Theoretical analysis of word production deficits in adult aphasia

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The cognitive analysis of adult language disorders continues to draw heavily on linguistic theory, but increasingly it reflects the influence of connectionist, spreading activation models of cognition. In the area of spoken word production, ‘localist’ connectionist models represent a natural evolution from the psycholinguistic theories of earlier decades. By contrast, the parallel distributed processing framework forces more radical rethinking of aphasic impairments. This paper exemplifies these multiple influences in contemporary cognitive aphasiology. Topics include (i) what aphasia reveals about semantic-phonological interaction in lexical access; (ii) controversies surrounding the interpretation of semantic errors and (iii) a computational account of the relationship between naming and word repetition in aphasia. Several of these topics have been addressed using case series methods, including computational simulation of the individual, quantitative error patterns of diverse groups of patients and analysis of brain lesions that correlate with error rates and patterns. Efforts to map the lesion correlates of nonword errors in naming and repetition highlight the involvement of sensorimotor areas in the brain and suggest the need to better integrate models of word production with models of speech and action.

1. Introduction

Aphasia in adults is caused by stroke, trauma or degenerative pathology that compromises brain networks for language. Arguably, the most pervasive symptom of aphasia, within and across etiologies, is the inability to produce known words in a timely and accurate manner. The word production deficit is readily detected by tests of picture naming, where it manifests in hesitations, word-finding gaps and/or commission errors. The focus of this paper is the model-driven analysis of naming errors in patients and what this reveals about the functional architecture of word production.

Recent decades have witnessed a change in both the models and methods of cognitive aphasiology. In place of the traditional box-and-arrow diagrams, contemporary models feature connectionist networks, with units corresponding to localist or distributed representations that are primed and retrieved through the mechanism of spreading activation. Computer-implemented models are necessarily highly specific in their representational and processing commitments and so have taken on an important role in theorizing about aphasia, as they have in normal language research.

The research designs used to collect aphasia data have also changed. The single-subject approach has evolved into case series methods, in which multiple individuals are studied on the same set of tasks with the goal of understanding why the patients differ from one another. In small case series studies, each individual can be studied intensively with multiple different tasks, in the manner of single case studies. Large case series studies trade depth of assessment for the opportunity to test a larger, more diverse, and sometimes more representative, sample of patients. The objective is then to explain the behavior of interest by analysing patterns of covariation within and across tasks (see [1]; and ensuing commentary in Cognitive Neuropsychology, 28/7, 2011). Data generated with case series methods have been simulated computationally. They have also been used in conjunction with advanced lesion-mapping methods to localize the lesions ever so closely related to the observed errors.
that explain the pattern of symptom variation. We will see examples in the sections that follow.

2. The two stages of word production

During the 1970s, linguistics-inspired analysis of speech error corpora produced a theory of sentence production that greatly influenced research in aphasia [2]. An enduring legacy of that theory is the idea that words are retrieved in two, discrete, serial stages, one concerned with selecting a word or ‘lemma’ [3] from the mental lexicon, the second with attaching form to the selected word. The first stage is responsive to meaning and grammar; the second to phonological structure and content [4–8].

The two-stage assumption has been implemented in localist connectionist models, where nodes at one level of the network stand in one-to-one correspondence with words. Within this class of models, some accept the discreteness of the stages [9], whereas others postulate an interactive flow of information between them [7,10]. They also differ in how many levels of representation are proposed and how they are characterized (for review [11,12]).

Along with experimental and speech error evidence from normal speakers, data from aphasia have contributed to the development and evaluation of competing models. In aphasia, error production tends to be high even in single-word production tasks like picture naming, and this has enabled researchers to use error data from these simple, controlled paradigms to address theoretical issues. A case in point is the debate about interactivity.

Table 1 shows some of the error types that researchers study. Error classification is invariably theory-driven, so the categories and definitions of error types vary somewhat from study to study. The definitions in the table are those that my colleagues and I have used in the case series studies referenced in this article.

Most individuals with aphasia produce a variety of these error types in their naming performance. However, much interest has centered on the occasional selective presentation, e.g. where one patient produces mostly phonological errors (formal errors and nonwords) and another, of comparable severity, produces mostly semantic errors. Double dissociations like these might be viewed as supporting the two-stage theory and, more specifically, the proposal that the semantic and phonological stages are discrete, i.e. non-interacting.

On the other hand, in studies of naming errors aggregated from multiple patients the interactive account has received support. The evidence here is that mixed errors and formal errors, both of which involve substitution of a word phonologically related to the target, occur at significantly higher frequencies than the discreteness account predicts [14–16]. This indicates that phonological information does not operate exclusively at the phonological stage of production; rather, it is accessed prior to lemma selection and plays a role in which lemma gets selected.

A recent study adds to the evidence for interaction by showing that a phonological variable, phonological neighbourhood density, can bias lemma selection in favour of the correct target [17]. This study corroborated previous evidence that naming accuracy is higher for words with many than few phonological neighbours [18–21]. Its novel contribution was to show this neighbourhood density effect in a patient whose error pattern was indicative of a problem at or before lemma selection (i.e. the patient’s errors were predominantly of the semantic type). That a stage-1 production problem was ameliorated by a variable that operates at stage 2 is strong evidence for interaction across the stages.

As noted earlier, computational modelling has come to play an increasingly important role in aphasia theorizing. In studies that simulate individual patients’ error patterns with implemented models, the best fits have been obtained with models that incorporate interaction. Importantly, though, the most successful of these models contain features designed to set limits on the interactive flow of information [11]. The next section describes in detail one such model and the evidence that supports it.

3. The interactive two-step model of lexical access

This model, developed by Dell and co-workers [13,22], has been used to fit data from patients in numerous studies of word production deficits [13,23–30]. Researchers interested in applying the model to new data can access the automated data-fitting program at http://langprod.cogsci.illinois.edu/cgi-bin/webfit.cgi.

The model contains a three-level lexical network consisting of semantic features, words, and phonemes (figure 1) and weighted connections that transmit activation both top-down and bottom-up. All activation is positive; there is no inhibition. All words are CVC syllables, with phonemes marked for syllable position. This is a model of a stable system after learning has been completed: connection weights are comparable and fixed for all words and all phonemes and the model does not represent variations in lexical frequency

<table>
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<th>Table 1. Taxonomy of error types (from [13]).</th>
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<td>Semantic—Real word response that is a synonym, category coordinate, subordinate, or strong associate of the target (e.g. pig → ‘sheep’; church → ‘building’; shoe → ‘Nike’; apple → ‘worm’).</td>
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<tr>
<td>Mixed—Real word response that qualifies as a semantic error and that meets the criterion for phonological similarity*(e.g. cow → cat; skirt → shirt).</td>
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<td>Formal—Real word response that meets the criterion for phonological similarity*(e.g. dog → dock; pirate → tire).</td>
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<tr>
<td>Unrelated—Real word response that is neither semantically nor phonologically similar to the target (e.g. banana → camp; well → car).</td>
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<td>Nonword—String of phonemes that does not constitute a word in the language (e.g. camel → /kæməl/ (‘kah-mer’); piano → /pɪənəʊ/ (‘pih-nah-no’). Most such errors pass the phonological similarity criterion*, and, depending on the study, this can be a requirement for inclusion in the category.</td>
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*Response and target must share at least one phoneme in corresponding syllable or word position of two phonemes in any position, not counting unstressed vowels.

http://rstb.royalsocietypublishing.org/ Downloaded from on June 12, 2017
(but see [30]). A naming trial begins with external activation supplied to the target’s semantic features. Activation flows freely for a designated number of time steps, after which the most activated word is selected. This completes step-1. Step-2 begins with external activation supplied to the selected word, designed to simulate grammatically triggered phonological encoding. This activation propagates down to the phonemes of the selected word and back up from each phoneme to all words in the lexicon that contain that phoneme in that syllable position. Activation reverberates throughout the network until, after a fixed number of time steps, the most activated segments from the onset, vowel and coda segment clusters are selected, terminating step-2 and the trial, and defining the model’s response.

As noted, activation flows bidirectionally during each of the two steps of lexical access. However, the design of the model was influenced by experimental evidence showing that early in a word retrieval episode, semantic influences dominate, whereas later in the episode, phonological influences dominate [31]. In keeping with this, the model’s default activation parameters were set so that step-1 selection is primarily influenced by top-down activation from semantics. Phonological feedback during step-1 has its primary impact on the cohort that competes for word selection, where it privileges candidates that share the target’s phonemes. This effect of feedback explains the above-chance incidence of mixed and formal errors, noted above.

Now consider step-2: in a model where the reciprocal feedback between semantics and phonology fully integrates these two information sources, the phonemes of words in the semantic neighbourhood of the target would be expected to enter into the competition for selection at step-2, along with those of the target. The interactive two-step model exhibits little competition of this kind, for the reason that the external activation supplied to the selected word at the start of step-2 boosts its segments over those of other words in the semantic cohort [11]. The weakness of competition for phoneme selection is key to why having many phonological neighbours helps rather than hinders phonological access [19,20].

By design, then, the interactive two-step model operates in a way that is generally modular (semantic and phonological information sources are largely restricted to step-1 and step-2, respectively) but locally interactive (feedback from later to earlier levels influences the competition for selection) [22]. The model has been tested for its ability to simulate individual differences in the naming response patterns of diverse groups with aphasia [13,23,32]. The naming response pattern is the proportional breakdown of total naming responses into correct responses and the five error types shown in table 1. According to the model, semantic, mixed, formal and unrelated errors are the product of incorrect word selection at step-1. Nonword errors result from faulty segmental selection at step-2.

Here is how the simulations worked: the model was first implemented to account for the normal naming pattern (mostly correct with a small number of semantic errors), and then assumptions were made about how aphasia alters the normal system [13,23,32]. The semantic-phonological account [32] proposes that aphasia alters the weight of connections in the lexical network, weakening the flow of activation between semantics and words (s-weight lesion) or between words and segments (p-weight lesion), or both. In a computational case series study with 94 patients, Schwartz et al. [23] found that the semantic-phonological account explained 94.5% of the variance in the individual naming response patterns.

As discussed by Schwartz et al. [23], the key reason why the semantic-phonological account is able to explain diverse, individual naming response patterns in aphasia is that it allows errors generated at step-1 (lexical errors) and those generated at step-2 (sublexical errors) to dissociate. My colleagues and I extended the evidence for the distinctiveness of these error generators in a series of studies that mapped the neural correlates of particular error types and model-driven weight parameters [33–37]. The key finding, for present purposes, is that the neural correlates of semantic errors (the prototypical step-1 error type) and nonword errors (step-2) were different and non-overlapping. As these lesion-mapping studies will be mentioned again later, some details are in order.

The method we used is called voxel-based lesion-symptom mapping (VLSM) [38]. Each of our studies started with case series behavioral data, in which the N’s ranged between 64 and 106. After tracing each patient’s lesion on the high-quality structural brain scan obtained under a research protocol,
4. Alternative accounts of semantic errors

The interactive two-step model of word retrieval has engendered considerable debate. One aspect of the debate centres on the model’s assumptions about interactivity. Rapp & Goldrick [11] proposed an alternative model, the restricted interaction account (RIA). While RIA, too, implements the two-step assumption in a localist connectionist architecture, it differs from Dell’s model in a few key respects, among which are that feedback from phonology reverberates between the phonological and lexical levels but does not extend to the semantic level. Rapp and Goldrick compared the computational fit of these two models, and some others, with the individual data from three contrasting aphasia cases and found that RIA produced the best fits. Additional arguments and evidence about the interaction assumptions in these two models can be found in the earlier studies [27,28,39].

The Dell and Schwartz model-fitting studies have been criticized for their single task focus. The concern is that by failing to consider patients’ performance on tasks other than oral naming, we missed the opportunity to independently verify the model’s account of where the deficit resides and, more importantly, to obtain evidence that might falsify model assumptions [39]. This is a valid criticism. For example, the model assumes that all semantic errors arise from weak s-weights and so does not recognize the possibility that semantic-level processing itself could be impaired. Yet, further study of patients who produce semantic errors in naming has shown that some do have semantic-level deficits (e.g. confusion between semantically similar items in comprehension); others, however, do not [11,40–44]. This indicates that the model is flawed in its assumption that all semantic errors are errors of lexical selection. RIA [11] potentially escapes this problem by allowing for damage (in the form of noisy activation) to the model’s semantic level, in addition to its lexical and phonological levels.

In a more radical departure from Dell’s model, it has been proposed that all semantic errors in production are the consequence of damage at the semantic level. The motivation for this is the parallel distributed processing (PDP) account of language production, which rejects the two-step assumption altogether. In the PDP framework, there is nothing in the structure of the system that corresponds to individual words. What appear to be lexical effects are properties that emerge from the functional interplay between semantics and phonology, which are fully interactive [45–48].

Semantics and phonology, in this account, are ‘primary systems’, each comprised representations that were learned in childhood under pressure to concurrently produce, comprehend and imitate language [46,49]. Having been shaped in this way, these representations are amodal in the sense that they support all manner of language tasks and modalities in adulthood. It follows, then, that patients’ accuracy and error patterns in naming might be predictable from their performance on other tasks. This prediction was tested by Lambon Ralph et al. [47] in a paper entitled, ‘Anomia is simply a reflection of semantic and phonological impairments’. It reported case series data from 21 patients with wide-ranging anomia, including evidence that the variance in accuracy in naming was predicted by a linear combination of composite semantic and phonological
measures (60–70% of the variance explained). Moreover, examination of the bivariate correlations between composite scores and error types showed that consistent with expectations, greater semantic deficit was associated with more semantic errors and omissions, whereas greater phonological deficit was associated with more nonwords.

In recent years, the debate between the two-step and PDP camps has moved into the cognitive neuroscience arena. Details are beyond the scope of this paper, but we can see some of what is at issue by looking back at figure 2. Recall that the area mapped in purple is where lesion status correlated with semantic errors in our VLSM of naming errors. It includes an area in the left temporal lobe that stretches from the mid-part of the middle temporal gyrus forward to the temporal pole. This area sits within the anterior temporal lobe (ATL), a region made famous by the ‘distributed-plus-hub’ theory of semantic memory [50]. The ATL is the hypothesized hub, proposed as the storage site for the amodal semantic representations that support language in multiple modalities [49]. While there are semantic hubs in both hemispheres, the one on the left is thought to play an especially strong role in production by virtue of its proximity to left-lateralized phonological areas [51].

At first blush, our finding that semantic errors in naming localize to the ATL would appear to accord well with this primary systems account. However, there is an important wrinkle to the story. The VLSM of semantic errors statistically controlled for errors that might have arisen at the semantic level by regressing out scores on comprehension measures. As my colleagues and I have argued [33,35], the evidence therefore favors the two-step account over the primary systems account by identifying a brain locus for production only semantic errors, exactly as would be expected if semantic errors arose in the process of accessing lemmas from semantics. Within the wider cognitive neuroscience literature, support can be found for both these theoretical positions [52–57].

Referring again to figure 2, we see that the lesion map for semantic errors (purple area) also includes a region in the left prefrontal cortex. It is possible that lesions to this area generate semantic errors by compromising cognitive control mechanisms involved in language production. There is substantial evidence that damage to left ventrolateral prefrontal cortex impacts aphasics’ ability to resolve competition for semantic and lexical selection under experimental conditions that exaggerate competition [58–60]. Our prefrontal finding may indicate that this competition-resolving mechanism plays a critical role even in a simple naming task.

5. The interactive two-step account of word repetition

Competing models of word production are challenged to explain other types of production tasks in addition to naming, most notably word repetition. Naming and repetition deficits frequently co-occur in aphasia, but they can also dissociate. Some have used such evidence as a means to distinguish phonological deficits that originate within the lexicon from those that originate in a post-lexical phonological/phonetic stage of processing. One proposal is that an impairment in lexical-phonological processing will impair naming, but not word repetition, because repetition can be performed without lexical mediation, as is the case when one repeats a nonword. On this view, comparable performance in naming and word repetition is evidence of a post-lexical phonological deficit [12]. Space precludes further consideration of the lexical/post-lexical distinction (see [61] for an interesting perspective). However, the relationship between production mechanisms in repetition and naming will be considered in some detail.

Most researchers would agree that while a phonological deficit of some sort lies at the heart of patients’ difficulties with repetition, lexical and semantic factors also play a role, even at the level of single words [62,63]. This motivates the effort to explain repetition within a word production model that includes semantic, lexical and phonological representations, and interactive activation between them. The rest of this section describes efforts to apply the interactive two-step model to the task of single-word repetition. For an account of repetition that adopts the primary systems view, see Ueno et al [55].

Dell and co-workers [24,30,64] carried out a series of computational studies of repetition in which they modified the basic naming model to instantiate different accounts of the relationship between naming and repetition, and then fit the alternative models to word repetition data from individual subjects or case series data. As a general feature, the implemented models included separate units and connections for phonological input and output processing (65); see [66] for a different approach), and the input processes were assumed to be intact, both in the model and in the patients whose data were simulated.²

Two repetition models were compared. Both commit to the proposition that word repetition involves retrieval of lexical-phonological representations via the second step of the lexical access model. The lexical-only account of repetition proposes that this is the sole mechanism by which the adult speaker repeats words. By contrast, the summation dual-route account, which was inspired by Hillis & Caramazza [67], proposes that phonological access in repetition is accomplished jointly by the lexical route and a second, non-lexical route. Figure 3 presents schematic diagrams of these two repetition models along with the naming model.

The lexical-only model makes the strong prediction that repetition can be accurately predicted from naming performance at the individual level. Simulating a patients’ repetition performance under the lexical-only model started by setting the naming model’s s- and p-weights in accordance with that individual’s naming response pattern. Each simulated repetition trial began with external activation supplied to the target lemma, implementing successful word recognition. As in naming, the activation was allowed to reverberate among the phonological, lexical and semantic units until eventually the most active phoneme units were chosen to represent the repetition response. Errors and accuracy were tabulated across trials to generate the predicted outcome for the patient.

To implement the summation dual-route account, a non-lexical route was grafted on to the interactive two-step model [24]. This route consisted of a single input node outside the model, which connected to the model’s output phonemes via connections whose weights represent the strength of the non-lexical route (figure 3). This parameter (‘nl’, for non-lexical) was estimated separately for each patient according to how accurately the patient performed a nonword repetition test. To run the summation dual-route model for a particular patient, the s-weight, p-weight and nl parameters were set based on the patient’s naming and nonword repetition, respectively.
On each trial, external activation was applied to the external input node, triggering non-lexical-route processing, and also to the target lexical node, triggering lexical-route processing. After a designated time period, the activation in the output units from both routes was summed to produce the response. Once again, errors and accuracy were tabulated across trials to arrive at model predictions, and these were compared with the actual word repetition scores of the patient.

Figure 4, from [64], shows that the lexical-only model did quite well in predicting word repetition accuracy. For most of the 59 patients studied, the deviation between predicted and obtained proportions was less than 0.20 (dotted lines in the figure). However, there were seven patients, represented as points above the upper dotted line, whose repetition was better than the lexical-only model predicted. These patients were more accurately fit by the summation dual-route model. However, for the majority of patients, the summation dual-route model predicted higher accuracy than was actually observed (see also [24]).

Nozari et al. [30] took a different approach to comparing the lexical-only route and summation dual-route repetition models. Their study was based on the well-founded assumption that the frequency-sensitivity of word repetition can be used to gauge the involvement of the lexical route. The study is complicated, and so I will limit my discussion to the major findings, which relate to the incidence and frequency-sensitivity of nonword errors in word repetition, compared with picture naming.

The investigators first analysed the naming and repetition responses of 59 diverse aphasic speakers using hierarchical, multinomial logistic regression analyses. This revealed that patients produced significantly fewer nonword errors in repetition than naming, but the frequency-sensitivity of nonword errors was as great in repetition as it was in naming. The latter is a striking finding, and strong indication that the lexical route is routinely used in repeating words. In the computational portion of the study, Nozari et al. adjusted lexical connection weights to implement frequency differences, and then ran the two repetition models, along with the naming model, to compare frequency-sensitivity across the models. The counterintuitive finding here was that the two repetition models yielded frequency effects that were equally strong and comparable to the naming model. The frequency effect in the dual-route repetition model was not diminished by the presence of the non-lexical route, because
effects of the two routes were additive (summed). This meant that the two routes combined without any loss; when the non-lexical route was added, the lexical route contributed as much as when the non-lexical route was absent.

Whereas the involvement of the non-lexical route did not diminish the size of the model’s frequency effect, it did reduce the model’s production of nonword errors and, consequently, made its repetition more accurate. With the summation dual-route model, as with the patient data, nonword errors were less numerous in repetition, compared with naming, but these errors were equally sensitive to frequency across tasks. In this study, then, the summation dual-route model provided the best account of the overall findings. At the same time, in summarizing findings for individual patients in this and previous comparisons of the repetition models, Nozari et al. emphasize that there are important individual differences in whether and to what degree the non-lexical route is added when repeating words. What accounts for these individual differences? Clues can be found in a recent meta-analysis that showed that patients who repeat by the lexical-only route tend to have better comprehension, whereas dual-routers have better phonological working memory [68].

6. Brain localization for nonword errors in naming and repetition

A central claim of the interactive two-step model of lexical access is that impairment in the step-2 mapping between lexical and phonological units is a cause of nonword errors in both naming and word repetition. VLSM offered interesting possibilities for testing this claim.

Earlier, I mentioned the VLSM analysis we ran to identify the neural correlate of nonword errors in picture naming [36]. The results of that analysis, which figure 2 displayed in blue, are reproduced in figure 5a using a more informative colour scale. The accompanying figure 5b shows the results of a VLSM of nonword errors in word repetition, based on same group of 103 patients. Importantly, for the latter analysis, scores on nonword repetition were regressed out to control for possible impairments in non-lexical-route processing (input or output) that might have influenced the word repetition data. This amounted to statistically isolating the contribution of lexical-route damage to production of nonword errors in word repetition.

Although the errors mapped in the two VLSM analyses derive from different tasks, according to the model they share a common functional locus and so should converge on the same region of brain. Confirming this prediction, the maps in figure 5a,b are strikingly similar. In both cases, the significant or nearly significant voxels localized to the anterior parietal lobe (supramarginal and post-central gyri), extending forward into pre-central and pre-motor cortices.

These fronto-parietal regions have been implicated in many prior neuroanatomical studies of phonological processing [69,70], including a large lesion-mapping study of phonological errors in acute aphasia [71]. On the other hand, in neuroimaging research aimed at identifying the neural substrate of lexical-phonological retrieval, the area most commonly identified is located more posteriorly, in temporal and temporo-parietal cortices inclusive of Wernecke’s area [53,72,73]. It was unexpected that the VLSM of nonword errors would fail to pick out this temporal lobe region while identifying strong effects in fronto-parietal regions most often associated with the planning and regulation of speech and action.

What lessons should we draw from this? Perhaps, we should be looking to theoretical accounts of language production that are more grounded in sensorimotor processes, for example, the theory of articulatory phonology that postulates that units that enter into phonemic speech errors are actually units of articulation [74]; see also [75]. Certainly, we need a better understanding of the mechanisms that transform static lexical representations into a temporally extended, ordered sequence of articulatory gestures. The PDP framework seems to offer an interesting way forward [46,55]. Also, there may be useful convergence with research looking at how the brain solves the serial-ordering problem in other, related domains, such as sign-language production and the gestures that accompany spoken speech [76].

7. Conclusion

This paper has presented a selected review of research on word production deficits in adult aphasia, emphasizing the
interactive two-step model of lexical access and some of the patient studies it has inspired. I hope to have conveyed something of the progress and promise of contemporary aphasiology, with its convergence of cognitive, computational and neuroimaging methods. A next frontier, I believe, is to better understand why the impact of lexical and phonological access deficits is sometimes greatly magnified when the task calls for production of multiword sequences [77,78]. New cognitive models of phonological and grammatical sequencing [79–81] may point the way forward.

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Endnotes

1 Task differences are also important to the primary systems account. Production/naming is considered the most vulnerable to semantic damage, because it rests solely on the mapping from semantics to phonology. By contrast, other tasks provide a second, non-semantic avenue for activating phonology (e.g., orthographies-to-phonology mapping in reading; acoustic-to-phonology mapping in repetition).

2 "Input processing was tested in the patients who contributed to these studies, and those who did not achieve high accuracy on phoneme discrimination and other auditory input tasks were treated separately."

3 "The data were collected for a published study [57] that mapped voxels that correlated with model parameters derived from patients’ repetition performance (i.e., p-weight, nl weight parameters). Here, in a re-analysis of the repetition data, we analysed the lesion correlates of the errors themselves."

References