

A division into three subtypes of apraxia of speech: ideomotor, kinetic and ideational.

Introduction

The precise nature of AOS remains elusive despite of lots of research (Maas, Robin, Wright & Ballard, 2008). Also the definitions that are used are not sufficiently clear (Ziegler, 2008). Based on a review of the recent literature on the underlying deficit in AOS we propose a division into three subtypes of AOS.

Apraxia of Speech: a programming deficit

AOS is universally defined as an impairment in the phonetic encoding (Darley, 1968, Ballard & Robin, 2007, Ziegler, 2008). However, there has been discussion about the exact boundaries of this definition to other speech and language disorders.

For one there has been discussion about whether AOS can be separated from a linguistic impairment. At one point it was suggested that a linguistic deficit might be the underlying disorder of AOS. Recently however most arguments to support this assumption were undermined (Aichert & Ziegler, 2004, Peach & Tonkovich, 2004, Laganaro & Alario, 2006). Certain symptoms of AOS, like a syllable frequency effect and substitutions, were initially explained as a result of a deficit at the phonological level. Closer inspection revealed that these symptoms can also be explained as result of a phonetic underlying deficit. Furthermore, there are several pure forms of AOS documented in subjects without linguistic deficits (Square, Roy, & Martin, 1997, Ziegler, 2005). Moreover, symptoms like distortions, lengthening of phonemes and intersyllabic segmentation cannot be explained on the basis of an underlying linguistic deficit, because these are never seen in pure aphasic patients (McNeill, Pratt & Fossett, 2004).

Secondly, there has been discussion whether or not AOS is due to an overall deficit in the motor programming of the articulators or specifically in the motor programming of speech. Yet, there is no convincing evidence for AOS to be a general motor programming deficit (Ziegler, 2008). This is confirmed by the fact that there is documentation available of patients with pure forms of either of these deficits (Peach & Tonkovich, 2004, Duffy, 2005). Also subjects are described in which the severity of both disorders differs immensely, which makes the assumption of one underlying disorder unlikely (Ziegler, 2003).

Finally, there has been discussion on the role of motor planning. There is general consensus that modification of motor planning does influence the characterization of AOS (Van der Merwe, 1997, Ballard, Granier & Robin, 2000, McNeil et al, 2004). Lengthened sound segments and inter segment durations enable subjects to create more time to accomplish a disturbed motor programming system (McNeil, Robin, & Schmidt, 1997). There is however no convincing evidence that impaired motor planning independently causes AOS. Discussions about preventive adaptation processes at other levels of speech and language processing such as in agrammatic aphasia and in stuttering, has led to the idea that modification of motor planning might be seen as a similar preventative adaptation that subjects either consciously or unconsciously apply (Ruiter, Kolk, & Rietveld, 2010, Namasivayam & Van Lieshout, 2008). Consequently, we define AOS as a pure deficit in the motor programming deficit of speech. Preventive adaptations that are made at the level of motor planning influence the symptomology of AOS and can be referred to as indirect symptoms of AOS.

Three types of AOS

The above described specification of AOS concerns the delineation of the definition with respect to related disorders. Recently, the discussion about the motor programming deficit itself is more upcoming (Knock, Ballard, Robin & Schmidt, 2000, Ballard & Robin, 2007, Maas et al, 2008). It is argued that AOS is an impairment in the activation or selection of a generalized motor program (GMP) and/or in the ability to correctly set the parameters specific to a situation (Ballard, et al, 2000). Others describe AOS as damage to the mechanism to develop a motor program and to efficiently integrate feedback (Ballard & Robin, 2007). AOS is also seen as an impairment of the preprogramming stage or in the process that assigns serial order to multiple programs in a sequence (Maas, et al, 2008)

In the literature the symptoms of AOS are broadly divided into three categories: 1) initiation problems, 2) poorly formed phonemes and 3) sequencing problems. Initiating problems occur as pauses, groping and restarts (Duffy, 2005, Ziegler, 2008). Poorly formed phonemes surface in distortions, where a phoneme is recognizable as the target phoneme, or in substitutions where change of features results in another phoneme (Den Ouden, 2004, Ziegler, 2008). Sequencing problems are reflected in the interchange of phonemes of syllables in an utterance (Square, et al, 1997, Haynes, Pindzola en Emerick, 1998, Ziegler, 2008).

Table 1 about here

Although AOS is often described as a specific type of apraxia in addition to the known types of limb apraxia, the categorization of AOS symptoms seems to parallel the classification of limb apraxia (Knollman-Porter, 2008). Limb apraxia can be divided into ideomotor, kinetic and ideational apraxia (Pearce, 2009). In ideomotor apraxia, the concept of the movement is intact, but cannot be converted into a motor program. The patient has difficulty to initiate movements on demand, while the same movements can often be performed as part of an automatic or emotional movement. In kinetic apraxia the initiated movement is correct, but the emphasis is on the clunky, not smooth execution of the movement. Ideational apraxia, finally, is a disorder in the sequence of movements. The movements stored in a kind of motor program are executed in the wrong order because they are not properly called.

AOS in which initiation problems are most salient seems to be like a form of ideomotor apraxia.

If initiation is facilitated by e.g. emotion or a cue, a correctly articulated word is often the result. This type of AOS seems to be an impairment in the access to the GMP. AOS in which poorly formed phonemes are most salient seems to be like a form of kinetic apraxia. The correct utterance is initiated, the correct phonemes are selected, but the articulatory movements to produce well-formed phonemes, consonant clusters or syllables are not executed correctly. The GMP of an articulation unit itself seems to be damaged.

Finally, AOS in which errors in sequencing are most salient seems to be like a form of ideational apraxia: the patient seems to have difficulty in choosing the correct order of the GMPs. Table 1 provides an overview of the suggested different types of AOS and the symptoms that occur with these types.

Final Remarks

In this paper we propose a division into three subtypes of AOS. We believe that this division provides a significant step forward towards a more specific and clinically useful diagnosis of AOS. We also believe the major impact on the therapy for AOS. The ideomotor AOS patients for example, need to regain access to the GMPs whereas kinetic AOS patients have to relearn the exact execution movement.

Currently we are working on a therapeutic instrument for AOS, amongst other things based on this new insight. We hope to present the development process of this instrument in the near future.

AOS types	Deficit	Symptoms
1. Ideomotor apraxia of speech	Deficit in the access to the GMP (initiating problems)	<ul style="list-style-type: none"> ➤ Visual and audible groping restarts ➤ Repetition of initial phonemes ➤ Hampered speech ➤ Decreased speech rate
2. Kinetic apraxia of speech	Damage to a GMP of an articulation unit itself	<ul style="list-style-type: none"> ➤ Distortions ➤ Substitutions ➤ Poor intelligibility
3. Ideational apraxia of speech	Errors in sequence of the GMPs	<ul style="list-style-type: none"> ➤ Interchange of phonemes of syllables in utterances

Table 1: suggested subdivision of AOS types

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