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Apraxia of Speech

SUMMARY

This article is devoted to pure apraxia of speech (AOS). It focuses on the characteristics of the speech disorder and the types of accompanying articulation errors. The paper presents the types of AOS, including the most recent reports on the mechanisms underlying the disorder. The author describes the relationship between AOS and oral apraxia, as well as elements of differential diagnosis between AOS, dysarthria and aphasia. The disorder is rarely or erroneously diagnosed in clinical practice due to its unrecognised mechanism and uniqueness caused by its selective nature, among other reasons. AOS and aphasia are usually concurrent disorders.

Key words: apraxia of speech, primary progressive apraxia of speech, primary progressive aphasia, aphasia, dysarthria

Apraxia of speech (AOS) is a type of motor speech disorder involving disturbance in motor speech planning or programming (Duffy 2013, 269–270). The pure form of AOS should be distinguished from dysarthria and aphasia (Staiger, Ziegler 2008, 1202). Although it was described in the second half of the 1960s by F.L. Darley (Josephs et al. 2012, 1523; Duffy 2013, 270), the concept first appeared in 1900, when H. Liepmann referred to a patient's problems with motor aphasia as the apraxia of the language muscles (*Apraxie der Sprachmuskeln*, as quoted in Ziegler, Staiger 2016, 987). In the early 20th century, the problem was also referred to as the apraxia of the glosso-labio-pharyngeal structures (Ogar et al. 2005, 427) or phonetic disintegration (Josephs et al. 2012, 1523). Today, the term "apraxia of speech" is widely known and used in clinical practice (Ogar et al. 2005, 428). The previously used terms "cortical dysarthria" and "aphemia" can be considered synonyms for AOS (Josephs et al. 2012, 1523). According to J.R. Duffy

(2013, 273), many more synonyms[1] were most likely used to refer to AOS (its pure form) or considered AOS to occur in other speech disorders (concurring disorders, such as aphasia). These include: articulatory dyspraxia, verbal apraxia, anarthria, afferent motor aphasia, apraxic dysarthria, efferent motor aphasia, expressive aphasia, pure motor aphasia, Broca's aphasia and subcortical motor aphasia.

1. THE CHARACTERISTICS

The distinguishing features of AOS are still debatable and some of them can also be found in the speech of people with aphasia or dysarthria. This is due to the rare occurrence of the pure form of AOS and its widespread co-occurrence with aphasia and dysarthria. Therefore, despite the existing list of articulatory characteristics helpful in the study of adult AOS (Dabul 2000; Wambaugh et al. 2006), researchers continue to discuss which symptoms are typical for AOS (Ogar et al. 2005, 431; Staiger et al. 2012, S1558).

Researchers consider the following to be the most important features of AOS: 1) articulatory errors (distortions and substitutions); 2) slowed speech rate; and 3) an unusual prosody (Wambaugh et al. 2006; Ogar et al. 2005, 428; Staiger et al. 2012, S1544). Among the articulatory errors in AOS are phonetic paraphasias[2], which are difficult to differentiate from errors occurring in dysarthria. However, features such as additions and substitutions rarely occur in the latter. In aphasia, there are phonemic paraphasias, but they are not perceptually distorted, as is the case in AOS. Therefore, in his typology of errors occurring in AOS, Duffy (2013, 278) uses the ubiquitous term "distortion". Its further differentiation allows him to distinguish errors, such as: distorted substitutions, distorted perseverative substitutions (e.g. "nanana" instead of "banana"), distorted anticipatory substitutions (e.g. "popado" instead of "potato"), distorted epentheses (added sounds), etc. As can be seen, there are different ways of describing errors in AOS as the mechanism of the disorder has not been sufficiently understood yet.

Among the basic features of AOS are rate and prosody disorders, although they are also considered to be secondary features in relation to articulatory difficulties, or even a form of compensation for these difficulties (Duffy 2013, 279). The rate of speech in patients with AOS is slower, regardless of articulation errors, especially for long utterances (more than one syllable in length). There is clear

[1] J. R. Duffy mentions as many as 25 synonyms (2013, 273).

[2] The phonetic, not phonological nature of errors in AOS has been confirmed by studies on voice onset time (VOT) in AOS. It is a functional tool for studying acoustic aspects of speech disorders, making it possible, among others, to determine if the errors are phonetic (involve deficits in temporal motor planning) or phonemic (involve phonological planning deficits) (Auzou et al. 2000, 146).

syllable segregation (statements are divided into syllables) and errors of stress assignment, resulting in monotonous intonation. Perception of a foreign accent may appear in monolingual patients. For example, some Polish-speaking patients with AOS (whom I diagnosed at the Department of Neurology and Cerebrovascular Disorders in Bierkowski Hospital in Poznań) gave the impression of speaking with an English accent or the accent of people from the Eastern Borderlands (patients did not have this origin). An outsider, a non-specialist, may consider such a patient to be a foreigner who has a different accent, speaks slowly and chants, i.e. looks for an articulatory posture.

The evident problems of patients with AOS also include fluency disorders[3], which take the form of silent or audible repetition of syllables or sounds, prolongation or visible and audible groping for articulatory postures (Duffy 2013, 279).

Many factors influence the character of apraxic utterances. Patients find it most difficult to articulate complex syllables (consisting of many phonemes), consonant clusters within syllables, rare syllables and words (frequency effect) and long words (consisting of many syllables). According to I. Aichert and W. Ziegler (2004, 154), numerous segmental errors appear even in utterances of patients with a mild form of AOS.

2. DIFFERENTIAL DIAGNOSIS

AOS should be differentiated from aphasia (Broca's and conduction aphasias) and dysarthria, although this is done rarely in clinical practice because AOS often concurs with these disorders. AOS reflects speech planning deficits at a higher level than dysarthria (Ziegler et al. 2012, S1487). It is also not synonymous with aphasia, but may accompany it, being the most common variant of the disorder in clinical practice. This article describes its isolated variant, i.e. pure AOS.

The lesion locus allows it to be distinguished from other motor speech disorders. It almost always affects the left hemisphere of the brain (frontal, parietal and subcortical lobes), accounting for more than 6.9% of people who have communication disorders (Duffy 2013, 269–270). Due to the lesion locus, patients with AOS may have deficits in the right side of the body, such as weakness and spasticity, sometimes apraxia of the limbs, consisting of the inability to perform deliberate movements of the limbs, which cannot be explained by motor disorders, hypertonia, as well as sensory and coordination disorders. These disorders, in turn, can affect both writing and non-verbal (gestures) communication skills (Duffy 2013, 272).

Non-verbal oral apraxia or buccofacial apraxia, which is not synonymous

[3] In non-fluent aphasia, fluency disorders include agrammatic speech, reduced length of utterance, i.e. speech deficits.

with AOS, should also be mentioned. It is an inability to imitate or perform volitional (arbitrary) movements of the articulatory muscles, including facial muscles, on demand (during coughing, blowing, smacking, clicking, whistling, etc.), which cannot be explained by comprehension disorders and sensory or motor deficits[4] and can be executed involuntarily (unconsciously) while eating (Drummond 2006, 115). The brain substrates of oral apraxia and AOS are not identical, although both disorders may, and do, concur. For example, in a study conducted by Duffy (2013, 275), oral apraxia was revealed in 77% of 92 patients[5] with AOS.

Table 1. Distribution of co-occurring aphasia, dysarthria, oral apraxia and AOS (based on Duffy 2013, 275–276)

AOS	+ Aphasia	+ Dysarthria	+ Oral apraxia
	65%	30%	77%

For comparison, in the same group of patients, aphasia occurred in 65% of participants in the experiment, meaning that aphasia did not occur in 35% of people with AOS. We know that both disorders (AOS and aphasia) can also co-occur. In turn, dysarthria was diagnosed in 30% of patients (see Table 1). Thus, only 4% of participants (exactly four patients) had pure AOS. Three of them suffered from neurodegenerative diseases suggesting that pure AOS is more common in degenerative diseases than in strokes and traumas. However, as far as the aetiology of AOS with aphasia is considered, it most often manifests itself as a result of strokes.

1.1. AOS and aphasia

AOS occurs regardless of disorders of language functions, such as: comprehension, reading, writing and naming. In contrast to aphasia, which takes the form of multimodal speech deficits, isolated AOS only concerns verbal expression (Duffy 2013, 269). Of course, it may, and does, concur with aphasia. Therefore, clinically we may deal with the primary form of AOS, i.e. with a deficit revealed as a result of specific aetiology, or with the secondary form of AOS, i.e. with disorders that occur after concurring aphasia subsides. This often leads to diagnostic errors, as a result of which AOS is qualified as dysarthria (due to the lack of difficulties with reading, writing, comprehension and naming). AOS and aphasia can also persistently co-occur. The similarity of aphasia and AOS may result in

[4] Patients with oral apraxia try to perform movements on command, but they do it awkwardly: they grope for correct movements with effort, commit errors and are often perplexed, frustrated, or amused by these difficulties. They sometimes repeat the command aloud and then try to execute it (Duffy 2013, 276).

[5] Patients were examined between 1999 and 2008 at Mayo Clinic.

qualifying the latter as motor or conduction aphasia (and less often as transcortical, sensory or anomic aphasia).

The frequent concurrence of both disorders and the rare occurrence of pure AOS make the differential diagnosis very difficult. It is also not facilitated by neuroanatomical characteristics[6] (frontal and left temporoparietal lobes supplied by the blood vessels of the left middle artery) and aetiological characteristics (predominance of strokes), which are not significantly different. Also, the types of occurring errors are not easy to differentiate (Duffy 2013, 366). In aphasia, there are mainly linguistic and phonological errors (in phonemic paraphasias). In AOS, errors result from disorders in motor speech planning and programming and are, therefore, phonetic because they take the form of substitutions, additions, transpositions, omissions, i.e. phonetic paraphrases. For this reason, the similarity of aphasia and AOS may result in confusing the latter with motor or conduction aphasia (and less often as transcortical, sensory or anomic aphasia). According to researchers, errors in conduction aphasia reflect a language deficit in the selection of the phonemes for speech. Apraxic speakers, on the other hand, can select the correct phonemes, but have trouble with their motor execution. Patients with conduction aphasia typically speak with near normal prosody, whereas halting, effortful speech with abnormal prosody is characteristic for AOS. It does not change the fact that both disorders are similar at the sound level of errors made (Ogar et al. 2005, 429).

Articulation, prosody and speech rate are distorted in patients with AOS. Syllables are clearly segregated and there are prolonged inter-word intervals. For comparison, rate and prosody are normal in fluent aphasias, such as sensory aphasia. People with AOS articulate substitutions and distorted words with effort and hesitation. There are no speech distortions and phonemes are specific to a given language in aphasic utterances, which are also articulated with hesitation and effort. Patients with AOS are aware of articulation errors and try to correct them in contrast to patients with aphasia without AOS, who less frequently correct phonological errors. According to researchers, apraxic errors are closer to articulatory postures than phonological errors made by people with aphasia without AOS. Frequent repetitions in AOS also result in less variability in the structure of words obtained compared to aphasic utterances (Duffy 2013, 367).

In general, when differentiating aphasia and AOS, it is helpful to assess language functions other than motor ones because there are no disorders of comprehension, reading, writing and naming in pure AOS. Therefore, the comparison of the quality of spoken and written language is very helpful in the differential

[6] Pure AOS "is more often associated with posterior frontal or insular lesions than with lesions in the temporal or parietal lobes, whereas aphasia without AOS tends more often to be associated with temporal or temporoparietal lesions" (Duffy 2013, 367).

diagnosis of AOS and aphasia (Polanowska, Pietrzyk-Krawczyk 2016, 501).

As the specifics of AOS and aphasia are noticeable, it is not surprising that forms of therapeutic impact should also take account of this aspect. Researchers suggest that therapy facilitating the communication of people with aphasia without AOS is not effective in AOS treatment and, vice versa, therapy for AOS is not effective in the treatment of aphasic disorders without AOS (Duffy 2013, 367).

1.2. AOS and dysarthria

As shown above, AOS often co-occurs with aphasia. Dysarthria, in turn, rarely concurs with aphasia. This does not change the fact that dysarthria may co-occur with AOS. Most often, AOS can be confused with its ataxic, hyperkinetic and spastic variants. Diagnostic difficulties also increase if we want to confirm the co-occurrence of AOS and dysarthria. The similarity of AOS and dysarthria is related to the fact that patients with AOS have weak facial and lingual muscles on the right side. Both types of disorders have a similar aetiology. AOS often occurs as a result of haemorrhagic strokes, which are also the cause of dysarthria. Both disorders occur in neurodegenerative diseases, but AOS does not occur in Parkinson's disease or in multiple system atrophy. Dysarthrias occur more frequently due to subcortical than cortical lesions, and AOS is more often caused by cortical damage (Duffy 2013, 363–364).

In contrast to dysarthria, AOS is not associated with functional disorders of the articulatory muscles (Duffy 2013, 269). Patients do not have paralysis or paresis of these muscles and do not show problems with non-language skills such as: chewing, swallowing and coughing (Polanowska, Pietrzyk-Krawczyk 2016, 500). The speech disorder cannot be explained by the possible occurrence of weakened lingual or facial muscles and is inadequate to the degree of reduction in muscle tone and the motility of the articulators. Therefore, utterances in AOS (during repetitions) are inconsistently distorted and may be periodically similar to or even convergent with the articulatory posture related to a word sought, and after a while they become very disturbed. The motor deficit results here from disturbed planning or programming, and not from the performance capabilities of the muscle apparatus. Automatic speech is a bit more efficient in AOS. The quality of speech is affected by the frequency, length and semantic features of words (Duffy 2013, 365). Dysarthric speech is uniformly disturbed, utterances correspond to a patient's current articulation possibilities, such as tongue, palate or lip paresis. Trial and error groping and attempts at self-correction are common in AOS, but dysarthric speakers rarely grope for the correct articulatory postures or attempt to correct errors (Ziegler, Staiger 2016, 988). To some extent, they also differ in the types of errors made. In dysarthria, either simplifications or distortions of speech sounds are observed. Errors occurring in AOS, such as distortions, substitutions, additions, repetitions and prolongations, are similar to aphasic errors. Errors in

AOS are very diverse[7], irregular and unpredictable[8] (Staiger et al. 2012, S1544; Hickok 2012, 139; Ziegler, Staiger 2016, 988). They are observed especially in utterances that have not been previously trained or made. This phenomenon testifies to the instability of particular articulatory systems rather than an inability to articulate sounds (Staiger et al. 2012, S1545, S1558).

Differential diagnosis can be facilitated by the assessment of oral apraxia, which may co-occur with AOS, but is rare in the case of dysarthria. Furthermore, the scale of disorders in dysarthria is much greater because it affects all elements of speech (breathing, phonation, articulation, prosody), whereas AOS is mainly associated with articulatory and prosodic disorders.

AOS is most often differentiated from ataxic, spastic and hyperkinetic dysarthrias (Duffy 2013, 365). Diagnosis is the most difficult in the first type of dysarthria because it is also characterised by articulatory and prosodic disorders. In the case of articulatory deficits in ataxic dysarthria, substitutions are not frequent, but articulatory irregularities are significantly more common. Indicators taking account of oral diadochokinesis can help differentiate both disorders. Alternating motion rates are normal in AOS, which is not the case in ataxic dysarthria (e.g. repeating /pʌ/, /tʌ/, /kʌ/ at a rate of 5 to 7 repetitions per second, Duffy 2013, 81). Some problems occurring in AOS, however, are not present in ataxic dysarthria. These include distorted sequential motion rates when repeating a syllable sequence (e.g. /pʌtʌkʌ/), which reflect possible speech planning or programming deficits. Moreover, the invariability of disorders in automatic and propositional speech is characteristic for ataxic dysarthria rather than for AOS, in which automatic speech is better than propositional speech.

It is somewhat easier to make a differential diagnosis of hyperkinetic and spastic dysarthrias. In the first type, there are visible involuntary movements that are absent in AOS. In contrast to spastic dysarthria, dysphonia and hypernasality (Ziegler, Staiger 2016, 988), as well as dysphagia, drooling and pseudobulbar affect do not occur in AOS (Duffy 2013, 365).

3. TYPES OF AOS

According to Duffy (2013, 274), certain subtypes of AOS can be distinguished, but the differentiation is not obvious and is unambiguous due to different criteria used. For example, the researcher claims that one of these subtypes may

[7] Researchers disagree about this distinctive feature in the diagnosis of AOS (see literature on this topic and Table 1 in: Staiger et al. 2012, S1545; Duffy 2013, 283–284).

[8] Error variability can be understood as: 1) inconsistency of error occurrence in the same utterances during repetition; 2) inconsistency of error type occurring in the same utterances in repetition tasks.

be AOS co-occurring with Wernicke's or conduction aphasia, where phonological deficits are observed. The second subtype may include dysarthria co-occurring with AOS. However, this condition is referred to as AOS co-occurring either with aphasia or with dysarthria rather than as pure AOS.

Other researchers take account of anatomical correlates associated with the occurrence of AOS. Due to different lesion loci, they come to the conclusion that there is probably more than one type of this disorder. Therefore, it should be considered a heterogeneous disorder, enabling distinction between the subtype with lesions in the left parietal lobe and the more common subtype with lesions in the left frontal lobe. Speech disorders associated with the so-called parietal AOS involve groping for initiation and within utterances, numerous off-target approximations of phonemes and occasional syllable segregation. Temporoparietal patients with AOS produce a greater percentage of polysyllabic sequencing errors and a smaller percentage of monosyllabic articulation errors than frontal patients (Ogar et al. 2005, 430). The suggested typology requires further research, especially since anatomical correlates related to the occurrence of AOS are not limited to the mentioned structures. Studies indicate a correlation between AOS and lesions in the temporoparietal and frontal lobes (posterior left superior frontal gyrus) and the subcortical structures, such as left insular cortex and basal ganglia (Ballard et al. 2014, 4).

The authors of another typology (Feiken, Jonkers 2012, 2) have noticed that the symptoms of AOS described in the literature can be assigned to three subtypes characterised by slightly different deficits, such as: 1) difficulty initiating utterances resulting in pauses, repetitions and groping for articulatory postures; 2) difficulty forming phonemes resulting in distortions or substitutions; and 3) difficulty sequencing or ordering a series of phonemes resulting in the exchange of phonemes within syllables. In their opinion, each of the described characteristics is observed in ideomotor, kinetic and ideational apraxia, respectively (see Table 2).

This typology refers directly, and is parallel, to the classification of limb apraxia. Ideomotor limb apraxia involves difficulty turning a plan for intended movement into a motor programme. Consequently, patients are unable to initiate this movement on demand, yet they can perform it as an automatic motor action. In the kinetic type of limb apraxia, an initiated movement is correct, but it is non-fluent and difficult to perform. The ideational subtype, in turn, involves an inability to execute complex sequences of motor actions.

These three subtypes of AOS co-occur and overlap very often. Symptoms of each of them are usually observed in patients (Polanowska, Pietrzyk-Krawczyk 2016, 501).

Table 2. Subtypes of AOS according to Feiken and Jonkers (2012, 3)

Subtypes of AOS	Disorders	Symptoms
Ideomotor	Difficulty initiating utterances (deficits in the access to the motor programme).	Repeated attempts to grope for articulatory postures (visual and audible groping for a particular sound). Repeating initial phonemes. Hampered speech. Slower speech rate.
Kinetic	Deficits in the scope of the motor programme for articulation.	Distortions, substitutions, poorly understandable utterances.
Ideational	Errors in sequence of the motor programme.	Replacing phonemes in the syllables that make up the speech.

4. MECHANISM

Contemporary researchers most often[9] describe AOS from three perspectives, which are also a manifestation of different approaches to this phenomenon. The first one is a disconnection of phonology from motor execution. The second is a disturbance of learned motor routines. The third is an incoordination of spatially and temporally patterned speech movements (Ziegler et al. 2012, S1497–S1498).

The most popular theories describing the basics of AOS include those indicating planning or programming disturbance, which involves the structures of the left hemisphere of the brain, especially the frontal and parietal lobes and associated subcortical structures (Duffy 2013, 270). Importantly, it is still discussed whether AOS results from a disorder of speech motor programming or speech motor planning (Feiken, Jonkers 2012, 1). A speech act (see Figure 1) involves two stages. The first is represented by the abstract form of a word that is not yet phonologically formed (the so-called lemma). It can be observed in the tip-of-the-tongue state (TOT syndrome). If we fail to retrieve a word from memory, we know what word we are looking for and sometimes we can give some information about its structure, but we are unable to find its phonological form. This is the second stage known as phonological. The conceptual and articulatory systems are somehow outside the speech production process (cf. Hickok 2012, 137).

Researchers suggest that AOS patients know what they want to say and how it should sound, but cannot translate it into appropriate articulatory movements (aimed at speech production). A disconnection of phonology from motor execution is, therefore, suggested. In the early 20th century, H. Liepmann (as quoted in Ziegler et al. 2012, S1487) described this phenomenon as parallel to ideoki-

[9] The characteristics of other theories regarding the mechanism of AOS can be found in Ballard et al. (2014, 1).

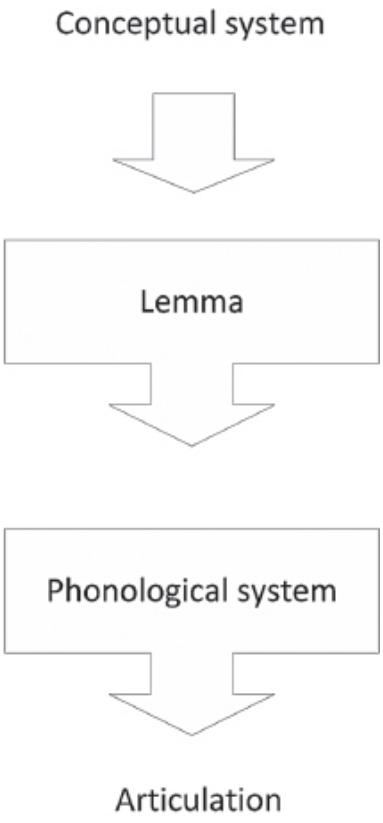

Figure 1. Two-stage speech production model (Levelt 1989; Levelt et al. 1999)

netic apraxia. In his opinion, AOS patients, like those with limb apraxia, have a basic functional motor system and access to more abstract, e.g. phonological, speech representations. According to the researcher, these two stages are disconnected in AOS.

The latest approaches consider how the auditory-based (phonological) representations of words are transformed into articulatory postures. For example, a dual-stream model of speech processing (Hickok, Poeppel 2007, 395) consists of two streams: ventral and dorsal, i.e. auditory and motor (Hickok 2012). In the model, a dorsal stream maps acoustic speech signals to frontal lobe articulatory networks (around the posterior end of the Sylvian fissure at the temporoparietal junction)[10], i.e. transforms sound forms into articulatory movements. In turn, a ventral stream processes speech signals for comprehension and is, thus, of a semantic nature.

The function of the auditory dorsal stream is the so-called sensory-motor integration or, more precisely, auditory-motor function, which occurs at the temporoparietal junction. This is where articulation gestures planned in the auditory space are transformed into their motor representations, i.e. acoustic speech signals are mapped to their motor representations. This is confirmed by deficits characteristic of conduction aphasia, in which patients retain their comprehension abilities; they are fairly fluent, but manifest repetition, naming and loud-reading disturbance due to persistent phonemic paraphasia (Hickok, Poeppel 2004, 93). In the hierarchical state feedback control model developed by G. Hickok (2012, 139–140), these two disorders (AOS and conduction aphasia) affect the same level of hierarchical motor control but in different components of the circuit: AOS affects access to motor phonological codes and conduction aphasia affects internal state feedback control.

[10] Due to its damage, patients show deficits characteristic for conduction aphasia with numerous phonemic paraphasias. The differential diagnosis between AOS and conduction aphasia is not easy. In AOS, speech is more effortful, often interrupted (Hickok 2012, 139).

Descriptions of the AOS mechanism also take account of a disturbance of learned motor routines, i.e. "mental images" of how an intended word, or – according to other researchers – syllables (a mental syllabary, i.e. a memory store comprising the phonetic plans of the most frequently occurring syllables in a speaker's language) should be articulated. In this approach, AOS results from a deconstruction of stored procedural knowledge of how particular syllables are articulated (Aichert, Ziegler 2004, 156; Staiger, Ziegler 2008, 1202; Ziegler et al. 2012, S1488).

Researchers also point to an incoordination (ataxia) of spatially and temporally patterned speech movements as the source of AOS. Skills in patterned speech movements are acquired (exercised, preserved) in childhood and adolescence (Ziegler et al. 2012, S1497–S1498).

5. AETIOLOGY

As Duffy (2013, 272) indicates, AOS can be caused by any process that damages the function of the dominant hemisphere in the field of speech planning or programming. AOS most often occurs in neurodegenerative diseases and strokes, accounting for over 80% of occurrences (Duffy 2013, 274).

Table 3. Distribution of disorders in which AOS occurs as a primary deficit in percentage points (based on Duffy 2013, 274)

Aetiology	Type of disorders	Percentage points
Neurodegenerative diseases	primary progressive aphasia (PPA), primary progressive apraxia of speech (PPAOS), cortico-basal dementia (CBD), amyotrophic lateral sclerosis (ALS), progressive supranuclear palsy (PSP), degenerative diseases of the central nervous system	54%
Vascular diseases	strokes, arteriovenous malformations (developmental defects), undetermined vascular mechanism	28%
Cancer	tumours within the left hemisphere of the brain, always involving the frontal lobe	5%
Traumas	neurosurgical interventions within the left frontal lobe	3%
Other	unspecified aetiology, epilepsy, demyelinating diseases, liver transplants, developmental disorders	10%

AOS observed in neurodegenerative diseases is progressive (see Table 3). It is known as primary progressive apraxia of speech (PPAOS) (Josephs et al. 2012, 1524).

In the differential diagnosis of primary progressive aphasia (PPA) developed by R. Vandenberghe (2016, 5), the agrammatic/non-fluent variant of PPA is treated as an isolated disorder (Josephs et al. 2012, 1531; Silveri et al. 2014, 58; Vandenberghe 2016, 5). Thus, it occurs as 1) an isolated form of non-fluent PPA accompanied by 2) an isolated form of agrammatism and 3) a mixed variant taking the form of agrammatism and AOS (Figure 2). This PPA typology clearly suggests that PPAOS is a separate entity, although it often co-occurs with aphasia in neurodegenerative diseases (Josephs et al. 2012, 1523; Silveri et al. 2014, 58), corresponding to the subtype known as agrammatism and AOS. A patient may, therefore, be diagnosed with: PPA, PPAOS or PPA and PPAOS (Duffy 2013, 275).

According to K.A. Josephs et al. (2012, 1524–1525), PPAOS is difficult to distinguish for two reasons. Firstly, PPAOS is difficult to differentiate from aphasia and is often treated as PPA. Secondly, even if diagnosed, AOS is eventually classified as aphasia or dysarthria in clinical practice (Josephs et al. 2012, 1523–1524; Josephs et al. 2006, 1395). Because PPAOS is a progressive deficit, aphasia may occur due to disease progression. It should be remembered, however, that the literature contains descriptions of patients in whom the isolated form (without accompanying dysfunctions) persisted for 8–10 years (Gerstner et al. 2007, 15).

In summary, PPAOS is considered to be an isolated deficit, although some researchers include it in the agrammatic variant of PPA and treat it as one of its subtypes. Similar to the non-fluent variant of PPA, PPAOS belongs to mainly neurodegenerative frontal syndromes, in which executive dysfunctions are revealed. Neither spatial nor visual perceptual problems have been observed in patients with these disorders (Josephs et al. 2012, 1533).

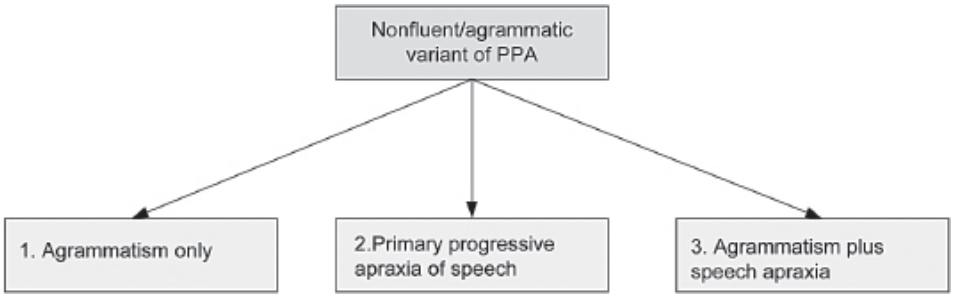

Figure 2. Subtypes of agrammatic (non-fluent) PPA according to Vandenberghe (2016, 7; Figure 5)

Despite these similarities, neuroanatomical patterns of grey and white matter loss are different to patterns described in non-fluent PPA patients (Josephs et al. 2012, 1531; Whitwell et al. 2013, 251), which differentiates it from this type of disorder. In AOS, the loss occurs in the posterior superior frontal lobe and supplementary motor area; in PPAOS, atrophy affects the pre-motor cortex (lateral) and supplementary motor area; and in non-fluent PPA patients, it affects Broca's area or the posterior inferior frontal lobe.

In addition to neurodegenerative diseases, strokes are among the common causes of AOS (28% of patients), observed in the acute phase of stroke in hospitals. This condition is known as AOS. Less commonly, AOS manifests itself as a result of tumours and injuries of the left frontal lobe (a total of 8%). Demyelinating diseases (including multiple sclerosis) sporadically lead to AOS. It also rarely occurs as a result of metabolic and inflammatory diseases (Duffy 2013, 275, 272).

6. CONCLUSIONS

AOS belongs to those motor speech disorders that are particularly interesting to researchers due to its uniqueness and a kind of scientific elusiveness, among other reasons. Many clinicians publish case studies in this area, others deal with a broader range of issues regarding neuroanatomical correlates, typology, terminology, models explaining the specifics of AOS and its therapies.

Due to the unrecognised mechanism of this disorder, various diagnostic guidelines can be found in the literature (see e.g. differentiation of errors), slightly different definitions indicating a distorted ability to coordinate sequential, articulatory movements needed for speech production (Wertz et al. 1984 as quoted in Ogar et al. 2005, 427), articulatory disorders resulting from disturbance in programming and sequencing of articulatory muscle movements (Darley and Aronson 1975 as quoted in Ogar et al. 2005, 428), as well as speech programming or planning disturbance, etc. (Duffy 2013).

It is worth noting that AOS, both due to vascular events and neurodegeneration, is a rare deficit (Duffy 2013, 269) and is often confused with aphasia (or PPA) and dysarthria, with which it most often co-occurs. Due to the lack of accompanying comprehension, reading and writing disorders, it is diagnosed as dysarthria. In turn, due to the absence of paresis or paralysis of the articulatory muscles, it is classified as motor aphasia. To emphasise its isolated form, i.e. independent of deficits typical of aphasia and dysarthria, it is often called pure AOS. The causes of AOS may be vascular disorders but also injuries and cancers. It very often appears as the first syndrome of neurodegenerative diseases such as non-fluent PPA or CBD (Ogar et al. 2005, 427).

AOS requires special attention due to its specificity and the fact that it rarely occurs in its pure form. Its clinical manifestations are often unnoticed, affecting the quality of therapies proposed to patients.

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