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## Apraxia of speech and the study of speech production impairments: Can we avoid further confusion? Reply to Romani (2021)

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### Abstract

We agree with Cristina Romani (CR) about reducing confusion and agree that the issues raised in her commentary are central to the study of apraxia of speech (AOS). However, CR critiques our approach from the perspective of basic cognitive neuropsychology. This is confusing and misleading because, contrary to CR's claim, we did not attempt to inform models of typical speech production. Instead, we relied on such models to study the impairment in the clinical category of AOS (translational cognitive neuropsychology). Thus, the approach along with the underlying assumptions is different. This response aims to clarify these assumptions, broaden the discussion regarding the methodological approach, and address CR's concerns. We argue that our approach is well-suited to meet the goals of our recent studies and is commensurate with the current state of the science of AOS. Ultimately, a plurality of approaches is needed to understand a phenomenon as complex as AOS.

### Keywords

Apraxia of speech; clinical diagnosis; methodological approach; speech/language production

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We are pleased that our recent papers on apraxia of speech (AOS; Mailend et al., 2019; 2021) have inspired a conversation on methodological and theoretical issues in AOS and cognitive neuropsychology more generally. We welcome this opportunity to respond to the Action Editor's commentary (Romani, 2021; henceforth CR), who commended our experimental approach, but also critiqued our assumptions, participant selection criteria, and interpretation. While we agree with CR on many points, her criticism appears to stem largely

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from incorrect characterization of the purpose and claims of our studies. Thus, we first clarify our research goals and areas of agreement to sharpen the focus of discussion. We then structure our response along the pillars of our approach to situate our research among the available approaches (including those proposed by CR), address CR's concerns, and stimulate thinking about AOS and other neurogenic speech/language disorders.

## Goals and agreements

We whole-heartedly agree with CR's admonishment to reduce confusions. In this spirit, we first address an important confusion related to our goals: Contrary to CR's abstract, our goal was *not* "to specify models of speech production based on the performance of aphasic speakers" ("basic" cognitive neuropsychology). Rather, we aimed to understand speech<sup>1</sup> planning impairments in people with AOS ("translational" cognitive neuropsychology, as defined on *Cognitive Neuropsychology's* homepage). This distinction is important because it influences the research approach: The former employs an impairment as a tool to address questions and hypotheses about *unimpaired* function with the goal of building theories of cognitive processes (Caramazza, 1984; Coltheart, 2017); the latter employs the underlying theory and associated experimental paradigms as tools to study *impaired* function. Thus, although we framed our hypotheses about speech motor planning impairments (i.e., AOS) within a current, detailed, and well-supported model of speech production, this should not be confused with an effort to validate this model or adjudicate between different models. CR admits that no such claims appear in our paper, but charges us with overinterpretation nonetheless because a reader "may incorrectly interpret" or "wrongly infer" (p. 4) that we made such claims.

We agree that, despite the detail offered by some current speech production models, many aspects remain underspecified, limiting the precision of our hypotheses about speech disorders. Given that the goal of translational science is not to evaluate theoretical assumptions, it is constrained by the theory within which it operates. This makes it even more important to choose models that are sufficiently detailed and well-supported as an interpretive anchor point from which to understand data from impaired speakers.

We also agree that the notion of AOS as currently defined is unsatisfactory, and that understanding the underlying impairment(s) will require specific behavioural patterns that go beyond broad, clinically-defined labels such as AOS or aphasia. We are sympathetic to the likely existence of AOS subtypes. In fact, our agreement in these matters is what drives much of our research, as we often note explicitly (e.g., Maas et al., 2014, 2015; Maas & Mailend, 2012; Mailend et al., 2019, 2021; Mailend & Maas, 2013, 2021; Terband et al., 2020).

Further, the approach in our recent studies was selected to meet specific aims, but unquestionably, multiple methods are needed to understand a phenomenon as complex as

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<sup>1</sup>-CR introduces additional confusion when suggesting that our goal is to "specify the nature of the language processing impairment" (p. 1). We focus on *speech production, not language processing*. We do not view AOS as a type of aphasia, contrary to what CR occasionally implies (e.g., "... in participants with AoS, but not in control participants or in aphasics [sic] with other classifications" [p. 2]).

AOS (Maas & Mailend, 2012; Mailend & Maas, 2021). Thus, we agree on the potential value of a data-driven approach in which many measures are obtained from a large broad-spectrum sample (including multiple clinical diagnoses) to extract coherent patterns or possible subtypes (Mailend & Maas, 2021). However, because “pure” impairments are rare and processes at motor and phonological levels likely influence one another (Ziegler et al., 2012), clusters of features may be observed for many reasons. Such clusters may capture meaningful theoretical entities, as suggested by CR, or they may simply reflect interdependence between phonological and motor processes, or processes frequently co-damaged in stroke or neurodegeneration.

Large-scale, data-driven methods are not new in the field (e.g., Darley et al., 1969; Kertesz & Phipps, 1977), and more recent promising examples of large-scale predictive or exploratory studies in motor speech disorders exist (e.g., Ballard et al., 2016; Den Ouden et al., 2018; Haley et al., 2017; Laganaro et al., 2021; Ziegler et al., 2017).<sup>2</sup> For such clusters to be theoretically interpretable relative to underlying processes, however, the input measures for such analyses should be independently motivated processing measures.<sup>3</sup> We view our work in this context: To systematically examine methods and measures that, on independent grounds, capture the processes and mechanisms of speech production. This informs interpretation of any clusters that may emerge and facilitates a process-oriented approach to assessment, diagnosis, and treatment (Maas et al., 2015; Mailend et al., 2021; Terband et al., 2019, 2020). Ultimately, convergence from divergent sources will strengthen the basis of our understanding.

## Our approach: Pillars, merits, issues

Our goal is to understand the nature of speech production impairments, including speech motor planning impairments. Fundamentally, our approach is deductive: It uses a model to formulate *specific* and *competing* hypotheses about underlying impairments, which are then tested using experimental paradigms from the basic speech production literature. CR’s proposed approach is more inductive: It involves observing, categorizing, and interpreting speech sound errors in people with speech/language difficulties, and looking for patterns of co-occurring errors with possible theoretical coherence. Each approach rests on core assumptions or pillars. Next, we lay out the pillars that undergird our approach, and address CR’s main points (see Maas & Mailend, 2012; Mailend & Maas, 2021, for further discussion).

### Pillar 1: Theory

We start with a model of unimpaired speech production for theoretical continuity with the broader speech production literature. The choice of model is guided by several criteria: It must (a) capture the pertinent processing stages (here, speech motor planning), (b) be based on independent empirical support (i.e., not based on clinical data), and (c) be sufficiently detailed to allow the formulation of specific competing hypotheses.

<sup>2</sup>.Most of these studies relate findings to clinically defined groupings, likely out of recognition that such research must make contact with the clinical realm.

<sup>3</sup>.See Schwartz (1984) for similar arguments relative to aphasia.

We explicitly justified our choice of model, recognizing other models. We used the DIVA/GODIVA model<sup>4</sup> (Bohland et al., 2010; Guenther, 2016; Guenther et al., 2006), a well-supported detailed model of speech motor planning and control, because it meets these criteria.

Contrary to CR, we see no problem with relying on models “whose complexity and/or distinctive features go beyond what is required to explain the results” (p. 5). In a *translational* cognitive neuropsychology study, such additional details may be irrelevant but do not detract from the interpretation based on the pertinent elements of the model. In fact, we view such additional details as a strength rather than a weakness, because it means that novel predictions can be generated and tested. For instance, our account does not hinge on the neural regions associated with components of the DIVA/GODIVA model, but it makes predictions about lesion locations that can be tested. Similarly, the computational detail of DIVA/GODIVA enables computer simulations to compare with human data (see Terband et al., 2020, for an example).

## Pillar 2: Experimental paradigms and measures

We test our hypotheses with experimental paradigms and measures from the basic speech motor planning literature. This pillar ensures empirical continuity with the broader literature on speech motor planning, which generally does not rely on error analysis in typical speakers. Replication of expected patterns with unimpaired speakers serves as a check on our paradigm and provides an interpretive anchor point for the AOS data.

With “linguistic analyses of perceived errors” (CR, 2021, p. 3), such continuity is more tenuous: Because typical speakers rarely make errors, error *patterns* cannot usually be compared against typical speaker data. Error analysis is also problematic for other reasons (Maas & Mailend, 2012). Most germane here is the ambiguous relationship between error types and underlying processing stages: disruptions at the phonological level can cause “phonetic” errors, and disruptions at the speech motor planning level can create “phonological” errors (CR; Galluzzi et al., 2015). Simply calling some errors “phonetic” and others “phonological” does not make them evidence of phonetic or phonological problems (e.g., Kent & Rosenbek, 1983). This is not to suggest that we cannot learn from error analysis – without any doubt the field has learned a lot from it. Rather, like any approach, error analysis has its weaknesses, which our approach aims to avoid.

One difficulty with studying AOS is finding a task that taps processing at the level of speech motor planning. CR acknowledges that our findings are compatible with our interpretation of AOS as a speech motor buffer retrieval deficit. However, she claims that we overinterpreted our findings because we failed to consider alternative interpretations. Although we did discuss several alternative interpretations, CR considers the assumption that our tasks *primarily* tap speech motor planning processes unwarranted, and suggests that a phonological interpretation should be favoured over the speech motor planning account.

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<sup>4</sup>The model that motivated our study was *not* the DIVA/FLF model, as CR states. The FLF model (Van der Merwe, 2021) is an entirely different model. This failure to distinguish these models not only mischaracterizes our work, but ironically, creates unnecessary confusion of the sort CR argues against.

Both interpretations rest on assumptions (“auxiliary hypotheses”; Caramazza, 1986) and therefore may be wrong. However, our speech motor interpretation is supported by several sources of evidence. First, phonological overlap in non-initial segments (as in our studies) typically creates *facilitation* (Damian & Dumay, 2007; Schnur et al., 2006; Schriefers et al., 1990; Sevald & Dell, 1994; Wheeldon, 2003; Wilshire et al., 2016);<sup>5</sup> we observed *interference*. Second, phonological effects disappear in delayed production tasks, suggesting that phonological planning is completed by the go-signal and not reflected in reaction time (RT) (Damian & Dumay, 2009; Grainger et al., 2000; Laganaro & Alario, 2006; Mailend & Maas, 2013). Third, we observed a significantly greater switch cost only for the group with an independently established clinical diagnosis of speech motor planning impairment (AOS). Further, using the correlational approach suggested by CR, we observed no correlation between the interference effect and measures of phonological processing (Mailend et al., 2019).

In contrast, CR’s alternative interpretation requires all of the following – not widely held – assumptions: (a) The delayed RT interval includes phonological encoding, (b) predictable overlap in non-initial segments produces phonological interference, (c) our participants with AOS had phonological impairments that were more severe than those in the no-AOS group, and (d) this combination of assumptions is more plausible than our interpretation. In our view, the current weight of evidence, logic, and parsimony favours our interpretation. Nevertheless, we acknowledged the theoretical possibility that the observed effect arose at the level of phonological planning and refer readers to our papers for further discussion of this possibility.

CR also argues that in addition to RT, we should have measured the timing of the second word in the two-word sequence, in particular inter-word intervals. Unlike in Rogers and Storkel (1999), our task was specifically designed to measure RT rather than inter-word intervals because our hypotheses generated clearly distinct predictions for RT. The choice of second-word timing measure requires additional assumptions about the interaction between speech planning and ongoing articulation (e.g., inter-word interval assumes that all planning of Word 2 occurs after articulation of Word 1 is complete). Furthermore, by controlling our materials to have the same word in the first position across conditions, we avoided potential confounds associated with using different acoustic landmarks to obtain timing measures. Given the measurement confound and lack of distinct predictions, adding such analyses would change our specific hypothesis-driven study, with a well-justified *a priori* outcome measure and theoretically coherent pattern of findings, into a “fishing expedition” that would only add confusion.

In sum, although one can disagree with our assumptions (such is science), we argue that our interpretation of the task and findings is more plausible and parsimonious than the account favoured by CR.

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<sup>5</sup>In a few studies, interference effects were attributed to the phonological level (e.g., Breining et al., 2016; O’Seaghdha & Marin, 2000). O’Seaghdha and Marin did find fragile inhibition effects for rhyme-overlap pairs in RT but only for high-frequency words; they found facilitation for low-frequency words. Our words were generally in the lower frequency range. Breining et al. reported phonological interference when overlap is unpredictable with respect to word position. In our task, the overlap was predictable (always shared rhyme).

### Pillar 3: Clinical population

Determining clinical groups is a difficult task for any population that lacks an accurate and reliable diagnostic instrument for classifying group membership, such as people with AOS. We recruit individuals who have been assigned this label clinically based on a collection of speech features that are widely thought to reflect speech motor planning impairments (e.g., Duffy, 2019; McNeil et al., 2009). This classification is derived from perceptually determined features, including speech sound distortions, abnormal prosody, and slow speech with segmented syllables. We obtain independent judgments from at least two experts, and quantify severity based on the Apraxia of Speech Rating Scale (ASRS; Strand et al., 2014), which is becoming a *de facto* standard in the AOS literature, having been adopted across AOS research groups (e.g., Basilakos et al., 2015; Bislick, 2020; Den Ouden et al., 2018; Haley et al., 2019; Hybbinette et al., 2021; Utianski et al., 2018; Wambaugh et al., 2020).

Given the translational goals, clinical diagnosis has clear advantages over the alternative proposed by CR, which is to disregard clinical diagnosis altogether in favour of a novel grouping. In particular, clinical classification provides continuity with the broader AOS literature and links our findings to the clinical domain which motivates our work. Assuming such a link without establishing it creates confusion. For example, Galluzzi et al. (2015) defined people with AOS based solely on the rate of “phonetic” errors: those with 10% or more of such errors were stipulated to have AOS.<sup>6</sup> This novel basis for defining AOS led to inclusion of individuals who, by current widely-accepted criteria, do not have AOS (e.g., people with typical or fast speech rate<sup>7</sup>). We see no problem in defining a new population, but using an existing term to label this new population is confusing and misleading, and complicates synthesis of the literature. Terminological confusion has plagued the early AOS literature, when “Broca’s aphasia” was often used to refer to AOS (McNeil et al., 2000; Rosenbek, 2001). We should avoid repeating such mistakes, and clearly distinguish terms for different entities (e.g., AOS vs. “articulatory impairment”; Buchwald, 2017).

CR also critiques clinical classification on grounds of circularity. However, our approach avoided such circularity: Our clinical classification was not based on performance on our task or RT measures. There is however a clear problem of circularity in the approach proposed by CR, which abandons clinical diagnosis in favour of a behaviour that is ambiguous with respect to processing level (“phonetic” errors). CR suggested that acoustic or kinematic variables may help identify the level of impairment. However, we note that, with the exception of the studies by Buchwald and colleagues (Buchwald et al., 2017; Buchwald & Miozzo, 2011, 2012), in all referenced studies the phonetic or phonological origin of the examined variable was either based on association with clinically defined groups or simply assumed. That is, the indicator was taken to reflect problems at the motor level because it was prevalent in a group of speakers with AOS (or worse, nonfluent aphasia)

<sup>6</sup>Recent studies show that high rates of phonetic errors can also occur in people with aphasia without AOS (Bislick & Hula, 2019; Haley et al., 2017). Thus, it is unclear how to relate this novel classification to the clinical entity of AOS as currently defined in the literature (Buchwald, 2017).

<sup>7</sup>Of course, speech rate can be slowed for several reasons, included for reasons mentioned by CR. That is why clinical diagnosis of AOS requires a *constellation* of features (rather than a single feature), amongst which slowed speech rate is a necessary (though not sufficient) component.



as determined by clinical evaluation. In sum, CR's solution points back to the very problem it aimed to solve.

Further, in order to understand speech motor planning impairments, it is critical to examine hypotheses about such impairments in people who are likely to have them. Imperfect though it may be, we recognize the value of clinical expertise in identifying patterns of behaviour that may reflect speech motor planning impairments. Thus, contrary to Galluzzi et al. (2015), who "believe that not much should be taken from the clinical classification of the patients [*sic*]" (p. 69), we believe that people diagnosed clinically with AOS are more likely to exhibit speech motor planning impairments than those without this diagnosis, and thus represent a logical population and a reasonable place to start to find convergence with our – independently established – experimental paradigms and measures of speech motor planning.

#### **Pillar 4: Aphasia control group**

We examine patterns of performance in speakers with AOS as well as speakers with aphasia without AOS, to assess divergent validity of our experimental measure: People without AOS (but with language production impairment) are *not* expected to show the exaggerated switch cost because their speech motor planning is presumed intact. Thus, this pillar places constraints on interpretation. For example, we showed that our groups did not differ in verbal working memory or the number of sound errors on the experimental task, suggesting that verbal working memory or rate of "phonological" errors cannot explain our group differences.

#### **Pillar 5: Group and individual analysis**

We present group data *and* individual analyses, in recognition of the imperfect nature of clinical diagnosis and the possibility of AOS subtypes. This pillar provides transparency beyond group mean and enables inspection of possible AOS subtypes.

CR takes issue with our interpretation because not all individuals conform to their group pattern. She suggests that 3/7 of our AOS participants did not show reliable interference effects and that 4/10 participants without AOS did. However, CR uses different standards for each group: she requires interference in *all* conditions for speakers with AOS, but in only one condition for speakers without AOS. There is legitimate room for disagreement here about the strength of the empirical support, but it is confusing and misleading to apply different standards to the groups.

Of course, the clearest possible finding would have been if *all* of our participants with AOS and *none* of the participants without AOS showed the interference pattern. The fact that this is not what we observed could be due to (1) the accuracy of the diagnosis, (2) sensitivity of our task, and/or (3) the possibility that the clinical category of AOS encompasses different subtypes (only one of which was captured by our task). Available data do not allow us to distinguish between these possibilities, and further research will be needed. Nevertheless, the interference effect was clearly more robust in the AOS group than in the aphasia group when applying the same standard.

Finally, we note that, while some of this variation may be due to imperfect clinical diagnosis, as CR suggests, this is far from unique to our work. In fact, individual variation is quite evident even when one defines groups based on CR's proposed criterion of 10% or more phonetic errors (see Galluzzi et al., 2015, Tables C1-D1<sup>8</sup>). This underscores the need to present both group and individual analyses. The challenge is to determine whether these patterns reflect meaningful differences in underlying impairment profile.

### **Pillar 6: Convergence and parsimony**

We agree with CR about the importance of converging findings, in particular, convergence from divergent sources. This applies both to the support for a given hypothesis and to the broader methodology. Regarding the former, the Program Retrieval Deficit hypothesis found converging support, and *predicted* RT and error data in three different experimental paradigms (Mailend et al., 2019, 2021; Mailend & Maas, 2013), without requiring novel assumptions with a weak or ambiguous theoretical and empirical basis.

Regarding the latter, to the extent that two converging measures of a construct are established independently, the validity of both measures is strengthened ("convergent validity"). In our case, clinical AOS diagnosis and the experimental paradigms were both argued to reflect speech motor planning *on independent grounds*. The observed convergence between them therefore supports both our interpretation of the task and the notion that even imperfect clinical diagnosis of AOS does capture relevant aspects of speech motor planning impairment.

### **Conclusion**

We have explained the foundations of our approach and argued that our interpretation is more plausible and parsimonious than the alternative proposed by CR. Our interpretation rests on conventional assumptions that maintain continuity with theoretical, empirical, and clinical domains. It provides a parsimonious and unified account for converging findings from divergent sources that were predicted *a priori*. In contrast, CR's alternative offers only a post-hoc explanation that requires adoption of several not widely held assumptions with a weak or ambiguous theoretical and empirical basis, and does not relate to the clinical entity (and label) of AOS as widely understood.

Disagreements about methodology and preferred theories are inevitable in the competition of ideas that is science and will undoubtedly remain. Theories and models offer ways to conceptualize, formulate, and test new hypotheses about complex phenomena, including communication disorders such as AOS. Theories serve their purpose if they stimulate new thinking, drive methodological innovation, and generate scientific evidence, regardless of which one will turn out to be more right in the end. We are pleased that our work generated such a strong response and may lead to development of detailed alternative hypotheses whose predictions can be tested in future studies. This means that it has served one of its main purposes: To stimulate thought and generation of competing ideas. We look forward to

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<sup>8</sup>Galluzzi et al. (2015) do not statistically analyse whether individual participants reflect their group pattern, but descriptive data indicate that many do not.



future developments that advance our understanding of speech production impairments and produce rigorous, effective, and theoretically coherent diagnostic tools and treatments for the people who live with such impairments.

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