

**Psycholinguistic effects, types of impairments and processing levels in word production
Can we reduce confusions?**

A commentary to Mailend, Maas, Beeson, Story & Forster (2021)

Cristina Romani
Aston University

Address correspondence to:
Cristina Romani, Ph.D.
College of Health and Life Sciences
Aston University, Aston Triangle
Birmingham B4 7ET - England
e-mail C.Romani@Aston.ac.uk
Tel: 00 44 121 – 204 4081

Key words: Apraxia of Speech; methodological issues, diagnosis, phonological errors,
phonological interference

This commentary highlights common difficulties faced by the literature that aims to specify models of speech production based on the performance of aphasic speakers, taking as a springboard a recent study by Mailend et al. (2021). These include: 1) Difficulties with theoretical assumptions which link psycholinguistic effects unequivocally to one processing level; 2) Difficulties using clinical classifications to localise experimental effects. 3) Difficulties making theoretical inferences given the controversial nature of the representations that characterize different processing levels. We argue that these difficulties could be ameliorated by studies in which: 1) The level of psycholinguistic effects is demonstrated with converging analyses; 2) Clinical classification is not taken as a starting point, but, instead, associations between clusters of symptoms are analysed; 3) The nature of processing levels associated with deficits is made clear and results are not over-interpreted as supporting models whose characteristics go beyond an explanation of the results.

This commentary discusses some of the difficulties faced by the current literature on speech production and apraxia of speech (AoS), taking examples from recent studies by Mailend Maas, Beeson, Story & Forster (2019; 2021). These studies are good examples of these difficulties precisely because of their commendable attempts to apply experimental paradigms to the study of aphasia and AoS in order to specify the nature of the language processing impairment. The aim of this commentary, therefore, is not to discourage these attempts, far from it, but to highlight methodological difficulties that the research area as a whole faces (cognitive neuropsychology as well as aphasiology) so that more concerted attempts to overcome them can be made.

Mailend et al. (2019, 2021) carried out two studies assessing interference effects in aphasic speakers with a clinical diagnosis of AoS. Interference effects were established by measuring speech onset RTs while reading monosyllabic words. In Mailend et al. (2019), participants prepared to say a single word (e.g., bill), but sometimes were asked to switch to reading a different word which could be more or less similar to the target (similar: pill or dill vs dissimilar: fill; where b-d only differ by a single phonological feature, while b-f differ by more features). Interference effects were measured by switch costs when a different word had to be read. In Mailend et al. (2021), participants prepared to say two words that were either identical (e.g., bill-bill), highly similar (bill-pill/ bill-dill), or less similar (bill-fill) and they had to produce them after a go signal. Interference effects were measured as differences in producing identical vs similar words. Across studies, results showed stronger interference in speakers with AoS (longer RTs for switching or producing a similar pairs) compared to control speakers or aphasic speakers without AoS.

According to Mailend et al., these results demonstrate that apraxia of speech is “a problem retrieving speech motor programmes in the face of simultaneously activated programmes”. Their results are, indeed, compatible with this interpretation. Phonologically similar words may activate similar articulatory plans and this could increase selection difficulties in a system where articulatory planning is already strained. However, the results of Mailend et al. are also compatible with alternative interpretations which are important to consider so that evidence for different possible impairments in AOS can be better assessed in the future.

The studies by Mailend et al. exemplify three main types of difficulties: 1) Attributing psycholinguistic effects to one processing level when the evidence under consideration is compatible with alternative levels being affected; 2) Using clinical diagnosis to determine the level of impairment when a diagnosis encompasses problems at more than one level; 3) overinterpreting results and/or interpreting results using models in which the functions of different processing components are not sufficiently clear. I will discuss these difficulties in turn.

1. Issues with theoretical assumptions. Mailend and colleagues define their experimental pairs as ‘phonetically’ similar, although they are equally phonologically similar (and also orthographically similar, for that matter). They assume that, in their paradigms, preparation eliminates any phonological contribution to interference effects. Since the phonology of the word/words is prepared, any delay in initiating speech would be due to articulatory planning. This assumption is unwarranted. In both studies, participants produced words after having read and activated a phonologically similar word. In Mailend et al. (2019), the phonology of the prime word would interfere with *phonological* activation of the target when this is different from the word prepared. In Mailend et al. (2021), there is no switching at the go signal. Potentially an articulatory program of the whole sequence can be prepared in

advance¹, but it may not be possible to prepare articulation in the same way we can prepare phonology (e.g., see Roelof, 2002) and/or buffer limitation may prevent this in AoS (see next). Either way, the *phonology* of the first word would interfere with the *phonology* of the second word causing errors and delaying production. Phonological interference would also increase competition at the articulatory level, but the source of the effect would be phonological not articulatory.

Mailend et al. (2021), wanted to disprove the hypothesis that difficulties in AoS arise from limitations in the capacity of an output buffer as suggested by Rogers and Storkel (1999). Like Mailend et al., Roger & Storkel also showed a detrimental influence of phonological similarity, when producing pairs of monosyllabic words, in participants with AoS, but not in control participants or in aphasics with other classifications. However, they measured the time lags between production of the two words/syllables, while Mailend et al. only measured onset RTs for the first word. Mailend et al. assumed that interference effects in onset RTS cannot reflect buffer capacity limitations. If participants with AoS have a buffer limited to one syllable, they claim, no interference effect should be seen because the production of the first word would be prepared and unaffected by the second word. This assumption, however, is also unwarranted. No effect would be seen in control speakers because a large buffer capacity allows retaining more distinct phonological representations and/or motor programmes. Instead, interference effects could be seen in participants with AoS because they cannot prepare the whole sequence and preparation/production of the first word is delayed by interference from the second work. To pinpoint the nature of the impairment and rule out a buffer impairment, Mailend et al. (2021) should have measured timing of the second word. If their participants with AoS had a normal capacity to keep articulatory plans activated in parallel (the role they envisioned for the output buffer), they should have been able to produce the second word of the pair with no delay because an integrated programme corresponding to the two target syllables would have been ready after initiation. Instead, longer offset-to-onset delays, especially for similar pairs, would point to capacity limitations as argued by Rogers and Storkel. This relates to our third point of ambiguity in theoretical frameworks and relationship to tasks.

2. Issues with clinical classification of aphasic participants. If one agrees that the similarity effects reported by Mailend et al. could be phonological, articulatory or both, the claim that they occur at the articulatory level comes to rest exclusively on the demonstration that they occur *selectively* in speakers with AoS who suffer from an articulatory impairment. This claim, however, is weakened by the fact that the identification of the processing component which is impaired is based on a clinical diagnosis rather than on behaviour, with clinical diagnosis being problematic for several reasons. There is a recognized lack of consensus in the criteria used to establish AoS, limited use of quantitative data, and inconsistency in attribution (see Haley et al., 2012; Maas et al., 2014). More importantly, there is a circularity of argument when participants are defined as having AoS based on pre-established criteria and then assessed to see how they perform on a new test with the aim to specify impairment. Performance on the new task will depend on the original categorization. If this does not select individual with pure impairments, any new characteristic will not be distinctive of AoS, but instead reflect average performance in an aphasic group with multiple impairments. The studies by Mailend et al. are an example of this methodological issue.

¹ Mailend et al. (2021) call switching both the condition where the participant prepare two non-identical words the condition where the participant has to change what was prepared at a go signal, but these two conditions are rather different in terms of cognitive functions/demands.

Diagnosis of AoS is particularly difficult because phonological and articulatory impairments are overlapping and difficult to distinguish (Haley et al., 2012; McNeil et al., 2016; Molloy & Jagoe, 2019) and pure impairments are rare. Mailend et al. classified aphasic speakers as having or not having AoS based on presence/absence of a slow speech rate, sound distortions, and impaired prosody. These are common criteria used to identify articulatory difficulties. However, slow speech is not a distinctive symptom; production can be slowed by difficulties at lexical, phonological or articulatory levels. Moreover, speakers with AoS commonly make high rates of phonological errors in speech production (as well as sound distortions/phonetic errors) and, without further analyses, it is difficult to establish whether these errors arise because of articulatory difficulties or because of independent phonological difficulties. In the studies by Mailend et al., additional analyses to establish the contribution of phonological impairment would have been crucial because their argument rests on excluding these contributions. Mailend et al. (2021) reported much higher rates of speech errors in participants classified as having AoS than in other aphasic participants as well as slower RTs at baseline (producing two identical words). This is consistent with, more severe phonological impairments in the AoS group. Since differences between conditions are generally exaggerated when speakers are slower/less able, a more severe phonological impairment could well explain stronger interference effects (more difference in producing two similar words) in this group.

Finally, Table 4 of Mailend et al. (2021) reports individual results. In the group *with* AoS, one participant demonstrated no interference at all across conditions and two more participants demonstrated an effect in only one condition (similarity in voicing/manner). Therefore, in 3/7=43% of the sample, there were either no or inconsistent effects. In the group *without* AoS, four participants (40% of sample) showed evidence of interference. Clearly, there was variability, but, more importantly, interference effects occurred even in speakers without articulatory difficulties reinforcing the point above that interference effects can result from phonological difficulties which could be more severe in the AoS group.

These considerations highlight the importance of establishing the nature of the impairment in a clinical population through experimental investigation rather than through an association with a clinical category. In the case of Mailend et al studies, it would have been important to quantify errors associated with phonological difficulties. The authors only state that errors involving single phonemes do not differ statistically in participants with and without AoS. Additionally, however, information about the properties of the errors would have been important. Spectrographic analyses and electro-magnetic tracking can help to identify errors that deviate from typical phonemic parameters (see Bartle-Meyer, Goozee, Murdock, 2009; Blumstein, et al., 1980; Bose & van Lieshout, 2012; den Ouden et al., 2018). Importantly several studies have demonstrated that error characteristics can be an important means to identify level of impairment. These studies have been based on acoustic/articulatory instrumental analyses (see Buchwald & Miozzo, 2011, 2012; Buchwald et al., 2017), but also on linguistic analyses of perceived errors that establish the proportion of errors that systematically simplify syllables and phonemes rather than semi-randomly pick between similar alternatives. Simplifications have been demonstrated to be a good indication of articulatory difficulties, co-occurring with phonetic errors (see den Ouden, 2002; den Ouden & Bastiaanse, 2003; Galluzzi et al., 2015; Romani & Galluzzi, 2005; Romani, et al 2002; 2011; 2017; see also see also for earlier demonstrations Nespoulous et al., 1984;1987). The point is that the nature and severity of contributing phonological impairments can and should be evaluated when the aim is to establish the cause of articulatory difficulties.

The circularity of testing groups of participants selected through pre-established criteria when the goal is to assess the nature of the impairment was well expressed in the 80s by Caramazza and colleagues (Caramazza, 1984; Caramazza & Badecker, 1989; McCloskey & Caramazza, 1988). While group studies of neuropsychological impairments may not always be problematic (see Shallice, 2015), having selection criteria which do not prejudice the outcome of the study is as important as ever. Clinical diagnosis is important in some contexts, but not when the purpose is to reach a better understanding of impairments and how they produce clusters of behavioural symptoms. If we want to improve our understanding, we should not assume that characteristics commonly associated with AoS (phonetic errors, phonological errors, longer word durations, dysprosodies, phonological simplifications, difficulties in initiating speech, groping, etc.) are a coherent set associated with a single source. Instead, we should empirically assess how strongly these characteristics co-occur or dissociate in different speakers and their relationship with results from new experimental tasks. This will allow us to establish whether AoS describes a single impairment or whether, instead, characteristics cluster in meaningful ways which identify different subtypes (e.g., reduction of resources, difficulties of selection, impaired knowledge of articulatory parameters, etc). In turn, because different clusters of symptoms may be associated with different neuro-anatomical sites, an understanding of subtypes will help in mapping functional components with brain-sites. Doing otherwise risks perpetuating confusion. The results of any given experimental study will depend on the composition of the clinical sample and the relative weight of different types of problems. This would mean, not only that those results cannot be replicated, but also that they will only be as meaningful and as coherent as the original classification, which can vary according to clinical practice.

3. Issues with theoretical inferences. It is important to stress that although we have focused on the nature of impairment in AoS, the issues we have raised relate more broadly to studies carried in cognitive neuropsychology. This is also true for the following observations related to theoretical models. Most of the points made here are not specific to the Mailend et al. study.

A first problem with theoretical interpretations of results is over-interpretation. For example, Mailend et al. do not directly make the claim that their results support the DIVA/FLF model (Guenter, 2016; Miller & Guenter, 2020 and Var der Merwe, 2020) *over* alternative models. However, given that alternative models are not cited, and that the authors say that the DIVA/FLF model is the motivation behind their studies, a reader may incorrectly interpret their results as support for this model. More generally, models are characterized by differences in content which, although are not addressed by empirical results, may be inferred to be supported when a study takes a particular model as the reference frame. For example, suprasegmental features are integrated with the phonological sequence at the level of phonological encoding in the model by Levelt et al. (1999). In contrast, they are specified at the later motor programming level in the FLF model and at the level of the ‘sequential structure buffer’ and ‘initiation map’ in the DIVA model and linked to the supplementary motor area. However, these differences lack empirical support and, in the case of Mailend et al. studies, are irrelevant to their interpretation of results. Moreover, differently from other models, The DIVA/FLF model has the ambition of linking processing stages to neuroanatomical sites and it specifies this mapping in great detail. However, again, these associations both lack empirical support, and are irrelevant for Mailend et al.’s interpretation of results. One should be careful in distinguishing the features of a model which are assumed in an investigation, from those which are important in interpreting results, and from those which are irrelevant.

A second issue concerns the clarity with which a model specifies what is represented at different processing stages. There is relatively broad agreement on the different stages required for speech production (semantics, phonological encoding, phonological output buffer, articulatory planning), but what is represented at the different levels is still not completely clear. The issue of the relation between phonological and articulatory representations is a particularly thorny one and a good example of more general difficulties. This is the backdrop that allows alternative interpretation of results, as we have seen in the case of the Mailend et al studies. For example, in the case of phonological encoding, it is not clear whether what is represented are sequences of phonemes, bundles of phonological features, or articulatory features and how these representations relate to a following level of articulatory planning. One possible set of distinctions might assume that, in the lexicon, words are represented in a concise form as sequences of unitary phonemes. At the following level, after words have been selected for production, phonemes are unpacked in terms of their features—which represent articulatory targets. This is the level often called phonological encoding.² After that, articulatory planning would specify how targets are realized as integrated action synergies in the context of previous and following targets. Articulatory plans could be either computed or accessed as pre-compiled routines as assumed by Mailend et al. (2021). However, this interpretation is not transparently shared, as highlighted by the variability in terms and levels described below.

To illustrate differences in terminology, the following representations have all been located at the level of motor planning in different models:

- Articulatory syllables (Levelt et al., 1999)
- Gestural scores (Tilsen, 2013) – as actions linked in synergy
- Core motor programs (FLF: Var der Merwe, 2020); ‘CMP contain ‘*spatial (place and manner of articulation) and temporal (relating to inter-articulatory synchronization) specification*’; ‘*sequential organization of movement*’ - Localized to ‘*prefrontal cortex, Area 6, the supplementary motor area (SMA), areas 5 and 7 (posterior parietal areas) and also Broca’s and Wernicke’s areas.*
- Generalized motor programs (Mass et al, 2008) – invariant aspects of movement patterns
- Speech sound maps (DIVA: Guenter, 2015; Miller & Guenter, 2020); ‘*well learned, highly coordinated spatio-temporal motor commands* – ‘*localized to the left ventral premotor cortex (vPMC) in the ventral precentral gyrus and surrounding portions of inferior frontal gyrus and anterior insula*’

In addition, even within one level in a single model, terms can conflict. The DIVA model uses the term ‘speech *sound maps*’ to refer to the *motor* planning level, which suggests representations in terms of sounds rather than gestures. The FLF of Van Der Merwe (2020) contrasts a level of motor *planning* with a level of motor *programming*; but the motor *planning* computes motor *programs*. Der Merwe (2020) also states that at the level of motor planning ‘*phonemes are changed into sounds which have a discrete place and manner of*

² This stage is motivated by the existence of aphasic speakers who display phonological difficulties across all spoken output tasks, even in tasks of repetition where a lexical representation is presented as a model and where, therefore, any problem with lexical access should be minimized (e.g., Joannette et al., 1980; Khon, 1984; Tippett et al., 2016).

articulation'. This, again, suggests a level corresponding more to phonological encoding than to motor planning.

The general point is that, given current theoretical uncertainties, we should carefully describe the processing stages and assumptions we use in our interpretations, but also make clear whether results are compatible with a range of models or, alternatively, exclude some possibilities. It is important not to give the false impression that results provide support for models whose complexity and/or distinctive features go beyond what is required to explain the results.

Conclusions. The general point of this commentary is not to dispute the possibility that some speakers with AoS may have difficulties selecting between competing articulatory programmes as argued by Mailend et al. This is plausible and Mailend et al., make an important contribution by suggesting it. Moreover, I applaud using psycholinguistic tasks and RT measures to address specific questions with patients, as they have done. However, at the moment, their conclusions do not rest on solid empirical grounds. The literature on AoS should strive to clarify assumptions and use detailed empirical data to characterise patients. This may require (counter the general trend) more extensive experimental investigations with better processing measures (e.g., articulatory difficulties might be better indexed by word durations and inter-word delays); better information on the impairments experienced by participants (e.g., quantifying properties of phonological and phonetic errors); and analyses that consider correlations among characteristics (e.g., phonetic errors may correlate with interference effects based on onset RTs, but they could also correlate with word durations). Moreover, research should build up from detailed analyses of the speech characteristics of the participants instead of working down from a priori syndrome classification criteria. This means relying more on correlation analyses and series of case studies than on analyses of average differences between clinically defined groups. This research approach will establish, in an evidence-based fashion, how different speech characteristics cluster together in different speakers and determine whether these clusters respect meaningful theoretical boundaries. Finally, the literature will benefit from all of us trying to be specific when referring to stages proposed by different models to explain impairments since the terminology is varied and possibly confusing. Equally, we should be conservative when invoking complex models to explain a set of results that could be compatible with several different models and where the complexity of the model is not directly relevant to the explanation.

In the interest of full disclosure, I note that I served as the action editor of Mailend et al. (2021). The main points raised in this commentary were brought to the attention of to the authors. They responded to the issues I raised in my review by highlighting the importance of sparking a debate in light of different methodological positions. This commentary was written in that spirit of constructive methodological debate. Interesting ideas and studies should be circulated recognizing that no study meets ideal standards.

REFERENCES

- Bartle-Meyer, C.J., Goozee, J.V., & Murdock, B.E., (2009). Kinematic analysis of consonant production in acquired apraxia of speech. *Journal of Medical Speech and Language Pathology*, 17 (2), 63-81.
- Blumstein, S.E., Cooper, W.E., Goodglass, H., Statlender, S., Gottlieb, J. (1980). Production deficits in aphasia: A voice-onset time analysis. *Brain and Language*, 9, 153-170.
- Bose, A., & van Lieshout, P. (2012). Speech-like and non-speech lip kinematics and coordination in aphasia. *International Journal of Language and Communication Disorders*, 47 (6), 654-672.
- Buchwald, A., & Miozzo, M. (2011). Finding levels of abstraction in speech production: evidence from sound-production impairment. *Psychological Science*, 22(9), 1113–1119. <https://doi.org/10.1177/095679761141772>
- Buchwald, A., & Miozzo, M. (2012). Phonological and motor errors in individuals with acquired sound production impairment. *Journal of Speech, Language, and Hearing Research*, 55, 1573-1586.
- Buchwald, A. Gagnon, B., & Miozzo, M. (2017). Identification and remediation of phonological and motor errors in acquired sound production impairment. *Journal of Speech, Language, and Hearing Research*, 60, 1726–1738.
- Caramazza, A. (1984). The logic of neuropsychological research and the problem of patient classification in aphasia. *Brain and Language*, 21, 9-20.
- Caramazza, A. & Badecker, W. (1989). Patient classification in neuropsychological research. *Brain and Cognition*, 10, 256-295.
- den Ouden, D.-B. (2002). Segmental vs syllable markedness: Deletion errors in the paraphasias of fluent and non-fluent aphasics. In E. Fava (Ed.), *Current Issues in Linguistic Theory* (Vol. 227, pp. 23–45). John Benjamins Publishing Company. <https://doi.org/10.1075/cilt.227.05oud>
- den Ouden, D.B. & Bastiaanse, R. (2003) Syllable structure at different levels in the speech production process: Evidence from Aphasia. In: J. van de Weijer, V. J. van Heuven & H. van der Hulst (Eds.) *The Phonological Spectrum. Vol. II: Suprasegmental Structure*. Current Issues in Linguistic Theory 234 (John Benjamins), pp. 81-107.
- den Ouden, D.B., Galkina, E., Basilakos, A., & Fridriksson, J. (2018). Vowel formant dispersion reflects severity of apraxia of speech. *Aphasiology*, 32(8), 902–921. <https://doi.org/10.1080/02687038.2017.1385050>
- Galluzzi, C., Bureca, I., Guariglia, C., & Romani, C. (2015). Phonological simplifications, apraxia of speech and the interaction between phonological and phonetic processing. *Neuropsychologia*, 71, 64–83. <https://doi.org/10.1016/j.neuropsychologia.2015.03.007>
- Guenther, F. H. (2015). *Neural control of speech*. The MIT Press.
- Joanette, Y., Keller, E., & Lecours, A. R. (1980). Sequences of phonemic approximations in aphasia. *Brain and Language*, 11(1), 30–44. [https://doi.org/10.1016/0093-934x\(80\)90107-8](https://doi.org/10.1016/0093-934x(80)90107-8)
- Kohn, SE. (1984). The nature of the phonological disorder in conduction aphasia. *Brain and Language*, 23(1), 97–115. [https://doi.org/10.1016/0093-934X\(84\)90009-9](https://doi.org/10.1016/0093-934X(84)90009-9)

- Haley, K. L., Jacks, A., de Riesthal, M., Abou-Khalil, R., & Roth, H. L. (2012). Toward a Quantitative Basis for Assessment and Diagnosis of Apraxia of Speech. *Journal of Speech Language and Hearing Research*, *55*(5), S1502–S1517. [https://doi.org/10.1044/1092-4388\(2012/11-0318\)](https://doi.org/10.1044/1092-4388(2012/11-0318))
- Levelt, W. J., Roelofs, A., & Meyer, A. S. (1999). A theory of lexical access in speech production. *The Behavioral and Brain Sciences*, *22*(1), 1–38; discussion 38-75. <https://doi.org/10.1017/s0140525x99001776>
- Maas, E., Gutiérrez, K., & Ballard, K. J. (2014). Phonological encoding in apraxia of speech and aphasia. *Aphasiology*, *28*(1), 25–48. <https://doi.org/10.1080/02687038.2013.850651>
- Mailend, M.-L., Maas, E., Beeson, P. M., Story, B. H., & Forster, K. I. (2019). Speech motor planning in the context of phonetically similar words: Evidence from apraxia of speech and aphasia. *Neuropsychologia*, *127*, 171–184. <https://doi.org/10.1016/j.neuropsychologia.2019.02.018>
- Mailend, M.-L., Maas, E., Beeson, P. M., Story, B. H., & Forster, K. I. (2021). Examining speech motor planning difficulties in apraxia of speech and aphasia via the sequential production of phonetically similar words. *Cognitive Neuropsychology*, *38*(1), 72-87. <https://doi.org/10.1080/02643294.2020.1847059>
- McCloskey, M. & Caramazza, A. (1988) Theory and methodology in cognitive neuropsychology: A response to our critics, *Cognitive Neuropsychology*, *5*(5), 583-623, DOI: [10.1080/02643298808253276](https://doi.org/10.1080/02643298808253276)
- McNeil, M.R., Ballard, K.J., Duffy, J.R., & Wambaugh, J. (2016). Apraxia of speech theory, assessment, differential diagnosis, and treatment: Past, present, and future. In P. van Liehout, B. Maassen & H. Terband (Eds), *Speech Motor Control in Normal and Disordered Speech: Future Developments in Theory and Methodology* (pp.195–221). ASHA Press.
- Miller, H. E., & Guenther, F. H. (2020). Modelling speech motor programming and apraxia of speech in the DIVA/GODIVA neurocomputational framework. *Aphasiology*, *35* (4), 424-441 <https://doi.org/10.1080/02687038.2020.1765307>
- Molloy, J., & Jagoe, C. (2019). Use of diverse diagnostic criteria for acquired apraxia of speech: A scoping review. *International Journal of Language & Communication Disorders*, *54*(6), 875–893. <https://doi.org/10.1111/1460-6984.12494>
- Nespoulous, J.-L., Joanette, Y., Béland, R., Caplan, D., & Lecours, A. R. (1984). Phonologic disturbances in aphasia: Is there a ‘markedness effect’ in aphasic phonetic errors? *Advances in Neurology*, *42*, 203-214.
- Nespoulous, J.-L., Joanette, Y., Ska, B., Caplan, D., & Lecours, A. (1987). Production deficits in Broca’s and conduction aphasia: Repetition vs. reading. In E. Keller & M. Gopnik (Eds), *Motor and Sensory Processes in Language* (pp. 53–79). New York: Psychology Press.
- Roelofs, A. (2002). Spoken language planning and the initiation of articulation. *Quarterly Journal of Experimental Psychology - Human Experimental Psychology*, *55* (2), 465-483.
- Rogers, M. A., & Storkel, H. L. (1999). Planning speech one syllable at a time: the reducedbuffer capacity hypothesis in apraxia of speech. *Aphasiology*, *13*(9–11), 793–805. <https://doi.org/10.1080/026870399401885>

- Romani, C., & Galluzzi, C. (2005). Effects of syllabic complexity in predicting accuracy of repetition and direction of errors in patients with articulatory and phonological difficulties. *Cognitive Neuropsychology*, 22 (7), 817–850. <https://doi.org/10.1080/02643290442000365>
- Romani, C., Olson, A., Semenza, C., & Granà, A. (2002). Patterns of phonological errors as a function of a phonological versus an articulatory locus of impairment. *Cortex*, 38(4), 541–567. [https://doi.org/10.1016/S0010-9452\(08\)70022-4](https://doi.org/10.1016/S0010-9452(08)70022-4)
- Romani, C., Galluzzi, C., Bureca, I., & Olson, A. (2011). Effects of syllable structure in aphasic errors: Implications for a new model of speech production. *Cognitive Psychology*, 62(2), 151–192. <https://doi.org/10.1016/j.cogpsych.2010.08.001>
- Romani, C., Galuzzi, C., Guariglia, C., & Goslin, J. (2017). Comparing phoneme frequency, age of acquisition, and loss in aphasia: Implications for phonological universals. *Cognitive Neuropsychology*, 34(7–8), 449–471. <https://doi.org/10.1080/02643294.2017.1369942>
- Shallice, T. (2015). Cognitive neuropsychology and its vicissitudes: The fate of Caramazza's axioms. *Cognitive Neuropsychology*, 32, 7-8, 385-411.
- Tippett, D.C., & Hillis, A. E. (2016). Vascular Aphasia Syndromes". In G. Hickok & S.L., Small (eds.). *Neurobiology of Language* (pp. 913–922). Boston: Academic Press.
- Tilsen, S. (2013). A Dynamical Model of Hierarchical Selection and Coordination in Speech Planning. *Plos One*, 8(4), e62800. <https://doi.org/10.1371/journal.pone.0062800>
- Van Der Merwe, A. (2020). New perspectives on speech motor planning and programming in the context of the four- level model and its implications for understanding the pathophysiology underlying apraxia of speech and other motor speech disorders. *Aphasiology*, 35 (4), 397-423. <https://doi.org/10.1080/02687038.2020.1765306>