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Underlying Cause(s) of Letter Perseveration Errors

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Abstract

Perseverations, the inappropriate intrusion of elements from a previous response into a current response, are commonly observed in individuals with acquired deficits. This study specifically investigates the contribution of failure-to activate and failure-to-inhibit deficit(s) in the generation of letter perseveration errors in acquired dysgraphia. We provide evidence from the performance of 12 dysgraphic individuals indicating that a failure to activate graphemes for a target word gives rise to letter perseveration errors. In addition, we also provide evidence that, in some individuals, a failure-to-inhibit deficit may also contribute to the production of perseveration errors.

Keywords

letter perseveration errors; dysgraphia; spelling; inhibition

1. INTRODUCTION

1.1 Perseveration errors: Underlying deficits?

Perseveration errors, the inappropriate repetition of a previous response in the place of the current target, are observed across a range of populations and tasks. While these errors have been reported with healthy young adults, in tasks such as serial recall (Conrad, 1960; Estes, 1991; Henson, 1999) and speeded naming (Moses, Nickels & Sheard, 2004), they are more commonly associated with brains that are immature (e.g. infants, Smith et al., 1999, early child language production, Stemberger, 1989), aging (e.g. healthy older adults, Foldi et al., 2003), brain damage (e.g. aphasia, Albert & Sandson, 1986), or other types of neural disruption (e.g. schizophrenia, Crider, 1997).

There are essentially two types of hypotheses regarding the mechanisms underlying perseveration errors. According to what we will call a “*failure-to-inhibit*” hypothesis, a previous response is selected in the place of the present target because it has remained abnormally active after production instead of being properly inhibited (e.g. Arbuthnott, 1996; Frankel & Penn, 2007; Hauser, 1999; Hudson, 1968; Diamond, Cruttenden & Neiderman, 1994; Stemberger, 1989; Yamadori, 1981). Alternatively, according to what we will refer to as a “*failure-to-activate*” hypothesis, perseveration errors arise when the

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current target receives abnormally little activation. Most theories of cognitive processing assume that recently produced responses remain active during the current trial, either because of residual activation (e.g. Dell, Svec & Burger, 1997) or incremental learning (e.g. Gotts & Plaut, 2004; Oppenheim, Dell & Schwartz, 2010). Therefore, given abnormally low activation for the current trial and residual activation from previous responses, perseveration errors are produced (e.g., Ackerman & Ellis, 2007; Cohen & Dehaene, 1998; Dell, Svec & Burger, 1997; Gotts, Incisa & Cipolotti, 2002; Gotts & Plaut, 2004; Hirsh, 1996; Martin & Dell, 2007; Martin, Roach, Brecher & Lowery, 1998; Moses, Sheard & Nickels 2007; Oppenheim, Dell & Schwartz, 2010). As Stark (2007) points out, these conflicting accounts of perseveration errors have a long history dating back to at least the middle of the 19th century. Much of the research to date has considered these two hypotheses as alternative accounts of perseveration errors, implicitly assuming that all individuals who persevere have the same underlying deficit. However, another possibility is that both hypotheses are correct, with some individuals suffering from one deficit, the other or both.

In this paper, the specific type of perseverations we will consider are the perseverations of letters from a previous written response into a subsequent response¹, produced by individuals with acquired dysgraphia as a result of neural insult. As an example, consider the misspelling of the target word “edge” as ERGE, in which the R is an intrusion into the current response. The fact that the R appears in a previous written response, FRENCE, indicates that it may be a perseveration from that previous response. We have recently reported on various dysgraphic individuals who produced significant numbers of letter perseveration errors in spelling-to-dictation (Fischer-Baum, McCloskey & Rapp, 2010; Rapp & Fischer-Baum, in press, McCloskey, Macaruso & Rapp, 2006), and there have been a number of other reports of similar error patterns in the literature (e.g., Basso, 2004; Hudson, 1968; Pickard, McAllister & Horton, 2010; Rohrer, Rossor & Warren, 2009; Schonauer & Denes, 1994; Tanaka, Yamadori & Murata, 1987; Varley et al., 2005). In this paper we examine if failure-to-activate, failure-to-inhibit or both types of failures are responsible for letter perseveration errors.

In recent work in the domain of verbal perseverations, the evidence appears to largely support a *failure-to-activate* account (e.g., Ackerman & Ellis, 2007; Cohen & Dehaene, 1998; Dell, Svec & Burger, 1997; Hirsh, 1996; Martin & Dell, 2004, 2007; Martin, Roach, Brecher & Lowery, 1998; Moses, Sheard & Nickels, 2007). A failure-to-activate hypothesis can fairly directly account for the verbal perseverations observed in some individuals. For example, Cohen and Dehaene (1998) reported on an individual with damage to the corpus callosum, but without damage to the language areas of the left hemisphere. As a result of the callosal lesion, the language areas of the brain were not activated normally from stimuli (either written words or pictures) presented in the left visual field and this individual was severely impaired in both reading and naming pictures presented in the left visual field. Consistent with the failure-to-activate hypothesis, many of the errors were perseverations of responses from previous trials.

A number of studies have made use of the following logic in attempting to adjudicate between the two hypotheses (e.g., Cohen & Dehaene, 1998; Martin & Dell, 2004, 2007; Moses, Nickels & Sheard, 2007). They have assumed that in cases in which there is a failure to properly activate the target element, perseveration errors will occur because previous

¹Letter perseveration errors in which the letter perseverations occur between spelling responses should be distinguished from letter perseveration errors in which the letter perseverations occur within the same trial (e.g. spelling EDGE as EDGGGE). See Caccappolo-van Vliet, Miozzo, Marder and Stern (2003), Neils-Strunjas, Shuren, Roeltgen and Brown (1998) and Venneri, Cubelli and Caffarra (1994) for discussions of these errors. In the Sandson and Albert (1984) taxonomy of perseveration errors, the between trials perseverations, which we are concerned with in this paper, are *recurrent* letter perseveration errors, as opposed to the within-trial *continuous* letter perseveration errors.

responses normally retain some activation and may be erroneously selected for production. Critically, however, other error types should occur as well, such as the intrusions of elements that did not appear in recent responses or omission errors, in which no response is produced. The prediction is that with increasing severity of the failure-to-activate deficit, the number of perseveration errors should increase, as should the number of non-perseverative intrusion errors and omission errors. In contrast, with a failure-to-inhibit deficit, both the previous responses and the current target are highly active. In that case, either a correct response or a perseveration error should be produced, but other types of errors are not predicted.² On the basis of this logic, for example, Martin and Dell (2007) reported correlations between perseverative and non-perseverative errors across a large set of aphasic individuals.

In this paper, we address the underlying causes of letter perseveration errors with an examination of the performance of a set of dysgraphic individuals selected because their spelling performance exhibited the hallmark features of a failure-to-activate deficit. As a framework for subsequent discussion, we begin by outlining the cognitive architecture of spelling, highlighting the level of representation at which we assume these letter perseveration errors arise as well as the upstream levels that provide input to that level. This theoretical framework will motivate the selection criteria for identifying individuals who are impaired at activating letters when spelling.

1.2 A framework for evaluating spelling and letter perseveration errors

Most of the data we report come from spelling to dictation (a word or nonword is dictated, and the participant produces a written spelling response). We also report results from a second spelling task – direct copy transcoding—in which participants see a printed word written in upper case or lower case and copy it into the opposite case, while the word remains visible. We describe a cognitive spelling theory that specifies the representations and processes required for both tasks (see Tainturier & Rapp, 2001, and Miceli & Capasso, 2006).

According to the spelling framework depicted in Figure 1, when a familiar word (e.g., “table”) is dictated, the corresponding phonological word form is activated in a phonological lexicon. This word form provides access to the word’s lexical-semantic representation, which in turn forms the basis for the retrieval of the corresponding orthographic word form in an orthographic lexicon. Some authors have also proposed a direct connection between the phonological and orthographic word forms (e.g., Patterson, 1986). The lexicons and semantic system correspond to long-term memory stores of knowledge of familiar words. Once an orthographic word form has been retrieved, it activates graphemic representations that specify the identities and ordering of the letters in the spelling of the word (e.g., T-A-B-L-E; see Fischer-Baum, McCloskey and Rapp, 2010, for an in-depth discussion of grapheme order representation). These graphemic representations are assumed to be abstract in the sense that they do not include information regarding the format of the letter to be produced. That is, the graphemic representation of TABLE includes the same T grapheme whether the word is to be typed, handwritten or spelled aloud. (Note that we use the terms *grapheme* and *graphemic* simply to refer to abstract letter representations, and not specifically to letter representations corresponding to single phonemes.) In terms of this investigation, it is important to note that we assume that disruption at the level of the orthographic word forms

²It is worth noting that the level of processing at which the failure-to-activate or failure-to-inhibit deficit occurs will affect both the types of perseveration errors observed and the other types of errors expected. If failures occur at a lexical level, whole word perseveration errors are predicted to occur and, under the failure-to-activate hypothesis, also other whole word errors. If the failure occurs at a phonological level, segmental perseveration errors are predicted to occur and, under the failure-to-activate hypothesis, also other segmental errors (see Moses, Sheard & Nickels, 2007 for a discussion of this point in the context of spoken production).

may result in a failure to activate the target graphemes from this stored knowledge about the spelling of the word.

The phoneme-to-grapheme conversion process (PGC process) uses (sublexical) knowledge of the relationships between the sounds and letters of English (e.g., /f/ is usually spelled with the letter F) to generate plausible spellings for phonological strings. For example, when an unfamiliar phonological string (corresponding to a novel word or nonword, such as /floʊp/) is dictated, no corresponding phonological word form or lexical-semantic representation is available, but the phoneme-to-grapheme conversion process can generate a plausible spelling (e.g., F-L-O-P-E). Phoneme-grapheme conversion deficits, or what we will call *sublexical* deficits, result in a failure to activate plausible target graphemes. It is also assumed that the PGC process is engaged by any phonological string whether it corresponds to a familiar word or not. As a result, a common grapheme level is activated by both the lexical and sublexical routes during the normal course of spelling. One function of the graphemic level of representation is to combine the information provided by the two routes (Rapp, Epstein & Tainturier, 2002; Folk, Goldrick & Rapp, 2002; Folk & Rapp, 2004).

Abstract graphemic representations provide the basis for activating format-specific letter representations. In oral spelling, letter-name representations are selected (e.g., /ti/ for the letter T) while, during written spelling, letter shapes corresponding to the intended case and font are selected. During the serially ordered selection of letter-shape or -name representations, the graphemic representations are assumed to be processed within a working memory system referred to as the graphemic buffer (Caramazza, Miceli & Villa, 1987) or orthographic working memory (see Buchwald and Rapp, 2010).

It is important to note, for the purposes of this paper, that abstract graphemic representations can be activated in tasks other than spelling tasks, including tasks that do not require the lexical or sublexical spelling routes. For example, in a direct copy transcoding task, participants see a word (or other letter string) in one case and copy it into the opposite case. This task relies on gaining access to abstract graphemic representations that are activated by the visually-presented letter string. That is, an abstract graphemic representation of each letter (e.g., lower-case letter *a*) must be activated (in addition to a case-specific letter representation) in order to provide access to the multiple formats available for letter production (in this task, the uppercase form: A). However, unlike in spelling, the abstract graphemic representations can be accessed by visual processes rather than by the lexical or sublexical routes. Furthermore, because the target remains present during the entire trial, this task does not depend on graphemic buffering during letter-shape selection, and a letter-by-letter transcoding strategy is possible. (Note: another similar task that does not depend on lexical and sublexical processes but requires graphemic representations and additionally requires graphemic buffering is the delayed copy transcoding task. It is identical to direct copy transcoding except that the target is removed from sight during letter production).

1.2.1 Letter perseveration errors arising at the graphemic level—In previous work, we argued that the letter perseveration errors produced by two dysgraphic individuals (LSS and CM) during spelling-to-dictation, arose at the level of graphemic representations (see Fischer-Baum, McCloskey & Rapp, 2010; McCloskey, Macaruso & Rapp, 2006). At this level of representation, graphemes from previous responses compete with the current target sequence. When a grapheme from the previous response is selected in place of a grapheme in the current target, a letter perseveration error occurs. Evidence that the letter perseveration errors produced by these two dysgraphic individuals arose at the graphemic level of representation came from several sources. First, LSS and CM perseverated at comparable rates in different spelling modalities (CM: written and typed spelling, LSS: written and oral spelling), indicating a deficit at a level of representation prior to format-

specific production processes. Second, cross-case perseveration errors occurred such that perseverations were in one case (e.g. an uppercase R in the error “edge” → ERGE) while the source of the perseveration had been produced in a different case (e.g. a lowercase r in the earlier response frence). Third, a phonological locus of the perseveration effect was ruled out with a task that required spelling lists of words that contained the sound /f/ spelled either with the letter F (e.g. AFRAID), or with some other combination of letters (e.g., PH in SPHERE). Perseverations of the letter F into subsequent responses occurred only when previous words contained the letter F (e.g. spelling DIRECT as DIFFENT immediately after producing AFRAID) and not when previous words with the /f/ sound were spelled with other letters. Taken together, this pattern of results clearly indicated a graphemic locus of the letter perseveration effect.

1.3 Predictions from different accounts of perseveration errors

From this previous work we can conclude, at least in spelling-to-dictation and for these two individuals, that graphemes from previous responses were occasionally more active than graphemes in the current target. As a result, previously produced graphemes were selected in the place of current target graphemes, and letter perseveration errors were produced. But this previous work did not examine why the graphemes from the previous trials were more active than the current targets. Perseveration errors could have arisen either because target graphemes failed to activate properly, or because previously produced graphemes failed to be properly inhibited, or a combination of both. These different underlying deficits predict different patterns of perseveration, outlined in Sections 1.3.1 and 1.3.2 below.

1.3.1. Failure-to-activate hypothesis—A failure-to-activate account posits that, in the normal system, previously produced graphemes retain some residual activation (Cohen & Dehaene, 1998) and, therefore, may be selected and produced in the place of underactivated target graphemes. According to the theory summarized in Section 1.2, given that in spelling-to-dictation both the lexical and the sublexical routes activate graphemes, then damage to both routes is necessary for perseveration errors to be produced. This is the case because an intact sublexical route can activate a set of plausible graphemes for the target word, while an intact lexical route can activate the graphemes associated in LTM with the target word. Therefore, if either the lexical or sublexical routes are intact, a dictated word will generate a set of robustly activated graphemes. However, if both the lexical and sublexical routes are impaired, then dictated stimuli may fail to properly activate graphemes. *The first prediction of the failure-to-activate hypothesis, therefore, is that individuals that have both lexical and sublexical spelling deficits may produce letter perseveration errors at above chance rates.*³ Note that this prediction holds for irregularly or inconsistently spelled words as well. The sublexical route is assumed to activate a set of plausible graphemes for any word, although some of those graphemes may be lexically incorrect. For example, in response to the target word “phone”, an intact sublexical route may strongly activate the grapheme F. Although the F is incorrect, because it is strongly activated it is, nonetheless, more likely to be produced than a previously produced grapheme that has only normal levels of residual activation.

³It may be necessary to make some additional assumptions in order for the failure-to-activate hypothesis to predict perseveration errors. If there is weak activation for the graphemes in both Trial 1 and Trial 2, and graphemes retain only residual activation after production, why should graphemes from Trial 1 ever be more active than graphemes in Trial 2? One possibility is that the deficit involves a variable failure to activate target graphemes. Because of this variability, on some trials the target graphemes in Trial 1 may be robustly activated, while the target graphemes in Trial 2 are only weakly activated, resulting in perseveration of graphemes from Trial 1 into Trial 2. Alternatively, the process of selecting a grapheme for production may result in an activation boost (see Dell, Svec & Burger, 1997 for a similar proposal in speech production). While initial activation may be low, as a result of a failure-to-activate deficit, once selected, graphemes from Trial 1 are robustly activated, and may perseverate into Trial 2.

Second, as indicated in Section 1.1, according to the failure-to-activate hypothesis perseverative and non-perseverative intrusion errors arise for the same reason; in both cases the target grapheme fails to be robustly activated and some other grapheme is produced in its place. For a perseveration error, it is a grapheme from a previously produced response; for non-perseveration errors, it is some other grapheme that is neither in the target nor in the immediately preceding response. *Since perseverative and non-perseverative intrusion errors share the same cause, the prevalence of two error-types should be correlated across individuals with varying degrees of severity of a failure-to-activate deficit.*

A third prediction of the failure-to-activate hypothesis links the severity of sublexical impairment to the proportion of intrusion errors that are perseverations. When a target grapheme is improperly activated, other graphemes can successfully compete for production. Graphemes produced in the previous responses constitute one set graphemes competing for production. In addition, graphemes not in the correct spelling but activated by the sublexical route are also competing for production. Thus, the severity of sublexical route impairment has clear consequences for whether phonologically plausible graphemes or perseverative graphemes are likely to intrude. Consider an attempt to produce the C in CAT after having correctly spelled TABLE. Both the previously produced T and the grapheme K (generated by the sublexical route for the /k/ sound at the beginning of CAT) compete for production with the target letter C. If the C is not selected, the T and K are likely intrusion errors. The likelihood that a phonologically plausible (K) or previously produced letter (T) will be intruded depends on the degree to which the sublexical system is impaired. The more impaired the sublexical system, the less active the phonologically plausible alternative (K) and, therefore, the greater the likelihood that the previously produced T will intrude in the place of the C. *Since greater sublexical impairment results in decreased activation of phonologically plausible graphemes, for a constant level of lexical impairment, the more severe the sublexical impairment, the more likely it is that an intrusion error will be a perseveration.*⁴

Fourth, according to the failure-to-activate deficit, perseveration errors in spelling-to-dictation arise because of an inability to activate graphemes rather than an impairment in processing the graphemes themselves. Therefore, perseveration errors are not predicted in situations in which graphemes are activated via processes that don't involve the lexical or sublexical routes. An example is the direct copy transcoding task described in Section 1.2. Although the task can be carried out via the lexical and sublexical processes, it can also be successfully accomplished using processes that activate graphemes from visually-presented letter-shapes (see Figure 1). Since there is no failure to activate graphemes in this task (unless there are additional deficits) *individuals with lexical and sublexical deficits should not produce perseveration errors in direct copy transcoding.*

1.3.2. Failure-to-inhibit hypothesis—The failure-to-inhibit hypothesis proposes that letter perseveration errors arise because graphemes are not inhibited after written production. The term *inhibition* has different meanings in the language processing literature. Here, when we refer to *inhibition*, we assume self-inhibition (a unit turning itself off), rather than lateral inhibition, in which the most activated unit reduces the activation of competing units (see Dell & O'Seaghdha, 1994 for discussion). Several mechanisms of self-inhibition have been suggested. For example, MacKay (1986) proposed a self-inhibition cycle, in

⁴A comparable prediction relating the level of lexical impairment to the proportion of intrusion errors that are perseverations is also possible. However, the details of this prediction are more difficult to specify. First, while it is clear which non-perseverative errors are predicted to be generated by the sublexical route (phonologically-plausible intrusion errors), it is less clear which non-perseverative errors should be generated by the lexical route. Second, it is much easier to measure the degree of sublexical deficit, as a nonword spelling task can only be carried out by a sublexical route, than the degree of lexical deficit, as both the lexical and sublexical routes contribute to word spelling.

which units are initially inhibited below a baseline level of activation⁵ followed by a rebound to above-baseline activation (see also Houghton, Glasspool and Shallice, 1994). By this account, a failure-to-inhibit deficit may correspond to a failure to initiate the self-inhibition cycle, or to an abnormally sharp rebound, resulting in just-activated units reaching abnormally high levels of activation after production and inhibition (see MacKay, 1986 for a discussion of this type of deficit in the context of stuttering). Another “self-inhibition” mechanism is simply the passive activation decay adopted in some spreading activation models, in which the activation of units decreases over time (e.g. Dell, 1986). A failure-to-inhibit deficit would, by this account, correspond to an abnormally low decay of activation for units from a previous response (see Stemberger, 1989 for a similar proposal with respect to spoken perseverations in child language).

The specific characteristics of the self-inhibition mechanism will not have a bearing on the predictions we examine. Therefore, we simply assume that during the course of normal processing, there are inhibitory processes that lower the activation levels of produced items prior to the onset of the next trial. A failure of these inhibitory processes results in abnormally high levels of activation for previously produced units during the processing of the current item. Under those circumstances, even if the graphemes corresponding to the dictated word are activated normally, they may sometimes be less active than the abnormally-inhibited graphemes from a previous response. The previously produced graphemes may then be selected and a perseveration error produced. Note that unlike the failure-to-activate hypothesis, the failure-to-inhibit disruption is specifically localized to a level of graphemic representation, leading to a different set of predictions than the failure-to-activate hypothesis.

First, if letter perseveration errors are caused only by a failure-to-inhibit deficit, then there should be no relationship between lexical and/or sublexical route failures and the rate of perseveration errors. That is, under a failure-to-inhibit hypothesis there is no reason why individuals with both lexical and sublexical deficits should perseverate significantly. Perseverations should only be observed in individuals with those deficits if they also suffer an additional failure-to-inhibit deficit.

Furthermore, a failure-to-inhibit deficit can only account for perseveration errors. Individuals with more severe failure-to-inhibit deficits should produce more letter perseveration errors, but not more non-perseverative errors. Therefore, *no relationship is predicted between the rate of perseverative and non-perseverative intrusions* and, additionally, there should be *no systematic relationship between the proportion of perseveration errors and the degree of sublexical impairment*. Finally, since the impairment affects the ability to inhibit graphemes after a response is produced, this deficit should affect all tasks that rely on the graphemic level of representation, predicting that *individuals who produce letter perseveration errors in spelling-to-dictation should also perseverate in tasks such as direct copy transcoding*.

Table 1 lists the predictions of each of the hypotheses under investigation. In the work we report on here, we identified a group of 12 individuals with both lexical and sublexical deficits in spelling to dictation. Unlike much of the previous research on this topic (typically involving verbal perseverations) in which participants are identified by the fact that they produce perseverations, we select participants on the basis of the fact that they exhibit characteristics of a failure-to-activate deficit. That is, to a greater or lesser degree, all the

⁵Note that the analyses presented here are not sensitive to this initial refractory period. Our analyses consider perseverations that occur between trials in spelling-to-dictation. The time between spelling responses (~10–15 secs) is much longer than MacKay’s proposed refractory period (~50ms).

participants in this study suffered from a failure-to-activate deficit. This allows us to evaluate the multiple predictions generated by the failure-to-activate hypothesis. Furthermore, we will also consider if a failure-to-activate deficit alone can explain the pattern of perseveration observed in all twelve individuals, or whether at least some of these individuals suffer from an additional failure-to-inhibit deficit. Finally, in our investigation of these questions, and unlike in most previous studies, we use chance analyses (e.g. Cohen & Dehaene, 1998) that allow us to distinguish between true perseverations errors and other errors in which the intrusion appears in the previous response(s) simply by chance.

2. MATERIALS, METHODS AND RESULTS

2.1. Participants

Participants were selected for this study only if their spelling performance indicated impairments to both the lexical and sublexical spelling processes. The hallmark characteristic of a lexical deficit is a lexical frequency effect (i.e., greater accuracy with high compared to low frequency words)⁶, while the hallmark characteristic of a sublexical deficit is a difficulty spelling nonwords. We identified all dysgraphic individuals who had been tested in our lab over a number of years who showed both significant frequency effects in word spelling and produced high rates of phonologically implausible spellings for nonwords.

All individuals identified in this manner had been evaluated with the Johns Hopkins University Dysgraphia Battery (Goodman & Caramazza, 1985), with high and low frequency words taken from one or more of the following lists: Length, Part-of-Speech, Concreteness and Phoneme-to-Grapheme Probability. Each of these lists allows for a comparison of high vs. low frequency words, controlling for relevant variables such as letter length, part of speech, concreteness and the regularity of sound-to-spelling correspondences. (Note that not all of the participants were administered all lists). Statistical significance of frequency effects was assessed with a one-tailed chi-square test considering correct and incorrectly spelled letters, (alpha level of $p < .05$). Nonword accuracy was assessed by calculating the number of phonologically plausible “whole word” responses as well as the number of plausible correct letters (5 control subjects between 72 and 82 years old were administered the same list of nonwords and produced phonologically plausible spellings on 97–100% of the trials).

In total, 12 individuals who previously participated in dysgraphia research in our lab were retrospectively identified as having both frequency effects and impairments in nonword spelling (see Table 2). In addition to the JHU Dysgraphia Battery, these individuals were tested on a number of other spelling and language tasks. Below we provide brief case descriptions for each of these individuals, reporting on them in order of their overall word spelling accuracy (from most accurate to least accurate). Many of these individuals have been described in other publications (AES: Rapp, Barriere & Fischer-Baum, 2006; BWN, Buchwald & Rapp, 2006, 2010; CM, McCloskey, Macaruso & Rapp, 2006; DHY, Buchwald & Rapp, 2010; DLE, Medina & Rapp, 2008; ELE (as case WR3), Goldrick, Folk & Rapp, 2010; GBT, Rapp & Lipka, 2008; LHT, Fischer-Baum, 2010; LSS, Fischer-Baum, McCloskey & Rapp, 2010; MMD, Rapp & Kane, 2002; VBR, Buchwald & Rapp, 2010).

2.1.1 VBR—VBR was a right-handed woman, with a high-school degree who suffered a CVA at the age of 51. Prior to the CVA, VBR was the president of a small company and an

⁶Individuals with lexical route impairment and intact sublexical processing also show regularity effects (i.e., greater accuracy on words with high compared to low probability phoneme-to-grapheme correspondences). But—as in the cases identified for this study—when the sublexical system is also damaged, a regularity effect is no longer predicted.

avid reader. MRI scanning revealed a large left hemisphere fronto-parietal infarct. VBR's spoken language production was severely impaired with 64% accuracy in single-word picture naming (21/33). Auditory word comprehension was very good, scoring at the 75th percentile on the PPVT-R (Dunn & Dunn, 1981). Further, she had no apparent perceptual difficulties in spoken-word recognition, as evidenced by nearly flawless performance on the PALPA (Psycholinguistics Assessments of Language Processing in Aphasia, Kay, Coltheart & Lesser, 1992) minimal pair discrimination tasks for words (99%, 71/72; PALPA 2) and nonwords (99%, 71/72; PALPA 1).

VBR was administered the Length, Part of Speech and Concreteness Lists from the JHU Dysgraphia Battery. Letter accuracy was greater for high- (477/487; 98%) than low frequency words (434/477; 91%, $\chi^2 = 21.16$, $p < .001$). Nonword spelling was severely impaired, with only 9% (3/34) phonologically plausible spellings and 67% (120/178) letter accuracy. In total, VBR spelled a total 317 words to dictation (74% word accuracy and 93% letter accuracy). Errors consisted primarily of phonologically implausible nonword errors (e.g. "execution" → EXTERATION; 53% of all errors), but also included: morphological errors (e.g. "bought" → BUY; 23% of errors), phonologically plausible errors (e.g. "cinch" → SINCH; 16% of errors), and other word errors (e.g. "residue" → REDUCE; 7% of errors).

2.1.2 DHY—DHY was a right-handed male, with a B.A. in Economics, who had been employed as a banker and marketing consultant prior to a CVA suffered in 2002, at the age of 35. The CVA resulted in a large left hemisphere lesion, affecting the posterior inferior frontal gyrus, the inferior postcentral gyrus, the supramarginal gyrus, the angular gyrus, and the superior edge of the superior temporal gyrus. DHY experienced moderate difficulties producing sentences in spontaneous speech, exhibiting some marked hesitations and word-finding difficulties, but his single-word picture naming was 100% correct (35/35). Auditory discrimination for words was within the normal range (97%, 70/72; PALPA 2) and only mildly impaired for nonwords (92%, 66/72; PALPA 1). Further, his single word auditory comprehension was excellent as assessed using the PPVT-R, where he scored in the 87th percentile.

From the JHU Dysgraphia Battery, DHY was administered the Length, Part of Speech, Concreteness and Phoneme-to-Grapheme Conversion Lists, exhibiting a clear frequency effect, with 94% (879/931) letter accuracy for high frequency words and only 87% (796/913) for low-frequency words ($\chi^2 = 28.08$, $p < .001$). Nonword spelling was severely impaired with only one phonologically plausible spelling (1/34; 3%) and 65% letter accuracy. In total, DHY spelled 545 word stimuli with 74% word accuracy and 92% letter accuracy. Errors consisted of phonologically implausible nonwords (52%), morphological (11%), other word (23%) and phonologically plausible (13%) errors.

2.1.3 AES—AES was an ambidextrous female, with an M.A. in Engraving, who suffered a CVA in 1997, at the age of 42. Prior to the CVA, AES had been employed as a manager for a federal agency. The CVA resulted in a large left hemisphere lesion affecting the inferior and middle frontal lobes, the superior temporal gyrus, supramarginal gyrus and extending posteriorly to the anterior portion of the angular gyrus. AES experienced moderate difficulties producing sentences in spontaneous speech, exhibiting hesitations and word-finding difficulties, but single-word picture naming was 90% correct (27/30). Auditory discrimination for words (100%, 72/72; PALPA 2) and nonwords (96%, 69/72; PALPA 1) were within normal range, as was her auditory lexical decision performance (99%, 158/160; PALPA 5). Further, her single word auditory comprehension was good as assessed using the PPVT-R, on which she scored at the 58th percentile.

AES was administered the Part-of-Speech and Phoneme-to-Grapheme Conversion Lists from the JHU Dysgraphia Battery. She was significantly more likely to correctly produce letters in high-frequency (465/478; 97%) than low-frequency words (425/485; 88%, $\chi^2 = 30.65$, $p < .001$). She plausibly spelled 10/34 nonwords (29%) with 72% letter accuracy. In total, AES spelled 433 words with 73% word accuracy and 91% letter accuracy. Errors were primarily phonologically implausible nonwords (43%) and also included morphological errors (28%), other word errors (17%), and phonologically plausible nonwords (12%).

2.1.4 BWN—BWN was a right-handed man, with a PhD, who suffered two CVAs 9 and 5 years prior to the onset of testing. Prior to the CVAs, he had worked as a school system administrator, and was president of the city chapter of a national public affairs organization. BWN suffered moderate difficulty in spoken language production, primarily characterized by reduced accuracy (68%, 177/260) in spoken picture naming (Snodgrass and Vanderwart, 1980 pictures). He suffered mild hearing loss and auditory discrimination difficulties with both words (89%; 64/72; PALPA 2) and nonwords (90%; 65/72; PALPA 1). Nonetheless, BWN's auditory word comprehension was very good, scoring in the 94th percentile on the PPVT-R.

BWN was administered the Length, Concreteness, Part-of-Speech and Phoneme-to-Grapheme Conversion Lists from the JHU Dysgraphia Battery. He was significantly more likely to correctly produce letters in high-frequency (756/768; 98%) than low-frequency words (744/777; 96%, $\chi^2 = 8.92$, $p < .01$). He plausibly spelled 24% (8/34) nonwords (77% letter accuracy). Across all testing sessions, BWN spelled 729 words with 71% word accuracy and 94% letter accuracy. His errors were primarily phonologically implausible nonword errors (79%), and he also produced phonologically plausible errors (14%) and other word errors (5%).

2.1.5 MMD—MMD was a right-handed woman who worked in a clerical position until retirement and suffered a CVA in November of 1998 at the age of 65. CT scanning indicated left posterior parietal and temporal lesions. She suffered only mild naming difficulties, correctly naming 233 out of 253 (92%) of the Snodgrass and Vanderwart (1980) pictures. Her auditory processing of words was quite good, with performance within normal range on: auditory discrimination (98%; 45/46), auditory lexical decision (93%, 205/220) and auditory word/picture verification (95%; 247/260).

MMD was administered the JHU Dysgraphia Battery Length List on several occasions and portions of the Phoneme-to-Grapheme Conversion List. She was significantly more likely to correctly produce letters in high-frequency (892/950; 94%) than low-frequency words (824/928; 89%, $\chi^2 = 14.86$, $p < .001$). She plausibly spelled 32% (11/34) nonwords (81% letter accuracy). In total, MMD spelled 2087 words with 64% word accuracy and 89% letter accuracy. Her errors were primarily phonologically implausible nonword errors (62%), and she also made phonologically plausible errors (21%) and other word errors (15%).

2.1.6 JCY—JCY was a right-handed man with a B.A. in English who suffered a left hemisphere CVA in 1997 at the age of 59. Prior to the CVA, JCY worked as a stocks and bonds broker. He suffered naming difficulties, correctly naming only 107 out of 197 (54%) of the Snodgrass and Vanderwart (1980) pictures. However, his auditory processing of words was within normal range on auditory discrimination of words (96%; 69/72; PALPA 2) and auditory lexical decision (95%, 171/180). Auditory word comprehension, as tested by the PPVT-R, was at the 18th percentile.

JCY was administered the Length, Part-of-Speech and Concreteness Lists. He was significantly more likely to correctly produce letters in a high-frequency (480/529; 91%)

than low-frequency words (446/524; 85%, $\chi^2 = 6.54$, $p < .01$). He plausibly spelled 27% (9/33) of nonwords (77% letter accuracy). In total, JCY spelled 311 words with 61% word accuracy and 89% letter accuracy. Errors were primarily phonologically implausible nonword errors (79%), although he also made phonologically plausible errors (12%) and other word errors (7%).

2.1.7 CM—CM was a right-handed man with a Ph.D. in electrical engineering. He worked as a university professor until suffering a stroke in September 1986 at age 59. A CT scan showed extensive cortical and subcortical damage in the distribution of the left middle cerebral artery. Auditory word comprehension appeared intact. In two administrations of the PPVT-R, CM placed in the 21st and 81st percentile of normal adults. However, his spoken word production was severely impaired. He was able to recite automatized sequences and repeat single words, but scored only 2/16 in repeating phrases. Confrontation and responsive naming were poor, and CM generated only 1 animal name in 90 seconds.

CM was administered the Length, Phoneme-to-Grapheme and Concreteness Lists of the JHU Dysgraphia Battery. He was more likely to correctly produce letters in high-frequency (421/542; 78%,) than low-frequency words (399/547; 78%, $\chi^2 = 3.03$, $p < .05$). He plausibly spelled 1/34 nonwords (3%; 52% letter accuracy). In total, CM spelled 3795 words with 55% word accuracy and 82% letter accuracy. His errors were primarily phonologically implausible nonword errors (60%), as well as other word errors (29%), phonologically plausible errors (6%) and morphological errors (5%).

2.1.8 ELE—ELE was a 65 year-old right-handed woman with a high school education who had retired from working as a home-health aide, when she suffered a left-hemisphere stroke in 2001 (additional data regarding her lesion are not available). She named only 74 out of 144 pictures of concrete objects correctly (51%) and exhibited some difficulties in auditory word comprehension, scoring 73% correct (186/254) on auditory word/picture verification.

ELE was administered the Length and Concreteness Lists of JHU Dysgraphia Battery. She was more likely to produce letter errors on low frequency words than high frequency words (High-Frequency: 182/293; 62% vs. Low-Frequency: 122/252; 48%, $\chi^2 = 9.77$, $p < .001$). She plausibly spelled 9% (3/34) of nonwords (66% letter accuracy). In all, ELE spelled 562 words with 39% word accuracy and 71% letter accuracy. Errors were primarily phonologically implausible nonword errors (65%), as well as other word errors (27%) and no responses (4%).

2.1.9 LSS—LSS was a left-handed man with a Master's degree in psychology who worked as a regional sales representative for a health services company until he suffered a CVA in July 2003 at age 54. CT scanning showed extensive cortical and subcortical damage in the distribution of the left middle cerebral artery. Auditory word discrimination was within normal range (92%, 66/72; PALPA 2), while auditory comprehension was moderately impaired (7th percentile on the PPVT-R). On the Philadelphia Naming Test (Roach et al., 1996), LSS correctly named only 112/175 (64%) of the picture stimuli.

LSS was administered the Length, Concreteness and Part-of-Speech Lists of the JHU Dysgraphia Battery. He was more accurate in producing letters in high vs. low frequency words (High-Frequency: 350/529; 66% vs. Low-Frequency: 248/524; 47%, $\chi^2 = 37.29$, $p < .001$). He failed to plausibly spell any nonwords (0/34) (40% letter accuracy). In all, LSS spelled 1636 words, with 20% word accuracy and 57% letter accuracy. Errors were primarily phonologically implausible nonword errors (82%), although he also produced other word errors (15%).

2.1.10 DLE—DLE was left-handed man with a degree in Mechanical Engineering who had worked as an engineer before suffering a left middle cerebral artery infarct in 2001 at the age of 67. MRI revealed extensive damage to the inferior frontal gyrus, insula and much of the precentral gyrus; damage extended posteriorly to the post-central gyrus, the supramarginal gyrus and the anterior portion of the angular gyrus, and posterior/superior portions of the superior temporal gyrus. His spoken production was moderately-severely impaired, correctly naming only 47/90 (52%) of line drawings of common verbs and nouns (Zingeser & Berndt, 1990). His ability to discriminate auditorily presented words (71/72; 98%; PALPA 2) was within normal range as was his auditory word comprehension (66th percentile of normal adults, according to the PPVT-R).

DLE was administered the Length and Concreteness Lists of the JHU Dysgraphia Battery on several occasions. He showed a clear frequency effect in terms of letter accuracy (High-Frequency: 283/618; 46% vs. Low-Frequency: 229/576; 40%, $\chi^2 = 4.19$, $p < .05$). He produced only one plausible spelling of a nonword (1/34; 42% letter accuracy). In all, DLE spelled 616 words with 10% word accuracy and 50% letter accuracy. Errors were primarily phonologically implausible nonword errors (80%), as well as some other word errors (19%).

2.1.11 LHT—LHT was a right-handed man with a degree in engineering who suffered a stroke in 2000 at the age of 67. MRI scans showed extensive cortical and subcortical damage in the area of the distribution of the left middle cerebral artery. LHT suffered a severe spoken naming deficit, correctly naming 24 of the first 46 items in the Philadelphia Naming Test (52%). He was unimpaired at minimal pair discrimination, with both words (72/72; 100%; PALPA 2) and nonwords (70/72; 97%; PALPA 1). He was mildly impaired with auditory lexical decision (141/160; 88%; PALPA 5), and scored at the 18th percentile in auditory word comprehension on the PPVT-R.

LHT was administered the Length List of the JHU Dysgraphia Battery on several occasions, as well as the Concreteness and the Phoneme-to-Grapheme Conversion Lists. He showed a clear frequency effect in terms of letter accuracy (High-Frequency: 431/771; 56% vs. Low-Frequency: 325/746; 44%, $\chi^2 = 22.59$, $p < .001$). He produced no phonologically plausible spellings for nonwords (0/34; 44% letter accuracy). In all, LHT spelled 615 words, with 10% word accuracy and 53% letter accuracy. Errors were phonologically implausible nonword errors (85%), and also other word errors (14%).

2.1.12 GBT—GBT was a right-handed man with a Ph.D. in American History, who learned English at age 16 (native language was Hungarian) and worked as a college history professor. He suffered a stroke in 2007 at the age of 67. MRI indicated a lesion primarily affecting the left parietal-temporal lobes, with damage to the supramarginal and angular gyri as well as superior and middle temporal gyri. GBT suffered a moderate spoken naming deficit, correctly naming 60 of 90 (67%) of drawings of nouns and verbs (Zingeser & Berndt, 1990). He was unimpaired at minimal pair discrimination, with both words (72/72; 100%; PALPA 2) and nonwords (71/72; 98%; PALPA 1) and he scored in the 66th percentile on the PPVT-R.

GBT was administered the portions of the Length List of the Johns Hopkins Dysgraphia Battery on several occasions. He showed a clear frequency effect in terms of letter accuracy (High-Frequency: 89/180; 49% vs. Low-Frequency: 58/156; 44%, $\chi^2 = 4.62$, $p < .05$). He produced no phonologically plausible spellings of nonwords (0/12; 46% letter accuracy). In all, GBT spelled 342 words with 3% word accuracy and 52% letter accuracy. Errors were primarily phonologically implausible nonword errors (92%), but also included other word errors (6%).

2.2 Analysis 1: Do individuals with lexical and sublexical spelling deficits perseverate in spelling-to-dictation?

If the failure to activate graphemes is a source of letter perseveration errors, then individuals with both lexical and sublexical deficits should produce significant numbers of letter perseveration errors in spelling-to-dictation. In previous work (Fischer-Baum, McCloskey & Rapp, 2010; McCloskey, Macaruso & Rapp, 2006), we described a method for testing whether individuals produce letter perseveration errors at significantly above chance rates. We applied this letter perseveration analysis to the letter intrusion errors produced by each of the twelve dysgraphic participants to determine if each made more perseveration errors than would be expected by chance.

Letter intrusions may take the form of substitutions or insertions. For example, in the trial “head” → HEAT, the T is an intruded letter, as is the I in the insertion error “spend” → SPIEND. However in many cases it is ambiguous as to which letters are substitutions and which are insertions (e.g. “music” → MUNCIC). Given this ambiguity, we analyzed substitution and insertion errors together. We excluded from further analysis responses that were phonologically plausible spellings of the target word (e.g., JCY: “total” → TOTLE), or that were morphologically (e.g., AES: “poking” → POKE) or semantically (e.g., ELE: “cup” → MUG) related to the target. These responses were excluded because these errors could have sources other than a failure to properly activate graphemes. For example, they could reflect robust activation of incorrect graphemes from the sublexical route, as in the case of phonologically plausible spellings, or from the lexical route, as in the case of morphological and semantic errors. We also excluded whole word perseverations –errors in which the whole response perseverated from any previous response during a single testing session (e.g. “building” → FORWARD immediately after “forward” → FORWARD) because whole word perseveration errors might not arise from the graphemic level (Moses, Sheard & Nickels 2007).

For the remaining intrusions, the letter perseveration analysis evaluated whether intruded letters were more likely than expected by chance to be present in at least one of the three trials immediately preceding the error (trials E-1 to E-3). Table 3 shows an example of a perseveration error produced by LSS (UNDER → UNDEL) with the five immediately preceding responses (E-1 through E-5). First, a computer program identified the intruded letters produced by each of the twelve individuals. For each participant and for each intruded letter, a computer program then tabulated whether or not the letter was present in one of the three immediately preceding responses. For example, the intruded L in LSS’s error “under” → UNDEL was present in the E-1 response, MOLDEL and the E-2 response SHOULD. For LSS’s error “kitchen” → KITCHEM, the intruded M was not present in the response on trial E-1 (BELEAFE), although it did appear in the E-2 response (SYSTEM). In both cases, the intruded letter was tabulated as having appeared at least once in the three immediately preceding responses. From this tabulation, the program calculated the proportion of intruded letters that appeared in one of the three immediately preceding responses.

The results are reported (black bars) in Figure 2. For example, the .61 value plotted for AES in Figure 2 reflects the fact that 50 of AES’s 82 intruded letters (61%) were present in at least one of the three responses immediately prior to each error. The value of .86 plotted for LSS reflects the fact that 1996 of LSS’s 2299 intruded letters (86%) appeared in at least one of the three immediately preceding responses. Of the 12 individuals included in the analysis, AES had the lowest proportion of intrusion errors observed in the three immediately preceding responses and LSS had the highest.

The perseveration analysis program also estimated, for each participant, the likelihood of an intruded letter being present by chance in at least one of the three immediately preceding

responses. The chance estimates are based on the following rationale: If the occurrence of a letter intrusion is unrelated to the presence of the intruded letter in the immediately preceding responses, then the intruded letter should be just as likely to be found in responses occurring on trials distant from the intrusion trial. For example if the L intrusion on trial E in Table 3 (UNDER → UNDEL) had nothing to do with whether Ls were present on trials E-1, E-2 or E-3, then Ls should be just as likely to be present in responses that did not immediately precede the UNDEL response. The logic of this analysis is identical to the letter perseveration analyses described in previous work (Fischer-Baum, McCloskey & Rapp, 2010; McCloskey, Macaruso & Rapp, 2006).

This chance analysis was carried out separately for each of the 12 individuals. For each of the actual E-1 through E-3 responses (e.g., MOLDEL, SHOULD and CHEUIVE for the error “under” → UNDEL), regardless of whether they contained the letter that was intruded on trial E, a computer program created a pool of control responses. The control pools for each of the E-1 through E-3 responses, consisted of all responses in the participant’s spelling corpus that were produced outside the window of the five trials either preceding or following the intrusion error and that had the same number of letters as the actual preceding response. For example, the pool of control responses for the E-1 response MOLDEL included all other six-letter responses produced by LSS that were not produced within five trials of the actual intrusion response UNDEL (examples include FRENST, BOTTOM and LAUGUE). A Monte Carlo analysis used the control response pools to estimate the chance likelihood that the intruded letter should be found in the three previous responses. To do so, for each of the letter intrusions, the program randomly sampled a response from the control pools for each of the three preceding responses, and tabulated whether any of the control responses included the intruded letter. The program then computed the proportion of all sampled control responses that contained the intruded letter. This process—random sampling of the control responses—was carried out 10,000 times for the complete set of letter intrusions for each participant.

The means of the 10,000 control proportions, for each individual, are reported (white bars) in Figure 2. For AES, the plotted value indicates that, in her case, there was a .54 chance probability of an intruded letter occurring in one of the three preceding responses. For all participants, the average chance value was numerically lower than the observed value (the proportion of times the intruded letter actually appeared in at least one of the three immediately preceding responses). For 7 of the 12 participants (CM, DLE, ELE, GBT, LHT, MMD and LSS) all of the 10,000 chance analysis values were lower than the observed proportions. For two others (JCY and VBR) fewer than 500 of the 10,000 chance analysis values (<5%) were as high as or higher than the observed proportion. For the remaining three individuals (DHY, AES and BWN) between 500 and 1,000 of the 10,000 chance analysis values (<10%) were as high as or higher than the observed proportion. In other words, at a significance level of $p < .05$, nine out of the twelve individuals with both lexical and sublexical deficits produced significant numbers of letter perseverations. The remaining three individuals, DHY, AES and BWN, all produced relatively few intrusion errors compared to the other individuals in the sample, suggesting that the lack of a statistically significant letter perseveration effect (at $p < .05$) in these individuals was due to low power. In fact, at a significance level of $p < .1$, all 12 individuals produced more letter perseverations than would be expected by chance.⁷

⁷Note that this analysis differs from other previously reported letter perseveration analyses (Fischer-Baum, McCloskey & Rapp, 2010; McCloskey, Macaruso & Rapp, 2006) in that it considered whether an intruded letter was present in any one of the three previous responses, rather than considering each of the preceding responses separately. We pooled data across the three preceding responses as some of the individuals in this study were not tested as extensively as others, yielding fewer data points for analysis.

Since all dysgraphic individuals that we identified as having both lexical and sublexical deficits produced significant numbers of letter perseverations (at least at $p < .1$) this analysis clearly supports Prediction 1 (Table 1) of the failure-to-activate account of letter perseveration errors.

2.3. Analysis 2: Are perseverative and non-perseverative error rates correlated across individuals?

As discussed in Section 1.3.1, the failure-to-activate hypothesis predicts that since perseverative and non-perseverative intrusion errors share the same cause, a correlation should be observed between the rates at which the two error types are produced. In contrast, since a failure-to-inhibit deficit predicts only perseverative intrusions and a separate deficit is required to account for non-perseverative intrusion errors, no correlation is expected between these rates. To examine this prediction, for each of the 12 individuals, we calculated the percentage of all produced letters (both correct and incorrect) that were true perseverative intrusions and the percentage that were clear non-perseverative intrusions.

2.3.1. True perseverative intrusions—The set of intrusions in which the intruded letter appears in at least one of the three immediately preceding responses necessarily includes not only *true* perseveration errors but also what may be called *pseudo*-perseveration errors. Suppose, for example, that CM intruded an R into the response ERGE for reasons having nothing to do with the presence of Rs in prior responses. In that case, the intruded R was not a true perseveration, but rather a pseudo-perseveration. Therefore, the cases in which an intruded letter appears in one of the three previous responses constitute the set of *potential* perseveration errors – a combination of true and pseudo perseveration errors. This identifies a challenge in testing the prediction outlined above. The prediction concerns the relationship between true perseveration errors and non-perseverative intrusions, but the pseudo perseveration errors are, in fact, non-perseverative intrusions. The greater the severity of the deficit that generates non-perseverative intrusions, the larger the number of pseudo perseveration errors that will be produced. In that case, it would be unsurprising to observe a correlation between the number of potential perseveration errors and non-perseverative intrusions, even if no correlation existed between true perseverations and non-perseverative intrusions. To address this issue, it is critical to identify the number of true perseveration errors produced by each individual. We did so by using Equation 1 which provides a method for estimating the number of true perseveration errors (*True*) from a set of potential perseveration errors (*Potential*), making use of the chance probability that an intruded letter (IL) appeared is the previous response ($pChanceILinPrev$) and the total number of intrusion errors (*Int*). We refer interested readers to Fischer-Baum, McCloskey and Rapp (2010) for the full derivation of this equation.

$$Potential = True + (Int - True) \times pChanceILinPrev \quad (1)$$

For each of the 12 participants, we estimated the number of true perseveration errors. For example, AES produced 50 potential perseveration errors out of 82 total intrusion errors, with a $pChanceILinPrev$ of 0.537. Using these values in equation 1, we estimated that AES produced 12.9 true perseveration errors out of 50 potential perseveration errors. We then calculated the percent of all produced letters⁸ that were true perseveration errors. For AES, 12.9 out of 2743 total letters produced were true perseverations, or 0.5% of all letters produced. These values ranged from 0.4% for BWN to 24.5% for LSS. Appendix 1 provides

⁸This value of total number of letters produced only included those letters produced that were considered in the perseveration analysis above. Therefore, this value doesn't include letters produced in semantic errors or phonologically plausible errors, or letter produced in response to the first several trials of a given testing session.

the information used for calculating these values for each individual (number of intrusions, potential perseverations and *pChanceLinPrev*, perseveration proportion and total number of letters produced).

2.3.2. Non-perseverative intrusions—For each participant, we tabulated the number of intrusion errors for which the intruded letter did not appear in any of the three previous responses, and considered these intrusion errors to be non-perseverative intrusions. For example, AES produced 32 intrusion errors that were clearly not perseveration errors or 1.2% (32/2743) of all of her produced letters. The percent of all produced letters that were non-perseverative intrusions ranged from 0.8% for BWN to 12.9% for DLE (see Appendix 1 for the precise values for each individual).

The above analyses yielded, for each participant, the percentages of all letters produced that were true perseverative and non-perseverative intrusion errors. These values are plotted in Figure 3, revealing a significant positive correlation between perseverative and non-perseverative intrusions ($r(10) = .52, p < .05$). Individuals who were more likely to produce letter perseveration errors were also more likely to produce non-perseverative intrusions. We would like to make clear that this result is not a necessary outcome of this analysis. That is, with increasing deficit severity there were various possible outcomes. For example, the proportion of responses that were true perseverations could have increased without a change in the proportion of non-perseverative intrusions, the reverse pattern could have been observed, etc. However, what the analysis reveals is that, in fact, both types of errors increase with increasing deficit severity.

It is also worth noting that one participant (LSS) appears to be an outlier in Figure 3, producing many more perseverative than non-perseverative intrusions. The correlation between perseverative and non-perseverative intrusions was still found to be significant when this individual was removed from the analysis ($r(9) = .83, p < .001$), indeed the strength of the correlation was greater. We simply note this here and take up the issue of outliers later, in Section 2.5.1. Importantly, the observed positive correlation conforms to the prediction of the failure-to-activate hypothesis that perseverative and non-perseverative intrusions are generated by the same deficit, while it is not predicted by the failure-to-inhibit hypothesis that assumes that perseverative and non-perseverative intrusions are the result of separate deficits.⁹

2.4. Analysis 3: Is the degree of sublexical deficit correlated with the likelihood that an intruded letter is a perseveration?

A final test of the failure-to-activate hypothesis examines the relationship between the degree of sublexical disruption and the type of intrusion errors produced. Specifically, we examined whether the proportion of all intrusions that are true perseverations (rather than proportion of all produced letters, as in the previous analysis), a value we will call *perseveration proportion*, is a function of the severity of the sublexical deficit. Degree of sublexical impairment was quantified as the error rate (letters incorrect) in nonword spelling. Perseveration proportion was calculated by dividing the number of true perseverations, computed from the previous analysis, by the number of total intrusions made by each individual.¹⁰

⁹The only scenario under which the failure-to-inhibit hypothesis would predict a correlation between perseverative and non-perseverative errors is one in which the severity of the two deficits responsible for the two error types is linked by some common factor, for example lesion size.

¹⁰Note that these perseveration proportions are computed over intrusion errors that occurred only in word spellings (rather than nonword spellings).

The results for the 12 participants (see Figure 4) reveal a significant positive correlation between perseveration proportion and degree of sublexical deficit ($r(10) = .63, p < .05$). That is, the greater the sublexical impairment, the greater the likelihood that intrusion errors were perseverations. In this way, the results further support the failure-to-activate hypothesis over the failure-to-inhibit hypothesis. As in the previous analysis, LSS appears to be an outlier with a higher perseveration proportion than would be expected based on the severity of his sublexical impairment. Again, the correlation was still significant when this outlier data point was removed ($r(9) = .55, p < .05$).

2.5. Analysis 4: Outliers and the possibility of an additional failure-to-inhibit deficit

Thus far we have confirmation of Predictions 1–3 (Table 1) of the failure-to-activate hypothesis regarding the source of perseveration errors. These results are difficult to reconcile with the hypothesis that letter perseveration errors only arise from a failure-to-inhibit deficit. However, the results do not require the conclusion that a failure-to-activate is the *only* deficit that produces letter perseveration errors. Thus, it is important to consider if any of these individuals suffer from an additional failure-to-inhibit deficit. Note that, we cannot determine whether a failure-to-inhibit deficit alone can produce letter perseveration errors given that all of the participants in this study were selected specifically because they exhibited the key characteristics of a failure-to-activate deficit: a frequency effect in word spelling accompanied by a nonword spelling deficit.

To examine whether any of the participants suffered from an additional failure-to-inhibit deficit, we carried out a two-part analysis. First, we determined whether there were any statistically significant outliers in the previous analyses. These would be individuals who produced more perseveration errors than would be predicted by their rate of non-perseverative intrusions, and/or those with higher perseveration proportions than would be predicted by their degree of sublexical damage. These patterns could not be explained with only a failure-to-activate deficit. Second, we looked for positive evidence for a failure-to-inhibit deficit, specifically examining performance on the task of direct copy transcoding. As discussed in Section 1.3.2, if perseveration errors arise because of a failure to inhibit graphemes at the graphemic level, then significant numbers of perseverations are predicted on all tasks that rely on the graphemic level, including spelling-to-dictation and direct copy transcoding.

2.5.1. Outlier detection—Visual inspection of Figures 3 and 4 suggests that the same participant (LSS) is an outlier (see also Appendix 1). In Analysis 2, approximately a quarter of all letters produced by LSS were true perseveration errors, a value nearly twice the magnitude of the next highest value in the group (LHT: 13.9%). However, only 4.5% of the letters LSS produced were non-perseverative intrusions, ranking him 8th among the 12 individuals. That is, LSS produced many more perseveration errors than would have been expected from his rate of non-perseverative intrusions. Similarly, in Analysis 3, although LSS had a comparable level of sublexical impairment (60% error by letter) as a number of other individuals (LHT: 56%; DLE: 58%), a much larger proportion of his intrusion errors were perseverations. Specifically about three quarters of LSS's intrusion errors were perseverations, approximately twice the magnitude of LHT's and DLE's perseverative proportions.

We carried out an analysis to evaluate whether LSS, or any of the other eleven individuals, was a significant outlier in either Analysis 2 or 3, using a Jackknifed Mahalanobis distance analysis corrected for small sample size and multiple comparisons (Penny, 1996). The results of this analysis indicate that LSS exceeded the critical value (Bonferroni-corrected

$\alpha=.0042$, critical value = 29.1) in both Analysis 2 (MD = 57.1) and Analysis 3 (MD = 38.3). No other outliers were detected using this technique.

It is unlikely that LSS's high rate of perseveration was the result of measurement error given that LSS' perseveration errors came from a corpus of approximately 1600 word spellings that included approximately 2300 letter intrusions. The 99% confidence interval for LSS's perseveration proportion falls between .71 and .74. On this basis, it is clear that LSS is more likely to produce perseveration errors than any of the other individuals tested, making it likely that LSS's perseverations did not all have the same source as those of the other individuals in the sample.

2.5.2. Perseverations in direct copy transcoding—Prediction 4 (Table 1) indicates that if LSS suffered a failure-to-inhibit deficit at the grapheme level, he should also produce letter perseverations in direct copy transcoding. To examine this prediction, LSS and four other individuals from the previous analyses (CM, DLE, ELE, LHT) were administered a direct copy transcoding task.

LSS was asked to copy transcode 84 words and 40 nonwords. He performed well on this task, correctly transcoding 676/688 (98%) of all letters. This high accuracy indicates a mild deficit. Nonetheless, even with these few errors, we can determine if LSS suffered from a failure-to-inhibit deficit. Of his twelve letter errors, one was a deletion error (hungry → HUNGY) and the remaining eleven were intrusion (substitution) errors (e.g. *length* → CENGTH). We analyzed these errors to determine if they appeared in immediately preceding responses more often than would be expected by chance.

First, we found that 82% of the intruded letters appeared in at least one of the three immediately preceding responses. Second, this observed value of 82% was compared to chance using the analysis described in Section 2.2. The mean value generated by that analysis indicated that, on average, only 49% of intruded letters should appear by chance in at least one of the three immediately preceding responses. In fact, fewer than 5% of the 10,000 chance analysis runs generated a value as high or higher than the observed value (82%). From this we can conclude that, despite his relatively good performance in this task, LSS produced statistically significant numbers of letter perseveration errors, providing positive evidence that he suffered from an additional failure-to-inhibit deficit. However, LSS produced many fewer letter perseveration errors in direct copy transcoding than spelling-to-dictation. Below, we discuss how this difference is predicted by assuming that both failure-to-inhibit and a failure-to-activate deficits affect spelling-to-dictation.

The four other individuals that performed direct copy transcoding (CM, DLE, ELE, LHT) all performed very well on this task (Letter accuracy: CM: 100%; DLE: 99.3%; ELE: 97.5%; LHT: 99.5%) and none produced significant numbers of letter perseveration errors. Specifically, CM and LHT produced no intrusion errors and DLE and ELE's intruded letters did not appear in previous responses any more than would be expected by chance ($p>.3$). For these four individuals, there was no evidence of an additional failure-to-inhibit deficit and, on this basis, a failure-to-activate deficit alone appears to be sufficient to explain their pattern of letter perseveration errors.

2.6 Understanding LSS's multiple sources of letter perseverations errors

Analysis 4 revealed that LSS's high rate of letter perseveration cannot be explained by a failure-to-activate deficit alone and that his performance in direct copy transcoding was consistent with a failure-to-inhibit deficit. The data are consistent with one of two possibilities; either LSS only suffered from a failure-to-inhibit deficit or he suffered from a combination of a failure-to-inhibit and a failure-to-activate deficits.

There are several reasons why LSS's difficulties are best understood as resulting from a combination of failure-to-activate and failure-to-inhibit deficits. First, while LSS perseverated both in direct copy transcoding and spelling-to-dictation, he perseverated at much greater rates in the latter than the former. Approximately a quarter of all letters produced in spelling-to-dictation were perseveration errors. In direct copy transcoding, only 1% of all produced letters were perseveration errors. If LSS suffered only from a failure-to-inhibit deficit, this difference would not be predicted.

Second, LSS had both lexical and sublexical deficits. We reanalyzed LSS's word and nonword spelling accuracy (by letter) in the JHU Dysgraphia Battery (Goodman & Caramazza, 1995) excluding all potential perseveration errors. Even with the perseveration errors removed, LSS showed a frequency effect (HF words: 90% by letter, LF words: 80%, $\chi^2(1, N=1020) = 19.6, p < .001$) and an impairment in nonword spelling (79% accuracy by letter). Since LSS clearly suffered from both lexical and sublexical deficits, we can assume that a failure-to-activate deficit contributed to the perseveration errors produced in spelling-to-dictation.

Third, if we assume both deficits we can make sense of the fact that while LSS had a very high rate of perseveration errors in spelling to dictation (25% of all letters produced), his perseveration rate was low in direct copy transcoding (1%). This explanation assumes an interaction of the two deficits rather than a simple additive effect in which some perseverations were caused by a failure-to-activate while others resulted from a failure-to-inhibit. This combined failure-to-inhibit and failure-to-activate account requires that we assume that LSS' failure-to-inhibit deficit resulted in graphemes being inhibited less than they would be under normal circumstances. For example, we might assume that in an intact system a grapheme's activation normally drops to 20% after production. This would be an activation level higher, on average, than that of other graphemes not recently produced (15%), but still unlikely to be very competitive with upcoming target graphemes. However, with a failure-to-inhibit deficit, we could assume that graphemes retain, on average, 70% of their full activation levels. For direct copy transcoding, this high level of residual activation would rarely cause problems since, in the absence of an additional failure-to-activate deficit for this task, the activation of the target grapheme would always be nearly 100%, ensuring its selection. As a result, very few errors would be produced though when an error is produced it would very likely be a perseveration. This is because the activation of previously produced graphemes would be much higher than the activation for graphemes that had not been recently produced. In contrast, in spelling to dictation, when the same high levels of residual activation occur in the context of weak activation of the target grapheme (due to the failure-to-activate deficit), it would be much more likely that previously produced letters would be selected instead of the target. In sum, we expect many more perseveration errors to occur in spelling to dictation for individuals suffering from both deficits than for individuals with comparable lexical and sublexical deficits who did not suffer from additional failure-to-inhibit impairments.

2.7. Investigating LSS' failure-to-inhibit deficit: Perseverations across cognitive domains

A number of proposals in the literature suggest that the ability to properly inhibit previous information is a domain-general cognitive ability. Friedman and Miyake (2004) argued for a common mechanism underlying the ability to resist intrusions from the past; this mechanism may be responsible for inhibiting previous responses in spelling-to-dictation as well as in other tasks, like picture naming or serial recall. Disruptions to this mechanism have been proposed to underlie the cognitive impairments observed in aging (Hasher & Zacks, 1988), attention deficit hyperactivity disorder (Shallice et al., 2002) and post-traumatic stress disorder (Uddo et al., 1993).¹¹ LSS provides an opportunity to test this domain-general inhibitory control hypothesis. The results of Analysis 4 indicate that LSS was impaired at

properly inhibiting graphemes after production, a deficit that led to perseverations in both spelling-to-dictation and direct copy transcoding. If his inability to properly inhibit graphemes reflects a more general impairment of inhibitory control, then we would expect a similarly, abnormally high, proportion of errors to be perseverations in other tasks as well. Over the course of investigation, LSS was administered a number of other tasks: (1) For spoken naming: the Philadelphia Naming Task (PNT), (2) For verbal working memory: Digit Span, and (3) For visuo-spatial memory: Benton Visual Retention Task (BVRT). Below we report his pattern of perseveration in these tasks.

2.7.1. Philadelphia Naming Task—LSS was administered the Philadelphia Naming Test (Roach et al., 1996) which involves producing a single spoken word to name each of 175 pictures of objects. Responses were categorized as follows: Correct responses, semantic errors, formal errors, mixed errors, nonword errors, unrelated errors, no responses and descriptions. LSS correctly named 112/175 (64%) of the pictures. His 63 errors consisted of 35 no responses (56%), 12 semantic errors (19%), 6 unrelated errors (10%), 5 formal errors (8%), 2 mixed errors that are both semantically and formally related (3%) and 3 descriptions (5%).

LSS produced 11 whole word intrusion responses that were not semantically related to the target (formal errors and unrelated errors).¹² For these errors, we determined that 8 out of the 11 (73%) appeared in one of the three immediately preceding responses. For example, LSS responded “pen” to a picture of a comb, three trials after correctly naming the picture of the pen. A chance analysis was irrelevant in this case as LSS’ intruded responses in this task never appeared outside of the window of the three immediately preceding responses. Therefore, we concluded that 8/11 (.73) of LSS’ non-semantic word intrusion responses were true perseverations errors from one of the three immediately preceding responses. LSS rarely produced phoneme intrusion errors in this task, and therefore we were unable to analyze the proportion of phoneme intrusions that were perseverations.

2.7.2. Immediate serial recall of digits—In digit recall task, digits were spoken by an experimenter at a rate of approximately one per second. At the end of the list, the experimenter indicated that LSS should begin his recall. Lists varied in length from 4 to 8 digits, making use of the numbers 0–12. Over the course of eight sessions, LSS was asked to recall between 20 and 40 lists per sessions, for a total of 255 lists.

Overall, LSS correctly recalled 41% of the lists and 76% of the items (in the correct position). He was more accurate, by item, for shorter lists than for longer lists (Length 4 and 5: 93%; Length 7 and 8: 64%, $\chi^2(1) = 112.77, p < .0001$). His errors consisted of deletions (e.g., [2 9 5 7 3 10] → “2 9 7 3 10”; 6%), insertions (e.g., [4 9 5 8 3 2] → “4 9 5 8 9 3 2”; 5%), substitutions (e.g., [9 9 6 3 8] → “9 9 7 3 8”; 9%) and movements (e.g., [1 9 4 5 8] → “1 9 5 4 8”; 5%), though most of his responses included combinations of these different error types (75%). For the perseveration analysis, a computer program first tabulated the number of LSS’s intrusion errors (both insertions and substitutions) that appeared in at least one of the three immediately preceding responses. The program identified 117 intruded digits. Of those, 106 appeared in at least one of the three immediately preceding responses (92%). A chance analysis determined that, on average, the intruded digit should appear in at least one of the three immediately preceding responses 72% of the time. The observed number of perseverations exceeded the chance values in all 10,000 of the chance analysis

¹¹These results are quite interesting in light of the present case, as, LSS suffered PTSD premorbidly.

¹²Unlike his unrelated errors, LSS’s semantic errors did not appear to be perseveratory. For only one of the 12 semantic or mixed errors was his response a repetition from any prior response (CAKE → “happy bread”, with a correct production of BREAD at E-141).

runs, demonstrating that LSS was producing more digit perseveration errors in immediate serial recall than would be expected by chance ($p < .0001$). His perseveration proportion in this task was calculated to be .66.

2.7.3. Perseveration in the Benton Visual Retention Task—In the Benton Visual Retention Task (BVRT) (Sivian, 1992), a geometric design is presented for 10 seconds and then covered from view. Subjects are then asked to draw the design. LSS was administered all three forms of the BVRT to determine if he produced significant numbers of perseverations. We scored each administration both for number of completely correct items and number of errors produced.

In the first form, LSS correctly reproduced 3 designs, making a total of 10 errors. In the second, he correctly reproduced 4 designs, making a total of 11 errors. In the third, he correctly reproduced 3 designs, making a total of 11 errors. His performance was stable across administration and in each case he was well below the performance expected by age-matched controls (Sivian, 1992).

LSS produced a number of intrusion errors that appeared in one of the three immediately preceding responses. Figure 5 shows an error with the three preceding responses. In the error response, LSS intruded a half-circle figure with a horizontal line. That same response appeared in both E-1 and E-3. We analyzed all of LSS's errors that could not simply be attributed to distortions, rotations or movements of the target figure. In all, 16 figures were intruded. Of those 16 intruded figures, 13 were possible perseverations from at least one of the three immediately preceding responses (13/16: 81%). A chance analysis indicated that, by chance, only 38% of the intrusion responses should appear in at least one of the three immediately preceding responses. The observed value exceeded the all 10,000 runs of the Monte Carlo analysis. As a result, we can conclude that LSS produced perseverations in the BVRT at rates greater than would be expected by chance. From the observed and chance rates a perseveration proportion of .70 was calculated for this task.

2.8 Domain-general perseveration deficit?

LSS produced significant numbers of perseveration errors on non-spelling tasks, perseverating words in spoken naming, digits in immediate serial recall and objects in a visual retention task. This pattern is consistent with a domain-general failure-to-inhibit deficit; according to this hypothesis, the cognitive mechanism responsible for inhibiting responses of many types after production was impaired in LSS, leading to perseveration errors in each of these domains.

However, we urge some caution in interpreting these results. It is unlikely that a failure-to-inhibit deficit is the sole cause of LSS's poor performance in these tasks. Additional spoken production deficits are required to explain why, for example, LSS was unable to name more than a third of the pictures in the PNT, often producing no response rather than perseverating the previous response. Similarly, many of LSS's errors in the BVRT were distortions, rotations or movements rather than whole figure intrusions. An additional visuo-spatial processing deficit is probably required to explain why LSS produced these other error types. What may be surprising, however, is the fact that, in all of these tasks, when LSS produced an intrusion error, it was quite likely that the intrusion error was a true perseveration from one of the three immediately preceding responses. In all of the tasks, between 65–75% of his intrusion errors were true perseveration errors from at least one of the three immediately preceding responses (Spelling-to-dictation: 73%; Direct copy transcoding: 65%; PNT: 73%; Immediate Serial Recall: 66%; BVRT: 70%). These perseveration proportions are generally higher than those commonly reported elsewhere. In the analyses of perseveration errors in aphasia (Martin & Dell, 2007), participants typically

produce twice as many non-perseverative as perseverative errors, while LSS produced perseverative errors three times as often as non-perseverative ones. Estes (1991) reports that approximately 60% of all digit intrusion errors in a serial recall task appear in E-1, while for LSS, 83% of his digit intrusions appeared in E-1. While perseverations may be common in both spoken production and immediate serial recall, LSS appears to have an abnormally high perseveration proportion, though further work would be required to demonstrate that LSS truly is an outlier in these tasks.

As discussed above, an abnormally high perseveration proportion is a hallmark of a failure-to-inhibit deficit. In the normal case, persisting activity results in previously produced items being more likely than items not produced recently to intrude in the place of the target. With a failure-to-inhibit deficit, abnormally high persisting activity boosts the advantage that previously produced items have over items not recently produced. This change results in a higher perseverative proportion. Therefore the pattern observed in LSS – significant numbers of perseveration errors and a high perseveration proportion across a number of cognitive domains – supports the hypothesis of a domain-general failure-to-inhibit deficit, presumably in addition to domain-specific deficits in spelling, spoken production, verbal WM and visuo-spatial processing.

3. GENERAL DISCUSSION

3.1 Summary of findings

In the present study, we examined the nature of the underlying deficit(s) that lead to the production of letter perseveration errors by dysgraphic individuals. Applying logic similar to that used in recent work investigating the underlying cause(s) of verbal perseveration errors in picture naming (e.g. Cohen & Dehaene, 1998; Dell, Svec & Burger, 1997; Martin & Dell, 2004, 2007; Moses, Nickels & Sheard, 2007), we investigated the patterns of letter perseveration errors produced by a set of twelve dysgraphic individuals. We examined the hypotheses that perseveration errors arise because of a failure-to-activate the letters in the current trial or because of a failure to inhibit letters from previous response, or because of both.

All of the individuals tested had deficits to both the lexical and the sublexical spelling routes, a situation that could be expected to result in a failure to robustly activate target graphemes and, therefore, allowing us to test the predictions of the failure-to-activate hypothesis of perseveration errors. The results provided clear support for the predictions of the failure-to-activate hypothesis; namely, we found that: (1) all of the individuals produced letter perseverations at significantly above chance rates, (2) there was a significant correlation between the rate of perseverative and non-perseverative intrusions and (3) there was a significant correlation between the degree of sublexical deficit and the proportion of intrusion errors that were perseverations.

We also found that the failure-to-activate hypothesis alone could not explain the pattern of perseveration observed in all individuals. LSS produced perseveration errors at rates greater than would be expected by either his degree of sublexical impairment or his rate of non-perseverative intrusion errors. We argued that LSS suffered an additional failure-to-inhibit deficit. As predicted by that hypothesis, we found that LSS produced a significant number of letter perseveration errors in direct copy transcoding, a task that relies on the level of grapheme representation but does not require lexical or sublexical activation. Furthermore, we presented a set of results indicating that LSS' failure-to-inhibit deficit may have resulted from an impairment to a general cognitive function rather than a function that is specific to the level of grapheme representation. This conclusion was based on the finding that LSS produced significant numbers of perseveration errors in various additional tasks – spoken

production, immediate serial recall of digits and visual-spatial memory – and in each case his intrusion errors were very likely to be perseverations. Given a similar pattern of perseveration errors across tasks, it is possible that LSS's inhibition deficit was general, rather than specific in nature.

3.2. Failure-to-inhibit deficits

The current study was designed in such a way that we were unable to identify any individuals whose perseverations were caused by a failure-to-inhibit deficit alone. This is because all the study participants were selected precisely because they had both lexical and sublexical deficits, resulting in a failure to activate target graphemes and thus allowing us to evaluate the predictions of the failure-to-activate hypothesis. Therefore, at a minimum, all individuals in the sample perseverated because of a failure-to-activate deficit. Whether a failure-to-inhibit deficit alone can cause perseveration errors clearly remains an open question for future research. The ideal case would be an individual whose errors in spelling both words and nonwords are entirely perseverative intrusions, who shows no frequency effects (indicating no lexical impairment) and produces comparable numbers of letter perseverations in all tasks that rely on the graphemic level (e.g., both spelling-to-dictation and direct copy transcoding). As with LSS, one important issue will be to determine whether such an individual's failure-to-inhibit is specific to the spelling system or reflects a more general cognitive impairment.

3.3. Further predictions

In this investigation, we focused on patterns of perseveration errors across dysgraphic individuals to test the predictions of failure-to-activate and failure-to-inhibit hypotheses. It may be possible to test these hypotheses by examining which attributes of the target (e.g. whole word frequency, sublexical consistency) predict a higher rate of perseveration. For example, lexical impairments affect low-frequency words more than high-frequency words. As a result, in cases such as those which we examined in this study, graphemes in low-frequency words should receive abnormally low activation relative to graphemes in high frequency words. This, in turn, should lead to greater rates of perseveration for lower frequency words. A similar case may be made for sublexical consistency. Some phoneme-to-grapheme mappings are highly predictable (for example, the phoneme /l/ is always spelled with the letter L) while for others, a single phoneme can be spelled with many different graphemes (the phoneme /i/ can be spelled as EE, EI, EA, IE, EY, etc). Graphemes with inconsistent phoneme-to-grapheme mappings may receive less activation from the sublexical route than graphemes with consistent phoneme-to-grapheme mappings (Jones, Folk & Rapp, 2009). Therefore, it may be more likely for perseverations to occur in the place of graphemes with low phoneme-to-grapheme consistency. We were unable to test these fine-grained predictions given that the items in the stimulus lists were not controlled for all of the relevant variables required for these detailed analyses. However, these types of analyses may provide critical converging evidence for the failure-to-activate hypothesis, and carefully designed experiments should be carried out to test these predictions.

3.5. Perseveration errors and implications for normal spelling

In addition to helping us understand the causes of letter perseveration errors in dysgraphia, the findings we have reported also have implications for our understanding of the operation of the normal, intact spelling system. We have presented evidence that letter perseveration errors should occur when the correct target grapheme receives little activation from the lexical and sublexical routes. However, this account requires previously produced graphemes to normally persist at above baseline activation on subsequent trials (Cohen & Dehaene, 1998).

Consistent with this assumption, normal effects of grapheme persistence have been revealed in other written production tasks. Damian and Stadthagen-Gonzalez (2009: Experiment 1) showed significant priming effects in single word writing; it took participants significantly less time to initiate a written response in a block in which all responses shared an onset than in blocks in which responses started with different letters. They interpreted this result to suggest priming at the grapheme level. Previously produced graphemes were more easily initiated than graphemes that were not previously produced. One account of this priming effect is that graphemes from a previous trial retain some residual activation after production. This residual activation allows the grapheme to be more easily selected on a subsequent trial, speeding up the time to initiate a response. Thus, both the reaction time effects in single word writing and the letter perseveration errors can be explained, in part, by assuming persisting activation after production at the level of grapheme representations.

Our investigation also indicates that in at least some individuals a failure-to-inhibit deficit contributes to letter perseveration errors. By this account, in the intact system, some mechanism normally inhibits graphemes from a prior response before initiating a subsequent trial. Stemmer (2009, see also Houghton, Glasspool and Shallice, 1994) argues that this self-inhibitory mechanism is critical for cognitive processing precisely because it prevents previously produced items from being repeated. When this mechanism fails, the graphemes from previous responses remain relatively more active and are occasionally perseverated. Furthermore, our results suggest that, at least in some individuals, the failure-to-inhibit deficit may be due to an impairment to a general inhibitory mechanism, rather than an inhibitory mechanism specific to the grapheme level in spelling. This is consistent with other proposals of a general inhibitory mechanism. Using correlations of individual performance on a battery of inhibition tasks like the Brown-Peterson task and Cued Recall, Friedman and Miyake (2004) argued that a common mechanism underlies the ability to resist intrusions from the past. Jonides and Nee (2006) suggested that the left inferior front gyrus is involved in resolving this competition between the previous and current trials. Their results converge nicely with the proposals of Thompson-Schill and colleagues (January, Trueswell & Thompson-Schill, 2009; Novick, Kan, Trueswell & Thompson-Schill, 2010; Novick, Trueswell & Thompson-Schill, 2005; Schnur et al., 2009; Snyder, Feigenson & Thompson-Schill, 2007; Thompson-Schill, D'Esposito & Kan, 1999; Thompson-Schill et al., 2002), who argued that the left inferior frontal gyrus acts as a general mechanism responsible for resolving competition in a number of tasks: picture naming, blocked cyclic naming, resolving semantic ambiguity, the Stroop interference paradigm, and a working memory probe task that includes negative probes from recent lists. Alternatively, a failure-to-inhibit deficit may have a neurochemical cause. McNamara and Albert (2004) argued that depletion of neurotransmitters from the prefrontal cortex – specifically dopamine and acetylcholine – leads to impairments in inhibitory control and therefore verbal perseverations.

3.6. Multiple underlying causes of perseveration errors

As we noted at the beginning of this paper, perseveration errors are commonly observed in individuals with neural impairments and, therefore, it is not surprising that understanding the deficits that give rise to these perseveration errors has been a research focus in many cognitive domains. In particular, much of the recent work on verbal perseverations in aphasia has concluded that perseveration errors arise from a failure-to-activate deficit, based on data from case studies and case series (e.g., Ackerman & Ellis, 2007; Cohen & Dehaene, 1998; Gotts, della Rocchetta & Cipolotti, 2002; Hirsh, 1996; Martin & Dell, 2007; Moses, Sheard & Nickels, 2007). While the findings of our investigation provide strong additional evidence for a failure-to-activate account of perseveration errors, they also provide a note of caution in interpreting results in spoken production and other domains. Our findings indicate

that, as in dysgraphia, it is possible that failure-to-inhibit deficits may contribute to perseveration errors observed in other domains. Identifying these deficits will require the careful analysis of the performance of individuals who deviate from patterns predicted by failure-to-activate deficits alone.

Highlights

> Letter perseverations are common when dysgraphic individuals spell. > Perseverations can be caused by a failure to activate target graphemes. > An additional failure-to-inhibit deficit results in high rates of perseveration. > This failure-to-inhibit deficit may reflect a domain-general mechanism.

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Figure 1.
Schematic depiction of the cognitive spelling mechanisms involved in the tasks of spelling to dictation and also direct copy transcoding

Figure 2.

For each of the 12 study participants, the proportion of intruded letters that appeared in one of the three immediately preceding responses (black bars) and the mean proportion expected by chance from 10,000 chance analysis runs (white bars). The total number of intruded letters analyzed for each participant is reported in parentheses below their initials.

Figure 3. Rates of perseverative and non-perseverative letter intrusion errors for the 12 study participants.

Figure 4. Perseveration proportions and nonword letter accuracy for the 12 study participants.

Trial	Target	Response
E
E-1
E-2
E-3

Figure 5.
For the Benton Visual Retention Test (BVRT; Sivian, 1992), example of an intrusion error on Trial E with the responses for the three immediately preceding trials (E-1 to E-3).

Table 1

Contrasting predictions of the failure-to-activate and failure-to-inhibit hypotheses of letter perseveration in spelling-to-dictation

Prediction	Failure-to-activate	Failure-to-inhibit
<i>Prediction 1:</i> Individuals with lexical and sublexical deficits produce perseverations	□	□
<i>Prediction 2:</i> Correlation between rate of perseverative and non-perseverative errors	□	□
<i>Prediction 3:</i> Correlation between sublexical deficit and perseveration proportion	□	□
<i>Prediction 4:</i> Perseveration errors in both spelling-to-dictation and direct copy transcoding	□	□

Table 2

Letter Accuracy for high and low frequency words and nonwords on spelling lists from the Johns Hopkins Dysgraphia Battery (Goodman & Caramazza, 1995). These patterns indicate both lexical and sublexical deficits for each participant.

	High Freq.	Low Freq.	Nonword
VBR	0.98	.91***	0.67
DHY	0.94	.87***	0.65
AES	0.97	.88***	0.72
BWN	0.98	.96**	0.77
MMD	0.94	.89***	0.81
JCY	0.91	.85**	0.77
CM	0.78	.73*	0.52
ELE	0.62	.48***	0.66
LSS	0.66	.47***	0.40
DLE	0.46	.40*	0.42
LHT	0.56	.44***	0.44
GBT	0.49	.36*	0.46

High Frequency > Low Frequency,

*
 $p < .05$,

**
 $p < .01$,

 $p < .001$

Table 3

Example of an intrusion error (trial E) with five preceding responses

Trial	Target	Response
E	UNDER	UNDEL
E-1	MOTEL	MOL <u>DE</u> L
E-2	SHOULD	SHOU <u>L</u> D
E-3	VULGAR	CHEU <u>P</u> IVE
E-4	CHEAP	CHEAP
E-5	CERTAIN	ABSEVE

Appendix 1

Individual participant data including number of words spelling, accuracy (both by word and by letter) as well as values derived from the letter perseveration analysis.

	Words Spelled	% Cor Words	% Cor Letters	Letters Produced	Intrusions	Potential Perseverations	pChancedL inPersev	True Perseverations (% Letters)	Non-perseverative intrusions (% Letters)	Perseveration Proportion
VBR	317	74.1%	93.0%	1337	44	30	0.58	10.97 (0.8)	14 (1.0)	0.25
DHY	545	74.1%	91.6%	2624	118	79	0.61	17.97 (0.7)	39 (1.5)	0.15
AES	433	72.5%	91.1%	2743	82	50	0.54	12.92 (0.5)	32 (1.2)	0.16
BWN	729	70.5%	93.7%	3043	48	34	0.62	11.14 (0.4)	14 (0.5)	0.23
MMD	2087	64.4%	89.3%	7926	426	264	0.54	76.39 (1.0)	162 (2.0)	0.18
JCY	311	61.1%	88.6%	1904	84	52	0.49	21.73 (1.1)	32 (1.7)	0.26
CM	3795	55.4%	82.3%	15896	2488	1849	0.58	970.54 (6.1)	639 (4.0)	0.39
ELE	562	39.0%	71.0%	1737	481	309	0.53	113.27 (6.5)	172 (9.9)	0.24
LSS	1636	20.0%	57.1%	6808	2299	1996	0.52	1668.93 (24.5)	303 (4.5)	0.73
DLE	616	10.6%	49.8%	1906	713	467	0.57	146.64 (7.7)	246 (12.9)	0.21
LHT	615	9.5%	52.7%	2736	1255	1000	0.71	380.26 (13.9)	255 (9.3)	0.30
GBT	342	2.9%	51.7%	1438	487	357	0.56	191.28 (13.3)	130 (9.0)	0.39