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NEUROBEHAVIORAL GRAND ROUNDS

Temporal processing deficits in letter-by-letter reading

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Abstract

Theories of the cognitive impairment underlying letter-by-letter reading vary widely, including prelexical and lexical level deficits. One prominent prelexical account proposes that the disorder results from difficulty in processing multiple letters simultaneously. We investigated whether this deficit extends to letters presented in rapid temporal succession. A letter-by-letter reader, G.M., was administered a rapid serial visual presentation task that has been used widely to study the temporal processing characteristics of the normal visual system. Comparisons were made to a control group of 6 brain-damaged individuals without reading deficits. Two target letters were embedded at varying temporal positions in a stream of rapidly presented single digits. After each stream, the identities of the two letters were reported. G.M. required an extended period of time after he had processed one letter before he was able to reliably identify a second letter, relative to the controls. In addition, G.M.'s report of the second letter was most impaired when it immediately followed the first letter, a pattern not seen in the controls, indicating that G.M. had difficulty processing the two items together. These data suggest that a letter-by-letter reading strategy may be adopted to help compensate for a deficit in the temporal processing of letters (*JINS*, 2007, *13*, 110–119.)

Keywords: Acquired dyslexia, Pure alexia, Cerebrovascular accident, Visual perception, Reading, Case reports

INTRODUCTION

In the acquired dyslexia known as letter-by-letter reading, patients individually identify the letters of a word in order to pronounce it. Although the letters are sometimes spoken aloud, this strategy is usually inferred from an abnormally large increase in reading latency as the number of letters in a word increases, a finding known as the word length effect. Letter-by-letter reading is sometimes accompanied by surface dyslexia (e.g., Bowers et al., 1996; Friedman & Hadley, 1992; Patterson & Kay, 1982), and this combined syndrome has been termed letter-by-letter surface alexia (Friedman & Hadley, 1992) or type 2 letter-by-letter reading (Patterson & Kay, 1982).

Many different accounts of the cognitive deficit that causes letter-by-letter reading have been proposed. Some theories argue that the disorder arises from damage to early, prelexical systems that affects the visual processing of letters and that may not be specific to orthographic materials (e.g., Behrmann et al., 1998a; Behrmann & Shallice, 1995; Farah & Wallace, 1991; Friedman & Alexander, 1984; Kinsbourne & Warrington, 1962). Other theories have adopted the view that the disorder results from a more central, lexical-level deficit, such as in the orthographic system (Warrington & Langdon, 1994, 2002; Warrington & Shallice, 1980) or in accessing phonological codes (Arguin et al., 1998; Bowers et al., 1996). In each of these accounts, letter-by-letter reading develops as a strategy that somehow compensates for the underlying impairment.

Although it is possible that these different theories of letter-by-letter reading represent heterogeneity of the disorder, Behrmann et al. (1998b), in a comprehensive literature review, observed that a prelexical perceptual or letter processing impairment was evident in almost all cases. One of the most common manifestations of this impairment has been seen in the difficulty with rapid processing of multiple visual stimuli, a deficit also known as simultanagnosia (Farah & Wallace, 1991; Kinsbourne & Warrington, 1962; Levine & Calvanio, 1978). It is thought that this deficit prevents patients from processing the letters in words simulta-

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neously, and thus forces them to rely on the letter-by-letter strategy to recognize words.

In a now classic study, Kinsbourne and Warrington (1962) described the first experimental investigations of a prelexical visual deficit in letter-by-letter reading. In a series of tachistoscopic tasks, they demonstrated that identification thresholds for two items (letters, geometric forms, or objects) presented simultaneously or in rapid temporal sequence were increased in 4 letter-by-letter readers relative to normal and brain-damaged controls. They concluded that the underlying problem in letter-by-letter reading was an increased "refractory period" between the processing of multiple visual stimuli. Although this study was the first to establish an association between prelexical visual deficits and letter-by-letter reading, it provided little detailed theory concerning the specific nature of the visual deficit, as well as its relationship to the reading disorder.

More recently, Behrmann and Shallice (1995) also suggested that a letter-by-letter reader was impaired at serial identification of two letters presented in rapid temporal sequence. To assess this ability, they used a rapid serial visual presentation (RSVP) paradigm in which items were displayed individually in rapid succession in the same spatial location. Two letters were embedded at varying positions in a stream of digits. After each item stream was presented, the subject was required to report the identities of the two letters. Behrmann and Shallice found that their letter-by-letter reader was often able to report the identity of the first letter, but that she required more time (i.e., more intervening digits) than the normal control subject to identify the second letter as well. Specifically, when the stimulus onset asynchrony (SOA) between the two letters was 100 ms or 400 ms, the letter-by-letter reader was worse than the control subject at reporting the second item; however, when the SOA was increased to 800 ms, their performances did not differ.

RSVP paradigms have been used extensively for studying temporal processing in the normal visual system. Individuals without neurological damage also show a transitory processing decrease in which identification of the second of two targets is reduced if it is presented within approximately 400 ms of the first (Duncan et al., 1994; Raymond et al., 1992; Shapiro et al., 1994). This phenomenon is known widely as the "attentional blink" (Raymond et al., 1992). In some RSVP tasks, however, a U-shaped trend is observed (see Visser et al., 1999, for review), in which accuracy of report of the second target (T2) is relatively high when it immediately follows the first target (T1), decreases substantially when it is the second or third item following T1, and then improves in the later item positions. This pattern has been referred to as "lag-1 sparing" (with lag-1 referring to a T2 item immediately following T1; Potter et al., 1998). This sparing is thought to occur when temporally contiguous items (i.e., T1 and T2) with similar processing requirements (e.g., determination of letter identity) enter the visual short-term memory system together, before the closing of an attentional gate (Visser et al., 1999).

Given that the version of the RSVP task used by Behrmann and Shallice (1995) included only three widely spaced SOA conditions (i.e., 100, 400, 800 ms) and only 1 normal control subject was tested, their conclusion that the letter-by-letter reader was uniquely impaired on this task may not be fully warranted; a processing decrease up to the 400-ms SOA would be expected even in normal individuals based on previous attentional blink results. As well, several neurological patient groups without reading deficits have shown attentional blinks that were protracted in duration (Shapiro et al., 2000), suggesting that reduced performance on this task cannot be assumed to be uniquely associated with letter-by-letter reading unless brain-damaged subjects without reading deficits are tested as controls. In addition, the task methodology used by Behrmann and Shallice did not allow for full examination of the RSVP phenomena (i.e., both attentional blink and lag-1 sparing). In fact, lag-1 sparing has not been examined previously in cases of dyslexia or in most other neurological conditions (Shapiro et al., 2000). Examination of this phenomenon in letter-by-letter reading may be particularly relevant to our understanding of this condition given that lag-1 sparing is thought to occur when individuals are able to process rapidly presented stimuli together (Visser et al., 1999), an ability suggested to be deficient in letter-by-letter reading (Farah & Wallace, 1991; Kinsbourne & Warrington, 1962; Levine & Calvanio, 1978).

The purpose of this study was to examine temporal processing phenomena in letter-by-letter reading as measured by the size of the attentional blink and associated lag-1 sparing. We administered an RSVP task known to produce both attentional blink and lag-1 sparing effects in normals (Chun & Potter, 1995; Visser et al., 1999) to a patient with letter-by-letter surface alexia. Comparisons were made to a group of 6 brain-damaged individuals without reading deficits to control for generalized effects of brain damage on processing speed. This methodology allows for impaired measures of performance in the experimental task to be more confidently assumed to be uniquely associated with the reading deficit, rather than due to other nonspecific effects of brain damage. It was expected that a temporal processing deficit would be manifested by increased attentional blink duration and/or the absence of lag-1 sparing.

SUBJECTS

Patient G.M.

G.M. was a 37-year-old man who presented at hospital with headache, confusion, blurred vision, and dysphasia in June of 1992. A computed tomography report indicated that G.M. had a 4-cm hyperdense lesion in the temporal–occipital region of the left cerebral hemisphere in keeping with a cerebral hemorrhage. Angiography found that the source of the bleeding was a ruptured arteriovenous malformation and incidentally revealed a left basilar tip aneurysm. He underwent neurosurgery for excision of the arteriovenous

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malformation in November of 1992, and for elective clipping of the basilar tip aneurysm in September of 1994.

Following his cerebral hemorrhage, G.M. had a moderate fluent aphasia for which he received speech-language therapy. He also had an upper-right quadrant visual field defect that had since resolved according to perimetry testing. He returned to full-time work as a foreman at an industrial plant in the fall of 1993. G.M. reported that he was completely unable to recognize words or letters for several months after his brain injury. His ability to recognize letters gradually improved, and he developed a letter-by-letter strategy (naming the letters aloud) to identify words. Over time, he became able to read words without explicitly naming each letter, although he reported that he continues to use this strategy silently. Even with the use of this strategy, he had difficulty naming some words accurately.

G.M. is right-handed and a native speaker of English. He has a grade 10 education and reported a normal rate of achievement in school. Although he was not an avid reader premorbidly, he denied any difficulties with reading and had read newspapers and magazines regularly.

G.M. was 42 years old when the current data were collected. His speech was fluent, but he displayed occasional word-finding problems in spontaneous speech. He appeared to have no difficulty with oral comprehension.

Control Subjects

Six individuals who had previously experienced a stroke served as controls in the current study. Demographic and neurological information is provided in Table 1. Subjects were approximately matched to G.M. in age and education. Only Subject 3 had returned to his premorbid occupation; the other control subjects had stroke-related disabilities that prevented them from working outside the home. Subjects varied considerably in the location of their brain lesions. All subjects had normal or corrected to normal visual acuity. None of the subjects had visual field defects. All subjects were native speakers of English, with the exception of Subject 3 who was raised by French-speaking parents but was schooled entirely in English and spoke English exclusively outside his original family home. All subjects gave informed consent before inclusion in this study. The study was conducted in accordance with standards of the university and hospital ethics committees and the Helsinki Declaration.

Neuropsychological Assessment

G.M. and the control subjects were administered the Neurobehavioral Cognitive Status Examination (COGNISTAT; Kiernan et al., 1987) and the reading subtest of the Wide Range Achievement Test (WRAT-3; Wilkinson, 1993) in order to obtain a general indication of their current levels of cognitive functioning and screen for reading difficulties. Test results are provided in Table 2. G.M. obtained perfect scores on all subtests except the naming subtest, which fell in the impaired range. In the control subjects, the domains and severity of cognitive impairment varied, with Subjects 2 and 4 scoring in the severely impaired range on the memory subtest. G.M.'s accuracy of reading single words orally on the WRAT-3 was impaired, and his score was at the 3rd percentile, equivalent to a grade 5 reading level. In contrast, the control subject reading scores all fell within the average range.

Table 1. Characteristics of control subjects

Subject	Age	Education	Gender	Premorbid handedness	Premorbid occupation	Etiology	CT or MRI scan results	Time post-stroke
1	58	Grade 11	M	R	School custodian	Infarction	Right internal capsule lesion, extending into basal ganglia	9 months
2	51	Grade 11	M	R	Insurance underwriter	Infarction	Right medial frontal cortex lesion, as well as old ischemic changes in left frontal cortex	1 year, 7 months
3	50	Grade 11	M	R	Hair dresser	Infarction	Left putamen and external capsule lesion	9 months
4	37	Grade 12	M	R	Office clerk	Infarction	Left frontal-temporal lesion, as well as old ischemic lesions in right subcortical areas	2 years, 4 months
5	48	Grade 12	M	R	Mechanic	Infarction	Left medulla infarct	11 months
6	42	Grade 10	F	R	Postal clerk	Hemorrhage and aneurysm clipping	Right frontal-parietal lesion	5 years, 2 months

Note. CT = computed tomography; MRI = magnetic resonance imaging.

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Table 2. Performance on cognitive screening measures for G.M. and control subjects

		COGNISTAT				
Subject	WRAT-3 (%ile, Gr level)	Orientation (Max = 12)	Attention (Max = 8)	Comprehension (Max = 6)	Repetition (Max = 12)	Naming (Max = 8)
G.M.	3, Gr 5	12 (Average)	8 (Average)	6 (Average)	12 (Average)	6 (Mild)
1	34, HS	12 (Average)	8 (Average)	6 (Average)	11 (Average)	8 (Average)
2	63, Post-HS	11 (Average)	2 (Mod-Sev)	6 (Average)	12 (Average)	8 (Average)
3	32, HS	12 (Average)	8 (Average)	5 (Average)	7 (Moderate)	5 (Mild)
4	55, Post-HS	12 (Average)	7 (Average)	6 (Average)	12 (Average)	8 (Average)
5	45, Post-HS	12 (Average)	8 (Average)	6 (Average)	12 (Average)	8 (Average)
6	32, HS	12 (Average)	8 (Average)	6 (Average)	12 (Average)	7 (Average)

	COGNISTAT				
Subject	Construction (Max = 5)	Memory (Max = 12)	Calculation (Max = 4)	Similarities (Max = 6)	Judgment (Max = 5)
G.M.	5 (Average)	12 (Average)	4 (Average)	6 (Average)	5 (Average)
1	5 (Average)	11 (Average)	2 (Mild)	6 (Average)	5 (Average)
2	5 (Average)	1 (Severe)	4 (Average)	6 (Average)	5 (Average)
3	5 (Average)	11 (Average)	4 (Average)	6 (Average)	5 (Average)
4	5 (Average)	0 (Severe)	4 (Average)	6 (Average)	5 (Average)
5	5 (Average)	12 (Average)	4 (Average)	6 (Average)	5 (Average)
6	5 (Average)	12 (Average)	4 (Average)	6 (Average)	5 (Average)

Note. WRAT-3 = Wide Range Achievement Test; Gr = grade; HS = high school; Max = Maximum score.

G.M. was administered several additional tests to further document his cognitive abilities. He showed anomia on the Boston Naming Test (Kaplan et al., 1983) on which he correctly named only 34 of 60 objects without cueing ($T = 16$; Heaton et al., 1999), although he named an additional 16 items with phonetic cues. He was able to provide accurate verbal descriptions of all objects that he was unable to name (e.g., igloo: "made of snow, Eskimo live in it"; abacus: "used for counting in ancient times"), indicating that he recognized the objects perceptually and had access to related semantic information.

Writing was assessed with Subtest 35 from the Psycholinguistic Assessment for Language Processing in Aphasia (PALPA; Kay et al., 1992). G.M. correctly spelled to dictation 20 of 30 regular words and 2 of 30 exception words, characteristic of surface dysgraphia. His errors consisted primarily of regularizations (e.g., colonel: "curnel"); homophone confusions (e.g., quay: "key"); visually similar letter substitutions (e.g., flannel: "blanel"); phonetically inappropriate letter insertions, particularly with vowels (e.g., pump: "poump"); and phonetically legitimate alternative spellings (e.g., effort: "efert").

On letter identification tests, G.M. obtained perfect scores in naming upper-case and lower-case letters (PALPA subtest 22), as well as in matching upper-case and lower-case forms (subtest 19). G.M. also performed well on the Visual Object and Space Perception Battery (Warrington & James, 1991), indicating that he did not have any obvious visuoperceptual impairments. He obtained nearly perfect scores on the shape screening, incomplete letters, dot counting, position discrimination, cube analysis, object decision, and number location subtests, and his scores on the silhouette (18/30) and progressive silhouette (11/20) subtests were lower, but fell above the 5th percentile cutoff scores for normal performance.

Reading Assessment

A detailed reading assessment was conducted to specify the nature of G.M.'s dyslexia. We presented 240 individual words in lower case font using a Macintosh Powerbook with SuperLab software. The following variables were orthogonally crossed to create 24 conditions (each with 10 words): word length (4, 5, 6, and 7 letters); frequency (high: > 100; medium: 20–99; low: < 20; Kucera & Francis, 1967); and regularity (regular, irregular). Specific frequency counts were matched across the length and regularity conditions within each frequency band, and the word length conditions were matched on regularity. Words were presented in random order. G.M. and control subjects were asked to read each word aloud as quickly and accurately as possible. Reaction time (RT) were measured by an Apple microphone and voice key from the onset of each word.

As can be seen in Figure 1, G.M.'s reading latencies were very slow and dramatically increased as word length increased. A regression line plotted with RT against word length revealed a linear fit ($r = .94$; $p < .06$) with a slope of 1230 ms/letter. Interestingly, the slope of the word length

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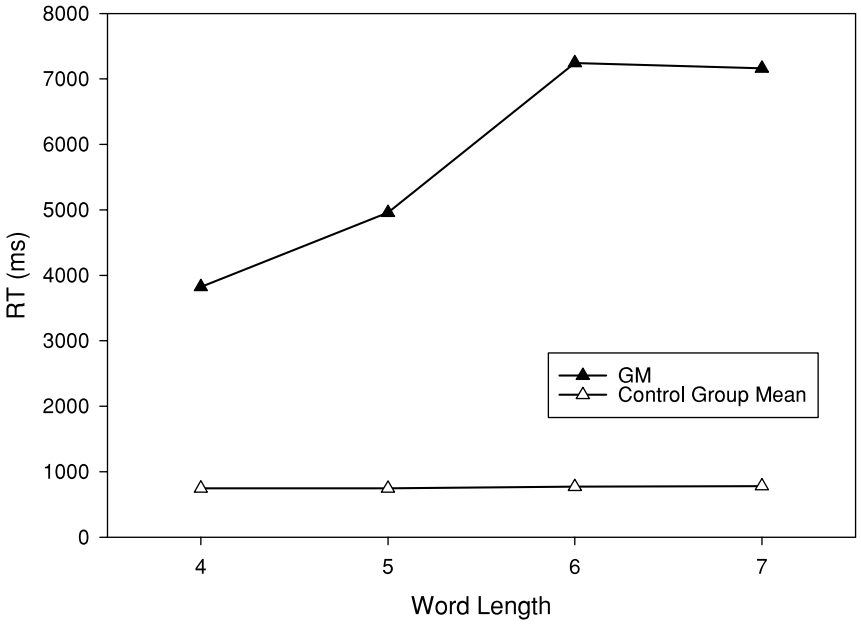

Fig. 1. Mean pronunciation reaction times (RTs) for correct trials as a function of word length for G.M. and the control group.

function levels off for 7-letter words, suggesting that G.M. may have been able to guess their identities without processing the final letter (Farah, 1999). This word length effect is the defining characteristic of letter-by-letter reading. In contrast, the controls showed only minimal effects of word length.

This description of the data was supported by statistical analyses. The latency data for G.M. and the control subjects were analyzed with three-way analyses of variance (ANOVAs) with length (4, 5, 6, and 7 letters), frequency (high, medium, and low), and regularity (regular and irregular) as factors. The analysis of G.M.'s data used individual items as independent samples. The analysis of the control data used individual subject means. Trials in which reading errors were made, the voice key was triggered erroneously (G.M.: $<$ 1%; controls: $<$ 3% of trials), or the RT was greater than 2 *SD* from the mean of a particular condition for a subject (G.M. and controls: $<$ 4% of trials) were excluded from the RT analyses. G.M.'s pronunciation latencies increased as word length increased [$F(3,148) = 9.59$; $p < .001$]. He was also faster at naming regular words (4789 ms) than irregular words [7260 ms; $F(1,148) = 17.44$; $p < .001$]. The pronunciation latencies of the control subjects were not significantly affected by length [$F(3,30) = 2.41$; $p = .11$], and the slopes of their word length functions (individual slopes ranged from 8 ms/letter to 23 ms/letter, mean = 12.6 ms/letter) were consistent with those found previously in normal readers (Butler & Hains, 1979; Henderson, 1982). The controls were faster at naming words of high frequency (726 ms) and medium frequency (740 ms) compared with low frequency words [817 ms; $F(2,30) = 5.90$; $p = .02$].

As can be seen in Figure 2, the accuracy of G.M.'s reading was strongly affected by the regularity of the words; he had much more difficulty reading irregular words (62% correct) than regular words [90% correct; $\chi^2(1) = 25.06$; $p < .001$]. This impaired reading of irregular words is diagnostic of surface dyslexia. Although frequency did not significantly affect the reading accuracy of either regular words [$\chi^2(2) = 1.67$; $p = .44$] or irregular words [$\chi^2(2) = 2.56$; $p = .28$], his performance was worst with the low frequency irregular items, consistent with that reported in some other surface dyslexics (e.g., Behrmann & Bub, 1992; Patterson & Hodges, 1992). The accuracy of G.M.'s reading was not affected by word length [75%, 77%, 78%, and 75% for 4-, 5-, 6-, and 7-letter words, respectively; $\chi^2(3) = .25$; $p = .97$]. Control subjects were highly accurate in word pronunciation (97% correct overall). Accuracy was greater than 93% in all regularity by frequency word groupings, with the least accurate control subject obtaining greater than 92% correct.

G.M.'s reading errors were also in keeping with a diagnosis of surface dyslexia, and included regularizations (e.g., racquet: "rack-qu-et"), visual–phonological word errors (e.g., decade: "decent"), and visual–phonological nonword errors (e.g., machine: "mayhin"). Thus, G.M.'s overall pattern of reading was indicative of letter-by-letter surface alexia or type 2 letter-by-letter reading.

IDENTIFICATION OF LETTERS IN RAPID SERIAL VISUAL PRESENTATION

Method

The experiment was run on a Macintosh Powerbook computer using custom software (Raymond et al., 1992). Each trial consisted of a series of successively presented digits in which two upper case letters were embedded as targets. The stimuli that were presented on a given trial were randomly selected from sets of eight single digits (0 and 1 excluded)

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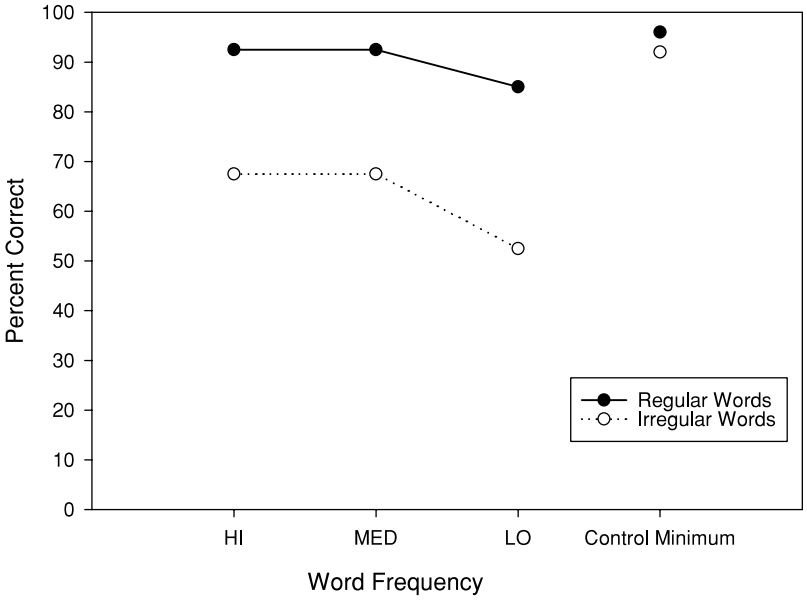

Fig. 2. G.M.'s pronunciation accuracy as a function of regularity and frequency, as well as data for the least accurate control subject.

and 24 letters (O and I excluded). The number of digits presented before the first target letter (T1) varied randomly between 7 and 15. T1 was always followed by a sequence of 10 items, and the second target letter (T2) appeared an equal number of times at each of these 10 serial positions. Each item was presented for 100 ms, with an interstimulus interval of 67 ms (presentation rate = 6 items/s). Thus, the SOA between T1 and T2 ranged between 167 and 1670 ms. Subjects initiated each trial by pressing a computer mouse button. The mouse press caused a central fixation point to disappear and the stream of stimuli to be displayed. Subjects were instructed to report the two letters aloud at the end of the trial. After the experimenter entered the responses into the computer, the fixation point returned and subjects could initiate the next trial when ready. G.M. completed 30 trials at each of the 10 intervals between T1 and T2 (for a total of 300 trials). Since the controls were available for a limited time for testing, only 20 trials at each of the 10 intervals were administered (for a total of 200 trials). The trials were presented in random order. Twenty-four practice trials were administered before data collection.

Results

Following typical RSVP procedure, our analyses included only those trials in which T1 was correctly reported to ensure that subjects were attending to the trial. T1 accuracy as a function of SOA is provided in Table 3. Temporal processing performance was then assessed using the percentage of trials in which T2 was correctly reported given that T1 was reported (T2IT1) as a function of SOA. These data are plotted for G.M. and controls in Figure 3.

A χ^2 analysis of G.M.'s data showed that the accuracy of T2IT1 was dependent on SOA ($\chi^2(9) = 85.67$; $p < .001$). Pairwise comparisons between the longest SOA (1670 ms) and each of the other nine SOA conditions revealed that accuracy at the 1670-ms SOA was significantly greater than accuracy at all SOA conditions from 167 ms to 668 ms ($p < .003$ for all comparisons). Performance at the 835-ms SOA and longer did not differ significantly from performance at the 1670-ms SOA condition ($p > .2$ for all comparisons). Although this statistical analysis suggests that the function reached a maximum asymptote at the 835-ms SOA, the function does not appear to asymptote until the 1169-ms SOA based upon visual inspection.

In the analyses of grouped control data, a one-way repeated measures ANOVA on the conditional report of T2IT1 showed a significant effect of SOA [$F(9,45) = 21.32$; $p < .001$]. Pairwise comparisons of performance between the 1670-ms SOA and each of the other nine SOA conditions showed that the T2IT1 percentages were significantly lower from 167 ms to 501 ms. No comparisons were significant at longer

Table 3. Mean percent correct for T1 as a function of SOA for G.M. and control subjects

	T1 % Correct	
SOA (ms)	G.M.	Controls
167	65.5	79.3
334	75.0	94.8
501	89.3	94.7
668	89.7	88.8
835	80.0	96.6
1002	89.3	94.8
1169	77.8	96.4
1336	68.9	95.0
1503	81.5	94.9
1670	75.0	94.9

Note. SOA = stimulus onset asynchrony.

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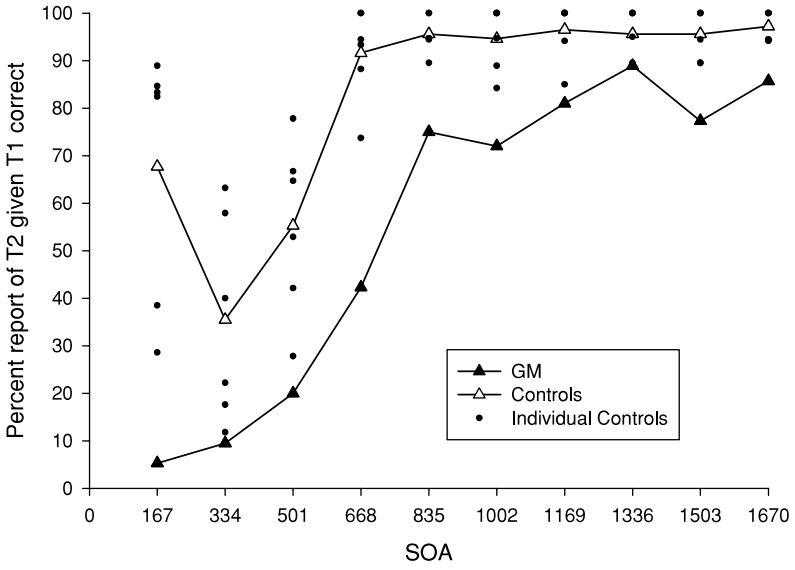

Fig. 3. Percentage of rapid serial visual presentation (RSVP) trials in which the second letter (T2) was correctly reported, given that the first letter (T1) was correctly reported, as a function of increasing stimulus onset asynchrony (SOA) for G.M. and controls (group mean and individual subjects).

SOAs, suggesting that, by the 668-ms SOA, the function had increased to reach an asymptote. In addition, T2IT1 accuracy was significantly higher at the 167-ms SOA compared with the 334-ms SOA ($p < .01$). Although the accuracy of letter report at each SOA was highly variable among subjects, the performance of every individual in the control group conformed to the overall pattern of results described above. That is, their performances reached an asymptote at the 668-ms SOA and T2IT1 accuracy was greater in the 167-ms SOA than in the 334-ms SOA condition (difference in T2IT1 accuracy between 167-ms and 334-ms SOA ranged from 16 to 66%).

DISCUSSION

We tested the hypothesis that letter-by-letter reading is associated with difficulty in rapid temporal processing of multiple letters using an RSVP task. Both G.M. and the control subjects had difficulty reporting the identity of T2 when it was presented in close temporal proximity to T1. In the normal literature, this transitory processing deficit is referred to as the "attentional blink" (Duncan et al., 1994; Raymond et al., 1992; Shapiro et al., 1994). G.M.'s performance improved progressively as the interval between T1 and T2 increased until it reached a maximal level of accuracy at about the 1169-ms SOA. In contrast, T2 reached a maximal asymptote by the 668-ms condition in each of the 6 control subjects. This difference indicates that G.M.'s attentional blink is protracted in duration in that he required an extended period of time after he had encoded the first letter before he was able to reliably identify the second letter, relative to the brain-damaged control group, as well as to normal subjects previously tested on this type of RSVP task (Chun & Potter, 1995).

The second important aspect of these results is that G.M. was most likely to miss T2 when it immediately followed T1 (167-ms SOA), and his performance generally improved as SOA increased. This pattern is distinct from that seen in the controls in which T2 accuracy was relatively high when it immediately followed T1 (167-ms SOA), decreased when T2 was the second (334-ms SOA) or third (501-ms SOA) item following T1, and then improved in the later item positions. This U-shaped function, referred to as "lag-1 sparing" (Potter et al., 1998), was seen in each of the 6 control subjects but was absent in G.M.

The final aspect of the results to note is that G.M.'s ability to identify rapidly presented letters, regardless of the interval between them, was worse than the controls. G.M.'s report of T1 and T2 at all SOA conditions fell below that of the least accurate control subject.

Several different theories have been developed to explain the underlying mechanism of the attentional blink. According to one theory, known as the interference model, the attentional blink results from interference between the target items in visual short-term memory when they need to be retrieved at short intertarget intervals (Raymond et al., 1995; Shapiro et al., 1994; Shapiro & Raymond, 1994). Alternatively, a second theory, known as the two-stage model, proposes that after targets are detected (stage 1), they enter a limited capacity stage required for identification (stage 2). The attentional blink is due to a processing bottleneck in that, at short intertarget intervals, stage 2 resources are busy processing T1 when T2 is presented. As a result, T2 is likely to decay in stage 1 as it awaits access to stage 2 (Chun & Potter, 1995).

According to these models, G.M.'s prolonged attentional blink could be due to increased interference between targets in visual short-term memory (i.e., based on an interference model) or disturbed processing of targets in stage 2 (i.e., based on a two-stage model). The overall pattern of results, however, may be more consistent with the two-stage model. In this model, the duration of stage 2 processing increases as the difficulty of target processing increases. Thus, increasing target processing difficulty in normals, by

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visual masking of T1, results in a greater attentional blink (Giesbrecht & Di Lollo, 1998; Seiffert & Di Lollo, 1997). If G.M. had difficulty with the perceptual encoding of letters, as is suggested by his overall lower report of T1 and T2 at all SOA conditions relative to controls, then his stage 2 processing of T1 would be slowed and T2 would likely be lost from stage 1 even at longer intertarget intervals. Thus, G.M.'s increased attentional blink and overall lower report of T1 and T2 at all SOA conditions is consistent with a deficit in the perceptual encoding of letters. Our account of G.M.'s protracted attentional blink also appears compatible with the proposal that the attentional blink in normals reflects a limitation in creating the T2 representation, rather than in consolidating the T1 representation (Raymond, 2003).

G.M. also differed from the control subjects in terms of his lack of lag-1 sparing. In both the interference model and the two-stage model, lag-1 sparing is thought to occur when temporally contiguous items enter the visual short-term memory system together, before the closing of an attentional gate (Chun & Potter, 1995; Shapiro & Raymond, 1994; Visser et al., 1999). If T2 immediately follows T1 in the RSVP stream (i.e., lag-1 condition), both targets can be reported and the attentional blink is prevented. Thus G.M.'s failure to show lag-1 sparing suggests that his visual short-term memory is limited in capacity such that he is only able to process one item at a time. The perceptual encoding deficit that is hypothesized to underlie G.M.'s prolonged attentional blink is unlikely to cause his lack of lag-1 sparing, given that visual masking of T1 in normals produces a greater attentional blink but does not affect lag-1 sparing (Seiffert & Di Lollo, 1997). These data suggest that independent processes underlie the production of the attentional blink and lag-1 sparing and that both of these processes (i.e., perceptual encoding and visual short-term memory) are impaired in G.M.

Therefore, G.M. appears to have deficits in perceptual encoding and visual short-term memory that impair his temporal processing abilities. It is not surprising that G.M. was impaired on multiple components of the RSVP task, given the large size of his brain lesion. It is important to note though that G.M. had no difficulty on unspeeded tests of letter identification, suggesting that his deficits are only observable in more challenging tasks, such as RSVP, that demand the rapid letter processing skills intrinsic to reading. Further research is clearly required to determine whether G.M.'s RSVP deficits are observable with numbers and symbols or are specific to letters.

Deficits in perceptual encoding and/or in visual short-term memory could potentially underlie G.M.'s letter-by-letter reading. A perceptual encoding deficit might challenge G.M.'s ability to process the multiple letters in words, requiring him to compensate by focusing his resources serially on the individual constituent letters. A visual short-term memory deficit that limited G.M. to processing only one item at a time could also prevent him from processing the letters in words simultaneously and restrict him to using a letter-by-letter procedure. This specific type of deficit, demonstrated in G.M. by the absence of lag-1 sparing, has not been associated previously with letter-by-letter reading. Lag-1 sparing, in fact, has been examined in few neurological conditions (Shapiro et al., 2000). Thus, our results suggest that measurement of this RSVP phenomenon may represent an easily interpretable and novel approach for assessing the capacity of visual short-term memory.

Our data cannot determine whether one or both of these hypothesized deficits (i.e., in perceptual encoding or visual short-term memory) are responsible for G.M.'s letter-by-letter reading. Our comparison to a brain-damaged control group, however, allows us to more confidently associate these RSVP deficits with his dyslexia, rather than to other nonspecific effects of brain damage. Thus, these data highlight the value of comparing data from single cases to this type of control group, an approach not commonly used in previous investigations of acquired dyslexia.

This account of G.M.'s dyslexia is consistent with many previous reports suggesting that letter-by-letter reading results from a perceptual or letter processing impairment (Behrmann et al., 1998b). It is important to note, however, that a prelexical deficit does not necessarily underlie all cases of letter-by-letter reading. For example, Warrington & Langdon's (1994, 2002) letter-by-letter reader was unimpaired on tasks of rapid letter identification and concluded to have a lexical-level deficit. Moreover, Hanley and Kay (1996) found that the variation in letter processing abilities in 2 letter-by-letter readers did not explain their variation in reading speed. They suggested that deficient letter processing was not a complete explanation of the disorder and an additional lexical-level deficit was present in 1 of their cases. It seems most likely that letter-by-letter reading represents a general strategy adopted to compensate for a variety of difficulties in reading-dependent processes. In fact, children learning to read also show significant word length effects (LaBerge & Samuals, 1974; Seymour & Porpodas, 1980). The nature of the cognitive deficit underlying the disorder presumably relates to the area of neural damage. For example, G.M.'s temporal–occipital lesion is consistent with damage to a visual short-term memory system for letters involving links between visual association cortices supporting orthographic representations and temporal cortices supporting phonological processes (Plaut et al., 1996).

In conclusion, the results of this study suggest that deficits in perceptual encoding and/or visual short-term memory are responsible for the temporal processing impairments of a letter-by-letter reader and may underlie the dyslexia in this particular case. Our study improves on previous methodology to allow the specific components of temporal processing that may be impaired in letter-by-letter reading to be teased apart and, given our comparison to a brain-damaged control group, a specific association to be made between the temporal processing and the reading deficit. Further work is required to clearly delineate the different variants of letter-by-letter reading and their anatomical correlates.

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