Dyscravia: Voicing substitution dysgraphia

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We report a new type of dysgraphia, which we term *dyscravia*. The main error type in dyscravia is substitution of the target letter with a letter that differs only with respect to the voicing feature, such as writing "coat" for "goat", and "vagd" for "fact". Two Hebrew-speaking individuals with acquired dyscravia are reported, TG, a man aged 31, and BG, a woman aged 66. Both had surface dysgraphia in addition to their dyscravia. To describe dyscravia in detail, and to explore the rate and types of errors made in spelling, we administered tests of writing to dictation, written naming, and oral spelling. In writing to dictation, TG made voicing errors on 38% of the words, and BG made 17% voicing errors. Voicing errors also occurred in nonword writing (43% for TG, 56% for BG). The writing performance and the variables that influenced the participants' spelling, as well as the results of the auditory discrimination and repetition tasks indicated that their dyscravia did not result from a deficit in auditory processing, the graphemic buffer, the phonological output lexicon, the phonological output buffer, or the allographic stage. The locus of the deficit is the phoneme-to-grapheme conversion, in a function specialized in the conversion of phonemes' voicing feature into graphemes. Because these participants had surface dysgraphia and were forced to write via the sublexical route, the deficit in voicing was evident in their writing of both words and nonwords. We further examined whether the participants also evinced parallel errors in reading. TG had a selective voicing deficit in writing, and did not show any voicing errors in reading, whereas BG had voicing errors also in the reading of nonwords (i.e., she had dyslegzia in addition to dyscravia). The dissociation TG demonstrated indicated that the voicing feature conversion is separate for reading and writing, and can be impaired selectively in writing. BG's dyslegzia indicates that the grapheme-to-phoneme conversion also includes a function that is sensitive to phonological features such as voicing. Thus the main conclusion of this study is that a separate function of voicing feature conversion exists in the phoneme-to-grapheme conversion route, which may be selectively impaired without deficits in other functions of the conversion route, and without a parallel deficit in reading.

1. INTRODUCTION

Thiz baber brezents a new dybe ov dyscravia. This is the way the participants in this study might write "this paper presents a new type of dysgraphia". The predominant error in this dysgraphia is voicing error: the substitution of a voiced letter with a voiceless one (writing *p* instead of *b*), and of a voiceless letter with a voiced one (writing *b* instead of *p*). The main challenge of this study is to find the location of the deficit in the spelling process that leads to this type of error, and to describe the nature of this newly identified spelling impairment.

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Before describing the possible impairment loci which cause various dysgraphias, we start with a short introduction of the model we assume for writing, presented in Figure 1.

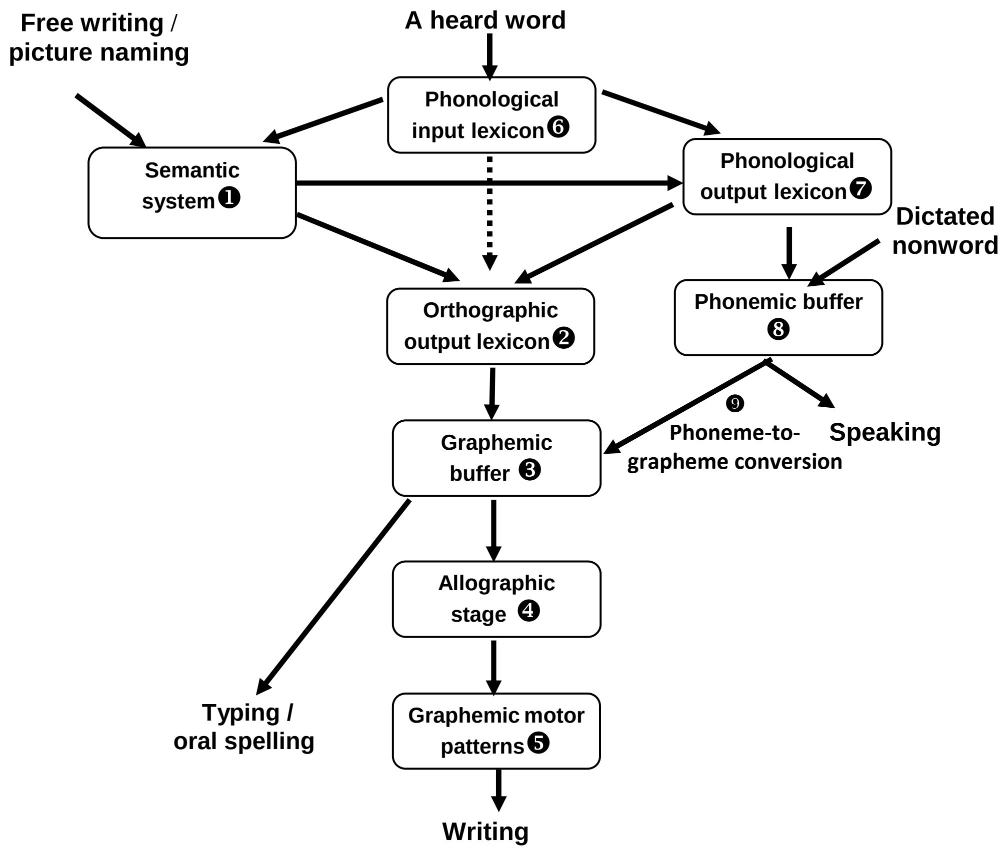

Figure 1. Single word writing model

To write a word, in free writing or to dictation, two routes are available – a lexical route and a sublexical route. The most efficient route is the lexical route, which uses the orthographic output lexicon (marked (2) in Figure 1), in which orthographic representations of words are stored and activated. This lexicon can be accessed either from the semantic system (1), in free writing, or from the phonological input lexicon (6) (possibly via the phonological output lexicon (7)), in writing to dictation. The graphemes selected from the lexicon are held for a short time in the graphemic buffer (3), a graphemic short term memory store, and are then transmitted to the allographic stage (4), where the abstract letter forms are stored. The final stage is the motor execution of writing, namely, the graphemic motor pattern stage (5), in which the specific motor patterns for the specific letters are activated in order to perform the hand movements required for writing. Only items that exist in the orthographic output lexicon can be written via this lexical route, and hence nonwords and new words cannot be written through this route.

The other route, the sublexical route, passes through the phonemic buffer (8) directly to the graphemic output buffer (3) using phoneme-to-grapheme conversion (9). The products of the phoneme-to-grapheme conversion, like those of the lexical route, are maintained in the graphemic output buffer. This sublexical route is used mainly for writing new words and nonwords – letter sequences that do not exist in the orthographic lexicon. It is also used when the lexical route is impaired, as is the case in surface dysgraphia. Words that have more than a single possible conversion from phonemes to graphemes, and words that do not obey standard phoneme-to-grapheme conversion rules, namely, words with homophonic letters and irregular words, may be written incorrectly via this sublexical route.

In recent years, several distinct types of acquired dysgraphia have been identified, each type resulting from a selective impairment to a different part of the spelling process (Ellis, 1993; Miceli & Capasso, 2006; Tainturier & Rapp, 2001). A deficit to the lexical route results in *surface dysgraphia* (Romani, Ward, & Olson, 1999; Tainturier & Rapp, 2001; Temple, 1985; Weekes & Coltheart, 1996), in which the writer is forced to write via the phoneme-to-grapheme-conversion route, causing regularization errors (writing *det* instead of *debt*), and homophonic letter substitution errors (writing *sity* instead of *city*). *Phonological dysgraphia* (Barry, 1994; Ogden, 1996; Rapcsak & Beeson, 2002) results from an impairment to phoneme-to-grapheme conversion, with intact lexical route, causing an inability to write new words and nonwords, whereas the writing of words that are already stored in the orthographic output lexicon remains intact. *Deep dysgraphia* (Bub & Kertesz, 1982; Cipolotti, Bird, Glasspool, & Shallice, 2004; Hillis, Rapp, & Caramazza, 1999; Raman & Weekes, 2005; Weekes, 2006) entails impairment in both nonword writing and semantic errors in word spelling. *Graphemic buffer dysgraphia* (Caramazza & Miceli, 1990; Posteraro, Zinelli, & Mazzucchi, 1988; see Miceli & Capasso, 2006 for a review) is a selective impairment at the graphemic output buffer, which causes letter identity errors (substitutions), letter additions and deletions, and errors of letter position within the word (letter transpositions), both in real words and in nonwords. The writing of individuals with graphemic buffer dysgraphia is affected by word length, because the buffer is a temporary store with limited capacity (Caramazza, Miceli, Villa, & Romani, 1987; but see Sage & Ellis, 2004 for a different view). Selective impairments within the graphemic buffer stage have also been identified, leading to selective letter identity (Cotelli, Abutalebi, Zorzi, & Cappa, 2003; Kay & Hanley, 1994; Kokubo, Suzuki, Yamadori, & Satou, 2001; Posteraro et al., 1988; Shallice, Rumiati, & Zadini, 2000; see Miceli & Capasso, 2006 for a review) or selective letter position errors (Gvion & Friedmann, in press).

Neglect dysgraphia is also assumed to result from a deficit at the graphemic buffer level (Baxter & Warrington, 1983; Caramazza, & Hillis, 1990; Hillis & Caramazza, 1995). Until today no specific dysgraphia has been reported that selectively causes voicing substitutions (But see the report of patient DR in the dysgraphia rehabilitation study by Luzzatti, Colombo, Frustaci, & Vitolo, 2000, who made many devoicing errors in writing, as well as some other substitutions, and Graham, Patterson, & Hodges, 2000, who reported individuals with semantic impairments who made voicing errors in spelling in addition to other types of errors; see also an abstract by Tainturier, 1996 briefly reporting a patient with voicing errors in writing[1]).

Because all the components of the lexical and sublexical routes, including the graphemic buffer, are shared by the various spelling outputs such as writing, typing, and oral spelling (Caramazza et al., 1987; Tainturier & Rapp, 2001) a deficit in these components causes errors in all orthographic output modalities. This is in contrast to selective impairments to later stages of spelling – the allographic store and the graphemic motor patterns for hand-writing. Deficits in the late components may affect only hand-writing but not oral spelling, which does not require the activation of a letter form.

Given these components of the spelling process and the possible selective impairments in them, the current study aims at assessing which of them can cause voicing errors in writing, and whether a modification to the model is required.

Because voicing is a purely acoustic-phonetic feature and skilled writing relies on pure orthographic representations, the relation between voicing and its orthographic implementation is not obvious. Voicing is a laryngeal feature that specifies the glottal features of the segment. [+voice] segments are produced by bringing together the vocal cords in such a way to be set in vibration when air passes between them. [-voice] sounds are

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[1] Many other studies of graphemic buffer dysgraphia, surface dysgraphia, allographic dysgraphia, and phonological dysgraphia reported individuals who made letter substitutions in spelling, usually in addition to other types of errors, however, none of them referred to the relation in voicing between the target and the substituting letter (Behrmann & Bub, 1992; Blanken, Schafer, Tucha, & Lange, 1999; Chialant, Domoto-Reilly, Proios, & Caramazza, 2002; Cholewa, Mabtey, Heber, & Hollweg, 2010;Cipolotti, Bird, Glasspool, & Shallice, 2004; Delazer, Lochy, Jenner, Domahs, & Benke, 2002; Glasspool, Shallice, & Cipolotti, 2006; Graham, Patterson, & Hodges, 1997; Hanley & Peters, 2001; Kirk, Blonder, Wertman, & Heilman, 1991; Miceli & Capasso, 2006; Miceli, Capasso, Benvegnù, & Caramazza, 2004; Neils, Roeltgen, & Greer, 1995; Ogden, 1996; Panton & Marshall, 2007; Rapcsak & Beeson, 2002; Rapp, 2005; Rapp, Epstein, & Tainturier, 2002; Rapp & Kong, 2002; Schiller, Greenhall, Shelton, & Caramazza, 2001; Ward & Romani, 2000; Zesiger, Martory, & Mayer, 1997), although some of them give examples that show voicing substitution errors (Annoni, Lemay, de Mattos Pimenta, & Lecours, 1998; Caramazza & Miceli, 1990; Caramazza, Miceli, & Romani, 1987; Folk, Rapp, & Goldrick, 2002; Iribarren, Jarema, & Lecours, 2001; Miceli, Capasso, Ivella, & Caramazza, 1997; Miceli, Silveri, & Caramazza, 1985; Miozzo & De Bastiani, 2002; Posteraro, Zinelli, & Mazzucchi, 1988; Sage & Ellis, 2004).

characterized by lack of vibration due to either separated vocal cords or increase in their tension (Ladefoged, 1993). Voiced consonants have significantly shorter voice onset time (VOT) than the parallel voiceless consonant, namely, the time that elapses between the plosive release and the voicing - the onset of vocal fold vibration, is shorter. Phoneme pairs that differ with respect to voicing are (voiced phoneme first): b-p, g-k, d-t, z-s, v-f.[2] In the current line of studies we explore in what way such a phonological feature is processed in the spelling model.

In the following description of the experimental investigation, the participants are presented in Section 2, Section 3 investigates the characteristics of the participants' writing errors in various tasks. Section 4 empirically evaluates the possible loci of the impairment that is responsible for the manifestation of *dyscravia*, and suggests the most probable source of this impairment. We then conclude in Section 5 by assessing the reading of the participants, to examine whether parallel errors exist also in reading.

2. PARTICIPANTS

The participants in this study were TG, a 31 years old man, and BG, a 66 years old woman, who were referred to the clinic with dyslexia and dysgraphia following a left hemisphere lesion.

TG, a right handed native speaker of Hebrew, sustained a traumatic brain injury in a motorcycle accident at the age of 16, following which he was referred to the rehabilitation center. Since the injury he stopped going to high school, and has not worked on a regular basis. He reported good premorbid reading and writing skills. According to a language assessment using the Hebrew version of the WAB (Kertesz, 1982; Hebrew version by Soroker, 1997) and BAFLA - a test battery for agrammatic comprehension and production (Friedmann, 1998), TG was diagnosed with non-fluent mild *agrammatic aphasia*. He had characteristic agrammatic speech: short, non-fluent, with mainly simple sentences, and ungrammatical production of complex sentences and Wh-questions. His sentence comprehension was also characteristic of agrammatism, with poor comprehension of reversible object relatives and topicalized sentences, and relatively good comprehension of simple active SVO sentences and subject relatives (using a sentence-picture matching test, described in Friedmann & Shapiro, 2003). TG's *phonological short term memory* was tested

[2] A useful tool for identifying voicing phoneme pairs is whispering them – the whisper takes out the voicing, leaving phonemes that differ only with respect to voicing identical.

using the FriGvi working memory battery (Friedmann & Gvion, 2002; see Friedmann & Gvion, 2003a, and Gvion & Friedmann, 2007, 2008a for a detailed description of the test battery). His working memory assessment showed limited word spans in all the recall tasks: word span for phonologically dissimilar 2-syllable words was 3 (normal age-matched average = 5.57, *SD* = 0.75); word span for phonologically similar 2-syllable words was 2 (normal age-matched average = 4.58, *SD* = 0.51). He showed significant length effect: he had a span of 2 for 4-syllable words (normal age-matched average = 4.45, *SD* = 0.50), and span of 3 for 2-syllable words. Crucially, he did not make voicing errors in any of these working memory tasks. He also had poorer-than-normal performance in recognition spans. He was tested using a matching word order span, a test in which the participants hear two word-lists containing the same 2-syllable phonologically and semantically unrelated words and are asked to judge whether the order of the words in the two lists was the same. TG's span in this task was 4 (normal age-matched average = 6.33, *SD* = 0.98). TG's lexical retrieval was examined using SHEMESH, a picture naming test of 100 colored objects (Biran & Friedmann, 2004). TG named correctly 87 of the 100 pictures, producing 5 close semantic paraphasias, 5 phonological errors (only one of which included devoicing of a voiced phoneme with an immediate self-correction), 1 formal paraphasia, 1 no response, and 1 circumlocution.

BG, a right handed woman, was at the time of the assessment one month post left temporal hemorrhage following a fall down a staircase. She had lived in Israel for 57 years and spoke, read, and wrote Hebrew fluently. She had a bachelor degree, worked in a biological lab and had no premorbid reading or writing disorders. According to language assessment using the Hebrew version of the WAB (Kertesz, 1982; Hebrew version by Soroker, 1997), she was diagnosed with mild anomic aphasia. On the *SHEMESH* naming test, she named correctly 69/100 of the pictures, producing 27 circumlocutions and 4 close semantic paraphasias. BG had normal phonological short term memory, as tested with the FriGvi battery. On the recall task of phonological dissimilar 2-syllable words, BG's span was 5 (normal age-matched average = 4.86, *SD* = 0.78); her 2-syllable pseudo-word span was 3 (normal age-matched average = 3.40, *SD* = 0.46). She also made no voicing errors in the working memory tasks.

3. An Assessment of TG’s and BG's Writing

3.1. An assessment of writing and voicing errors to dictation

To assess the participants’ spelling, and specifically to learn about their voicing errors in spelling, we first tested writing to dictation. A list of words with a potential for voicing substitution errors, namely words that contain at least one of the phonemes */v, f, b, p, d, t, g, k, s, z/* was dictated to the participants[3]. TG wrote 214 words to dictation, and BG wrote 270. They were requested to repeat the word before writing, to make sure that they heard the words accurately. Their repetition was flawless, and did not include any voicing substitutions.

The word lists were constructed in such a way as to detect various types of dysgraphia. To detect surface dysgraphia, we included words that could not be written correctly solely on the basis of phoneme-to-grapheme conversion, but required lexical information to be written correctly, because of the inclusion of phonemes that could be mapped to one of several graphemes, or because of irregular spelling. Most of the words in the lists had such a property and thus were sensitive for detecting surface dysgraphia: 165 for TG and 258 for BG; These words included 55 potentiophones for TG, and 62 for BG. (Potentiophones are words that, when written via phoneme-to-grapheme conversion, might result in another existing word, like *now* for *know*; Friedmann & Lukov, 2008). To detect graphemic buffer dysgraphia, the lists included words of varying length, from 2 to 11 letters (M = 4.65, SD = 1.68 for TG; M = 4.60, SD = 1.51 for BG). Words were of various lexical categories (nouns, verbs, adjectives, and function words) to allow for the detection of deep dysgraphia, and many of the words could lead to another existing word following a transposition of middle letters (104 such words for TG, 142 for BG) or a neglect error, to allow for the detection of letter position dysgraphia and neglect dysgraphia respectively.

In the scoring of errors as voicing errors, we were conservative, and scored errors that could either be classified as surface errors or as voicing errors, as surface errors. This mainly included cases in which a consonant assimilated to the voicing of the following consonant as is the case with the word *safta* (grandma), which is written *savta*, but is usually pronounced with assimilation of the *v* to the unvoiced *t* as *safta*. In this case, if our participants spelled the word with an *f*, it was not counted as voicing error but rather as surface error. (A relevant

[3] Four of these phonemes could be mapped into 2 homophonic Hebrew letters, and three pairs of phonemes could be mapped to the same letter. The phonemes *r* and *x* could count as a voicing pair for some Hebrew speakers, however, TG only made only 3 such errors experimentwise, and BG made none, so we did not include these phonemes in the letters with a voicing error potential throughout the article (neither did we include them in the non-voicing letters).

English example is the letter *s* in *dogs.* If the participant wrote the word with *z, dogz*, we would score this error as a surface error, rather than a voicing error).

3.1.1. Results

TG wrote correctly only 69 of the 214 words dictated to him (32%) and BG wrote correctly 204 of the 270 words (76%). The predominant errors in the writing of both participants was voicing substitution errors and surface dysgraphia errors. TG made 81 (36%) voicing substitution errors out of the total of 228 words he wrote incorrectly[4], and 94 (41%) surface dysgraphia errors; BG made 45 (51%) voicing substitution errors out of her 88 errors, and 38 (43%) surface dysgraphia errors. Voicing errors occurred for both participants in both the morphological affixes and the consonantal root.[5]

In addition, BG made only 4 substitutions that were not voicing or homophonic, and one letter omission. TG made 25 substitutions that were not voicing or homophonic substitutions, 15 omissions, 6 additions, and 5 transpositions, errors which indicate that he had also a mild graphemic buffer dysgraphia.

None of the participants made semantic substitutions or morphological errors, indicating that none of them had deep dysgraphia. Neither of them made errors that indicate left or right neglect dysgraphia. In their responses that included voicing errors, both participants wrote more nonwords than words: TG had 67 nonlexical responses and 31 lexical; BG had 26 nonlexical and 13 lexical responses.

3.2. Do voicing substitution errors occur across tasks?

To further learn about *dyscravia* we also tested TG and BG's writing using a written picture naming task. Colored pictures from the *SHEMESH* test (Biran & Friedmann, 2004) and from a homophone and potentiophone picture test (*KRUV*, Gvion & Friedmann, 2008b) were presented for written naming, 106 pictures for TG, and 96 for BG, the names of all pictures were words with a potential for voicing errors.

[4] The total number of errors is larger than the number of misspelled words, because the participants sometimes made several errors in the same word, either voicing errors on more than one letter (such as writing "bik" instead of "pig"), or different error types in the same word, typically one voicing substitution error and one surface dysgraphia error (writing "kad" instead of "cat").

[5] For example, the letter ת, t, got converted to ד, d, both when it served as the future inflection morpheme (תחבר-דחבר), and when it served as a consonantal root letter (מפתה-מפדה).

3.2.1. Results

TG wrote accurately only 54 of the 106 picture names (51%). The error pattern was similar to the one he demonstrated in writing to dictation. Errors included 23 (38%) voicing substitutions, and 31 (51%) errors that indicate writing via phoneme-grapheme conversion, 5 other substitution errors, and 2 letter omissions.[6]

BG wrote correctly 85/96 (89%) of the picture names. Like TG, and like in her writing to dictation, she made predominantly voicing substitution errors and surface dysgraphia errors. She made 7 (54%) voicing substitution errors, and 6 (46%) surface dysgraphia errors.

Thus, for both participants, voicing errors occur not only in writing to dictation but also in written naming, when no auditory input is involved. In Figure 2 we assembled the types of errors evinced in their writing to dictation and written naming tasks that included words with voicing error potentials. The figure shows that the most prominent error types across tasks and for both participants are voicing substitutions and surface dysgraphia errors.

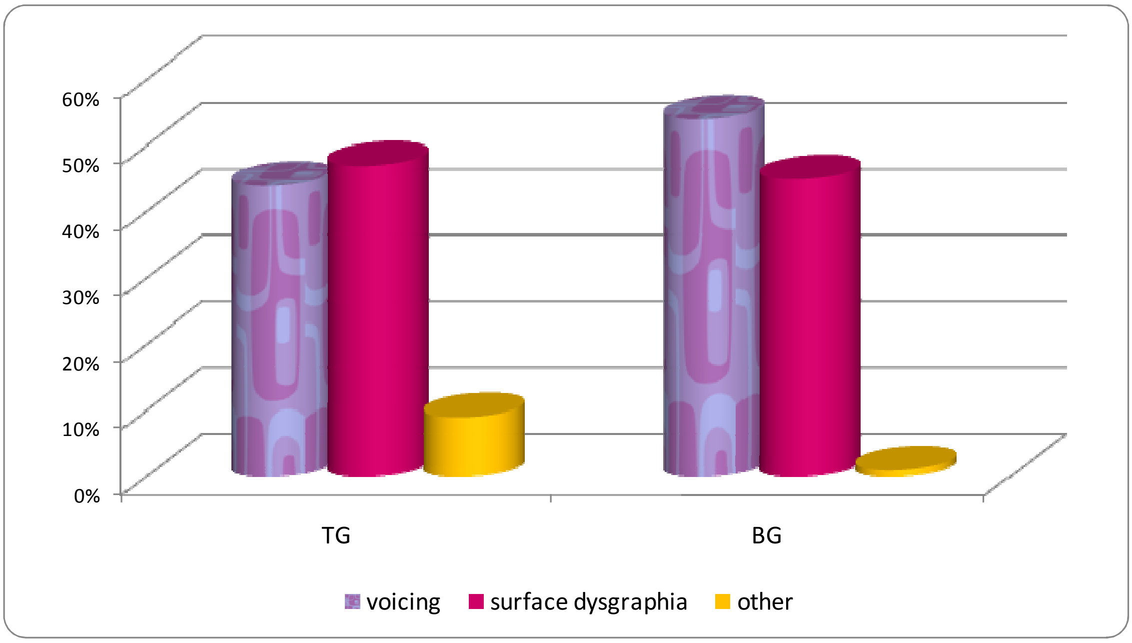

Figure 2. Types of errors in word spelling

3.3. Nonword writing

To assess nonword writing, we dictated to TG and BG nonwords of 2-6 letters (TG: 30 nonwords, mean length = 4.3, SD = 1.3; BG: 36 nonwords, mean length = 4.9, SD = 0.7), all with a potential for voicing errors. Notice, that if voicing errors result from a deficit at the

[6] TG and BG had paraphasias in oral naming (as reported in the Participants section). These were not present in the written naming task because they wrote the words without any time pressure and only after they reached the correct word.

orthographic lexicon, one would not expect voicing errors in writing nonwords at all. As in the word writing to dictation task, the participants were requested to repeat the nonword before writing them, and here, too, they showed flawless repetition, without any voicing substitutions.

3.3.1. Results

TG wrote correctly 14 (47%) of the nonwords. Again, voicing errors were the most evident error type in his writing. He made 13 (77%) voicing substitution errors, as well as 3 other substitutions and 1 transposition. BG wrote correctly 15 (42%) of the nonwords, making 20 voicing substitution errors and 3 other substitutions. The finding that for both participants voicing errors occurred not only in word writing but also in nonword writing excludes the lexical route as a possible locus of the deficit.

3.4. A comparison of voicing errors in word and nonword writing to chance

The prominence of voicing substitutions and the paucity of other substitutions indicate a selective deficit that is related to voicing. To statistically compare the rate of voicing substitutions to chance, we collected the number of voicing substitutions out of the total number of substitutions in word writing (section 3.1), written naming (section 3.2), and nonword writing (section 3.3). We compared this rate to 1/21, the rate that is expected for a random substitution to accidentally be the voicing counterpart of the target letter (as there are 22 letters in Hebrew). TG had 150 substitutions, 117 (78%) of them were voicing substitutions. BG had 74 substitutions, 67 (91%) of them were voicing substitutions. The rate of substitution with a voicing counterpart out of the total number of substitutions is significantly larger than chance, both for TG, $\chi^2 = 109.22$, $p < .0001$, and for BG, $\chi^2 = 166.33$, $p < .0001$.

Similarly, if the substitutions were guided by frequency and not by voicing, we would expect the most frequent letters to appear more often in the participants' substitutions. We took the most frequent consonant letter מ (m), which has a high frequency of 11.3% of the consonants (Schocken, 2008), and compared the rate of substitutions to *m* to the rate of substitutions to the voicing counterpart of the target letter. We included in this analysis the 8 target letters that have a voicing counterpart (the letters in Table 1), and counted the number of substitutions for a voicing counterpart and the number of substitution to *m*. If frequency were the determining factor for substitutions, we would have expected that substitutions to *m* would occur more than substitutions to the voicing counterpart of the target letter. However, this comparison indicated clearly that there were far more substitutions for a voicing counterpart (104 for TG, 65 for BG) than for *m* (1 for TG 0 for BG), albeit that the average frequency of the voicing counterparts was 3.9, far less frequent than *m*.

3.5. Writing of words without potential of voicing substitution error

To compare writing of words with and without letters with a potential for voicing substitutions, we dictated to the participants a list of words without such potential (words that contain the phonemes *n, m, l, y, h,* glottal stops, and vowels), 43 for TG and 30 for BG. These words were presented together with the words with a voicing error potential reported in section 3.1, in a random order. For these words as well, the participants repeated the words before writing them.

3.5.1. Results

As the words included no letters with voicing counterparts, the participants did not make voicing errors in writing this word list. This led to significantly better writing of these words without a voicing potential than words with such potential, $\chi^2 = 5.60$, $p = .02$ for TG, and $\chi^2 = 4.87$, $p = .03$ for BG.

TG wrote correctly 22/43 (51%) of the words without voicing substitution potential. He only had 19 surface dysgraphia errors, 1 letter addition, and 1 non-homophonic letter substitution. BG wrote correctly 28/30 (93%) of the words, with 2 surface dysgraphia errors.

The writing assessment reveals that both participants have voicing substitution errors and surface dysgraphia errors (in words that include letters with a voicing counterpart). In the following sections we report experiments we conducted in order to further explore the characteristics of the voicing substitution errors they made.

3.6. Is there a default? Is the direction of substitutions toward voiced or voiceless letters?

To further characterize dyscravia, we analyzed the direction of the substitutions, namely whether the unmarked feature [-voice] is the default, with [+voice] segments being replaced with [-voice] ones consistently, or whether both directions of substitution occur. We analyzed the errors on words and nonwords that include letters with voicing distinctions that TG and BG wrote (including writing to dictation and written naming). This analysis revealed that both participants made both directions of errors, with no consistently preferred direction, as detailed in Table 1.[7] Lumping together all the pairs presented in Table 1, we found no significant difference between devoicing and voicing, either for TG, $\chi^2 = 0.17$, $p = .68$, or for BG, $\chi^2 = 0$, $p < .99$. Phoneme frequency seemed to have some effect on the direction of errors within each phoneme minimal pair for TG, but not for BG, although both of them showed errors in both directions for each phoneme pair, as shown in Table 1. According to Schocken's (2008) Hebrew phoneme frequency, the frequencies of the consonants in Table 1 were: t = 10.4%, d = 3.7%, k = 4.7%, g = 1.8%, f = 2.0%, v = 3.9%, p = 1.4%, b = 3.6%.

Table 1

Voicing and devoicing errors

	t→d	d→t	k→g	g→k	f→v	v→f	p→b	b→p	Total voiceless→ voiced	Total voiced→ voiceless
TG	19/122 16%	17/49 35%	12/96 13%	23/62 37%	30/38 79%	8/60 13%	3/21 14%	2/31 6%	64/277 23%	50/202 25%
BG	13/181 7%	7/63 11%	14/103 14%	6/78 8%	12/81 15%	10/71 14%	2/50 4%	1/32 3%	41/415 10%	24/244 10%

The error rates in each cell are calculated out of the number of the target phoneme occurrences in the target words and nonwords that included voicing errors.

3.7. Do voicing substitutions occur at the graphemic or the phonological level?

When the letter b is written instead of p, for example, two explanations are possible. One is that the substitution is at the letter level; the other possibility is that the substitution happens at the phoneme level, which then gets mapped onto a grapheme, but the errors depend on the sound of the target phoneme. Hebrew suggests a natural way to decide between these options. In Hebrew, some letters are ambi-phonic, namely, they have several possible mappings to phonemes. For example, the letter כ can be the graphemic representation of either the phoneme *k* or *x*, and ו can be mapped to the vowels *o* or *u*, or to the consonant *v*. If the substitution happens at the phoneme level, it should obey the phoneme in the target word, namely – כ should be substituted into g only when it serves as the phoneme *k* in the target word, and to r when it serves as the phoneme *x*, but not vice versa. [A relevant example from English is the letter *c* – if the error is at the phoneme level (or at the phoneme activating a wrong grapheme), then *cat*, but not *city*, can be written with *g* instead of *c*.]

[7] The analysis presented in Table 1 did not include the pair s/z, because TG and BG seem to have a different voicing counterparts for s – TG made no s/z error but made 9 s/c errors; BG made no s/c errors but made 8 s/z errors). The analysis together with the s/z or s/c pair reached the same results, with no significant difference for any of the participants between voicing and devoicing errors.

The analysis of errors along these lines points to phoneme-based substitutions, as the letters got consistently substituted according to their phonemes. Namely, the letter כ was substituted only into g when the target phoneme was *k* (14 times, 7 for each participant), and to r when the target phoneme was *x* (once, for TG). Similarly, ו was never substituted when it appeared as a vowel, but when it appeared as the consonant v in the target word, it was substituted to f (albeit only once - וילון-פילון) (or with a surface error to ב).

3.8. Do phonological properties of the target words affect error rate and position?

To further characterize the voicing errors, we tested whether these errors occur more frequently in non-stressed syllables. We counted for each of the participants the voicing errors they made on words with more than one syllable, classifying them according to whether they occurred on stressed or on unstressed syllables in each word. We also analyzed the number of errors on stressed and unstressed syllables out of the number of stressed and unstressed syllables with a voicing error potential. This analysis indicated that for TG, out of 96 words he made voicing errors in, in 49 words he made errors on an unstressed syllable only, in 33 words he made errors only on the stressed syllable, and in 8 words he made errors on both the stressed and an unstressed syllable. Namely, he made 41/96 errors on stressed syllables (42%), and 57/155 errors on unstressed syllables (36%), rates that were not statistically different, $\chi^2 = 0.88$, $p = .35$.

BG also showed no effect of stress position: in 20 words she made errors on the unstressed syllable, and in 20 other words she made voicing errors on the stressed syllable, in 6 words voicing errors occurred on both the stressed and an unstressed syllable. Thus, for her, too, there was no difference between the rate of voicing errors on stressed syllables, 26/42 (62%), and her errors on non-stressed syllables, 26/52 (50%), $\chi^2 = 1.33$, $p = .25$.

4. What is the locus of the impairment ?

An impairment in which of the components of the spelling process can give rise to this pattern of voicing substitutions? In this section we explore the various sources one by one, to locate the deficit causing voicing errors in writing. One possible source might be auditory perception impairment, which does not distinguish voiced from voiceless consonants. In this case, we would expect voicing errors to occur only in writing to dictation but not in written naming. We would also expect errors in tasks that involve auditory perception even if they do not involve spelling, like auditory discrimination tasks and word repetition. Another source to consider would be the graphemic buffer. However, it is not clear how the graphemic buffer should be sensitive to phonological distinction like voicing, being a graphemic component, which is active after the phonemes have been processed and converted to graphemes. Furthermore, a selective deficit at the graphemic buffer is expected to cause random substitutions, not only substitutions between letters that form voicing minimal pairs. Substitutions that result from a graphemic buffer deficit should also be subject to a length effect, and to grapheme position effect. Deficits to the allographic level, in which the activation of the abstract letter forms occurs, can also lead to substitutions, but again, this level is even farther away from the phonemes, and the expected errors are graphic, rather than phonological. Furthermore, errors are expected only in handwriting. Finally, a deficit to the orthographic output lexicon that causes voicing errors is only imaginable if the entries in the lexicon were incorrectly represented. In this case, we would expect consistent errors for each word, and no errors in nonword writing. In the following sections we empirically tested these various possibilities, rule each of them out, and suggest that the only possible source for TG and BG’s dyscravia is in the phoneme-to-grapheme conversion.

4.1. Is the deficit located in auditory processing?

To examine whether the source of the voicing errors is an impairment in auditory discrimination, we tested auditory perception without writing, and writing without auditory perception. These tasks of auditory perception without writing included an auditory same-different judgment tasks, a word-to-picture matching task, and a word repetition task. We already reported in section 3.2 the results of spelling without auditory input, tested using written picture naming, which showed the same pattern of voicing errors as did the writing to dictation. We also reported that word repetition in working memory tasks and in repeating words to dictation were flawless.

4.1.1. Auditory perception

Each of the participants was tested using two tests of auditory discrimination. TG was tested using the *PALPA's* same-different judgment task on nonwords (PALPA 1). The nonwords in each pair differed in one phoneme, in either voicing, manner, or place of articulation. The nonword pairs were introduced auditorily, and TG was requested to judge whether the pairs were same or different. He performed flawlessly (40/40) on this task. He also performed flawlessly (26/26) on a same-different judgment task of auditory word pairs differing only in the voicing of one phoneme, such as *bay* and *pay*.

BG was tested using a same-different judgment task of words (*PALPA 2,* Kay, Lesser, & Coltheart, 1992; Hebrew version by Gil & Edelstein, 1999). The words in each pair differed in one phoneme, in either voicing, manner, or place of articulation. The word pairs were introduced auditorily and BG judged whether the pairs were same or different. She performed flawlessly on this task (20/20). She was also presented with pairs of words that started either with the same phoneme or with phonemes that differ in voicing. She was requested to judge whether the words started in the same phoneme or not. She performed very well also on this task, with 15/16 correct responses.

4.1.2. Word repetition

Another way to test voicing in auditory perception, which can also assess voicing in speech production, was a repetition task. The participants heard a list of 1-2 syllable words, each of the words included one or two phonemes that have a voicing counterpart, and were requested to repeat each word. TG repeated 41 words, and BG repeated 80 words. Each word was presented by the examiner only once.

Both participants performed very well on this task: TG repeated correctly 40/41 (98%) of the words, and his single error was not a voicing error, and BG repeated correctly 100% of the words. Recall also that the task of writing to dictation required the participants to repeat each dictated word and nonword before writing it, and in this task, too, their repetition was flawless, and without voicing errors.

Moreover, even in tasks that put a larger load on the phonemic buffer, the various span tasks, neither of them made any voicing errors, neither did they make voicing errors in spontaneous speech.

4.1.3. Interim summary

The data from the auditory discrimination tests and the word repetition tests indicate that TG and BG did not have auditory perception impairment. Taken together with the results of the written naming task (see section 3.2), which showed that voicing errors occurred even when the input was not auditory, these results clearly indicate that TG and BG did *not* have an auditory discrimination deficit. The results of the repetition tasks, together with the absence of voicing errors in the naming task, the FriGvi working memory tasks, and the repetition of the dictated words and nonwords in the writing to dictation task, indicate that the participants did not have a voicing deficit in phonological output either.

4.2. Is the deficit located in the graphemic buffer?

The graphemic buffer is assumed to hold temporarily active orthographic representations that were retrieved either from the orthographic output lexicon or assembled from the phoneme-to-grapheme converter, until spelling is completed. Firstly, a theoretical consideration renders it implausible that a deficit in the graphemic buffer would create voicing errors: the representations in the graphemic buffer are orthographic, rather than phonemic, in nature. This seems to preclude the possibility that voicing substitution errors may be a result of an impairment to this stage. In addition, it is clear that it cannot be the ordinary graphemic buffer dysgraphia, as the participants' spelling errors almost never included omissions, transpositions, or additions of letters, and the substitutions were very specific to voicing.

Nevertheless, we tried to examine the possibility that the deficit lies in the graphemic buffer, but in a way that only causes the voicing feature to fade out.

4.2.1. Length effect

Because the most prominent property of the graphemic buffer is its liability to length effect, we would expect this fading of the voicing feature to increase with word length, if it is indeed a result of a graphemic buffer deficit.

To assess the length effect, it is important to control for the fact that longer words may be more liable to voicing errors simply because they contain more letters that have a voicing counterpart. Thus, to analyze the effect of length and control the number of letters with a voicing error potential, we included in the analysis only words that include exactly 3 letters with a voicing counterpart (such as כסף [ksF, *kesef*, silver or money], and אוטובוסים, [AOtOBOsIM, *otobusim*, buses]). These words were 3-9 letters long (for this analysis we disregarded surface dysgraphia errors and counted them as correct).

As shown in Table 2, the number of letters in a word did not affect the rate of voicing errors for any of the participants.[8]

[8] Interestingly, TG did show a length effect when the analysis included all word types, not only the words with letters with voicing counterparts, and all error types but surface errors, including the classical graphemic buffer errors of substitutions and omissions (2 letter words - 72% correct, 3 - 45% correct, 4 - 40%, 5 - 37%, 6 - 26%, 7 letters or longer - 7%).

Table 2

Rate of voicing substitution errors as a function of word length

	3 letters	4 letters	5 letters	6 letters	7+ letters
TG	70%	50%	60%	60%	33%
BG	33%	33%	10%	20%	33%

4.2.2. Error positions

Another characteristic of graphemic buffer disorder is the tendency to make more errors at the end of the word (Schiller, Greenhall, Shelton, & Caramazza, 2001; Tainturier & Caramazza, 1996), and for some, in the middle of the word (Beeson & Rapcsak, 2002; Caramazza & Miceli, 1990; Gvion & Friedmann, in press; Jónsdóttir, Shallice, & Wise, 1996; Posteraro et al., 1988; Tainturier & Rapp, 2004). Had the deficit been due to a deficit at the graphemic output buffer, we would expect voicing errors to occur primarily at the end, or possibly in the middle. An analysis of voicing error positions (number of voicing errors out of the number of segments with a potential for voicing error in the first, middle, and final phonemes) indicated no position effect for any of the participants (TG: 15%, 21%, 11%, respectively, $\chi^2(2) = 5.29$, $p = .08$; BG: 8%, 10%, 12%, respectively, $\chi^2(2) = 1.19$, $p = .55$).

These results thus indicate, beyond the theoretical considerations, that the graphemic buffer was not the source of the voicing errors in spelling. Furthermore, had the decay-in-the-buffer theory been correct, it is reasonable to assume that the stressed syllables would be more resistant to decay than the unstressed ones. Nevertheless, as the results of section 3.8 indicate, there was no difference in the voicing error rate between stressed and unstressed syllables.

4.3. Is the deficit located in the allographic stage?

The allographic stage is a stage where it is assumed that abstract letter forms are stored. It is viewed as a stage in which the letter representation is selected by determining the shape, the case (lower vs. upper-case) and the type (print vs. cursive) of letters to be written. Representations processed at this level are thought to be physically or spatially coded. Typical errors that result from an impairment to the allographic stage are within-word case confusion errors (De Bastiani & Barry, 1989), showing dissociation between upper to lower-case writing (Menichelli, Rapp, & Semenza, 2008), and between print and cursive writing style (Venneri, Pestell, & Caffarra, 2002), substitutions of the target letter with a letter that is graphemic-motorically or visuo-spatially similar (Del Grosso Destreri et al., 2000; Zesiger, Martory, & Mayer, 1997), as well as letter omissions. Importantly, the performance in spelling tasks that do not involve the activation of the letter representation, such as oral spelling, is usually spared (Del Grosso Destreri et al., 2000; Zesiger et al., 1997).

Thus, we can rule out a deficit in the allographic representations as a source for the voicing errors on the basis of the fact that the target letters and the letters that were produced instead shared phonological similarities, but did not necessarily share physical or spatial similarity, as can be seen in example (1), which presents the voicing-substitution letter pairs in cursive (hand-) writing, as they were written by the participants.

(1) ב↔פ; ג↔ק or ג↔כ; ד↔ט or ד↔ת ; ס↔ז or ש↔ז

4.3.1. Oral spelling

A way to evaluate the allographic stage as a source for the voicing errors would be testing oral spelling, because it does not involve the recall of the letter form. If the deficit resides in the allographic stage, no voicing errors are expected in oral spelling. If, however, the deficit results from an earlier stage that is shared by the various output routes, voicing errors should occur in oral spelling as well.

We tested TG and BG's oral spelling by auditorily presenting to them words in which voicing substitutions create another existing word, and asking them to spell the words aloud (BG spelled 23 words, TG spelled the first letter of 28 words). Their error pattern was very similar to that witnessed in the other spelling tasks – writing to dictation and written naming: the most prominent error type was again voicing substitution. BG spelled correctly only 18/23 (78%) of the words. She made voicing substitution errors in all of the misspelled words, making 9 (82%) voicing substitution errors. In addition, she omitted 2 letters. TG named correctly the first letter of 22/28 (79%) of the words, and made 5 voicing substitution errors (83% of the errors) and one surface dysgraphia error. Thus, BG made even more voicing errors out of the total number of words in oral spelling than in written spelling, 39% in oral spelling vs. 17% in writing to dictation, $\chi^2 = 7.11$, $p = .007$. TG had a similar rate of voicing errors in oral spelling and written spelling to dictation, $\chi^2 = 0.31$, $p = .58$. He made 18% voicing errors in oral spelling (of first letters out of the number of first letters) compared to 22% of the letters with voicing counterpart in writing to dictation (100 voicing substitutions out of the 448 letters with voicing counterpart he wrote to dictation).

Thus, the finding that voicing errors occurred for both patients not only in writing but also in oral spelling indicates that the deficit resides earlier than the allographic stage. This, together with the pattern of errors that were phonologically rather than visually similar, indicates that the allographic stage is intact and is *not* responsible for dyscravia.

4.4. Is the deficit located in the orthographic lexicon?

Another source of the deficit that should be considered is the orthographic lexicon. However, one major empirical finding rules out the lexicon as the source of this deficit: the finding that voicing errors occurred also in spelling nonwords, which, naturally, are not written via the lexicon. Furthermore, the lexicon as a possible source for voicing errors is theoretically quite questionable, because the lexical entries in the orthographic lexicon should be graphemically, rather than phonemically, represented. It thus seems less probable that phonological errors and mis-activation of phonological, rather than orthographic, neighbors would occur in the orthographic lexicon.

We nevertheless entertained the possibility of impaired lexicon, and specifically that the representations in the lexicon are corrupted in a way that encodes graphemes incorrectly with respect to voicing. If so, we would expect the errors made for each word to be consistent. Namely, if the word *bear* is incorrectly coded in the lexicon as *pear*, we would expect *bear* to be consistently written incorrectly as *pear*. To test whether this is indeed the case, we asked the participants to write the same word several times, in different sessions. TG wrote 2-4 times 147 words that included at least one letter with a voicing counterpart; BG wrote 314 such words 2-4 times.

In the analysis of the words that were written at least once with a voicing error, for TG out of 109 such words, there was no word which was written incorrectly the same way every time – namely, when another spelling attempt included a voicing error, it was a different one; 20 of the words TG wrote at least once with a voicing error were written once correctly. For BG, there were 60 words that were written at least once with a voicing error, only four of which were written twice with the same error. Eleven of the words that were written at least once with a voicing error were also written at least once correctly.

For example, TG wrote the word צדף (CDF, *cedef*, shell) 3 times. He wrote it once with only one voicing error, צדב (CDv), once with two voicing errors, צתב (CTv), and once again with two voicing errors, but different ones, צטב (Ctv). BG wrote the word תוספת (TOsFT, *tosefet*, addition) once correctly and once as דוזבת (DOZvT). She wrote the word כסף (ksF, *kesef*, money) once as גשב (GSv, which involves two voicing errors and one surface error) and once as גשף (GSF, one voicing, one surface error).

Thus, we can conclude, on the basis of the finding that errors occurred also in the spelling of nonwords and not only in the spelling of word, that the deficit does not lie in the orthographic lexicon.[9] The inconsistent voicing errors in word writing also indicate that the deficit does not result from corrupted representations in the orthographic lexicon.

4.5. Interim summary: the locus of impairment causing dyscravia

In section 4 we ruled out a deficit in auditory discrimination, phonemic output buffer, graphemic output buffer, allographic stage, and orthographic lexicon as causing dyscravia.

We suggest that the deficit that causes the voicing errors in TG and BG’s writing is in the phoneme-to-grapheme conversion route. Given the selective nature of the errors, which only include voicing substitutions, it is not the whole conversion process that is impaired, but rather only a specific function. The results thus lead us to suggest that the conversion route is not a single entity converting whole phonemes to whole graphemes, but rather it receives (correct) phonemic feature bundles from the phonemic output buffer, and activates the graphemes that match these features. Within this route, there is a function that is responsible for the conversion of the voicing feature of phonemes into corresponding graphemes. A deficit in this function led to the voicing errors we saw in writing. Because the participants were writing via the sublexical, rather than the lexical, route, the deficit in phoneme-voicing to grapheme conversion caused voicing errors in writing both words and nonwords.

One type of support for the suggestion that dyscravia results from a deficit at the sublexical route comes from the analysis of the relation between the participants’ surface errors and voicing errors. We analyzed these two types of errors on the most extremely irregular words (words that are ambiguous beyond homophonic letters and the representation of vowels, and which include silent letters or a very infrequent conversion of letters, Friedmann & Lukov, 2008). BG wrote 29 such very-irregular words with a voicing error potential, 27 of them without errors, and two with a surface error (she had a low overall error rate, and her few surface errors usually occurred on homophonic letters rather than on silent vowel letters). On these words, she did not make any voicing errors. Writing these words without surface errors points to writing via the lexical route. Given the extreme irregularity of

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[9] We ran another test, in which we asked the participants to write single phonemes to dictation ("write the letter of the sound /b/"). Twelve phonemes with a voicing counterpart (/*b,p,f,v,s,z,c,sh,t,d,k,g*/), which can be mapped into 13 letters (בפוסשזצטתדקכג) were included in this test. TG wrote correctly only 9/12 (75%) letters, substituting or hesitating between the target letters and their voicing counterparts. BG wrote correctly 12 letters. We do not know, however, what can be concluded from this test, because a reasonable strategy to use in this case would have been to write a letter starting with the given sound, which in Hebrew consistently leads to the correct letters, even without using phoneme-to-grapheme conversion.

these words, it is not likely that these words were written correctly by chance or via the sublexical route. Namely, when she was able to write via the orthographic lexicon, she also made no voicing errors, and there was no case in which she wrote an irregular word correctly via the lexicon but made a voicing error. The fact that she was able to write these words correctly reflects the relatively mild surface dysgraphia that she has, and consequently, explains the relatively low rate of voicing errors she had in words in comparison to TG. Another analysis was done to inspect the other direction of relation between voicing errors and surface errors: we analyzed the words on which a voicing error occurred, and which also included additional (non-morphological) letter or letters that could be spelled incorrectly when writing via the sublexical route. This yielded 16 words, 7 of them were written with a surface error in addition to the voicing error, indicating sublexical route writing. (We would not have expected all words to be written with a surface error because random distribution would predict that some of the phonemes with two possible mappings would be written correctly via the sublexical route by chance).

TG showed a more significant disorder of writing via the lexical route. He made surface errors in writing 38 of 41 very-irregular words with voicing potential, and on 16 of the words he wrote with a surface error, he also made voicing errors. For example, the word סתיו (sTIV, *stav*, autumn) is quite irregular – all 3 consonants are ambiguously mapped from phonemes to graphemes, and it also includes the vowel /a/, which usually is mapped to no letter or to א, but in this word is mapped to י, which usually represents the vowel /i/. In writing this word, TG spelled it four out of four times with surface errors, two of these responses also included voicing errors (סדב, סדוו, סתוו, סתב – sDv, sDVV, sTVV, sTv).

These findings show the tight relation between the use of the sublexical route, as reflected in the surface errors, and the production of voicing errors, supporting the assumption that TG and BG’s dyscravia results from a deficit in the phoneme-to-grapheme conversion route.

Another type of evidence for the tight relation between writing via the sublexical route and voicing errors comes from the comparison of the voicing error rate and surface error rate in writing words and nonwords for the two participants. Whereas TG showed a similar error rate in writing words and nonwords to dictation (38% and 41% respectively), BG had significantly more errors in writing nonwords (17% voicing errors in words, 56% voicing errors in nonwords, $\chi^2 = 28.71$, $p < .0001$). The relatively low voicing error rate that BG demonstrated in writing words correlates with the relatively low surface error rate she had in writing words: she had 14% surface errors in writing words and 17% voicing errors in words, whereas TG had 44% surface errors and 38% voicing errors. Namely, each of them had a very similar surface error rate and voicing error rate in writing words. This result supports the claim that only when they wrote words via the sublexical could their voicing conversion deficit manifest itself. Because BG was often able to write via the lexical route, she also managed to write many words without voicing errors. TG, on the other hand, had a very high rate of surface errors, indicating that he was much more often forced to write via the sublexical route, and this resulted in his elevated voicing error rate in word writing. When the patients wrote nonwords, one could see that the basic error rate for BG was even larger than for TG (56% compared to 43%). This type of analysis thus suggests another type of support for the idea that the voicing errors occurred while writing via the sublexical route.

5. Do Voicing Errors Occur in Reading as Well?

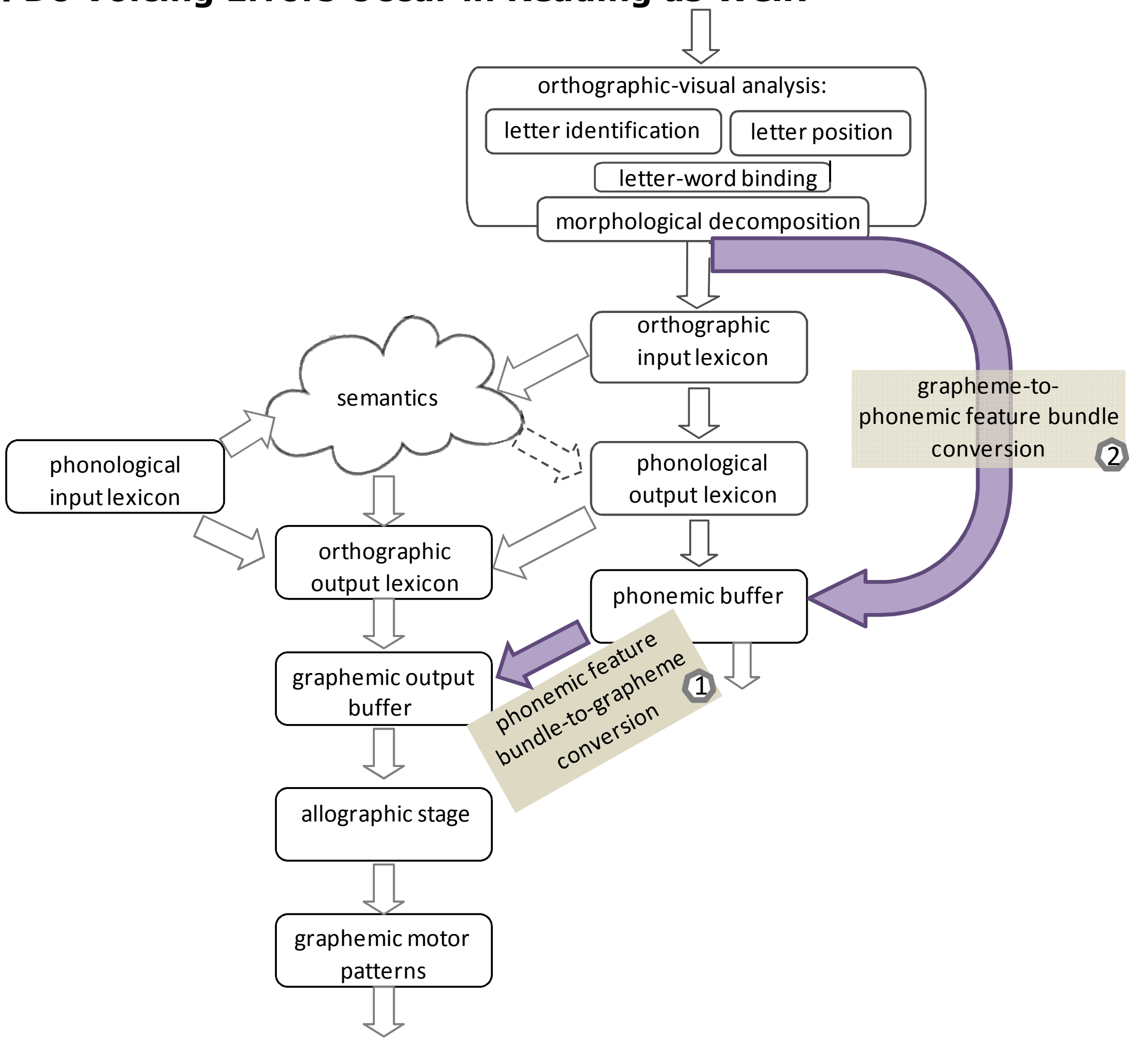

Figure 3. Single word reading and writing model

To explore the relation between writing and reading, and specifically, between the conversion of the voicing feature to graphemes in writing (❶ in Figure 3) and the conversion of graphemes to voicing features of phonemes in reading (❷ in Figure 3), we tested whether the *reading* of the participants with *dyscravia* also includes voicing errors.

Lists of written words (503 for TG, 347 for BG) and nonwords (30 for TG, 41 for BG) from the TILTAN battery (Friedmann & Gvion, 2003b) were presented to the participants for reading aloud. Sixty of the words for each participant were presented as 30 horizontally-presented pairs, the rest of the words were presented as a list one above the other. All words were presented on a white paper, without time limitation. The words, word pairs, and nonwords were presented in separate lists. The participants read the words aloud, and their reading was tape-recorded and transcribed by the two authors at the time of testing; After each session, the recorded responses were also transcribed fully.

The word list included words of various types that can reveal the different types of dyslexia: although Hebrew is a highly irregular orthography, in which no word can be read unambiguously via the sublexical route, for identifying surface dyslexia we included the words that are most sensitive to surface dyslexia: potentiophones, and words that are irregular beyond the underspecification of vowels; the list also included migratable words (words like *there*, which can be read, with a middle-letter migration, as another existing word, *three*) for identifying letter position dyslexia; words that can be read as other words by neglecting one side of the word, for identifying neglect dyslexia; words with many orthographic neighbors, for identifying visual dyslexia; function words and morphologically complex words, for identifying deep dyslexia and some additional types of dyslexia. The word pairs were included to identify attentional dyslexia. Nonwords were included for identifying phonological and deep dyslexia, as well as various peripheral dyslexias.

5.1. Results

The two participants differed considerably with respect to voicing errors in reading, as seen in Table 3.

Table 3

Error analysis in the word and nonword reading of TG and BG

	Total items	Correct	Voicing errors	Sibilant errors	Surface errors	Within-word migrations	Between-words migrations
TG							
Words	503	368 73%	2 0%	0 0%	60 12%	38 8%	14 3%
Nonwords	30	26 87%	0 0%	0 0%	-	3 10%	0 0%
BG							
Words	347	302 87%	10 3%	5 1%	18 5%	0 0%	1 0%
Nonwords	41	27 66%	12 29%	3 7%	-	1 2%	0 0%

TG made only 2 voicing errors out of 503 words he read (0.4%), and made no voicing errors in reading nonwords. TG's reading did include errors, but they were not of the type that was evident in his writing: he made 12% errors typical to surface dyslexia, and 8% errors of letter migrations within words, typical of letter position dyslexia (Friedmann & Gvion, 2001; Friedmann & Haddad-Hanna, in press; Friedmann & Rahamim, 2007) he also made 14 between-word migrations, typical of attentional dyslexia (Friedmann, Kerbel, & Shvimer, in press; Hall et al., 2001; Mayall & Humphreys, 2002; Saffran & Coslett, 1996; Shallice & Warrington, 1977; Warrington, Cipolotti, & McNeil, 1993), and 25 additional errors, which included mainly vowel letter errors (additions, omissions, and substitutions of vowels – indicative of a DIVL, difficulty in vowel letters). Thus, the patterns of TG's errors in reading and writing is completely different. Whereas in writing he makes a considerable number of voicing errors, in reading he makes no voicing errors; He makes letter position errors in reading, but not in writing (in writing he made 1% of such errors, which is within the normal range). The only parallel we found between his reading and writing related to the use of the sublexical route - he makes errors that result from using the sublexical, rather than the lexical, route in both reading and writing, indicating surface dyslexia and surface dysgraphia. Thus, his performance forms a clear dissociation between voicing conversion in reading and in writing.

BG's reading showed a different picture: she made very few voicing errors in reading words (3%), but when reading nonwords, and hence reading via grapheme-to-phoneme conversion route, she made 29% such errors. She also made sibilant substitution errors, which might indicate a deficit in an additional conversion function, one that is responsible for sibilants. This critical difference between words and nonwords supports the localization of voicing errors in the conversion route. Because BG reads words primarily via the lexical route (as indicated by the very small rate of surface dyslexia errors in her reading, 5%), the voicing deficit does not manifest itself in words (or manifests itself only to the small extent of her use of the sublexical route for word reading). However, when, she is required to read via the sublexical route, in the reading of nonwords, the voicing conversion deficit is manifested, leading to voicing errors in reading. Importantly, BG's voicing errors cannot be ascribed to an output deficit (impaired phonemic output buffer) because such errors did not occur in her spontaneous speech, repetition, or naming. Interestingly, patient DR, described in the rehabilitation study of Luzzatti et al. (2000) showed a similar pattern of devoicing errors in nonword reading, and devoicing in writing both words and nonwords.

Thus, whereas TG demonstrated a clear dissociation between impaired voicing in writing and intact voicing in reading, BG had voicing errors while reading via the sublexical route, as summarized in Figure 4.[10]

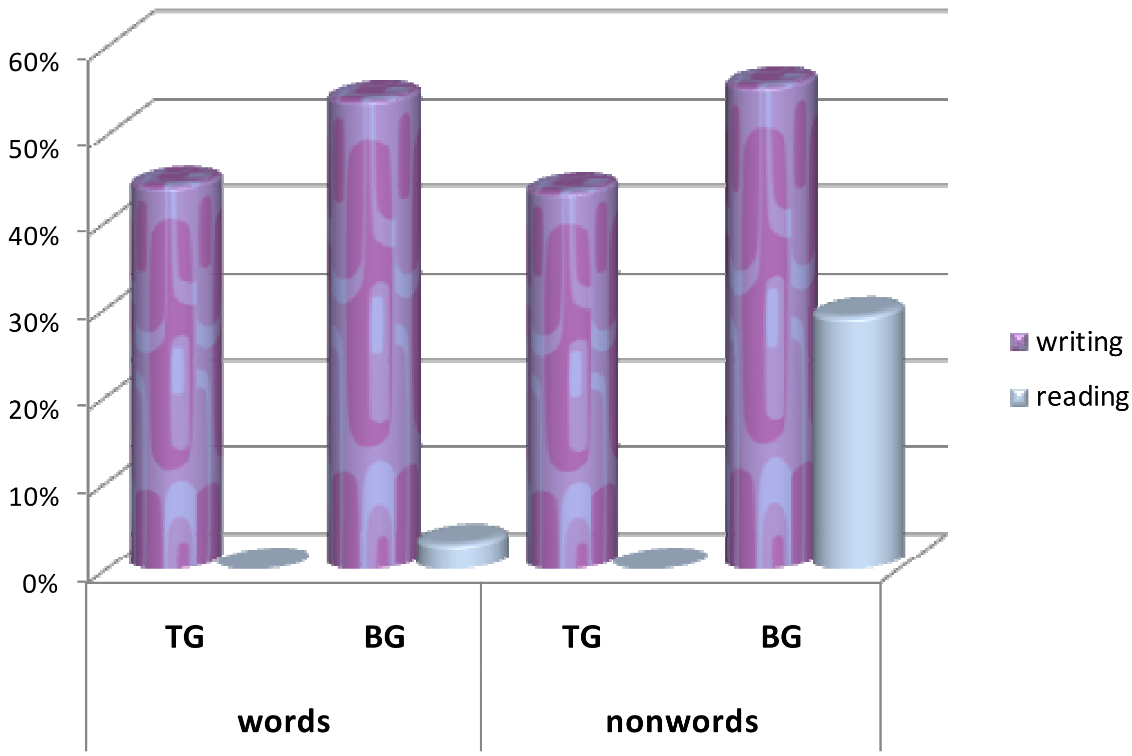

Figure 4. Voicing errors in writing and in reading

[10] BG's reading and writing reported in this paper are from the acute phase. In an additional testing a month and a half later (2.5 months post onset), BG showed some improvement in reading and writing. In reading 184 words she made 4 voicing errors (2%), 2 errors in sibilants, and 2 surface dyslexia errors. In reading 30 nonwords she made 3 voicing errors (10%) and 3 sibilant errors. In writing to 60 words to dictation she made 6 voicing errors (10%) and 4 surface errors. In writing 10 nonwords she made 2 voicing errors (20%) and 2 sibilants errors.

6. DISCUSSION

This study explored in detail the nature of voicing substitution errors in the writing of two individuals with acquired dysgraphia, and attempted to identify the functional source of the deficit.

6.1. Dyscravia: Voicing errors in writing, their origin, and implications for the writing model

The participants made voicing errors while writing words and nonwords, and in various tasks – writing to dictation, written naming, and oral spelling. Importantly, they showed good word and nonword repetition and good auditory discrimination, indicating intact phonological input and output. We also ruled out the orthographic lexicon and the graphemic output buffer as possible sources for the deficit, firstly because of theoretical considerations: both are orthographic creatures, which process graphemic, rather than phonemic representations, and it is therefore hard to see how an impairment in either of them can create voicing errors, which are phonological in nature. Beyond these theoretical considerations, we also ran tests and analyses that ruled out these components as possible sources of dyscravia: a deficit in the lexicon might have caused the lexical entries to be represented with voicing-incorrect graphemes. Hence, we would expect consistent errors in these ill-represented words. However, we asked the participants to write the same words multiple times, and they made various different errors in each of these words. Furthermore, a straightforward exclusion for the lexicon as the source of the deficit is that nonwords showed voicing errors just like words (and even in a higher rate for BG). We also conducted analyses to assess the possibility of a deficit at the graphemic buffer. We conjectured, that if the deficit is at the graphemic buffer, then it is due to decay of features, which should be more severe the longer the word is, and maybe more severe in non-stressed than stressed syllables. However, the results showed no length effect, and no difference between stressed and non-stressed syllables. There was also no position-sensitive effect, characteristic of graphemic buffer dysgraphia. Hence, the graphemic buffer is also not the source of dyscravia. The allographic stage was also ruled out on similar theoretical grounds – as it is also strongly graphemic, not phonemic. It was also ruled out on the basis of empirical results: voicing errors occurred also in oral spelling, suggesting that the deficit resides in an earlier stage in the process of writing.

We suggest that the deficit that causes voicing errors in these patients' writing is a deficit in the phoneme-to-grapheme conversion route, which has selectively impaired a specific function converting phonemes with a voicing distinctive feature to the appropriate graphemes. Because our participants also had surface dyslexia, their over-reliance on the sublexical route made this phenomenon evident also on words, and not only nonwords.

Thus, the participants' writing pattern resulted from a deficit in two locations: a deficit in the lexical route that causes writing via the sublexical route (surface dysgraphia), and another deficit in the phoneme-to-grapheme conversion, in the voicing conversion function (*dyscravia*).[11]

If indeed the locus of the impairment that causes voicing errors in spelling is in the conversion route, it will only be possible to detect *dyscravia* when writing via phoneme-to-grapheme conversion. Namely, dyscravia can be detected either for individuals with surface dysgraphia who, because of their dysgraphia, are forced to write words via the sublexical route, or for individuals without surface dysgraphia, when writing unfamiliar words and nonwords (whereas their writing of familiar words that are represented in the orthographic lexicon should not involve voicing errors).

Probably the most important outcome of this study is the light it sheds on the process of phoneme-to-grapheme conversion. It suggests that the conversion is not of a whole phoneme to a grapheme, but rather that the conversion is done feature-by-feature. One way to think of this conversion would be that the phonemes arrive from the phonemic output buffer as bundles of features, which then activate a grapheme that corresponds to the phonemic specification. This bundle might include not only voicing, but other phonemic features that are relevant for distinctions between letters, such as place of articulation, manner of articulation, etc. Thinking about the function of the phonemic output buffer, it might not be surprising that its output is phonemic feature bundles rather than whole phonemes. Notice, that the phonemic output buffer not only holds phonemes activated for a short term, but it is also where co-articulation and assimilation effects take place, so it has to be sensitive to voicing features.[12] Moreover, given that the phonemic buffer provides input to articulation mechanisms, which also need to receive information with respect to voicing and other phonemic features, it seems natural that its output will include these specifications. Some studies of skilled reading (using masked priming and ERP) also indicate the involvement of

[11] We also worked with two children who had developmental dyscravia (and surface dysgraphia). These children showed very similar error patterns in writing to the ones reported here for TG and BG.

[12] It might not be accidental that we see selective errors in voicing of all phonemic features. Voicing features are often involved in phonological processes such as voicing assimilation, and it may thus be that they are more prone to conversion errors than more stable and consistent phonological features such as place and manner of articulation.

sub-phonemic feature information and specifically, voicing, during word and nonword reading (Ashby, Sanders, & Kingston, 2008; Kinoshita, 1999).

Further dysgraphia cases with selective impairments to other phonological features might shed more light as to the features that are relevant for the conversion. For example, one open question is whether each of the phonological features can be selectively impaired, or whether the impairment follows the hierarchy suggested by Clements (1985) and McCarthy (1988). If the feature conversion impairment obeys this hierarchy, the voice feature can be impaired selectively, but an impairment at higher nodes in the hierarchy, like the supralaryngeal node, which governs both the Place and the Manner nodes, are expected to cause impairment in all the features below it.

Thus, the current study forms another example for how neuropsychological studies can have implications to the theory of the cognitive model, in this case – the normal writing model.

6.2. Voicing errors in reading: dissociation and a new type of dyslexia

Another two new findings of this research related to voicing errors in reading. Firstly, one of the participants, TG, showed a clear dissociation between writing and reading – whereas his writing showed a considerable rate of voicing errors, his reading did not include such errors. This dissociation suggests that the voicing conversion functions in reading and in writing are separate and hence, can be separately impaired.[13]

The other finding of this study was that a parallel dyslexia, *dyslegzia*, can exist. BG showed the same pattern of voicing errors when she read aloud via the grapheme-to-phoneme

[13] The dissociation found between voicing errors in writing but not in reading cannot be ascribed to writing being harder than reading, as a reviewer suggested. First, it is not clear why a harder task should cause voicing errors and not other errors, or why a harder task should cause the use of the sublexical route. Secondly, in Hebrew, using the phoneme-to-grapheme route is more taxing in reading than in writing. Because of the many degrees of freedom, which result from the underrepresentation of vowels in the orthography, and the phoneme-ambiguity of 9 letters, reading via the sublexical route has more degrees of freedom than writing. Whereas, for example, the three-consonant word שפן (SPN) has 288 possible ways to be read via the grapheme-to-phoneme conversion (6*6*2*2*2 = 6 possible vowels per each non-final consonant, 2 letters that could be mapped to 2 phonemes each, and 2 possible stress positions), it has a single way to be written via phoneme-to-grapheme conversion. Moreover, dissociation in the other direction has been found in Hebrew in another individual with a dyslexia in the grapheme-to-phoneme conversion route. AE, reported in Gvion and Friedmann (in press) had a phonological dyslexia, which caused 13% (55/429) morphological errors in reading, but he had only few morphological errors in spelling 4% (49/1093). TG in the current study had 8% letter position errors in reading, but not in writing (where he made 1% of such errors). If spelling had been simply more taxing for any difficulty in grapheme-to-phoneme conversion, we would expect the participants to have more morphological errors and more transposition errors in writing than in reading, but the pattern was just the opposite. Finally, even if spelling were more liable to using the sublexical route, and reading more liable to use the lexical route, we would still expect to see voicing errors in reading when TG read nonwords, because nonwords have to be read via the sublexical route. However, TG did not make voicing errors on nonword reading either.

conversion route. Namely, when she read nonwords, she made voicing errors, converting graphemes to their voiced or unvoiced counterpart phonemes. Again, given that she had no difficulties in repetition and auditory discrimination, and because her errors occurred only when reading nonwords, we suggest that her voicing dyslexia resulted from a deficit in the function that converts graphemes to voicing features of phonemes.

References

Annoni, J. M., Lemay, M. A., de Mattos Pimenta, M. A., & Lecours, A. R. (1998). The contribution of attentional mechanisms to an irregularity effect at the graphemic buffer level. *Brain and Language*, *63*, 64-78.

Ashby, J., Sanders, L. D., & Kingston, J. (2009). Skilled readers begin processing sub-phonemic features by 80 ms during visual word recognition: Evidence from ERPs. *Biological Psychology*, *80*, 84-94.

Barry, C. (1994). Spelling Routes (or roots or rutes). In G. D. A. Brown & N. C. Ellis (Eds.), *Handbook of spelling: Theory, process, and intervention* (pp. 27-49). Chichester: J. Wiley & sons.

Baxter, D. M., & Warrington E. K. (1983). Neglect dysgraphia. *Journal of Neurology, Neurosurgery, and Psychiatry*, *46*, 1073-1078.

Beeson, P. M., & Rapcsak, S. Z. (2002). Clinical diagnosis and treatment of spelling disorders. In A. E. Hillis (Ed.), *The handbook of adult language disorders*. New York & Hove: Psychology Press.

Behrmann, M., & Bub, D. (1992). Surface dyslexia and dysgraphia: Dual routes, single lexicon. *Cognitive Neuropsychology*, *9*, 209-251.

Biran, M., & Friedmann, N. (2004). *SHEMESH: Naming a hundred objects*. Tel Aviv University. (in Hebrew).

Blanken, G., Schafer, C., Tucha, O., & Lange, K. W. (1999). Serial processing in graphemic encoding: Evidence from letter exchange errors in a multilingual patient. *Journal of Neurolinguistics*, *12*, 13-39.

Bub, D., & Kertesz, A. (1982). Deep agraphia. *Brain and Language*, *17*, 146-165.

Caramazza, A., & Hillis, A. E. (1990). Spatial representation of words in the brain implied by studies of a unilateral neglect patient. *Nature*, *346*, 267-269.

Caramazza, A., & Miceli, G. (1990). The structure of the graphemic representation. *Cognition*, *37*, 243-297.

Caramazza, A., Miceli, G., Villa, G., & Romani, C. (1987). The role of the graphemic buffer in spelling: Evidence from a case of acquired dysgraphia. *Cognition*, *26*, 59-85.

Chialant, D., Domoto-Reilly, K., Proios, H., Caramazza, A. (2002). Preserved orthographic length and transitional probabilities in written spelling in a case of acquired dysgraphia. *Brain and Language*, *82*, 30-46.

Cholewa, J., Mantey, S., Heber, S., & Hollweg, W. (2010). Developmental surface and phonological dysgraphia in German 3rd graders. *Reading and Writing*, *23*, 97-127.

Cipolotti, L., Bird, C. M., Glasspool, D. W., & Shallice, T. (2004). The impact of deep dysgraphia on graphemic buffer disorder. *Neurocase*, *10*, 405-419.

Clements, G. N. (1985). The geometry of phonological features. *Phonology Yearbook*, *2*, 225-252.

Cotelli, M., Abutalebi, J., Zorzi, M., & Cappa, S. (2003). Vowels in the buffer: A case study of acquired dysgraphia with selective vowel substitutions. *Cognitive Neuropsychology*, *20*, 99-114.

De Bastiani, P., & Barry, C. (1989). A cognitive analysis of an acquired dysgraphic patient with an allographic writing disorder. *Cognitive Neuropsychology*, *6*, 25-41.

Del Grosso Destreri, N., Farina, E., Alberoni, M., Pomati, S., Nicchelli, P., & Mariani, C. (2000). Selective upper case dysgraphia with loss of visual imagery of letter forms: A window on the organization of graphomotor patterns. *Brain and Language*, *71*, 353-372.

Delazer, M., Lochy, A., Jenner, C., Domahs, F., & Benke, Th. (2002). When writing 0 (zero) is easier than writing O(o): A neuropsychological case study of agraphia. *Neuropsychologia*, *40*, 2167-2177.

Ellis, A. W. (1993). *Reading, writing, and dyslexia: A cognitive analysis*. Hove: Erlbaum.

Folk, J. R., Rapp, B., & Goldrick, M. (2002). The interaction of lexical and sublexical information in spelling: What's the point? *Cognitive Neuropsychology*, *19*, 653-671.

Friedmann, N. (1998). *BAFLA*: *Friedmann Battery for Agrammatism*. Tel Aviv University. (in Hebrew)

Friedmann, N., & Gvion, A. (2001). Letter position dyslexia. *Cognitive Neuropsychology*, *18*, 673-696.

Friedmann, N., & Gvion, A. (2002). *FriGvi*: *Friedmann Gvion battery for assessment of phonological Working Memory*. Tel Aviv University. (in Hebrew)

Friedmann, N., & Gvion, A. (2003a). Sentence comprehension and working memory limitation in aphasia: A dissociation between semantic and phonological encoding. *Brain and Language*, *86*, 23-39.

Friedmann, N., & Gvion, A. (2003b). *TILTAN: Battery for the diagnosis of dyslexias*. Tel Aviv University. (in Hebrew)

Friedmann, N., & Haddad-Hanna, M. (in press). Letter position dyslexia in Arabic: From form to position. *Behavioural Neurology*.

Friedmann, N., Kerbel, N., & Shvimer, L. (in press). Developmental attentional dyslexia. *Cortex*.

Friedmann, N., & Lukov, L. (2008). Developmental surface dyslexias. *Cortex*, *44*, 1146-1160.

Friedmann, N., & Rahamim, E. (2007). Developmental letter position dyslexia. *Journal of Neuropsychology*, *1*, 201-236.

Friedmann, N., & Shapiro, L. P. (2003). Agrammatic comprehension of simple active sentences with moved constituents: Hebrew OSV and OVS structures. *Journal of Speech*, *Language, and Hearing Research*, *46*, 288-297.

Gil, M., & Edelstein, C. (1999). *Hebrew version of the PALPA*. Ra'anana, Israel: Loewenstein Hospital Rehabilitation Center.

Glasspool, D. W., Shallice, T., & Cipolotti, L. (2006). Towards a unified process model for graphemic buffer disorder and deep dysgraphia. *Cognitive Neuropsychology*, *23*, 479-512.

Graham, N. L., Patterson, K., & Hodges, J. R. (1997). Progressive dysgraphia: Co-occurrence of central and peripheral impairments. *Cognitive Neuropsychology*, *14*, 975-1005.

Graham, N. L., Patterson, K., & Hodges J. R. (2000). The impact of semantic memory impairment on spelling: Evidence from semantic dementia. *Neuropsychologia*, *38*, 143-163.

Gvion, A., & Friedmann, N. (2007). Is phonological working memory involved in sentence comprehension? The difference between phonological and semantic reactivation. In Y. Falk (Ed), *The Israel Association of Theoretical Linguistics*, *23*, 1-54.

Gvion, A., & Friedmann, N. (2008a). FriGvi: A battery for the assessment of phonological working memory. *Language and Brain*, *7*, 161-181. (in Hebrew)

Gvion, A., & Friedmann, N. (2008b). *KRUV: Homophone and potentiophone selection test*. Tel Aviv University. (in Hebrew)

Gvion, A., & Friedmann, N. (in press). Letter position dysgraphia. *Cortex*. doi:10.1016/j.cortex.2009.09.008.

Hall, D. H., Humphreys, G. W., & Cooper, A. C. G. (2001). Neuropsychological evidence for case-specific reading: Multi-letter units in visual word recognition. *The Quarterly Journal of Experimental Psychology*, *54*, 439-467.

Hanley, J. R., & Peters, S. (2001). Allograph errors and impaired access to graphic motor codes in a case of unilateral agraphia of the dominant left hand. *Cognitive Neuropsycholgy*, *18*, 307-321.

Hillis, A. E., & Caramazza, A. (1995). Spatially specific deficits in processing graphemic representations in reading and writing. *Brain and Language*, *48*, 263-308.

Hillis, A. E., Rapp, B. C., & Caramazza, A. (1999). When a rose is a rose in speech but a tulip in writing. *Cortex*, *35*, 337-356.

Iribarren, I. C., Jarema, G., & Lecours, A. R. (2001). Two different dysgraphic syndromes in a regular orthography, Spanish. *Brain and Language*, *77*, 166-175.

Jónsdóttir, M. K., Shallice, T., & Wise, R. (1996). Phonological mediation and the graphemic buffer disorder in spelling: Cross-language differences? *Cognition*, *59*, 169-197.

Kay, J., & Hanley, R. (1994). Peripheral disorders of spelling: The role of the graphemic buffer. In G. D. A. Brown & N. C. Ellis (Eds.), *Handbook of spelling*: *Theory*, *process*, *and intervention* (pp. 295-315). Chichester: J. Wiley & sons.

Kay, J., Lesser, R., & Coltheart, M. (1992). *PALPA: Psycholinguistic Assessments of Language Processing in Aphasia*. Hove, UK: Erlbaum.

Kertesz, A. (1982). *Western Aphasia Battery*. Orlando, FL: Grune and Stratton.

Kinoshita, S. (1999, November). *The phonological nature of the masked onset priming effect*. Paper presented at the Second Lexical Processing Workshop, Tucson, AZ.

Kirk, A., Blonder, L. X., Wertman, E., & Heilman, K. M. (1991). Phonolexical agraphia: Superimposition of acquired lexical agraphia on developmental phonological dysgraphia. *Brain*, *114*, 1977-1996.

Kokubo, K., Suzuki, K., Yamadori, A., & Satou, K. (2001). Pure Kana agraphia as a manifestation of graphemic buffer impairment. *Cortex*, *37*, 187-195.

Ladefoged, P. (1993). *A course in phonetics* (3rd ed.). Fort Worth, TX: Harcourt, Brace, and Jovanovich.

Luzzatti, C., Colombo, C., Frustaci, M., & Vitolo, F. (2000). Rehabilitation of spelling along the sub-word-level routine. *Neuropsychological Rehabilitation*, *10*, 249-278.

Mayall, K., & Humphreys, G. W. (2002). Presentation and task effects on migration errors in attentional dyslexia. *Neuropsychologia*, *40*, 1506-1515.

McCarthy, J. J. (1988). Feature geometry and dependency: A review. *Phonetica*, *45*, 84-108.

Menichelli, A., Rapp, B., & Semenza, C. (2008). Allographic dysgraphia: A case study. *Cortex*, *44*, 861-868.

Miceli, G., Benvegnù, B., & Capasso, R. (1995). Selective deficit in processing double letters. *Cortex*, *31*, 161-171.

Miceli, G., & Capasso R. (2006). Spelling and dysgraphia. *Cognitive Neuropsychology*, *23*, 110-134.

Miceli, G., Capasso, R., Ivella, A., & Caramazza, A. (1997). Acquired dysgraphia in alphabetic and stenographic handwriting. *Cortex*, *33*, 355-367.

Miceli, G., Silveri, M. C., & Caramazza, A. (1985). Cognitive analysis of a case of pure dysgraphia. *Brain and Language*, *25*, 187-212.

Miozzo, M., & De Bastiani, P. (2002). The organization of letter-form representations in written spelling: Evidence from acquired dysgraphia. *Brain and Language*, *80*, 366-392.

Neils, J., Roeltgen, D. P., & Greer, A. (1995). Spelling and attention in early Alzheimer’s Disease: Evidence for impairment of the graphemic buffer. *Brain and Language*, *49*, 241-262.

Ogden, J. A. (1996). Phonological dyslexia and phonological dysgraphia following left and right hemispherectomy. *Neuropsychologia*, *34*, 905-918.

Panton, A., & Marshall, J. (2007). Improving spelling and everyday writing after a CVA: A single-case therapy study. *Aphasiology*, *1*, 1-20.

Posteraro, L., Zinelli, P., & Mazzucchi A. (1988). Selective impairment of the graphemic buffer in acquired dysgraphia: A case study. *Brain and Language*, *35*, 274-286.

Raman, I., & Weekes, B. S. (2005). Deep dysgraphia in Turkish. *Behavioural Neurology*, *16*, 59-69.

Rapcsak, S. Z., & Beeson, P. M. (2002). Neuroanatomical correlates of spelling and writing. In A. E. Hillis (Ed.), *The handbook of adult language disorders*. New York & Hove: Psychology press.

Rapp, B. (2005). The relationship between treatment outcomes and the underlying cognitive deficit: Evidence from the remediation of acquired dysgraphia. *Aphasiology*, *19*, 994-1008.

Rapp, B., Epstein, C., & Tainturier, M. J. (2002). The integration of information across lexical and sublexical processes in spelling. *Cognitive Neuropsychology*, *19*, 1-29.

Rapp, B., & Kong D. (2002). Revealing the component functions of the graphemic buffer. *Brain and Language*, *83*, 112-114.

Romani, C., Ward, J., & Olson, A. (1999). Developmental surface dysgraphia: What is the underlying cognitive impairment? *The Quarterly Journal of Experimental Psychology*, *52*, 97-128.

Saffran, E. M., & Coslett, H. B. (1996). "Attentional dyslexia" in Alzheimer's disease: A case study. *Cognitive Neuropsychology*, *13*, 205-228.

Sage, K., & Ellis, A. W. (2004). Lexical influences in graphemic buffer disorder. *Cognitive Neuropsychology*, *21*, 381-400.

Shallice, T., Rumiati, R. I., & Zadini, A. (2000). The selective impairment of the phonological output

buffer. *Cognitive Neuropsychology*, *17*, 517-546.
Shallice, T., & Warrington, E. K. (1977). The possible role of selective attention in acquired dyslexia. *Neuropsychologia*, *15*, 31-41.
Schiller, O. S., Greenhall, J. A., Shelton, J. R., & Caramazza, A. (2001). Serial order effects in spelling errors: Evidence from two dysgraphic patients. *Neurocase*, *7*, 1-14.
Schocken, R. (2008). *Characterization and evaluation of speech-reading support systems for hard-of-hearing people*. Unpublished doctoral dissertation, Tel-Aviv University, Tel-Aviv, Israel. (in Hebrew)
Soroker, N. (1997). *Hebrew Western Aphasia Battery*. Ra'anana, Israel: Loewenstein Hospital Rehabilitation Center. (in Hebrew).
Tainturier, M. J. (1996). Phonologically-based errors and their implications in the specification of phonology to orthography conversion processes. *Brain and Cognition*, *32*, 148-151.
Tainturier, M. J., & Caramazza, A. (1996). The status of double letters in graphemic representations. *Journal of Memory and Language*, *35*, 53-73.
Tainturier, M. J., & Rapp, B. (2001). The spelling process. In B. Rapp (Ed.), *The handbook of cognitive neuropsychology: What deficits reveal about the human mind*. Philadelphia: Psychology Press.
Tainturier, M. J., & Rapp, B. C. (2004). Complex graphemes as functional spelling units: Evidence from acquired dysgraphia. *Neurocase, 10*, 122-131.
Temple, C. M. (1985). Developmental surface dysgraphia: A case report. *Applied Psycholinguistics*, *6*, 391-406.
Venneri, A., Pestell, S. J., & Caffarra, P. (2002). Independent representations for cursive and print style: Evidence from dysgraphia in Alzheimer's disease. *Cognitive Neuropsychology*, *19*, 387-400.
Ward, J., & Romani, C. (2000). Consonant-vowel encoding and ortho-syllables in a case of acquired dysgraphia. *Cognitive Neuropsychology*, *17*, 641-663.
Warrington E. K., Cipolotti L., & McNeil, J. (1993). Attentional dyslexia: A single case study. *Neuropsychologia*, *31*, 871-885.
Weekes, B. S. (2006). Deep dysgraphia: Evidence for a summation account of written word production. *Brain and Language*, *99*, 21-22.
Weekes, B., & Coltheart, M. (1996). Surface dyslexia and surface dysgraphia: Treatment studies and their theoretical implications. *Cognitive Neuropsychology*, *13*, 277-315.
Zesiger, P., Martory, M.-D., & Mayer, E. (1997). Writing without graphic motor patterns: A case of dysgraphia for letters and digits sparing shorthand writing. *Cognitive Neuropsychology*, *14*, 743-763.