A neurophonetic perspective on articulation planning[1]

Wolfram Ziegler
EKN, Institute of Phonetics, Ludwig-Maximilians-University, Munich, Germany
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Abstract

This paper gives an overview of a model that predicts articulation ease for German phonological words on the basis of error data from patients with apraxia of speech (AOS). AOS is introduced as a clinical model of higher order motor processes for articulation. Word production accuracy in AOS is considered as a window into the structure of articulation plans as acquired through speech motor learning in childhood. The NLG model of apraxia of speech is explained. Applications in speech development and adult speech are outlined.

Keywords: articulation apraxia of speech

Neurophonetics

Neurophonetics is a research field that uses the knowledge and methodological tools developed in phonetics to study the neurological aspects of speaking. It covers investigations of the impact of brain dysfunctions on speech production in patients with neurologic conditions, but also of the neural substrates of speech in typical speakers.

Two different lines of neurophonetic research can be distinguished. First and foremost, phonetic thinking and methodologies are applied in the service of clinical neurology. An increasing number of studies is devoted to describing the speech patterns of patients with neurologic disorders, such as stroke, Parkinson's disease, or cerebellar ataxia. This research mainly aims at understanding the impact of neural dysfunction of specific aetiologies or localizations on the patients' speech characteristics. Applications of this research seek to establish physiologic, acoustic, or auditory-perceptual parameters that are sensitive and specific to neurologic speech impairments, with the ultimate goal of developing phonetically based clinical assessment tools (cf. Duffy 2019).

A second, less frequented line of neurophonetic research goes the opposite way, aiming to uncover general principles of speech production by investigating their breakdown due to dysfunctions of relevant brain networks. The study of disordered cognitive functions to learn more about the neural organization of "normal" cognition has long been a productive principle in cognitive neuropsychology, especially in neurolinguistics, though the "transparency" assumption underlying this principle has always been disputed. This assumption

claims that the impairment resulting from brain lesions is transparent for the structure of the underlying, unimpaired cognitive functions (Caramazza 1988). As a major precondition, this approach must be based on the solid foundations of a clinical model whose relationship to the cognitive function in question is sufficiently well established.

In this paper I will present a well-established clinical model of higher-order speech motor functions, *apraxia of speech* (*AOS*), and describe how investigations of this model may create evidence about the organization of typical speech. The disorder is considered to result from a dysfunction of the ability to plan the articulator movements required to produce the syllables, words, and sentences of the patient's native language.

A clinical model of articulation planning

Apraxia of speech occurs predominantly in patients who have suffered a left hemisphere stroke. Initially the patients are often completely unable to articulate, but within several hours or days their speech gradually recovers. Their articulation is usually slow, dysfluent, and effortful, and they produce errors such as substitutions, omissions and distortions of speech sounds, schwa intrusions in consonant clusters or cluster reductions, slowed or distorted transitions between speech sounds, and impaired coarticulation. They show visibly laborious groping movements of the articulators, with repeated attempts, false starts, and restarts in the initiation of utterances. The syndrome may manifest itself over a wide range of severity levels, from almost complete mutism to only mildly dysfluent speech with occasional articulation errors, and with different recovery dynamics across patients. For descriptions of the clinical pattern of AOS see e.g. Duffy (2019) or Ziegler (2008).

An important issue in the theoretical classification of this disorder is to differentiate it from other types of neurogenic sound production impairment. On the one hand, the symptoms of AOS are not explainable by "elementary" motor pathologies of the vocal tract muscles, such as paresis, ataxia, hypo- or hyperkinesia etc., which are summarized under the clinical term *dysarthria*. Unlike the dysarthrias, which are typically caused by bilateral brain lesions, AOS is a syndrome of the language-dominant hemisphere. The label "apraxia" historically relates to exactly this circumstance and characterizes the disorder as an impairment of "higher" motor functions. On the other hand, the syndrome is different from aphasic-phonological impairment, which is characterized by phoneme errors in essentially fluent speech, without any apparent signs of articulomotor involvement. Though these differential diagnostic considerations are far from trivial and have often been disputed, AOS is widely accepted as an autonomous clinical unit, and an enormous amount of research has been devoted to its clinical pattern and its neuro-anatomic substrate. For discussions see Ziegler, Aichert, and Staiger (2012).

Research interest in this condition has existed for almost 160 years. Broca's seminal case study of a man who lost "la faculté du langage articulé" after a lesion to the posterior part of the left inferior frontal gyrus (Broca, 1861) is generally acknowledged as the birth of systematic clinical brain-behaviour research. Broca pointed out, in the terminology of his time, that the patient was unable to articulate, although he had no obvious motor restrictions of the tongue and lips and no generalized language or cognitive impairment. He allocated the ability to articulate to the left frontal cortical region that bears his name until today, and characterized the speech impairment as a "loss of the memory of the procedures required for the articulation of words" (Broca 1861; p. 333).

Since Broca's time, the neuro-anatomic basis and the functional characterization of this condition have been discussed extensively, - probably more than any other neurologic speech or language dysfunction. Over decades, evidence has accumulated that lesions to left posterior inferior frontal cortex including the opercular part of Broca's area and the adjacent pre-motor and motor cortex, as well as subjacent anterior insular cortex, are responsible for the development of AOS. Moreover, in more recent years this region has been identified in numerous imaging studies of non-brain-damaged speakers as a higher-order speech motor centre. The dysfunction resulting from lesions to this cortical area has been characterized in varying terms as "apraxia of the language muscles", "phonetic disintegration of speech", a "programming deficit", a breakdown of the "functional coalitions" of articulation, or, more recently, a disorder of "phonetic planning" or "speech motor planning" (for a historical review see Ziegler et al. 2012).

Implied in this thinking is that the pathomechanism of AOS disrupts the language-specific motor patterns for the production of syllables, words and sentences as acquired during speech development. We have recently proposed a model that delineates how speech motor learning in childhood is mediated by subcortical structures and leads to an accumulation of a "knowledge base for articulation" in the left frontal cortex of the adult brain (Ziegler & Ackermann 2017). An obvious assumption, based on a wealth of knowledge about the learning-dependent plasticity of the human brain, is that speech motor patterns that are more strongly integrated through speech learning are represented more redundantly within the functional network of this brain region. By implication, such patterns are less vulnerable to a loss of cortical tissue in this area. Conversely, articulatory patterns that are less typical of the speaker's native language, or less cohesive, have less redundant neural representations and are therefore more vulnerable to cortical damage. For a more detailed explanation of this argument see Ziegler, Lehner, Pfab & Aichert (2020).

As a conclusion, the speech patterns of patients with AOS provide a window into the make-up of the acquired implicit knowledge about how the words and sentences of their language are articulated. The argument is similar to that

brought up in studies of speech errors in healthy speakers (e.g., Pouplier & Hardcastle 2005), but with the difference that a much larger corpus of data can be acquired from patients with AOS under relatively natural speaking conditions, and that the errors made by patients with AOS can be allocated to a rather circumscribed functional and neuro-anatomic source, i.e., a dysfunction of the acquired articulation planning processes located in the left posterior inferior frontal lobe.

Accuracy of apraxic word production as a yardstick of "articulatory ease"

For the reasons mentioned above, the errors made by patients with AOS reveal what is easy and what is difficult to articulate for adult native speakers of a given language. "Ease of articulation" is a disputed concept, at least for adult speech, because the highly overlearned nature of the articulation patterns of our native language entails that everything is equally easy for us to say (Ladefoged 1990). In the sense used here, a word is easy to pronounce if patients with apraxia of speech have relatively few problems producing it. More specifically, patients with only mild impairment may mostly produce it correctly, and only those with severe AOS make errors on it. Conversely, difficult phonetic patterns are those that provoke errors even in patients with mild AOS. Following the transparency assumption explained above, the susceptibility of words to apraxic failure mirrors the redundancy of the representation of its motor components in left inferior frontal cortex, which, in turn, is considered the neural substrate of the degree to which language-specific articulatory patterns were stabilized through speech motor learning during childhood.

In many of the earlier studies of AOS, error rates were related to phonemes, mostly with the finding that consonants are less error prone than vowels, plosives and nasals less than fricatives or affricates, voiceless obstruents less than voiced obstruents, etc. Other findings were related to syllable structure, e.g., that coda consonant errors are less frequent than onset errors, or simplex syllables are less vulnerable than complex syllables. Finally, at the supra-syllabic level a rather common finding was that the likelihood of an error increases with the number of syllables in a word. More recently, we could also demonstrate an effect of lexical stress, showing that in German AOS patients, trochaic words were "easier" to pronounce than iambic words (Aichert, Späth & Ziegler 2016). An overview of these findings including the corresponding references is listed in Table 1 of Ziegler et al. (2020).

The NLG-model

Considering that the hierarchies of articulatory planning requirements sketched above extend across all levels of the phonological architecture of words, an approach that integrates these levels is necessary to account for the interactions