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To Lump or to Split? Possible Subtypes of Apraxia of Speech

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Abstract

Background: The speculation that apraxia of speech (AOS) is not a unitary diagnosis, but consists of different subtypes instead, has been around for decades. However, attempts to empirically substantiate such a notion remain few and far between.

Aims: The primary objective of this article is to consider the different bases for identifying subtypes of AOS, review existing evidence regarding subtypes under each classification basis, and provide discussion and implications for future research.

Main Contribution: AOS subtypes have been proposed on the basis of clinical symptomatology, theoretical constructs, and an analogy to limb apraxia. Different possible subtypes of AOS are reviewed, along with their empirical support and limitations. Empirical evidence, particularly in the context of a progressive disease, supports the idea that AOS diagnosis may capture different underlying impairments of speech motor planning. Future research to advance our understanding of AOS should carefully consider the basis for subtype classification, and include large sample sizes to differentiate individual variability from possible subtypes.

Conclusions: Several proposed AOS subtypes have found some support in the literature. Further research is needed to determine the validity, coherence and utility of possible AOS subtypes for theoretical and clinical purposes.

Keywords

Apraxia of speech; subtype; classification; motor planning

Introduction

Since the early days of apraxia of speech (AOS), researchers have occasionally raised the possibility that the broad category of AOS could be “split” into subcategories (e.g., Buckingham, 1979; Collins et al., 1980; Croot, 2002; Duffy & Josephs, 2012; Kent, 1991; Rosenbek, 1991; Square et al., 1982). However, most of the AOS literature has proceeded from the assumption, explicit or implicit, that AOS represents a unitary disorder in the sense that there are no coherent or systematic subgroupings within this overall category, and thus

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people with AOS can be "lumped" together into a single category. For example, Darley et al. (1975: 262) noted that although people with AOS varied from one another in various ways, "… patients who present the type of articulatory disorder here described are more homogeneous than heterogeneous." An early focus on similarities rather than on differences among people with AOS is understandable, given that the notion of AOS as a separate clinical entity from aphasia and dysarthria was controversial and had to be established over many years (see Rosenbek, 2001, for an excellent historical overview). However, differences between people who receive the same diagnostic label of AOS exist – in clinical presentation, prognosis, response to treatment, etc. – and deserve explanation as well.

Inter-individual variability is generally acknowledged, but may not necessarily reflect distinct subtypes of AOS. Such individual differences between individuals with AOS may instead reflect differences in severity, in co-morbid impairments, in compensatory strategies, in pre-morbid speaking styles or language/dialect backgrounds, etc. Although these factors are important and interesting to consider, for the purpose of the present paper, we focus on the possibility that there may be different types of AOS (each of which may interact with these other sources of inter-individual variability). First, we discuss some reasons for and against "lumping" and "splitting." Next, we review a number of proposals that have been made regarding possible subtypes, organized by their general basis for such subtype classifications: analogy to limb apraxia, clinical symptomatology, and theory. The paper concludes with a summary and some suggestions for promising future directions to explore subtypes of AOS.

To Lump or to Split?

Considering the many complicating factors for identifying subtypes of AOS—the variability in symptoms, lesion size and location, and comorbid diagnosis—it is appropriate to first consider, what can be gained from pursuing the possibility of AOS subtypes. Because a debate about subtypes has played out in the aphasia literature (and is still playing in some circles; e.g., see Ardila, 2010 and commentaries; Brownsett et al., 2019; Caplan, 1993; Howard et al., 2010; Schwartz, 1984), it serves as a starting point for identifying reasons to consider. Reviewing Frederic Darley's contribution to the idea of subtypes in aphasia (e.g., Darley, 1982, 1983), McNeil and Kimelman (2001) offer four reasons for the attempt to classify this syndrome. These included (1) identifying distinct clinical entities that lead to accurate selection of a therapeutic approach, (2) predicting lesion location, (3) uncovering the underlying impairment, and (4) facilitating communication between professionals[1]. These considerations appear relevant also in the case of AOS (although predicting lesion location is of lesser concern nowadays, given the advent of sophisticated neuroimaging techniques; McNeil & Kimelman, 2001). Our interest in subtypes of AOS here stems

[1]Although Darley (1982, 1983) argued against the notion of aphasia subtypes (and AOS), emphasizing similarities among people with aphasia and explaining differences primarily as the result of concomitant impairments (e.g., apraxia of speech) (e.g., Darley, 1982: 48), this perspective was largely in reference to broad aphasia syndromes based on modalities (input, output) and tasks (naming, repetition, etc.). Certainly, such broad aphasia categories (Broca's aphasia, conduction aphasia, etc.) appear unlikely to capture any potentially different profiles in underlying processing operations (see also Schwartz, 1984). To determine whether subtypes exist with respect to differentially impaired processes, a different set of constructs and methods may be needed, rather than a classification according to syndromes.

primarily from the first one (treatment selection and candidacy) and the third one (underlying impairment).

On the theoretical side, current models of speech motor planning[2] are specific enough to provide the basis for detailed hypotheses about different impairments to the components and processes of the speech motor planning mechanism (e.g., Bohland et al., 2010; Guenther et al., 2006; Hickok, 2012; Hickok et al., 2011; Levelt et al., 1999; Parrell et al., 2019; Van der Merwe, 1997, 2020). A good reason to consider the possibility of subtypes within AOS, thus, is the possibility that they exist on theoretical grounds if our models of speech production accurately reflect the speech motor planning operations. Identifying theoretically predicted subtypes of AOS would, in turn, provide support to the existing theories and potentially point to more accurate clinical diagnosis and more targeted treatment approaches. A possibility remains, however, that such an attempt will not be fruitful even if specific impairments to the speech motor planning operations exist. For example, it is possible that interactivity between different stages of speech production and/or redundancy in the production system render the attempt to identify different subtypes practically impossible.

The other major reason to consider subtypes of AOS is a practical or clinical one. AOS subtypes become clinically relevant if they guide rehabilitation or prognosis for people who experience AOS. That is, subtypes have merit if identifying the specific impairment aids in choosing the most appropriate or efficient treatment approach (e.g., Collins et al., 1980) or predict likely outcomes for a given individual. This is particularly relevant with respect to impairment-oriented approaches to treatment. Of course, it is only fair to acknowledge that some treatments may be helpful to all people with AOS regardless of putative subtype and without understanding the specific mechanism of treatment, in particular if such treatments are not impairment-oriented but communication or participation-oriented. For example, interventions such as alternative and augmentative communication training (e.g., Lane & Samples, 1981; Lustig & Tompkins, 2002; Rogers, 2001) or script training (Youmans et al., 2011) may be applicable and effective regardless of AOS subtype, although even in some of these cases, the particular strategies that are most effective may depend on the underlying impairment. Alternatively, response to treatment may be predicted by other factors entirely, such as the severity of the impairment, nature of the underlying disease (acute or progressive), or, by way of adherence to the treatment protocol, the personality of the patient.

Despite some reasons for scepticism, entertaining the idea of subtypes in AOS has a clear potential for advancing the field both clinically and theoretically. The clinical entity of AOS currently includes cases of vastly different speech and lesion profiles and lacks a clear-cut boundary with its clinical neighbours, particularly aphasia with phonemic paraphasias and upper motor neuron dysarthria (e.g., Ballard et al., 2016; Cunningham et al., 2016; Den Ouden et al., 2018; Haley et al., 2012; Strand et al., 2014). Exploring the specific ways in which the operations of speech motor planning are impaired and how these deficits are

[2]Unless otherwise noted, we are using the term “speech motor planning” here as a general term for the set of processes and mechanisms that transform a linguistic representation into a code that guides or controls the movements of the physical speech apparatus.

manifested in a person's speech profile may help establish more meaningful boundaries within and around the syndrome. Whether this leads to clinical utility can only be determined by further study of the problem, but new knowledge about the architecture of speech production and its impairments will be gained regardless. Ultimately, what we can hope to achieve with a classification system of AOS is largely determined by the basis on which the classification is built and developed. To elucidate, we will next review existing attempts at identifying subtypes of AOS from the perspective of the classification basis.

Basis for splitting

Limb apraxia analogy

The term 'apraxia', signifying a problem in volitional control of skilled movement, is borrowed from the limb movement disorders literature, beginning with Hugo Liepmann's seminal work in the early 20th century (e.g., Liepmann, 1900). Possible similarities between apraxia and aphasia were noted even in the earliest discussions of apraxia. For example, the term 'apraxia', when first used by Steinthal in 1871 (cited by Pearce, 2009), was used by analogy to aphasia to distinguish it from paralysis (apraxia vs. paralysis; aphasia vs. anarthria). In addition, Liepmann (1911, as cited by Goldenberg, 2003: 513) suggested that there may be many different types of apraxia by noting that "there may be as many varieties of apraxia as aphasia". Given this history and the use of a shared term, it is natural to explore the potential similarities between different types of limb apraxia and AOS.

The most commonly acknowledged types of limb apraxia are ideational apraxia, ideomotor apraxia, and limb-kinetic apraxia (e.g., Buxbaum et al., 2008; De Renzi & Lucchelli, 1988; Ochipa & Gonzalez Rothi, 2000). Broadly speaking, ideational apraxia is viewed as resulting from a loss of the knowledge of plans for purposeful actions, for example how to use tools (e.g., Buxbaum, 2001; De Renzi & Lucchelli, 1988; Ochipa et al., 1992; Roy & Square, 1985). Errors often involve use of incorrect tools or inappropriate sequencing of otherwise well-executed movement components. Ideomotor apraxia is thought to reflect impairment of sensorimotor plans that encode spatial and temporal aspects of purposeful movement gestures, in particular transitive movements involving objects (e.g., Buxbaum, 2001; Ochipa & Gonzalez Rothi, 2000). Errors are often spatial in nature, for example involving inappropriate orientation or location of the hand for a given task. Finally, limb-kinetic apraxia reflects poor fine motor control, involving spatial and temporal distortions sometimes described as 'clumsiness' (e.g., Buxbaum, 2001; Ochipa & Gonzalez Rothi, 2000; Roy & Square, 1985).

Attempts have been made to apply this limb apraxia division to AOS (e.g., Feiken & Jonkers, 2012; Roy & Square, 1985; see McNeil et al., 2000, for discussion). For example, Feiken and Jonkers (2012) proposed ideational AOS as involving a problem in sequencing motor plans (resulting primarily in phoneme or syllable transposition errors), ideomotor AOS as involving a problem in accessing motor plans (resulting primarily in initiation difficulties), and kinetic AOS as involving damaged motor plans (resulting primarily in distortion and substitution errors). Feiken and Jonkers did not provide empirical support for their proposed subtype classification. However, as eloquently argued by McNeil et al. (2000), it is conceptually and empirically difficult or impossible to distinguish ideational and

ideomotor AOS from aphasic impairments at the lexical-semantic, syntactic, or phonological levels. For example, transposition errors are currently viewed as phonological rather than motoric in origin (e.g., McNeil et al., 2000; Wambaugh et al., 2006), and initiation difficulties and articulatory groping are no longer considered specific to AOS but also can be observed in conduction aphasia (e.g., McNeil et al., 2000; Wambaugh et al., 2006). Thus, even if such errors could also be explained as apraxic errors, the difficulty of ambiguous interpretation remains. Further, even in the limb domain, differentiating between ideational and ideomotor apraxia has been challenging and controversial, in part due to difficulties with inferring underlying cognitive impairments from error types (e.g., Buxbaum, 2001; De Renzi & Lucchelli, 1988). The prevailing consensus to date appears to be that AOS is most akin to a limb-kinetic form of apraxia (e.g., Code, 1998; McNeil et al., 2000). It is possible that a division analogous to the limb domain can be shown in the speech domain as well, although challenges remain with respect to specifying error types that can relatively unambiguously be attributed to different types of apraxia rather than aphasia.

Clinical symptomatology

Another dimension along which to divide AOS into different types is clinical symptomatology. One possible division relates to the clinical course and/or etiology. For example, AOS following a sudden injury such as a stroke may differ from AOS in progressive neurodegenerative disease (e.g., Duffy, 2006). Clearly, at the medical/neurological level of description, there are obvious and important differences between stroke-based AOS and progressive AOS, including differences in onset (sudden vs. gradual), course (recovery/chronic vs. deterioration), and lesion distribution. However, as AOS is a speech diagnosis rather than a medical diagnosis[3], the focus of the present discussion is on potential subtypes in terms of behavioural and cognitive levels of description. As argued by Duffy and Josephs (2012), it is reasonable to expect differences based on the aforementioned medical and neurological factors. Large-scale prospective comparative studies have not yet been conducted, but retrospective studies indicate that, by and large, the features of AOS are similar between stroke-based and neurodegenerative AOS, although one reported difference is a reduced number of syllables per breath group despite sufficient maximum phonation duration in progressive AOS, which has not been reported for stroke-based AOS (Duffy, 2006), although difficulties with respiratory control in stroke-based AOS have been reported (Keatley & Pike, 1976).

Patterns of error types is another example of clinical symptomatology that might distinguish types of AOS. The relatively recent development of a structured, specific, and quantified diagnostic rating scale for AOS has enabled such an inductive approach to discerning possible subtypes of AOS (Strand et al., 2014; Utianski et al., 2018). One specific example is the recent distinction between an ‘articulatory’ or phonetic subtype and a ‘prosodic’ subtype of AOS (Josephs et al., 2013; Takakura et al., 2019; Utianski et al., 2018). The phonetic subtype is characterized by a predominance (relatively greater prominence) of speech sound distortions and distorted substitutions and additions, whereas the prosodic subtype is

[3]Of course speech signs and accurate speech diagnosis are important for informing the medical diagnosis and management of AOS (e.g., Duffy, 2013).

characterized primarily by suprasegmental features (e.g., slow rate, segmentation, and/or abnormal prosody). Speech sound distortions and segmentation are among the main hallmark features of AOS, and this subdivision suggests that they may reflect different underlying impairments that could dissociate. We hasten to add that proponents of this division acknowledge that not all people with AOS fit clearly into one or the other subtype (referred to as AOS-NOS [Not Otherwise Specified]; Josephs et al., 2013; or Mixed AOS, Utianski et al., 2018) and instead exhibit features of both subtypes. For example, of the 37 participants with progressive variants of AOS reported by Josephs et al., 29 could be classified as either phonetic (n=11) or prosodic (n=18) with the remainder classified as AOS-NOS. Interrater agreement on classification into these subtypes is high in published studies to date (Josephs et al., 2013; Takakura et al., 2019; Utianski et al., 2018).

Although there are potentially many different error types that could be proposed to point to AOS subtypes using such an inductive classification approach, the proposed division into phonetic and prosodic subtypes receives some validation from their alignment with different clinical categories of progressive speech and language disorders (Josephs et al., 2013). For example, Josephs et al. reported that the prosodic subtype was more common than the phonetic subtype in primary progressive AOS and progressive agrammatic aphasia with AOS in which the AOS was more severe than the aphasia, whereas only the phonetic type (and AOS-NOS) were found in progressive agrammatic aphasia with AOS in which the aphasia was more severe. Importantly, overall AOS severity did not account for this pattern. Further support for this particular distinction between subtypes comes from neuroimaging data, which indicate that the prosodic subtype appears associated primarily with damage in superior premotor cortex (including supplemental motor area), which was not a common area of damage for the phonetic subtype (Josephs et al., 2013), although Utianski et al. (2018) did report this region to be involved in both subtypes. Utianski et al. also reported that the phonetic subtype (but not the prosodic subtype) revealed more widespread damage (including atrophy in the prefrontal cortex and cerebellum), although differences between subtypes were not significant when controlled for age. Again, overall AOS severity did not differ between subtypes, nor did language impairment, as the individuals in the Utianski et al. study did not have a clinical diagnosis of aphasia. Thus, there is some suggestion that these two subtypes may hold some predictive value with respect to clinical classification of progressive speech and language disorders (e.g., primary progressive AOS vs. progressive nonfluent aphasia) and neurological underpinnings, at least in a neurodegenerative context. Future studies that include postmortem pathology results may provide further evidence regarding the predictive value of these subtypes.

It should be noted that thus far, these two subtypes have been mostly proposed in the context of progressive variants of AOS, and Utianski et al. (2018) suggested that the phonetic subtype (and perhaps AOS-NOS) may be more common in stroke-based AOS. A recent study attempted to apply this classification to stroke-based AOS (Takakura et al., 2019). In this study, the eight participants with stroke-based AOS could be classified into phonetic (n=5) or prosodic (n=3) subtypes; the three participants with progressive AOS were all classified as AOS-NOS. Lesion patterns showed some differences between phonetic and prosodic subtypes in the stroke-based AOS group. Takakura et al. further noted that time since diagnosis may be associated with subtype, such that those individuals with longer

times since diagnosis were more likely to exhibit the prosodic subtype, suggesting that the prosodic variant may reflect compensatory strategies to some extent (Takakura et al., 2019; see also Darley et al., 1975; Kent & Rosenbek, 1982, for early suggestions that prosodic abnormalities may reflect compensatory strategies).

In sum, the proposal of phonetic and prosodic AOS subtypes is a promising example of classification based on patterns of clinical features. This particular example has gained some support in the context of progressive AOS, with high reliability of classification and emerging validation with respect to neural basis. It remains to be seen to what extent this classification predicts neuropathology, disease progression and prognosis, and treatment response – and to what extent this classification holds for stroke-based AOS. As noted by Duffy and Josephs (2012), the fact that the disease process in progressive AOS is not determined by vascular distribution means that a different pattern of damage to the speech motor planning network is likely. If different components of the complex neural circuitry for speech motor planning are damaged, then it stands to reason that the underlying impairments in cognitive terms will also be different (Duffy & Josephs, 2012; Josephs et al., 2013; see also Croot, 2002; Maas et al., 2015).

One potential challenge for this particular division lies in the implication for diagnosis of AOS overall: At present, diagnostic criteria for AOS include normal speech rate and normal prosody as exclusionary criteria (McNeil et al.1997; Wambaugh et al., 2006). This may mean that the phonetic subtype may be underdiagnosed if speech rate and prosody are relatively normal in this subtype. Although Utianski et al. (2018) clearly state that these subtypes are a matter of *relative* prominence, and report that most of their sample with the phonetic subtype of AOS do also exhibit reduced rate and/or abnormal prosody, two of their participants are in fact judged to have normal rate and prosody (similarly, one of the participants with the prosodic subtype is judged to have no phonetic errors). This suggests that the diagnostic criteria for AOS may require revision (if people with the phonetic subtype AOS and normal rate and prosody are to be retained in the category of AOS) or that the phonetic subtype may not be considered to be a form of AOS.

In terms of theory, this inductive approach to subtype classification based on predominance of error types does not clearly point to particular underlying speech motor planning deficits, because the observed error types could arise from disruptions at multiple different stages or mechanisms of the planning process. For example, distortions could reflect co-activation of multiple incompatible motor plans due to a selection problem at the phonological planning level (e.g., Goldrick & Blumstein, 2006), difficulties planning the transitions between articulatory configurations (a speech motor *planning* problem in the specific sense of the term as used in the four-level framework of Van der Merwe [1997, 2020]), or difficulties in temporal coordination of contracting multiple muscle groups (a speech motor *programming* problem in the specific terminology of the four-level framework of Van der Merwe [1997, 2020]). In this sense, the division into phonetic and prosodic subtypes is similar to the division of aphasia into the classic aphasia syndromes, which are inductively formed groups based on relatively broad observations of task performance (e.g., Kertesz & Phipps, 1977) rather than on patterns of specific underlying impairments (e.g., Caplan, 1993; Schwartz, 1984). There may be multiple different deficits that could result in a preponderance of

articulatory (or prosodic) errors, and a single underlying problem could possibly lead to both articulatory and prosodic errors. Some of the error patterns may reflect the primary deficit, whereas others may reflect compensatory strategies (e.g., Darley et al., 1975; Kent & Rosenbek, 1982; Takakura et al., 2019), and it is difficult to disentangle these possibilities. Ultimately, any division into subtypes ought to relate to a theoretical understanding of the underlying processes or mechanisms thought to be impaired, in order to advance our understanding of speech planning and its disorders and in order to optimize (impairment-oriented) treatment methods (see also Collins et al., 1980) and prognosis.

Theoretical conceptualization

A requirement for a theoretically conceptualized classification of AOS is a sufficiently nuanced theory of speech production and of speech motor planning operations in particular. Speech motor planning represents a translation from a linguistic code into a motor code (e.g., Van der Merwe, 2020). Unfortunately, the development of theories and models of language production (e.g., Dell, 1986; Fromkin, 1971; Garrett, 1975; Goldrick, 2006; Shattuck-Hufnagel, 1979) has historically proceeded relatively separately – in different disciplines and literatures, and often based on different methodologies – from the development of theories and models of speech motor control (e.g., Fairbanks, 1954; Guenther, 1994, 1995; 2016; Klapp, 2003; MacNeilage, 1970; Parrell et al., 2018; Perkell, 1969, 2012, 2013; Sternberg et al., 1978; Van der Merwe, 1997).[4] In part as a result of this relatively separate development of theories and models that focus on one or the other domain, the interface between these domains has been understudied and poorly understood (Hickok, 2012; Ziegler, 2002). Nevertheless, theory-driven subtypes of AOS have been proposed and within the last decades, sophisticated models of speech production have been developed which link the language and motor control domains and include more nuance for their interface at the level of speech motor planning.

One of the first attempts to derive subtypes of AOS from a theoretical account can be attributed to Alexander Luria—an influential neuropsychologist who, among several significant contributions, developed a comprehensive theory of human cognition in the middle of the last century (see Akhutina, 2016, for a recent overview of Luria's work in aphasia). Luria's theory of speech production (Luria, 1973, 1980) follows—much like currently prominent theories of speech motor control (Guenther, 2016; Hickok, 2012, Parrell et al., 2019; Van der Merwe, 1997, 2020)—the contemporary advancements of voluntary movement theories of his time (e.g., Bernstein 1947, cited by Luria, 1980). His theory of speech production, developed in a comprehensive framework of cognitive functions, views the planning and control of speech movements as a complex orchestration between several different functional units, which in turn, are subserved by specific networks of brain regions. Damage to the different networks is thought to impair the voluntary movements of speech but in qualitatively different ways.

[4]We are not suggesting that theories and models of language production always ignore speech motor control or vice versa, but rather that theories and models tend to focus primarily on one domain (either speech motor control or language), with relatively little attention to and detail about the other domain. For some exceptions and interesting discussion, we refer the interested reader to Folkins and Bleile (1990), Hickok (2012), and Levelt (1989).

Like current thinking (e.g., Guenther, 2016; Hickok, 2012), Luria's theory elaborates the complex nature of movement planning that precedes the final efferent impulses arising at the level of the primary motor cortex and stresses the importance of afferent information in planning and controlling speech movements. We will briefly review this theory next, admitting up front that the description to follow does not do justice to the sophisticated and nuanced nature of this theory. In broad strokes then, the act of speaking starts with an intent to speak. This intent specifies the end goal of the speaking act in linguistic terms. In service of realizing the goal, the theory divides speech motor planning into two broad functional systems of higher order speech motor control, each of which can be independently impaired and thus give rise to a specific type of impairment (i.e., subtypes of AOS). One of these units stresses the importance of somatosensory information in guiding speech movements. Knowledge of the current position in space (i.e., state estimation) is critical for selection of precise movement trajectories appropriate for achieving the intended goal. Luria termed this aspect of speech production the 'afferent' or 'kinesthetic' organization of speech movements, and localized it to the inferior part of the postcentral regions of the cortex. As predicted from its function, patients with a lesion to this brain region are expected to struggle primarily with the precision of articulation while other aspects of speech production, such as speech fluency, are presumed to be unimpaired if the brain areas responsible for representing the constellation of motor impulses and the smooth sequencing from one movement to another are intact. This syndrome is called afferent (or kinesthetic) motor aphasia[5] in Luria's writings.

The second important functional unit in this theory is the system for the temporal organization and sequencing of motor impulses. This unit is responsible for the timely selection and smooth transition between motor impulses in a stream of speech which typically comprises a rapid succession of simple motor acts that correspond to units of speech. Luria called this system the 'efferent' or 'kinetic' organization of speech and localized it to the inferior part of the premotor areas, including Broca's area. A lesion to these areas gives rise to a syndrome that Luria termed efferent (or kinetic) motor aphasia. People with efferent motor aphasia are typically able to produce isolated sounds with precision, but in combining syllables and words, the speech becomes halted and effortful with transition difficulties between units of speech. Perseverations are also common due to the inability to switch from one motor impulse to the next.

It is interesting to note the parallels between Luria's kinetic and kinesthetic subtypes of speech motor planning impairments and the two subtypes of primary progressive AOS (PPAOS) described by Duffy and colleagues (Josephs et al., 2013, Utianski et al., 2018). The primary symptom in afferent motor aphasia and the articulatory subtype of PPAOS is the

[5]The term *'motor aphasia'* may create confusion. Unlike contemporary theories, Luria's theory does not articulate a clear divide between processes that belong to the realm of language and those that belong to the realm of motor control, but rather views them as intricately and intimately linked. Nevertheless, it is clear from his descriptions and grounding in motor theory that *'motor aphasia'* refers to the impairment of the cognitive organization of speech movements, which are analogous to afferent and efferent apraxia when affecting the limb (Luria, 1980). Attempts have been made to draw parallels between Luria's aphasia classification and the Wernicke-Lichtheim classification commonly used in the Western world (e.g. Akhutina, 2016) but the parallels are imperfect and likely unjustified due to substantial differences in the theoretical basis and the purpose of the two classification systems. Luria's classification attempts to describe the different components that subserve the complex act of speaking and thus is not primarily designed to classify the complex syndrome that a given individual may present with.

lack of articulatory precision, whereas in efferent motor aphasia and the prosodic subtype of PPAOS, the primary impairment relates to the difficulty of smooth transitions from one chunk of movement to the next. In terms of implicated brain regions of damage, there is overlap and there are differences. Most notably, the involvement of the parietal cortex in afferent motor aphasia has not been implicated in the articulatory subtype of PPAOS (Utianski et al., 2018) unless aphasia is also present (Josephs et al., 2013). Both proposed subtype classifications are largely based on studying populations other than stroke—progressive disease in one case and traumatic brain injury and brain tumour in the other. The different constellation of symptoms compared to reports of stroke patients, where a clear division between prosodic and phonetic types has not emerged (but see Takakura et al., 2019, for a different perspective), highlights the problem of establishing subtypes based on symptoms that commonly co-occur in patients with a common underlying disease.

Modern attempts at explicitly deriving potential AOS subtypes from a specific theory or model are, to the best of our knowledge, rare. A recent exception is the proposal by Van der Merwe (2020, this issue) in the context of the Four-Level (FL) model. The FL model distinguishes between speech motor *planning* and speech motor *programming*, and AOS is viewed as a speech motor *planning* disorder, which could take several forms (possible subtypes) such as damage to the inverse internal model, damage to the forward internal model, or a damaged pathway for relying on an efference copy (see Figure 3[dummy] and related discussion in Van der Merwe, 2020, for details). However, formulating clear and empirically testable differential predictions associated with each potential impairment is difficult. For example, Van der Merwe notes that damaged inverse internal models and damaged forward internal models both predict the same observable speech features (e.g., distortions, abnormal prosody, slow speech, restarts). Moreover, many of these same speech features can also arise from damage at the speech motor *programming* level, although the effects of contextual factors such as utterance complexity may differentiate planning impairments from programming impairments (Van der Merwe, 2020).

Despite the relatively few explicit attempts at deriving AOS subtypes from specific theories or models, there is a body of literature that is focused on elucidating the nature of the speech motor planning impairment in AOS (e.g., Aichert & Ziegler, 2004; Deger & Ziegler; 2001; Maas et al., 2008, 2015; Mailend et al., 2019, 2020; Rogers & Storkel, 1999; Varley & Whiteside, 2001; Ziegler, 2005; Ziegler et al., 2020) framed in the context of current theories of speech production (e.g., Bohland et al., 2010; Guenther, 2016; Klapp, 2003; Levelt et al., 1999; Van der Merwe, 2020). This body of work has not yet led to empirically substantiated subtypes of AOS, but several observations from these studies illustrate how subtypes of AOS may emerge from such an approach, and how these subtypes may differ from those derived based on clinical presentation. We will use recent studies that have been guided by the DIVA model (Guenther et al., 2006), and its extension, GODIVA (Bohland et al., 2010) to illustrate these points.[6] We will first briefly review this neurocomputationally specified framework,

[6]Our focus on the DIVA/GODIVA models is not to suggest that other theories and models cannot inform thinking about possible AOS subtypes; clearly, they can. For example, possible AOS subtypes in the Klapp (2003) model might relate to deficits in INT (internal programming) vs. SEQ (sequencing) (e.g., Deger & Ziegler, 2002; Maas et al., 2008), and possible AOS subtypes in the Levelt et al.

focusing on aspects that are relevant to provide context to the current discussion (see also Miller & Guenther, 2020, for further discussion).

In very broad strokes, speech motor planning within DIVA/GODIVA starts with the activation of a representation in the speech sound map from corresponding phonological representations encoded in the previous stage (phonological encoding). A representation in the speech sound map corresponds to a speech unit (e.g., a syllable, although the unit may represent smaller or larger units of speech based on their frequency of occurrence). The speech sound map representations, in turn, activate (a) feedforward connections to the motor cortex, which represent predictive commands for previously learned motor routines that guide articulation (i.e., motor programs) and (b) feedback connections, which encode the expected auditory and somatosensory targets and generate error signals, should a mismatch between the expected and actual feedback be detected. While the feedback system has a critical role in development and finetuning of the feedforward system, adult speakers rely primarily on feedforward commands to speak. The feedback system acts as a monitor under typical production. Finally, speech unfolds via rapid sequential production of different motor routines and thus the theory also includes parallel activation of upcoming speech units and a mechanism for sequential selection amongst the simultaneously activated units.

Each of the above described speech motor planning operations within the DIVA/GODIVA model can theoretically be impaired and thus give rise to a specific impairment of speech motor planning. Several of these impairment mechanisms have been captured in specific hypotheses and experimentally tested (Ballard et al., 2018; Jacks, 2008; Maas et al., 2015; Mailend & Maas, 2013; Mailend et al., 2019, 2020). For example, damage to the speech sound map would compromise the stored knowledge about a given speech movement resulting in underspecified or absent speech motor programs for a given unit of speech. This hypothesis has been termed the Damaged Programs Hypothesis (Mailend & Maas, 2013) or Feedforward System Deficit Hypothesis (Maas et al., 2015). Alternatively, damage to the mechanism for parallel activation may result in a difficulty with selecting the intended speech motor program from a number of simultaneously activated units, giving rise to a different type of speech motor planning impairment (the Program Retrieval Deficit Hypothesis, Mailend & Maas, 2013; see Miller & Guenther, 2020, for a comprehensive description of speech motor planning impairments within the DIVA/GODIVA framework proposed thus far).

To illustrate the ideas that are relevant in light of the discussion of subtypes, the rest of the discussion will focus on a few studies that tested specific hypotheses of the underlying impairment in AOS within the DIVA/GODIVA framework. These experiments were designed for group-level analysis but, in order to corroborate the group-level findings, tests at the level of the individual were also performed. It is the individual results that proved particularly interesting from the perspective of possible subtypes of AOS.

(1999) theory might relate to deficits in direct vs. indirect processing routes (e.g., Aichert & Ziegler, 2004; Varley & Whiteside, 2001). However, review of all theories and models in relation to their possible AOS subtypes is beyond the scope of this paper. We chose to illustrate the potential for elucidating AOS subtypes within the DIVA/GODIVA models because they are computationally specified, well-supported by evidence from typical speakers, and have been used to guide recent experimental investigations of AOS that presented individual speaker data.

In a series of studies that tested the Program Retrieval Deficit Hypothesis reviewed above against competing alternatives (Mailend & Maas, 2013, Mailend et al., 2019, 2020), the group-level findings were consistent with the predictions of the Program Retrieval Deficit Hypothesis. The individual analysis corroborated the findings in most speakers with the clinical diagnosis of AOS, but there were also some participants whose individual data deviated from the group pattern. These discrepancies between group and individual analysis may have many reasons; among them is the possibility that the different individual patterns reflected a different speech motor planning impairment and by extension a different subtype of AOS. Considering that the experiments were not primarily designed to test the hypotheses at the level of the individual, it may be premature to attempt to pinpoint the reasons for the occasional discrepancies between individual- and group-level data. Nevertheless, this inconsistency raises a concern that meaningful individual differences may be masked by group-level analysis, underscoring the importance of examining individual data (see e.g., Aichert & Ziegler, 2012; Odell et al., 1990, 1991, for similar points). Furthermore, these data demonstrate that current models of speech production are sufficiently detailed to support investigations into different components that may be individually impaired in speakers with AOS and thus reflect a different subtype of a speech motor planning impairment.

The study of AOS is complicated by the limited number of participants with an isolated diagnosis of AOS in the absence of other speech/language diagnoses such as aphasia and/or dysarthria. Thus, including participants with aphasia without AOS is often necessary to control for the general effect of brain damage and the more specific effect of aphasia in the experimental results. In some studies (e.g., Mailend & Maas 2013; Mailend et al., 2020), individual-level analysis has identified participants with a clinical diagnosis of aphasia without AOS, whose data resembled the AOS group data. From these results alone, it cannot be determined whether the current criteria for diagnosing AOS (and in particular differentiating AOS from phonological impairments in aphasia) exclude some individuals whose speech motor planning processes are impaired (McNeil et al., 1990) or whether some clinical symptoms that indicate AOS diagnosis result from a disruption at a higher level, such as phonological retrieval (Buchwald et al., 2017; Maas et al., 2014). Consequently, without understanding the possible impairment mechanisms *within* a diagnostic class, it is difficult to determine the boundaries with its clinical neighbours *outside* that diagnostic class. Furthermore, these individual-level data substantiate the concern that subclasses derived inductively from clinical symptoms and those derived deductively from theoretical conceptualization do not necessarily overlap.

In sum, developing a detailed understanding of the impaired process(es) of speech motor planning in accordance with a well-specified theory of speech motor planning has been informative to the discussion of possible subtypes in AOS in many regards. First, current theories of speech production are specific enough at the level of speech motor planning to derive and test hypotheses involving impairments of a specific component or mechanism of motor planning. An added benefit of computationally specified models is that underlying impairments (either single isolated impairments or multiple combined impairments) can be simulated in order to generate expected patterns of behaviour (see Terband et al., 2020, for example with respect to AOS). The complexity of the system can make it difficult to derive

predictions on the basis of conceptual understanding alone, and counter-intuitive predictions may emerge that would not have otherwise been expected (see Terband et al., 2009, for an example in childhood apraxia of speech, where abnormal perseverative coarticulation was predicted by the computational simulation). Of course, as with any model, the quality of its predictions depends on the validity of both the theoretical and modelling assumptions. Second, the theory-driven approach has highlighted the importance of examining individual-level data and including a sample of speakers that represent not only the disorder of interest but also its clinical neighbours. Third, current theoretical accounts of speech motor planning support the notion that the impairment may not be unitary across all people who have received the diagnostic label of AOS. Although the theory-driven experimental approach may not lead to immediate clinical application, it may be able to help break the circularity of studying a phenomenon with the same tools with which it was defined. Instead, by independently defining and circumscribing the domain of speech motor planning and generating specific predictions for patterns of behaviour in experimental tasks associated with deficits in these presumed speech motor planning mechanisms – in particular patterns of behaviour that were not used to diagnose AOS – we may discover that some people who are clinically diagnosed with AOS do not fit the expected patterns and, conversely, that some people who do not have a clinical diagnosis of AOS may meet these expectations. While we would expect, and hope, that there would be considerable overlap between clinical diagnosis and theoretically derived characterization of speech motor planning impairments, there are likely to be some disagreements as well. Such disagreements may reflect imperfections of the "gold standard" of clinical judgment to diagnose speech motor planning problems (i.e. AOS), problems with the underlying theory or its derived hypotheses and predictions, and/or different subtypes of AOS. Either way, this model-driven approach to understanding AOS (and its possible subtypes) is likely to be most fruitful when including participants with a range of speech output difficulties, rather than only those diagnosed clinically with AOS, so that theoretically coherent patterns can be detected, separate from whether such patterns align with clinical diagnosis. Finally, finding evidence for an impairment that was predicted by a theory will help elucidate the architecture of speech production from a theoretical perspective, by offering a different source of evidence.

Future Directions

The potential utility of AOS subtypes, and the appropriate basis for classification, depends on the goal. For example, if the goal is to predict the likely course of progressive AOS, perhaps a division into phonetic and prosodic subtypes based on predominant error patterns is sufficient and appropriate. If the goal is to gain a deeper understanding of the underlying speech motor planning operations and impairments, then a theory-driven approach to identifying potential subtypes may be more appropriate. Ultimately, a clearer understanding is needed of the sources of inter-individual variability among people with AOS in terms of speech performance, treatment response, and recovery or disease progression. Whether, or to what extent, such variability can be captured by AOS subtypes (and if so, which ones) remains to be seen. We suggest here that consideration of theoretically motivated subtypes is a promising avenue for further exploration, especially with the emergence of increasingly specific and detailed theories and models of speech motor planning (e.g., Bohland et al.,

2010; Guenther et al., 2006; Hickok, 2012; Parrell et al., 2019). Below we outline a few important directions and considerations for future research on possible AOS subtypes.

First, research should be guided by well-developed and detailed theories and models of speech planning processes. Consideration of the various processes and mechanisms involved in planning and producing speech enables conceptualization of different possible impairments (see also Miller & Guenther, 2020). Recognizing the complexity of speech motor planning and control, rather than viewing speech motor planning and control as a single processing step, naturally suggests that the different mechanisms or processes could be selectively impaired (see also Croot, 2002). The complexity of speech motor planning, with multiple processes and mechanisms that interact in intricate ways, makes it difficult to generate specific predictions based on intuition and a conceptual understanding. For this reason, computational modelling will become more crucial to develop hypotheses and generate and test specific predictions about expected performance patterns. Computational simulations of specific deficits in particular components of the speech motor planning system can help constrain the search space and possible patterns of performance, which can be compared to behaviour of human individuals with AOS (see Terband et al., 2020, for an example of computational simulations compared to empirical data from people with AOS).

Second, tasks and measures must be developed that are sufficiently sensitive and specific to detect deficits in different components of the speech motor planning system. Given the well-known problems of categorical perception and the ambiguity of perceived errors relative to underlying processes (e.g., Liberman et al., 1957; Kent, 1996; Maas & Mailend, 2012; MacNeilage, 1982; A. S. Meyer, 1992), error analysis is likely to be insufficient to capture perhaps subtle yet important distinctions between different types of AOS. A more fruitful approach may be to reason from underlying processes to expected patterns of behaviour on more sensitive measures obtained in the context of particular experimental manipulations. The basic science of speech motor control has relied less on perceived speech errors than on measures such as acoustics (e.g., Houde & Jordan, 1998; Shiller et al., 2009; Villacorta et al., 2007), kinematics (e.g., Tremblay et al., 2003) and reaction times (e.g., Cholin et al., 2011; D. E. Meyer & Gordon, 1985; Rogers & Storkel, 1998). Furthermore, such measures are often obtained in the context of experimental paradigms in which participants produce speech under different conditions, such as acoustic or kinematic perturbations (e.g., Houde & Jordan, 1998; Tremblay et al., 2003; Villacorta et al., 2007) or specific order of presentation to induce interference (e.g., D. E. Meyer & Gordon, 1985; Rogers & Storkel, 1998). As we have argued elsewhere (e.g., Maas & Mailend, 2012; Maas et al., 2014, 2015; Mailend et al., 2019), an understanding of speech disorders should be framed in the context of models of speech production. Application of experimental paradigms and measures from the basic literature allows for cross-validation and facilitates integration with models of speech production (see Ballard et al., 2018; Jacks, 2008; Maas et al., 2015; Mailend et al., 2019, 2020, for recent examples).[7] Of course, ultimately, the findings from such paradigms

[7]We do not mean to imply that use of acoustic or physiological measures to study AOS is a new phenomenon. In fact, there is a rich literature in which AOS has been studied using acoustic (e.g., DiSimoni & Darley, 1977; Freeman et al., 1978; Hoit-Dalgaard et al., 1983; Kent & Rosenbek, 1983; Lozano & Dreyer, 1978; Ziegler & Von Cramon, 1986) and kinematic and physiological measures

and measures must be interpreted relative to the observable speech features for a full understanding.

Third, to the extent that specific tasks and measures can be developed that assess the integrity of different speech motor planning processes or mechanisms, large-scale studies are ultimately needed to determine whether theoretically coherent subtypes emerge in the population of people with AOS at large. Case study methodology may also be appropriate to detect double dissociations within the broader category of AOS (see also Croot, 2002). For further validation, relationships with other variables must be examined as well, including lesion location, disease progression, recovery and response to treatment. For this admittedly longer-term goal, large sample sizes and longitudinal designs are likely needed, as well as clinically feasible versions of these various tasks and measures to be used in such studies. In the meantime, development and testing of specific theory-based hypotheses regarding underlying impairments in AOS may proceed using smaller sample sizes and individual analyses in order to develop and refine experimental paradigms and measures that tap into different processes and mechanisms.

In sum, it remains to be seen whether there are discernible subtypes of AOS or whether all interindividual variability among people with AOS can be explained by overall severity or factors extraneous to AOS proper (e.g., comorbid aphasia, dysarthria, compensatory strategies). Nevertheless, the systematic, theoretically motivated search for potential subtypes along the lines sketched here is likely to advance our understanding of AOS and speech motor planning in general, which in turn may provide important insights for clinical management of AOS.

Conclusions

Despite decades of speculations that the AOS diagnosis may capture different types of underlying impairments, relatively few studies have focused on identifying the potential subtypes and their corroboration with empirical evidence. In this review, we explored the literature that may be relevant to the idea of possible subtypes in AOS from the perspective of different bases for classification. Some important conclusions emerge form looking at this problem from such a viewpoint. Currently, the most substantiated case for AOS subtypes comes from studies on progressive AOS where different subtypes are identified based on clinical presentations. The nature of these subtypes – phonetic and prosodic variants—have a significant resemblance to efferent and afferent subtypes of AOS described by Luria in the context of his classic theory of cognitive functions. Importantly, much of Luria's work also focused on patients with a different etiology than stroke, particularly head wounds and tumours. Considering that such subtypes have not yet emerged from AOS studies with stroke patients (although see Takakura et al., 2019), which undoubtedly has been the population of interest in most AOS studies to date, this similarity across decades and approaches highlights the importance of studying AOS in the context of different etiologies (see also Duffy & Josephs, 2012). That is particularly important if the goal is to identify subtypes that

(e.g., Fromm et al., 1982; Itoh et al., 1979; Keatley & Pike, 1976; Robin et al., 1989; Shankweiler et al., 1968; Sugishita et al., 1986; Towne & Crary, 1988). However, many of these studies are largely descriptive and not framed in terms of specific theories or models of speech production.

reflect underlying processes rather than commonly co-damaged impairments due to vascular distribution patterns.

Similarly, recent studies from our lab that have examined the underlying impairment in AOS from a theory-driven perspective (e.g., Maas et al., 2015; Mailend & Maas, 2013; Mailend et al., 2019) have shown that not all people with a clinical diagnosis of AOS show similar result patterns in experimental tasks that were designed to tap the speech motor planning processes. Furthermore, some speakers with aphasia without AOS did pattern with the majority of speakers in the AOS group. Although the sample sizes in these experiments were not sufficient to argue for subtypes over individual differences unrelated to AOS, this finding raises the concern that the clinical diagnosis based on current behavioural criteria is neither sufficiently sensitive nor specific to identify uniform groups in terms of underlying impairment.

In conclusion, we argue that there is sufficient evidence to question the uniformity of the motor planning impairment currently captured with the clinical diagnosis of AOS. Furthermore, subtypes solely based on perceptual clinical symptoms may be inadequate for identifying boundaries within the syndrome, given that many speech features may arise from different underlying deficits (whether different speech motor planning deficits or deficits at the phonological level). This is not to say that speech features, or patterns of speech features, could not be identified that separate particular AOS subtypes or AOS from phonological impairments, but rather that juxtaposing symptom-based subtypes with theory-driven experimentally demonstrated subtypes is a promising future direction in order to cross-validate such (patterns of) speech features, particularly when participants with various underlying etiologies are considered. Examining the possibility of subtypes within a theory of speech production has the potential to advance our understanding of speech motor planning, identify its potential impairment mechanisms, and serve as a triangulation point for delineating clinical groups that reflect a common underlying impairment. It is expected that such an approach may lead to a classification system that identifies impairments to specific processes rather than constellation of symptoms into syndromes (see Caplan, 1993, for a similar argument in aphasia, and Baker et al., 2001 and Terband et al., 2019, for paediatric speech disorders). Such a theoretically based and process-oriented understanding of AOS may naturally diminish problems with identifying the boundaries between AOS and aphasia and hold important promise for clinical practice.

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References

Aichert I, & Ziegler W (2004). Syllable frequency and syllable structure in apraxia of speech. Brain and Language, 88 (1), 148–159. [PubMed: 14698739]

Aichert I, & Ziegler W (2012). Word position effects in apraxia of speech: Group data and individual variation. Journal of Medical Speech-Language Pathology, 20 (4), 7–11.

Akhutina T (2016). Luria's classification of aphasias and its theoretical basis. Aphasiology, 30 (8), 878–897.

Ardila A (2010). A proposed reinterpretation and reclassification of aphasic syndromes. Aphasiology, 24 (3), 363–394.

Baker E, Croot K, McLeod S, & Paul R (2001). Psycholinguistic models of speech development and their application to clinical practice. Journal of Speech, Language, and Hearing Research, 44 (3), 685–702.

Ballard KJ, Azizi L, Duffy JR, McNeil MR, Halaki M, O'Dwyer N, Layfield C, Scholl DI, Vogel AP, & Robin DA (2016). A predictive model for diagnosing stroke-related apraxia of speech. Neuropsychologia, 81, 129–139. [PubMed: 26707715]

Ballard KJ, Halaki M, Sowman P, Kha A, Daliri A, Robin DA, ... & Guenther, F. H. (2018). An investigation of compensation and adaptation to auditory perturbations in individuals with acquired apraxia of speech. Frontiers in Human Neuroscience, 12, 510. [PubMed: 30618687]

Bohland JW, Bullock D, & Guenther FH (2010). Neural representations and mechanisms for the performance of simple speech sequences. Journal of Cognitive Neuroscience, 22 (7), 1504–1529. [PubMed: 19583476]

Brownsett SLE, Ramajoo K, Copland D, McMahon KL, Robinson G, Drummond K, ... & De Zubicaray G (2019). Language deficits following dominant hemisphere tumor resection are significantly underestimated by syndrome-based aphasia assessments. Aphasiology, 33(10), 1163–1181.

Buckingham HW Jr (1979). Explanation in apraxia with consequences for the concept of apraxia of speech. Brain and Language, 8, 202–226. [PubMed: 487070]

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 (6S), 1726–1738.

Buxbaum LJ (2001). Ideomotor apraxia: A call to action. Neurocase, 7 (6), 445–458. [PubMed: 11788737]

Buxbaum LJ, Haal KY, Hallett M, Wheaton L, Heilman KM, Rodriguez A, & Gonzalez Rothi LJ (2008). Treatment of limb apraxia. Moving forward to improved action. American Journal of Physical Medicine & Rehabilitation, 87 (2), 149–161. [PubMed: 18209511]

Caplan D (1993). Toward a psycholinguistic approach to acquired neurogenic language disorders. American Journal of Speech-Language Pathology, 2, 59–83.

Cholin J, Dell GS, & Levelt WJ (2011). Planning and articulation in incremental word production: Syllable-frequency effects in English. Journal of Experimental Psychology: Learning, Memory, and Cognition, 37 (1), 109–122.

Code C (1998). Models, theories and heuristics in apraxia of speech. Clinical Linguistics & Phonetics, 12 (1), 47–65.

Collins M, Cariski D, Longstretch D, & Rosenbek J (1980). Patterns of articulatory behavior in selected motor speech programming disorders. In Brookshire RH (Ed.), Clinical aphasiology 1980 (Vol. 10, pp. 196–208). BRK Publishers.

Croot K (2002). Diagnosis of AOS: Definition and criteria. Seminars in Speech and Language, 23 (4), 267–280. [PubMed: 12461726]

Cunningham KT, Haley KL, & Jacks A (2016). Speech sound distortions in aphasia and apraxia of speech: Reliability and diagnostic significance. Aphasiology, 30 (4), 396–413.

Darley FL (1982). Aphasia. WB Saunders Co.

Darley FL (1983). Aphasia: With and without adjectives. Clinical Aphasiology, 13, 281–284.

Darley FL, Aronson AE, & Brown JR (1975). Motor speech disorders. Saunders.

Deger K, & Ziegler W (2002). Speech motor programming in apraxia of speech. Journal of Phonetics, 30 (3), 321–335.

Dell GS (1986). A spreading activation theory of retrieval in sentence production. Psychological Review, 93, 283–321. [PubMed: 3749399]

Den Ouden DB, Galkina E, Basilakos A, & Fridriksson J (2018). Vowel formant dispersion reflects severity of apraxia of speech. Aphasiology, 32 (8), 902–921. [PubMed: 30297975]

De Renzi E, & Lucchelli F (1988). Ideational apraxia. Brain, 111 (5), 1173–1185. [PubMed: 3179688]

DiSimoni FG, & Darley FL (1977). Effect on phoneme duration control of three utterance-length conditions in an apractic patient. Journal of Speech and Hearing Disorders, 42 (2), 257–264.

Duffy JR (2006). Apraxia of speech in degenerative neurologic disease. Aphasiology, 20 (6), 511–527.

Duffy JR (2013). Motor speech disorders: Substrates, differential diagnosis, and management (3rd Edition). Elsevier, Mosby.

Duffy JR, & Josephs KA (2012). The diagnosis and understanding of apraxia of speech: Why including neurodegenerative etiologies may be important. Journal of Speech, Language, and Hearing Research, 55, S1518–S1522.

Fairbanks G (1954). Systematic research in experimental phonetics: 1. A theory of the speech mechanism as a servosystem. Journal of Speech and Hearing Disorders, 19 (2), 133–139.

Feiken JF, & Jonkers R (2012). A division into three subtypes of apraxia of speech: Ideomotor, kinetic and ideational. Paper presented at the Clinical Aphasiology Conference (Lake Tahoe, CA, May 2012).

Folkins JW, & Bleile KM (1990). Taxonomies in biology, phonetics, phonology, and speech motor control. Journal of Speech and Hearing Disorders, 55 (4), 596–611.

Freeman FJ, Sands ES, & Harris KS (1978). Temporal coordination of phonation and articulation in a case of verbal apraxia: A voice onset time study. Brain and Language, 6 (1), 106–111. [PubMed: 698778]

Fromkin VA (1971). The non-anomalous nature of anomalous utterances. Language, 51, 27–52.

Fromm D, Abbs JH, McNeil MR, & Rosenbek JC (1982). Simultaneous perceptual-physiological method for studying apraxia of speech. In Brookshire RH (Ed.), Clinical aphasiology 1982 (Vol. 12, pp. 251–262). BRK Publishers.

Garrett MF (1975). The analysis of sentence production. In Bower G (Ed.), Psychology of learning and motivation (Vol. 9, pp. 137–177). Academic Press.

Goldenberg G (2003). Apraxia and beyond: Life and work of Hugo Liepmann. Cortex, 39, 509–524. [PubMed: 12870824]

Goldrick M (2006). Limited interaction in speech production: Chronometric, speech error, and neuropsychological evidence. Language and Cognitive Processes, 21 (7–8), 817–855.

Goldrick M, & Blumstein SE (2006). Cascading activation from phonological planning to articulatory processes: Evidence from tongue twisters. Language and Cognitive Processes, 21 (6), 649–683.

Guenther FH (1994). A neural network model of speech acquisition and motor equivalent speech production. Biological Cybernetics, 72, 43–53. [PubMed: 7880914]

Guenther FH (1995). Speech sound acquisition, coarticulation, and rate effects in a neural network model of speech production. Psychological Review, 102, 594–621. [PubMed: 7624456]

Guenther FH (2016). Neural control of speech. MIT Press.

Guenther FH, Ghosh SS, & Tourville JA (2006). Neural modeling and imaging of the cortical interactions underlying syllable production. Brain and Language, 96 (3), 280–301. [PubMed: 16040108]

Haley KL, Jacks A, de Riesthal M, Abou-Khalil R, & Roth HL (2012). Toward a quantitative basis for assessment and diagnosis of apraxia of speech. Journal of Speech, Language, and Hearing Research, 55, S1502–S1517.

Hickok G (2012). Computational neuroanatomy of speech production. Nature Neuroscience Reviews, 13, 135–145.

Hickok G, Houde J, & Rong F (2011). Sensorimotor integration in speech processing: Computational basis and neural organization. Neuron, 69, 407–422. [PubMed: 21315253]

Hoit-Dalgaard J, Murry T, & Kopp HG (1983). Voice onset time production and perception in apraxic subjects. Brain and Language, 20 (2), 329–339. [PubMed: 6640283]

Houde JF, & Jordan MI (1998). Sensorimotor adaptation in speech production. Science, 279 (5354), 1213–1216. [PubMed: 9469813]

Howard D, Swinburn K, & Porter G (2010). Putting the CAT out: What the Comprehensive Aphasia Test has to offer. Aphasiology, 24 (1), 56–74.

Itoh M, Sasanuma S, & Ushijima T (1979). Velar movements during speech in a patient with apraxia of speech. Brain and Language, 7 (2), 227–239. [PubMed: 466393]

Jacks A (2008). Bite block vowel production in apraxia of speech. Journal of Speech, Language, and Hearing Research, 51 (4), 898–913.

Josephs KA, Duffy JR, Strand EA, Machulda MM, Senjem ML, Lowe VJ, Jack CR Jr., & Whitwell JL (2013). Syndromes dominated by apraxia of speech show distinct characteristics from agrammatic PPA. Neurology, 81 (4), 337–345. [PubMed: 23803320]

Keatley MA, & Pike P (1976). The Automated Pulmonary Function Laboratory: Clinical use in determining respiratory variations in apraxia. In Brookshire RH (Ed.), Clinical aphasiology 1976 (Vol. 6, pp. 98–109). BRK Publishers.

Kent RD (1991). Research needs in the assessment of speech motor disorders. In Cooper JA (Ed.), Assessment of speech and voice production: Research and clinical applications (pp. 17–29). National Institute on Deafness and Other Communication Disorders.

Kent RD (1996). Hearing and believing: Some limits to the auditory-perceptual assessment of speech and voice disorders. American Journal of Speech-Language Pathology, 5, 7–23.

Kent RD, & Rosenbek JC (1982). Prosodic disturbance and neurologic lesion. Brain and Language, 15 (2), 259–291. [PubMed: 7074345]

Kent RD, & Rosenbek JC (1983). Acoustic patterns of apraxia of speech. Journal of Speech and Hearing Research, 26 (2), 231–249. [PubMed: 6887810]

Kertesz A, & Phipps JB (1977). Numerical taxonomy of aphasia. Brain and Language, 4 (1), 1–10. [PubMed: 832101]

Klapp ST (2003). Reaction time analysis of two types of motor preparation for speech articulation: Action as a sequence of chunks. Journal of Motor Behavior, 35, 135–150. [PubMed: 12711585]

Lane VW, & Samples JM (1981). Facilitating communication skills in adult apraxics: Application of blissymbols in a group setting. Journal of Communication Disorders, 14 (2), 157–167. [PubMed: 6454701]

Levelt WJM, Roelofs A, & Meyer AS (1999). A theory of lexical access in speech production. Behavioral and Brain Sciences, 22, 1–38.

Liberman AM, Harris KS, Hoffman HS, & Griffith BC (1957). The discrimination of speech sounds within and across phoneme boundaries. Journal of Experimental Psychology, 54, 358–368. [PubMed: 13481283]

Liepmann H (1900). Das Krankheitsbild der Apraxie (Motorische asymbolie): Auf Grund eines Falles von einseitiger Apraxie. [The clinical presentation of apraxia (motoric asymbolia): On the basis of a case of unilateral apraxia] Monatschrift für Psychiatrie und Neurologie, 8, 15–44.

Lozano RA, & Dreyer DE (1978). Some effects of delayed auditory feedback on dyspraxia of speech. Journal of Communication Disorders, 11 (5), 407–415. [PubMed: 730833]

Luria AR (1973). The working brain: An introduction to neuropsychology. Basic Books.

Luria AR (1980). Higher cortical functions in man (2nd Ed.). Basic Books, Inc., Publishers.

Lustig AP, & Tompkins CA (2002). A written communication strategy for a speaker with aphasia and apraxia of speech: Treatment outcomes and social validity. Aphasiology, 16 (4–6), 507–521.

Maas E, Gutiérrez K, & Ballard KJ (2014). Phonological encoding in apraxia of speech and aphasia. Aphasiology, 28 (1), 25–48.

Maas E, Mailend ML, & Guenther FH (2015). Feedforward and feedback control in apraxia of speech: Effects of noise masking on vowel production. Journal of Speech, Language, and Hearing Research, 58 (2), 185–200.

Maas E, Robin DA, Wright DL, & Ballard KJ (2008). Motor programming in apraxia of speech. Brain and Language, 106, 107–118. [PubMed: 18417200]

MacNeilage PF (1970). Motor control of serial ordering of speech. Psychological Review, 77 (3), 182. [PubMed: 5454132]

MacNeilage PF (1982). Speech production mechanisms in aphasia. In Grillner S, Lindblom B, Lubker J, & Persson A (Eds.), Speech motor control (pp. 43–60). Pergamon Press.

Mailend M-L, & Maas E (2013). Speech motor programming in apraxia of speech: Evidence from a delayed picture-word interference task. American Journal of Speech-Language Pathology, 22 (2), S380–S396. [PubMed: 23695911]

Mailend ML, Maas E, Beeson PM, Story BH, & Forster KI (2019). Speech motor planning in the context of phonetically similar words: Evidence from apraxia of speech and aphasia. Neuropsychologia, 127, 171–184. [PubMed: 30817912]

Mailend ML, Maas E, Beeson PM, Story BH, & Forster KI (2020). Examining speech motor planning difficulties in apraxia of speech and aphasia via the sequential production of phonetically similar words. [Manuscript submitted for publication].

McNeil MR, Doyle PJ, & Wambaugh JL (2000). Apraxia of speech: A treatable disorder of motor planning and programming. In Nadeau SE, Rothi L. J. Gonzalez, & Crosson B (Eds.), Aphasia and language: Theory to practice (pp. 221–266). The Guilford Press.

McNeil MR, & Kimelman MD (2001). Darley and the nature of aphasia: The defining and classifying controversies. Aphasiology, 15, 221–229.

McNeil MR, Liss JM, Tseng CH, & Kent RD (1990). Effects of speech rate on the absolute and relative timing of apraxic and conduction aphasic sentence production. Brain and Language, 38 (1), 135–158. [PubMed: 2302543]

McNeil MR, Robin DA, & Schmidt RA (1997). Apraxia of speech: Definition, differentiation, and treatment. In: McNeil MR (Ed.), Clinical management of sensorimotor speech disorders (pp. 311–344). Thieme.

Meyer AS (1992). Investigation of phonological encoding through speech error analysis: Achievements, limitations, and alternatives. Cognition, 42, 181–211. [PubMed: 1582156]

Meyer DE, & Gordon PC (1985). Speech production: Motor programming of phonetic features. Journal of Memory and Language, 24 (1), 3–26.

Miller HE, & Guenther FH (2020). Modelling speech motor programming and apraxia of speech in the DIVA/GODIVA neurocomputational framework. Aphasiology, 1–18.

Ochipa C, & Rothi LJG (2000). Limb apraxia. Seminars in Neurology, 20 (4), 471–478. [PubMed: 11149703]

Ochipa C, Rothi LG, & Heilman KM (1989). Ideational apraxia: A deficit in tool selection and use. Annals of Neurology, 25 (2), 190–193. [PubMed: 2465733]

Ochipa C, Rothi LJG, & Heilman KM (1992). Conceptual apraxia in Alzheimer's disease. Brain, 115 (4), 1061–1071. [PubMed: 1393502]

Odell K, McNeil MR, Rosenbek JC, & Hunter L (1990). Perceptual characteristics of consonant production by apraxic speakers. Journal of Speech and Hearing Disorders, 55 (2), 345–359.

Odell K, McNeil MR, Rosenbek JC, & Hunter L (1991). Perceptual characteristics of vowel and prosody production in apraxic, aphasic, and dysarthric speakers. Journal of Speech, Language, and Hearing Research, 34 (1), 67–80.

Parrell B, Ramanarayanan V, Nagarajan S, & Houde J (2019). The FACTS model of speech motor control: Fusing state estimation and task-based control. PLoS Computational Biology, 15 (9), e1007321. [PubMed: 31479444]

Pearce JMS (2009). Hugo Karl Liepmann and apraxia. Clinical Medicine, 9 (5), 466–470. [PubMed: 19886110]

Perkell JS (1969). Physiology of speech production: Results and implication of quantitative cineradiographic study. Monograph, 53. MIT Press.

Perkell JS (2012). Movement goals and feedback and feedforward control mechanisms in speech production. Journal of Neurolinguistics, 25 (5), 382–407. [PubMed: 22661828]

Perkell JS (2013). Five decades of research in speech motor control: what have we learned, and where should we go from here? Journal of Speech, Language, and Hearing Research, 56, S1857–S1874.

Robin DA, Bean C, & Folkins JW (1989). Lip movement in apraxia of speech. Journal of Speech and Hearing Research, 32 (3), 512–523. [PubMed: 2779196]

Rogers MA (2001). Treatment research on augmentative and alternative communication for adults with apraxia of speech. Perspectives on Neurophysiology and Neurogenic Speech and Language Disorders, 11(4), 21–28.

Rogers MA, & Storkel HL (1998). Reprogramming phonologically similar utterances: The role of phonetic features in pre-motor encoding. Journal of Speech, Language, and Hearing Research, 41 (2), 258–274.

Rogers MA, & Storkel HL (1999). Planning speech one syllable at a time: the reduced buffer capacity hypothesis in apraxia of speech. Aphasiology, 13 (9–11), 793–805.

Rosenbek JC (1991). It's a poor sort of memory that only works backwards. Clinical Aphasiology, 20, 41–52.

Rosenbek JC (2001). Darley and apraxia of speech in adults. Aphasiology, 15 (3), 261–273.

Roy EA, & Square PA (1985). Common considerations in the study of limb, verbal and oral apraxia. In Roy EA (Ed.), Neuropsychological studies of apraxia and related disorders (pp. 111–161). North Holland: Elsevier.

Schwartz MF (1984). What the classical aphasia syndromes can't do for us, and why. Brain and Language, 21, 3–8. [PubMed: 6697169]

Shankweiler D, Harris KS, & Taylor ML (1968). Electromyographic studies of articulation in aphasia. Archives of Physical Medicine and Rehabilitation, 49, 1–8. [PubMed: 5635198]

Shattuck-Hufnagel S (1979). Speech errors as evidence for a serial order mechanism in sentence production. In Cooper WE & Walker ECT (Eds.), Sentence processing: Psycholinguistic studies presented to Merrill Garrett (pp. 295–342). Lawrence Erlbaum.

Shiller DM, Sato M, Gracco VL, & Baum SR (2009). Perceptual recalibration of speech sounds following speech motor learning. Journal of the Acoustical Society of America, 125 (2), 1103–1113.

Square PA, Darley FL, & Sommers RK (1982). An analysis of the productive errors made by pure apractic speakers with differing loci of lesions. In: Clinical Aphasiology: Proceedings of the Conference 1982 (pp. 245–250). BRK Publishers.

Sternberg S, Monsell S, Knoll RL, & Wright CE (1978). The latency and duration of rapid movement sequences: Comparisons of speech and typewriting. In Stelmach GE (Ed.), Information processing in motor control and learning (pp. 117–152). Academic Press.

Strand EA, Duffy JR, Clark HM, & Josephs K (2014). The Apraxia of Speech Rating Scale: A tool for diagnosis and description of apraxia of speech. Journal of Communication Disorders, 51, 43–50. [PubMed: 25092638]

Sugishita M, Konno K, Kabe S, Yunoki K, Togashi O, & Kawamura M (1987). Electropalatographic analysis of apraxia of speech in a left hander and in a right hander. Brain, 110 (5), 1393–1417. [PubMed: 3676707]

Takakura Y, Otsuki M, Sakai S, Tajima Y, Mito Y, Ogata A, Koshimizu S, Yoshino M, Uemori G, Takakura S, & Nakagawa Y (2019). Sub-classification of apraxia of speech in patients with cerebrovascular and neurodegenerative diseases. Brain and Cognition, 130, 1–10. [PubMed: 30622034]

Terband H, Maassen B, Guenther FH, & Brumberg J (2009). Computational neural modeling of speech motor control in childhood apraxia of speech (CAS). Journal of Speech, Language, and Hearing Research, 52, 1595–1609.

Terband H, Maassen B, & Maas E (2019). A model of speech development and disorders for diagnosis and treatment planning. Folia Phoniatrica et Logopaedica, 71, 216–227. [PubMed: 31269495]

Terband H, Rodd J, & Maas E (2020). Testing hypotheses about the underlying deficit of apraxia of speech through computational neural modelling with the DIVA model. International Journal of Speech-Language Pathology.

Towne RL, & Crary MA (1988). Verbal reaction time patterns in aphasic adults: consideration for apraxia of speech. Brain and Language, 35 (1), 138–153. [PubMed: 2460184]

Utianski RL, Duffy JR, Clark HM, Strand EA, Botha H, Schwarz CG, Machulda MM, Senjem ML, Spychalla AJ, Jack CR Jr., Petersen RC, Lowe VJ, Whitwell JL, & Josephs KA (2018). Prosodic and phonetic subtypes of primary progressive apraxia of speech. Brain and Language, 184, 54–65. [PubMed: 29980072]

Tremblay S, Shiller DM, & Ostry DJ (2003). Somatosensory basis of speech production. Nature, 423 (6942), 866–869. [PubMed: 12815431]

Van der Merwe A (1997). A theoretical framework for the characterization of pathological speech sensorimotor control. In McNeil MR (Ed.), Clinical management of sensorimotor speech disorders (pp. 1–25). Thieme.

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, 1–27.

Varley R, & Whiteside SP (2001). What is the underlying impairment in acquired apraxia of speech. Aphasiology, 15 (1), 39–49.

Villacorta V, Perkell JS, & Guenther FH (2007). Sensorimotor adaptation to feedback perturbations of vowel acoustics and its relation to perception. Journal of the Acoustical Society of America, 122 (4), 2306–2319.

Wambaugh JL, Duffy JR, McNeil MR, Robin DA, & Rogers MA (2006). Treatment guidelines for acquired apraxia of speech: A synthesis and evaluation of the evidence. Journal of Medical Speech-Language Pathology, 14 (2), xv-xv.

Youmans G, Youmans SR, & Hancock AB (2011). Script training treatment for adults with apraxia of speech. American Journal of Speech-Language Pathology, 20 (1), 23–37. [PubMed: 20739633]

Ziegler W (2002). Psycholinguistic and motor theories of apraxia of speech. Seminars in Speech and Language, 23 (4), 231–244. [PubMed: 12461723]

Ziegler W (2005). A nonlinear model of word length effects in apraxia of speech. Cognitive Neuropsychology, 22 (5), 603–623. [PubMed: 21038268]

Ziegler W, Aichert I, & Staiger A (2012). Apraxia of speech: Concepts and controversies. Journal of Speech, Language, and Hearing Research, 55 (5), S1485–S1501.

Ziegler W, Lehner K, Pfab J, & Aichert I (2020). The nonlinear gestural model of speech apraxia: clinical implications and applications. Aphasiology, 1–23.

Ziegler W, & Von Cramon D (1986). Disturbed coarticulation in apraxia of speech: Acoustic evidence. Brain and Language, 29 (1), 34–47. [PubMed: 3756460]