score of 33 (highest possible score = 57), which suggests moderate dementia; M.O.'s mMMSE score of 42 at the start of our study was indicative of mild dementia. Both patients also showed impaired nonword reading. This latter deficit spurred the current investigation, which was initiated in February 2002 for M.O. and May 2002 for I.B., and ended in March 2003 for both. The investigation obtained approval from Columbia University's ethical committee. I.B.'s testing was limited because she agreed to be evaluated only when she had a previously scheduled appointment at the university hospital. Neither patient participated in a language rehabilitation program. A comparison of the mMMSE scores of the patients at the beginning and end of our investigation reveals that I.B.'s cognitive abilities remained stable, but M.O.'s cognitive abilities declined (his score decreased from 42 to 33). (Note that in order to prevent a systematic effect of cognitive decline, we obtained the data for reading and phonological processing in the same testing sessions.)

IS THE PATIENTS' READING IMPAIRED?

The patients' performance in reading words aloud is summarized in Tables 2 and 3. We used several lists to compare the patients' accuracy in reading words with regular and irregular spellings (e.g., *pink*

TABLE 1

Neuropsychological Test Scores

Test	Range	Control subjects		Patients	
		Mean	*SD*	M.O.	I.B.
Attention					
Cancellations (seconds)[a]	0–240	63	27	120	47
Construction					
Rosen Drawing Test	0–5	3	1	3	5
Benton Visual Retention Test: Matching	0–10	9	1	9	10
Memory					
Selective Reminding Test					
Total Recall	0–72	43	9	21	22
Delayed Recall	0–12	6	3	0	0
Delayed Recognition	0–12	11	1	6	3
Benton Visual Retention Test: Recognition	0–10	8	2	8	6
Verbal short-term memory					
Digit Span (WAIS–R, age-scaled)	1–19	10	3	7	6
Language					
Boston Naming Test	0–15	14	1	13	8
Controlled Word Association (letters *c*, *f*, and *l*)	0–99th %ile	64	31	22	1
Category Fluency (Animals)	0–99th %ile	36	28	3	1
Sentence Repetition (BDAE)	0–8	7	1	7	7
Comprehension (BDAE)	0–6	6	1	5	5
Abstract reasoning					
Similarities (WAIS-R, age-scaled)	0–20	10	3	7	7
Identities & Oddities (Mattis DRS)	0–16	15	1	16	14

Note. Tests were administered in winter 2002, prior to the beginning of the current investigation. Control subjects were 155 nondemented elderly persons. The sources of the tests were as follows: Cancellations—Rosen (1993); Rosen Drawing Test—Rosen (1981); Benton Visual Retention Test: Matching—Benton (1955); Selective Reminding Test—Buschke and Fuld (1974); Benton Visual Retention Test: Recognition—Benton (1955); Digit Span—Wechsler (1981); Boston Naming Test—Goodglass (1983); Controlled Word Association—Benton and Hamsher (1976); Category Fluency—Benton and Hamsher (1976); Sentence Repetition—Goodglass and Kaplan (1983); Comprehension—Goodglass and Kaplan (1983); Similarities—Wechsler (1981); Identities & Oddities—Mattis (1988). BDAE = Boston Diagnostic Aphasia Examination; DRS = Dementia Rating Scale; WAIS–R = Wechsler Adult Intelligence Scale–Revised.

[a] Higher scores (i.e., time in seconds) reflect lower performance.

vs. *pint*). They responded accurately ($>$ 90% correct) to both types of words (χ^2s < 1). On the lists of irregular words compiled by Glushko (1979), the patients were as accurate as unimpaired readers (range: 91–100% vs. 88–92% correct). M.O. and I.B. were as accurate reading derived, inflected, and compound words as reading monomorphemic words of the same frequency and length ($\chi^2 < 1$). The patients' accuracy also did not vary as a function of variables such as concreteness, grammatical class, frequency, or word length. Of particular interest is the finding that neither patient encountered problems with functors (closed-class words, such as prepositions and determiners). In this respect, M.O. and I.B. differed from the group of phonological dyslexics reported by Friedman (1996), who were impaired in reading functors.

We administered two tasks to assess reading comprehension. In the first task, the patients were shown four written words and were instructed to point to the word spoken by the experimenter (e.g., *glass*)

TABLE 2

Number and Percentage of Correct Responses Provided by M.O. and I.B. in Reading Aloud Lists Including Regular and Irregular Familiar Words

Test	M.O.		I.B.	
	Regular	Irregular	Regular	Irregular
Johns Hopkins Dyslexia Battery	35/36 (97%)	34/36 (94%)	34/36 (94%)	34/36 (94%)
Coltheart, Besner, Jonasson, & Davelaar (1979)	39/40 (97%)	38/39 (97%)	38/40 (95%)	37/39 (95%)
Glushko (1979, Experiment 1)[a]	43/43 (100%)	39/43 (91%)	39/43 (91%)	41/43 (95%)
Glushko (1979, Experiment 3)[a]	41/41 (100%)	41/41 (100%)	40/41 (98%)	40/41 (98%)
Shallice, Warrington, & McCarthy (1983)	37/39 (94%)	72/76 (95%)	38/39 (97%)	71/76 (93%)

[a] Glushko (1979) reported accuracy data from unimpaired readers (college students); their scores with irregular words (means = 87.8% and 91.7% for Experiments 1 and 2, respectively) were comparable to those of M.O. and I.B.

TABLE 3

Number and Percentage of Correct Responses Provided by M.O. and I.B. in Reading Aloud Words With Different Characteristics

Characteristic	M.O.	I.B.
Concreteness		
Concrete	19/20 (95%)	19/20 (95%)
Abstract	18/20 (90%)	17/20 (85%)
Grammatical class		
Nouns	25/26 (96%)	26/26 (100%)
Verbs	24/26 (92%)	25/26 (96%)
Adjectives	25/26 (96%)	25/26 (96%)
Functors	25/26 (96%)	25/26 (96%)
Frequency		
High	25/25 (100%)	25/25 (100%)
Low	25/25 (100%)	25/25 (100%)
Word length		
4 letters	13/13 (100%)	13/13 (100%)
5 letters	13/13 (100%)	13/13 (100%)
6 letters	12/13 (92%)	13/13 (100%)
7 letters	13/13 (100%)	11/13 (85%)
8 letters	13/13 (100%)	13/13 (100%)
Morphological structure		
Prefixes		
Prefixed words	72/75 (96%)	69/75 (92%)
Monomorphemic controls	74/75 (98%)	71/75 (95%)
Inflection		
Inflected words	72/75 (96%)	72/75 (96%)
Monomorphemic controls	73/75 (97%)	70/75 (93%)
Compounding		
Compounds	71/75 (95%)	71/75 (95%)
Monomorphemic controls	74/75 (98%)	73/75 (97%)

Note. Items involving morphological structure were from Badecker, Hillis, and Caramazza (1990). Morphologically complex words and their monomorphemic controls were matched for frequency and number of letters. All other items were from the Johns Hopkins Dyslexia Battery (Goodman & Caramazza, 1986); in each of these lists, words were controlled for variables affecting reading (e.g., concrete and abstract words were matched for frequency, grammatical class, and length).

TABLE 4

Numbers of Plausible Responses Provided by M.O. and I.B. in Reading Nonwords Aloud

Word List	M.O.	I.B.
Woodcock & Johnson (1989): Word Attack	12/28 (43%)	12/28 (43%)
Friedman, Ferguson, Robinson, & Sunderland (1992)	27/69 (39%)	18/69 (26%)
Glushko (1979)	66/138 (48%)	58/138 (42%)
Kay & Patterson (1985)	47/80 (59%)	22/80 (27%)
Berndt, Haendiges, Mitchum, & Wayland (1996)[a]		
High grapheme-to-phoneme correspondence	27/32 (84%)	21/32 (66%)
Low grapheme-to-phoneme correspondence	4/20 (20%)	3/20 (15%)
Caccappolo-van Vliet, Miozzo, & Stern (in press)		
4-letter words	31/48 (64%)	21/48 (43%)
5-letter words	16/48 (33%)	20/48 (41%)
6-letter words	27/48 (56%)	30/48 (62%)
Total	74/144 (51%)	71/144 (49%)

[a] The difference in number of correct responses to nonwords with high versus low grapheme-to-phoneme correspondence was significant for both M.O., $\chi^2(1, N = 52) = 4.0$ ($p = .04$), and I.B., $\chi^2(1, N = 52) = 10.7$ ($p = .001$; Yates' correction applied).

rather than to a semantic foil (*cup*), a phonological-orthographic foil (*gloss*), or an unrelated foil (*bloom*). The second task had the same design with the exception that the target stimuli were pictures rather than spoken words. The responses of M.O. and I.B. were invariably correct in both tasks (12/12 and 20/20, respectively), a result that suggests that reading comprehension was fairly intact in both patients.

By contrast, the patients were impaired in reading nonwords aloud. For these tasks, the patients were informed that they would be presented with "invented" words. We adopted a lenient scoring procedure: We considered responses correct if they conformed to rules of English pronunciation (Venezky, 1970) or to pronunciations of parts of real English words with irregular spellings (e.g., *heaf* read as rhyming with *deaf*). Despite such lenient criteria, both patients' accuracy was below 60% on several lists (see Table 4 for a summary). The only exception was that accuracy was 84% for M.O. and 66% for I.B. on a list of nonwords with high grapheme-to-phoneme correspondence (GPC; e.g., *san* or *teep*), taken from Berndt et al. (1996). These results can be explained in various ways: For example, grapheme-to-phoneme rules may be more resistant to damage the more frequent they are; alternatively, if nonword pronunciations are derived by analogy to familiar words, correct responses are more probable for nonwords the higher their GPC. In a group study, Berndt et al. observed an advantage for high-GPC nonwords only in patients who scored high in word reading. M.O.'s and I.B.'s responses fit this pattern.

Nonwords were presented along with familiar words, except in two lists (Word Attack; Friedman, Ferguson, Robinson, & Sunderland, 1992). Accuracy, however, did not change as a function of list composition, as is evident from Table 4. Unimpaired readers perform much better with nonwords than these patients did. For example, Friedman et al. found that unimpaired readers scored above 80% on their list, whereas our patients scored lower than 40% on the same list. An analysis of patients' errors can be found in Table 5. Two results commonly observed in phonological dyslexia also appeared in our patients' errors. First, lexicalizations—reading nonwords as familiar words (e.g., *bip* → *hip*)—accounted for many of the errors committed by M.O. (21%) and I.B. (43%). Second, single-phoneme errors (substitutions, deletions, and additions) were far more common with vowels than with consonants (M.O.: 77 vs. 11; I.B.: 83 vs. 29), a result that reflects the greater variability in letter-sound correspondence of vowels in English. (Note that nonwords comprise more consonants than vowels.)

We examined the possibility that the nonword reading impairment stemmed from the verbal short-term memory (STM) deficit that also affected our patients. Various authors (e.g., Caramazza, Capasso, & Miceli, 1996; Derousné & Beauvois, 1985) have pointed out that if nonword letter-sound transcoding occurs serially, sounds are probably stored in the STM structure until the assembly of the whole sound sequence is completed. Reduced STM capacity like that experienced

TABLE 5

Numbers of Errors Produced by M.O. and I.B. in Reading Nonwords Aloud

Type of error	M.O.	I.B.
Lexicalizations (word responses)	53 (21%)	132 (43%)
Single-phoneme errors[a]		
Vowels	77 (30%)	83 (27%)
Consonants	11 (4%)	29 (10%)
Complex errors[b]	113 (45%)	42 (14%)
Omissions (failures to respond)	0 (0%)	20 (6%)
Total	254	306

[a]These errors consisted of nonword errors that involved the substitution of a single phoneme (e.g., *joon* → ‘‘zoon’’), deletion of a phoneme (e.g., *forch* → ‘‘orch’’), or addition of a phoneme (e.g., *sost* → ‘‘soist’’). [b]In these errors, more than one phoneme was produced incorrectly, as in *shan* → ‘‘charan’’ or *teus* → ‘‘teususs.’’

by our patients could give rise to nonword reading problems, and these problems should be more pronounced the longer the items. However, error rates did not increase from four-letter to six-letter nonwords (see Table 4), a finding that does not lend support to the hypothesis that an STM deficit underlies the nonword reading deficit of our patients.

IS THE PATIENTS’ PHONOLOGICAL PROCESSING IMPAIRED?

M.O. and I.B. were tested with a wide range of tasks that have revealed phonological deficits in past studies. The experimenter aurally presented the items and, depending on the phonological task, the patients repeated a word or nonword, or identified, produced, or manipulated certain phonemes. Instructions, material descriptions, and results are reported in Tables 6 and 7. Because of her limited availability, I.B. completed a few tasks only partially. Items were counterbalanced for the critical variables and were approximately matched in the tasks partially completed by I.B.

Despite our repeated attempts, I.B. failed to understand the instructions for the spoonerism task, in which proper names were to be repeated with swapped onsets (as in *John Kennedy* → *Kohn Jennedy*). This finding is of little surprise given the complexity of the task instructions and the severity of I.B.’s dementia. We discontinued the nonword triplet-repetition task with M.O. because of his frustration with consecutive failures. We are inclined to attribute these failures to M.O.’s severe verbal STM deficit. When he was assessed with the nonword triplet-repetition task, M.O.’s scaled digit-span score was 4. (Note that I.B.’s scaled score was higher: 6.) It is also likely that M.O.’s STM deficit affected his responses on the spoonerism task. In 12 of the

TABLE 6

Patients’ Accuracy in Phonological Tasks Involving Both Words and Nonwords

	M.O.		I.B.	
Task	Words	Nonwords	Words	Nonwords
Repetition	100%	97%	98%	97%
“I’m going to say a word/nonword; repeat it after me”	(120/120)	(116/120)	(93/94)	(70/72)
Syllable counting	97%	96%	86%	87%
“I’m going to say a word/nonword; tell me how many syllables it has”	(117/120)	(115/120)	(65/75)	(105/120)
Discrimination[a]	100%	100%	100%	100%
“I’m going to say two words/nonwords; tell me if they are the same or if they differ by one sound”	(60/60)	(80/80)	(60/60)	(80/80)
Phoneme identification[b]	97%	95%	98%	97%
“I’m going to say a word/nonword; does it have a __ sound?”	(116/120)	(114/120)	(73/75)	(73/75)
Initial-phoneme production[c]	100%	100%	100%	100%
“I’m going to say a word/nonword; tell me its first sound”	(50/50)	(50/50)	(25/25)	(25/25)
Final-phoneme production[c]	98%	100%	97%	100%
“I’m going to say a word/nonword; tell me its final sound”	(49/50)	(50/50)	(29/30)	(30/30)
Phoneme blending[d]	100%	92%	100%	90%
“I’m going to say the first sound and then the remaining sounds of a word/nonword; blend the two parts together and tell me the entire word/nonword”	(25/25)	(23/25)	(10/10)	(9/10)
Phoneme blending[d]	100%	96%	89%	89%
“I’m going to say the first part and then the last sound of a word/nonword; blend the two parts together and tell me the entire word/nonword”	(25/25)	(24/25)	(16/18)	(16/18)

Note. In some tasks, I.B. could be tested only with a subset of the items. Words and nonwords were one to four syllables long in the repetition and syllable-counting tasks; words of different length were shown an equal number of times. In the remaining tasks, items were monosyllabic. [a]Pairs of consonant-vowel-consonant words (e.g., *jog-fog*) and nonwords (e.g., *wep-weg*) were used. The paired words were different on 45 of 60 trials, and the paired nonwords were different on 60 of 80 trials. If the two words or nonwords on a trial differed, they differed by a single phoneme; this different phoneme appeared approximately equally often in the first, second, or third position. [b]The target phonemes were /b/, /k/, /r/, and /j/ and occurred in the initial, middle, and final positions of monosyllabic words and nonwords. The correct response was ‘‘no’’ on one third of the trials. [c]The target phonemes were consonants. [d]Different materials were used in the two phoneme-blending tests: first phoneme + rest of the word (e.g., *l* + *amp*) versus rest of the word + last phoneme (e.g., *was* + *p*). The responses were assembled words and nonwords such as *lamp* (*l* + *amp*) or *wasp* (*was* + *p*). Some of the items had complex, two-consonant onsets or codas.

TABLE 7

Patients' Accuracy in Phonological Tasks Involving Either Words or Nonwords

Task	M.O.	I.B.
Word-rhyme recognition[a]	100%	100%
"I'm going to say two words; tell me whether they rhyme or not"	(52/52)	(52/52)
Word-rhyme production	100%	95%
"I'm going to say a word; tell me a word that rhymes with it"	(65/65)	(62/65)
Initial-phoneme deletion[b]	95%	95%
"I'm going to say a word; take away the first sound and tell me what is left"	(95/100)	(95/100)
Final-phoneme deletion[b]	97%	94%
"I'm going to say a word; take away the final sound and tell me what is left"	(97/100)	(30/32)
Spoonerism[c]	98%	Failed
"I'm going to say a person's name; switch the initial letter of the first and the last name and say the resulting name aloud"	(125/128)	
Repetition of three nonwords[d]	Failed	98%
"I'm going to say three nonwords; repeat them in the same order"		(49/50)
Nonword completion[e]	98%	98%
"I'm going to say a nonword; I'll also show the written form of the nonword with one letter missing. Tell me what letter is needed to complete the written nonword"	(49/50)	(49/50)

Note. Except in the spoonerism task, all test items were monosyllabic.

[a] Rhyming and nonrhyming pairs occurred equally often. The final parts of words in rhyming pairs were not spelled identically (e.g., *port-court*). However, words in nonrhyming pairs could share some of their final letters (e.g., *pair-gain*). The materials were selected so as to prevent patients from responding on the basis of orthography. [b] An equal number of expected responses were familiar words (e.g., *cart* → ''art'') and nonwords (e.g., *sent* → *''ent''). [c] An example is *Ray Charles* → *Chay Rarles*. There were a total of 64 names; some were from Perin (1983), and others were names of American celebrities. We scored whether M.O. correctly swapped the two phonemes (hence the total $N = 128$). [d] Each triplet comprised three consonant-vowel-consonant nonwords (e.g., *vin dut bef*). [e] In this task, devised by Derousné and Beauvois (1985), the missing letter occurred in different positions of the monosyllabic words.

64 trials (19%), M.O. had problems repeating the final syllables of the second name, as when he responded "Pike Mia" (instead of *Pike Miazza*, for *Mike Piazza*) or "Zed Lezzlin" (instead of *Zed Leppelin*, for *Led Zeppelin*). Because the spoonerism task involves STM, and because the final syllables are stored longer than earlier syllables in STM, the final syllables are most likely to be affected by an STM deficit. However, M.O. always produced the first name correctly and did not have difficulty exchanging the word onsets.

Considering their limitations, it is even more remarkable that both patients scored, on average, higher than 95% correct in the various phonological tasks. Moreover, their performance was comparable for words and nonwords (for the individual tasks, $\chi s^2 < 1$). One could be concerned whether the nonperfect scores obtained in some tasks indicate impairment. To address this concern, we gathered control data for a few of the tasks in which the patients' scores were below 100%. Control subjects were tested only with nonwords and final phonemes, because we reasoned that these materials were potentially more taxing than words and initial phonemes. For each patient, we tested 2 control subjects matched for sex, age (± 5 years), and education. As can be seen in Table 8, the patients' scores were generally as good as those of

TABLE 8

Percentage Accuracy in Phonological Tasks: M.O. and I.B. Versus Their Respective Control Subjects

Test	M.O.	M.O.'s control subjects	I.B.	I.B.'s control subjects
Nonword tasks				
Repetition	97	96–100	97	97–100
Syllable counting	96	94–99	87	96–97
Phoneme identification	95	94–99	97	93–94
Final-phoneme production	100	100	100	90–93
Nonword completion	98	98–100	98	92–96
Phoneme blending				
Onset phoneme + rest of the word	92	96–100	90	88–92
Beginning of the word + final phoneme	96	96–100	89	89–96
Word tasks				
Word-rhyme production	100	100	95	88–97
Final-phoneme deletion	97	97–100	94	97

Note. Task materials and instructions are described in Tables 6 and 7. Each control group was tested with the same materials used for the corresponding patient.

the control subjects. The only exception was I.B.'s score in the syllable-counting task, which was slightly below the control subjects' norms (87% vs. 96%). I.B.'s score is in part accounted for by implausible responses such as "eight" and "nine syllables," which most probably reflect sporadic difficulties in following the task. Overall, M.O.'s and I.B.'s data for the phonological tasks do not indicate impairment. This conclusion is in line with the clinical observation that their speech was not punctuated by the phonological distortions frequently encountered in patients with acquired phonological deficits (Caplan, 1992).

CONCLUSIONS

M.O. and I.B., 2 patients with cognitive decline due to Alzheimer's disease, showed pure phonological dyslexia: They encountered severe problems in reading nonwords aloud despite a relatively spared ability to read familiar words aloud and to comprehend familiar words. Results on a number of phonological tests did not reveal a frank phonological impairment, and anomalies in the realization of word phonology were not detected in the patients' speech production. M.O. and I.B. provide another example of the dissociation between (impaired) nonword reading and (seemingly spared) phonological processing, a type of dissociation that has only rarely been documented in past studies. Our investigation of these 2 patients seems to escape some of the criticisms raised in response to previous studies. Concerns about accuracy of word reading, severity of impaired nonword reading, and effects of remediation programs that emphasize phonological awareness do not apply to our patients: Their word reading was extremely accurate across various types of words, their nonword reading was quite impaired, and they had not participated in any language rehabilitation programs. Nevertheless, we were unable to administer a handful of tasks because of the patients' reduced STM and restricted ability to understand and follow complex tasks. With this caveat in mind, one can conclude that phonological deficits were not detected in M.O. and I.B. This conclusion has critical implications for reading models and accounts of phonological dyslexia.

The results obtained with M.O. and I.B. can be readily accounted for by reading models that propose distinct mechanisms for familiar words and nonwords. The dual-route model (e.g., Berndt et al., 1996; Coltheart et al., 2001) is one example of a model of this kind. The selective nature of the patients' reading deficits can be explained if it is assumed that their brain damage affected only the nonlexical route, leaving the lexical route intact. Such a model also assumes that nonword reading and phonological encoding are supported by (partially) distinct mechanisms. If mechanisms for phonological encoding were spared, the model can also explain why performance in phonological tasks was unimpaired despite poor nonword reading. The dual-route model is also compatible with other features of our patients' data. For example, the tendency to produce words in response to nonword prompts would arise because patients resorted to using the intact lexical route. Moreover, if the nonlexical route was impaired, patients would be expected to respond more accurately to graphemes or graphemic clusters with a limited number of phonological realizations (e.g., typically consonants or high-GPC words) than to less univocal mappings, as we observed in M.O. and I.B.

More generally, the data of M.O. and I.B. suggest that phonological dyslexia is not a side effect of wide-ranging phonological deficits. Phonological deficits can accompany phonological dyslexia, but this is not necessary. In this respect, the data of M.O. and I.B. (and a few other patients) are at odds with the phonological-deficit hypothesis, which attributes difficulty in nonword reading to a phonological impairment whose impact extends to a variety of tasks, including reading. This hypothesis stemmed from theoretical perspectives as diverse as connectionism and evolutionary theory. Of course, one cannot rule out the existence of a phonological deficit in our patients with absolute certainty. If they have such a deficit, however, it must be very mild given that it was not detected by standard tests. So, at the very least, the question raised by the present data is how very mild phonological deficits lead to sizable nonword impairment. One also has to explain why, in other patients, nonword impairments of comparable severity are associated with sizable phonological deficits. In this respect, the comparison of M.O., I.B., and 3 of the patients reported by Berndt et al. (1996) is especially informative. Although all these patients performed similarly in nonword reading (accuracy range = 75–85% for high-GPC items and 10–55% for low-GPC items), only the patients of Berndt et al. were impaired in phoneme-blending, phoneme-deletion, and nonword-repetition tasks. Ultimately, if there is a way to reconcile the available data with the phonological hypothesis, it is by assuming that different phonological impairments can cause phonological dyslexia. Certain phonological impairments could yield more severe nonword reading deficits than others.

Finally, it remains to be explained how phonological deficits leave word reading unimpaired or have less severe effects on words than nonwords. This is a critical issue for models that do not incorporate mechanisms specifically involved in nonword reading. The computer simulations of Harm and Seidenberg's (1999) connectionist model remind us that as the severity of the phonological damage increases, the effects extend to irregular words. How a severe nonword deficit could coexist with spared word reading presents another challenge for models that subscribe to the phonological-impairment hypothesis. Computer simulations could be useful for exploring the effects of various types of phonological impairments; naturally, existing implementations of connectionist models (e.g., Harm & Seidenberg, 1999) are promising starting points for investigating this issue.

The specific reading deficits of M.O. and I.B. provide another example of how selective cognitive deficits can appear in dementia along with widespread memory and attention decay. The selective nature of such deficits invites the conclusion that, at least in certain brain areas, dementias can be associated with relatively narrow lesions. The lesions of our patients were in areas that are critical for nonword reading. It is significant that phonological dyslexia is almost invariably accompanied by phonological impairment in patients with more extensive brain lesions caused, for example, by strokes. The discrepancy between this pattern of deficits and the one exhibited by M.O. and I.B. perhaps reflects differences in the brain damage of these two patient groups. Nonword reading and phonology may be processed in contiguous brain regions that both tend to be lesioned by the relatively massive damage caused by strokes. We do not know how frequently relatively local lesions appear in degenerative diseases, but if this is a fairly common event, the investigation of other cases similar to M.O. and I.B. will lead to a better understanding of the causes of phonological dyslexia and will provide further data for evaluating reading models.

Acknowledgments—This work was supported by National Institute of Aging Grant T32AG00261. Michele Miozzo was supported by a grant from the Keck Foundation. We thank patients M.O. and I.B. and their families and Kristen Geiger for comments.

REFERENCES

Badecker, W., Hillis, A.E., & Caramazza, A. (1990). Lexical morphology and its role in the writing process: Evidence from a case of acquired dysgraphia. *Cognition*, *35*, 205–243.

Beauvois, M.-F., & Derousné, J. (1979). Phonological alexia: Three dissociations. *Journal of Neurology, Neurosurgery and Psychiatry*, *42*, 1115–1124.

Benton, A., & Hamsher, K. (1976). *Multilingual Aphasia Examination*. Iowa City: University of Iowa.

Benton, A.L. (1955). *The Visual Retention Test*. New York: Psychological Corp.

Berndt, R.S., Haendiges, A.N., Mitchum, C.C., & Wayland, S.C. (1996). An investigation of nonlexical reading impairments. *Cognitive Neuropsychology*, *13*, 763–801.

Bisiacchi, P.S., Cipolotti, L., & Denes, G. (1989). Impairment in processing meaningless verbal material in several modalities: The relationship between short-term memory and phonological skills. *Quarterly Journal of Experimental Psychology*, *41A*, 293–319.

Buschke, H., & Fuld, P.A. (1974). Evaluating storage, retention, and retrieval in disordered memory and learning. *Neurology*, *24*, 1019–1025.

Caccappolo-van Vliet, E., Miozzo, M., & Stern, Y. (in press). Phonological dyslexia without phonological impairment? *Cognitive Neuropsychology.*

Caplan, D. (1992). *Language: Structure, processing, and disorders*. Cambridge, MA: MIT Press.

Caramazza, A., Capasso, R., & Miceli, G. (1996). The role of the graphemic buffer in reading. *Cognitive Neuropsychology*, *13*, 673–698.

Coltheart, M. (1985). Cognitive neuropsychology and the study of reading. In M.I. Posner & O.S.M. Marin (Eds.), *Attention and performance: Vol. XI* (pp. 3–27). Hillsdale, NJ: Erlbaum.

Coltheart, M., Besner, D., Jonasson, J.-T., & Davelaar, E. (1979). Phonological encoding in the lexical decision task. *Quarterly Journal of Experimental Psychology*, *31*, 489–507.

Coltheart, M., Rastle, K., Perry, C., Ziegler, J., & Langdon, R. (2001). DRC: A dual route cascade model of visual word recognition and reading aloud. *Psychological Review*, *108*, 204–256.

Derousné, J., & Beauvois, M.-F. (1985). The "phonemic" state in the nonlexical reading process: Evidence from a case of phonological alexia. In K.E. Patterson, J.C. Marshall, & M. Coltheart (Eds.), *Surface dyslexia: Neuropsychological and cognitive studies of phonological reading* (pp. 399–457). London: Erlbaum.

Farah, M., Stowe, R.M., & Levinson, K.L. (1996). Phonological dyslexia: Loss of a reading-specific component of cognitive architecture? *Cognitive Neuropsychology*, *13*, 849–868.

Friedman, R.B. (1996). Phonological text alexia: Poor pseudoword reading plus difficulty reading functors and affixes in text. *Cognitive Neuropsychology*, *13*, 869–885.

Friedman, R.B., Ferguson, S., Robinson, S., & Sunderland, T. (1992). Dissociation of mechanisms of reading in Alzheimer's Disease. *Brain and Language*, *43*, 400–413.

Funnell, E. (1983). Phonological processing in reading: New evidence from acquired dyslexia. *British Journal of Psychology*, *74*, 159–180.

Glushko, R.J. (1979). The organization and activation of orthographic knowledge in reading aloud. *Journal of Experimental Psychology: Human Perception and Performance*, *5*, 674–691.

Goodglass, H. (1983). *The assessment of aphasia and related disorders* (2nd ed.). Philadelphia: Lea & Febiger.

Goodglass, H., & Kaplan, E. (1983). *Boston Diagnostic Aphasia Examination (BDAE)*. Philadelphia: Lea & Febiger.

Goodman, R., & Caramazza, A. (1986). *The Johns Hopkins Dyslexia Battery*. Baltimore: Johns Hopkins University.

Harm, M.W., & Seidenberg, M.S. (1999). Phonology, reading acquisition, and dyslexia: Insights from connectionist models. *Psychological Review*, *106*, 491–528.

Harm, M.W., & Seidenberg, M.S. (2001). Are there orthographic impairments in phonological dyslexia? *Cognitive Neuropsychology*, *18*, 71–92.

Kay, J., & Patterson, K.E. (1985). Routes to meaning in surface dyslexia. In K.E. Patterson, J.C. Marshall, & M. Coltheart (Eds.), *Surface dyslexia: Neuropsychological and cognitive studies of phonological reading* (pp. 79–104). London: Erlbaum.

Mattis, S. (1988). *Dementia Rating Scale (DRS)*. Odessa, FL: Psychological Assessment Resources.

Mayeux, R., Stern, Y., Rosen, J., & Leventhal, J. (1981). Depression, intellectual impairment, and Parkinson disease. *Neurology*, *31*, 645–650.

Patterson, K. (2000). Phonological alexia: The case of the singing detective. In E. Funnell (Ed.), *Case studies in the neuropsychology of reading* (pp. 57–83). Hove, England: Erlbaum.

Perin, D. (1983). Phonemic segmentation and spelling. *British Journal of Psychology*, *74*, 129–144.

Plaut, D.C., McClelland, J.L., Seidenberg, M.S., & Patterson, K. (1996). Understanding normal and impaired word reading: Computational principles in quasi-regular domains. *Psychological Review*, *103*, 56–115.

Rosen, W.G. (1981). *The Rosen Drawing Test*. Bronx, NY: Veterans Administration Medical Center.

Rosen, W.G. (1993, May). *Atypical response patterns on a target detection test: Signs of malingering?* Paper presented at the Ninth Annual Symposium in Forensic Psychology, Santa Fe, NM.

Seidenberg, M.S., & McClelland, J.L. (1989). A distributed, developmental model of word recognition and naming. *Psychological Review*, *96*, 523–568.

Shallice, T., & Warrington, E.K. (1980). Single and multiple component central dyslexic syndromes. In M. Coltheart, K.E. Patterson, & J.C. Marshall (Eds.), *Deep dyslexia* (pp. 119–145). London: Routledge.

Shallice, T., Warrington, E.K., & McCarthy, R. (1983). Reading without semantics. *Quarterly Journal of Experimental Psychology*, *35A*, 111–138.

Temple, C.M., & Marshall, J.C. (1983). A case study of developmental phonological dyslexia. *British Journal of Psychology*, *74*, 517–533.

Venezky, R.L. (1970). *The structure of English orthography*. The Hague, The Netherlands: Mouton.

Wechsler, D. (1981). *Wechsler Adult Intelligence Scale–Revised*. New York: Psychological Corp.

Woodcock, R.W., & Johnson, M.B. (1989). Woodcock-Johnson Psycho-Educational Battery–Revised. Allen, TX: DLM Teaching Resources.

(Received 7/11/03; Revision accepted 9/24/03)