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Case report

Post-stroke pure apraxia of speech – A rare experience

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ABSTRACT

Apraxia of speech (AOS) is a motor speech disorder, most typically caused by stroke, which in its "pure" form (without other speech-language deficits) is very rare in clinical practice. Because some observable characteristics of AOS overlap with more common verbal communication neurologic syndromes (i.e. aphasia, dysarthria) distinguishing them may be difficult. The present study describes AOS in a 49-year-old right-handed male after left-hemispheric stroke. Analysis of his articulatory and prosodic abnormalities in the context of intact communicative abilities as well as description of symptoms dynamics over time provides valuable information for clinical diagnosis of this specific disorder and prognosis for its recovery. This in turn is the basis for the selection of appropriate rehabilitative interventions.

1. Introduction

In 1969, Fred Darley argued for distinguishing the term "apraxia" from other verbal communication neurologic disorders (aphasia, dysarthria) due to the observation of brain-damaged patients with impairment restricted to specific speech subsystems not crossing other communication modalities [1]. This impairment, called acquired apraxia of speech (AOS), is usually defined as a "disorder of learned volitional actions associated with breakdown in planning or programming movements needed for speech" [2]. It is assumed that problems in sequencing the spatiotemporal and force aspects of movement (muscle tone, resistance, and absolute force, direction, speed, range, and rate of motion) distort the positioning of the speech musculature in a coordinated and well-timed manner for volitional speech production [2,3]. Therefore AOS is predominantly a disorder of articulation and prosody (tune and rhythm), manifested especially in actions requiring volitional control of speech [2–4].

AOS most frequently emerges as a consequence of a stroke (93%) [5], although it is increasingly recognized in neurodegenerative diseases (e.g. motor neuron disease), and may be also a result of tumors or a trauma [6]. Pinpointing a singular structural pathology underlying AOS remains controversial. The majority of focal lesions were reported within the vasculature of left middle cerebral artery with the most common damage to the left posterior inferior frontal gyrus (inter alia to posterior Broca's area and adjacent cortex), precentral gyrus of the anterior insula, other frontal and

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temporoparietal cortex, as well as to left subcortical structures, particularly the basal ganglia [6,7].

There is limited information about the prevalence of AOS. This is partly due to problems in definition and delineation of AOS from more common speech and language disorders. In Duffy's study [8], AOS was the main cause of communication problems in 7.6% of patients with acquired neurologic motor speech disorders. However, AOS frequently co-occurs with non-fluent aphasia (linguistic disorder with agrammatism and naming difficulties which in a severe form is almost always accompanied by AOS) or overlaps with conduction aphasia (linguistic deficits with phonemic paraphasias and repetition difficulties) or dysarthria (neuromuscular disorders affecting execution of oromotor and speech functions).

In addition to accompanying communication disorders, post-stroke AOS may co-occur with contralesional hemiparesis, sensory deficits and other forms of apraxia. Among the latter, oral apraxia (sometimes called nonverbal apraxia) happens quite frequently. It affects voluntary but non-verbal movements of the vocal tract (larynx, lips, tongue, palate) either on command or during imitation, despite the appropriate strength and range of motion. Examples of tasks are: to protrude tongue, the tongue raised to the top teeth, the lips round [1].

AOS very rarely occurs in a ''pure'' form (without coexisting aphasia or dysarthria), which provides an understanding of its symptoms. Isolated AOS is characterized by slow rate of speech with a tendency to break words into individual syllables [4]. The most common abnormalities are: (1) trial-and-error groping of articulatory movement with attempts to self-correct, (2) persistent dysprosody in which there are no extended periods of normal rhythm, stress and intonation, (3) articulatory inconsistency on repeated productions of the same words, (4) difficulty initiating utterances [9], and (5) typical self-awareness of speech problems [1–3].

The present study aims at describing AOS symptoms and their dynamics in a patient with no other difficulties in speech and language abilities. Prospective characteristics of AOS provide valuable information for clinical diagnosis of this rare disorders and prognosis for recovery from it.

2. Case report

2.1. *Patient's demographic and clinical characteristics*

A 49-year-old right-handed man, native Polish speaker with secondary education (he worked as a professional driver), was hospitalized after having experienced sudden inability to speak and right-sided facial asymmetry. His medical history revealed a hypertension, hyperthyroidism (Graves-Basedow disease), transient ischemic attack with temporary paresis of the right upper limb five years earlier, inferior wall myocardial infarction seven years earlier, and chronic nicotine addiction. He had negative histories of psychological disorders and neurological conditions other than stroke. No prior speech or language disturbance was reported.

On admission, the patient was conscious, could not speak and even emit voice but his non-verbal responses on the yes–no questions (e.g. relating to taken medications and their doses) were quick and correct. He had minor right central facial paresis (the National Institutes of Health Stroke Scale – NIHSS for facial nerve item was scored 1) and mild right arm weakness (NIHSS motor arm item was scored 0). ECG examination revealed supraventricular tachycardia, which passed in atrial flutter after antiarrhythmic treatment and 24 h later stabilized as sinus rhythm. A CT revealed no fresh brain pathology (hypodensity or hemorrhage), apart from a small old lesion in the right occipital lobe.

The patient was treated with intravenous thrombolysis with no significant change in the clinical picture.

CT re-examination of the brain, performed on the second day of hospitalization, showed an area of vascular ischemic damage localized within the left frontal lobe, near fronto-parietal junction (Fig. 1). NMR performed a week from onset indicated a mild hemorrhagic transformation of the left hemispheric infarct located on the boundary of the left frontal and parietal lobes (Fig. 2).

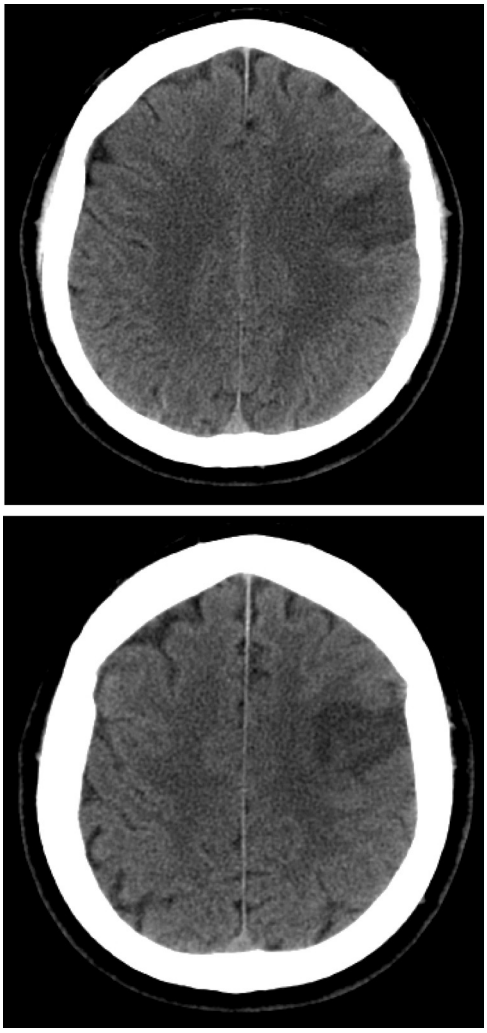

Fig. 1 – CT shows left hemispheric damage located within the frontal lobe, near fronto-parietal junction.

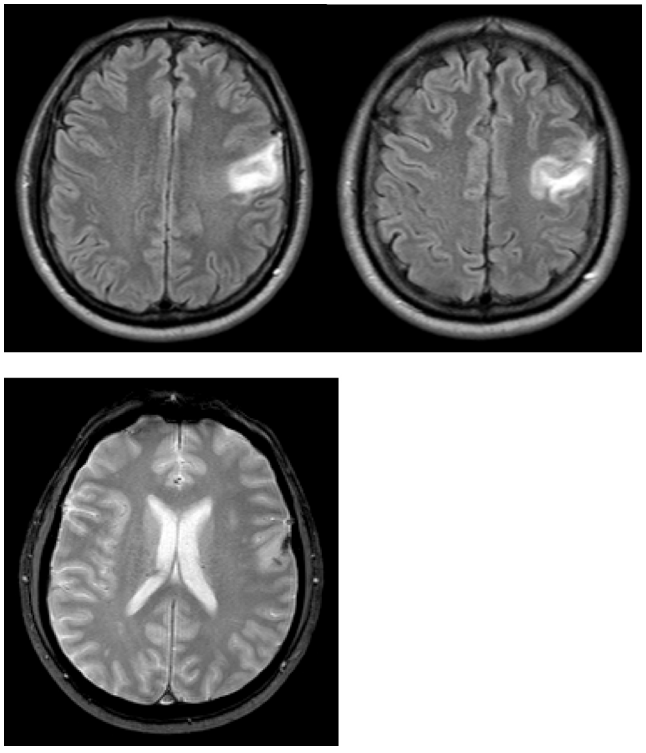

Fig. 2 – NMR shows mild hemorrhagic transformation of the left hemispheric infarct located on the boundary of the left frontal and parietal lobes.

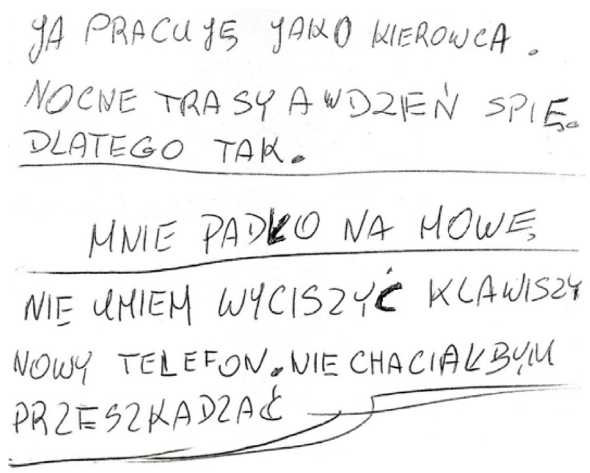

Fig. 3 – Written communication with another patient on the first day of hospitalization (in English: "I work as a driver. Night trips and I sleep during the day. That is why". "It affects my speech". "I cannot mute the keys. A new phone. I don't want to disturb").

2.2. *Speech and language skills, their dynamics and therapy*

For the first three days of staying in the intensive care unit, the patient was unable to say anything, although his vocal folds could function well for reflexive cough or laughter. All his attempts to speak failed and led to nervousness and anger at himself. At the same time, he spontaneously used written communication by briefly and quickly (therefore, sometimes quite casually) answering questions (Fig. 3). He was also able to read fast, understand written information and perform complex letter-sound analysis of presented words. His speech comprehension was unimpaired (e.g. good performance of the Revised Token Test [10] which requires processing of complex commands containing abstract concepts).

These disparities in forms of verbal communication were the basis for further evaluation symptoms, their determinants and dynamics, and also for choosing the proper form of therapy.

The examination started as soon as the patient began to speak single words, which allowed for an assessment of all communication modalities. For a differential diagnosis and monitoring of functional changes with the passage of time from stroke, the patient was tested three times: one week, one month and three months since stroke with the use of three following tests: the second edition of Frenchay Dysarthria Assessment – FDA-2 [11], the short form of the Boston Diagnostic Aphasia Examination – BDAE [12,13], and the second edition of Apraxia Battery for Adults – ABA-2 [14]. These tests allow to assess different aspects of verbal communication behaviors: articulatory and prosodic features of speech, linguistic abilities analyzed in tasks requiring speaking (e.g. formulating and repetition of utterances, reading aloud) or not (comprehension of speech, written language, silent reading with understanding), as well as movements of articulators in non-speech tasks. To collect quantitative data from all tests, FDA-2 descriptive scale ranging from "a" (normal function) to "e" (no function) was converted into a numerical scale where "e" corresponded to 0 points and "a" corresponded to 4 points.

Between the first and the fourth week after stroke (the period between the first and the second measurement), the patient participated in speech therapy aimed at enhancing natural processes of spontaneous recovery and speech relearning, as well as in psychoeducation and emotional support sessions.

One year after the stroke, the quality of patient's speech was assessed clinically during open conversation about his problems with speaking in everyday life and at work.

2.2.1. *Examination one week post-stroke*

In FDA-2 we noted slow articulatory movements and numerous speech errors. There was also a slight right-sided asymmetry of lips with occasional air leakage. Apart from these symptoms, no neuromuscular disorders were detected that could prevent vocalization and articulation (correct reflexes, swallowing, breathing, tongue mobility, agility and efficiency of the soft palate evaluated in non-verbal trials, lip seal enough for articulation, correct quality and phonation length).

In BDAE, the patient correctly performed all tasks of auditory comprehension, silent reading and understanding of written language, as well as various tasks evaluating writing (word-picture matching, writing under dictation, formulation of free written narrative; Fig. 4 shows patient's written

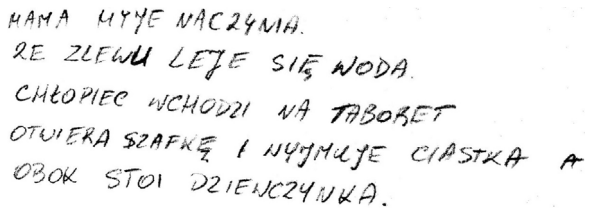

Fig. 4 – Patient's description of the "cookie-theft" picture from the BDAE test one week after stroke (in English: "The mum is washing dishes. Water is sloping from the sink. The boy is climbing on a stool. He is opening the cabinet and taking out cookies and the girl is next to him").

description of a story-picture). However he had serious problems with tasks requiring the spoken language. In these tests, his speech production accuracy was slightly higher if implemented utterances were rather automated (e.g. producing numerical sequence) than volitional (producing and repetition of words and sentences, naming of visually presented objects, reading aloud) but generally distorted.

ABA-2 showed the correct performance of subtests designed to detect limb apraxia and oral apraxia. Severe abnormalities were detected in all trials focused on different types of speech behaviors (oral diadochokinesis, ability to sequence the correct numbers of syllables in the proper order, change in articulation over successive trials, automatic speech, spontaneous speech and reading). The patient had problems with initiating (increased latency) and fluent maintenance of utterances (prolonged performance of words, abnormally long vowels and consonants, inter- and intraword pauses). Among numerous articulatory errors, the most frequent were: distortions and omissions with effortful trial-and-error groping of movements to achieve verbal targets. On the other hand, perseverations were occasional.

2.2.2. *Early speech therapy*

After the first examination completion, the patient began a three-week speech therapy. It was conducted by a professional speech therapist (I P-K) and comprised 15 (five times a week) 30-min sessions of progressive articulatory and prosodic exercises. They included imitation the movements necessary for vocalization of different speech sounds, repetition of sound, words, and short phrases supplemented by tactile and visual cues, as well as practicing utterances with exaggerated intonation of speech. Diversity of speech exercises performed during therapy allowed us to notice that the imitative and reading aloud accuracy was better than spontaneous accuracy and that the patient benefited from watching and listening to models of practiced utterances.

2.2.3. *Examination one month post-stroke*

One month since onset, apart from a slight right-sided asymmetry of lips the general neuromuscular condition of patient's vocal-speech apparatus was enough and a voice quality was normal (FDA-2). Although his speech was seriously dysprosodic (low speech rate with prolongation of word segments and inappropriate pauses, syllabication, reduced sentences stress patterns), he significantly recovered in terms of articulation. ABA-2 showed improvement in the ability to initiate and perform utterances (lower average time to initiate words and time needed to produce a set of words in ABA-2). FDA-2, ABA-2, and BDAE subtests showed sufficient speech intelligibility (near-normal articulatory precision of conventional stereotypical phrases and automatic speech, repetition and reading aloud of single words, occasional errors in spontaneous speech and repetition of sentences). At this stage we noted predominantly substitutions errors, and transpositions of sounds or syllables, which occurred in the long and/or phonetically complex utterances, as well as occasional trial-and-error behavior (ABA-2).

2.2.4. *Examination three months post-stroke*

Three months post-stroke, the patient still exhibited slightly lower right corner of the lips (FDA-2), but the quality of his speech was much better. In fact, abnormalities concerned mainly speech prosody. He spoke with reduced stress patterning for words and sentences, sometimes monotonously and with tendency to divide words into syllables. He committed a few minor articulatory errors in long words (FDA-2, ABA-2, BDAE). In his own opinion, speech difficulties appeared mainly when he was trying to speak quickly or when he was agitated. Slow and syllabic speech helped him avoid them.

Table 1 summarizes the results of speech and language tests administered one week, one month and three months after stroke. Individual subtests were categorized with respect to whether their execution required speaking (production and repetition of utterances, reading aloud) or not (non-verbal responses, silent reading, writing).

2.2.5. *One year post-stroke*

During the interview with the patient, conducted one year after the stroke, we observed signs of inappropriate prosody, but they were less severe than in the previous assessment. The disturbances included slight articulatory prolongation and reduction of linguistic and emotional stress patterns resembling speech of foreigner. In patient's opinion, his speech did not change significantly since that time.

3. Discussion

The study presents a case of a right-handed patient with AOS which was accompanied by no other speech-language deficits or apraxic signs in non-speech tasks. Isolated deficits of selective components of speech following stroke in the left (language dominant) hemisphere are very rare in clinical practice but also very informative. Such configuration of symptoms is possible because the oral movements (either speech or non-speech) and language functions are controlled by separate but cooperating systems [3].

In our patient AOS manifested itself by an apraxic mutism (lasting for a few days) and then by various articulatory and prosodic abnormalities. Typically for AOS, the patient exhibited greater ability to produce accurate automatic-reactive and well-practiced speech than volitional-purposive speech. In terms of the latter, repetition and reading aloud prompted by additional visual cues was better than self-generated speech.

Table 1 – The results of speech and language tests conducted one week, one month and three months post-stroke.

Test		1 week post-stroke	1 month post-stroke	3 months post-stroke
Subtests not requiring speaking				
FDA-2	Max. score			
Reflexes: cough, swallow, dribble/drool	12	12	12	12
Respiration at rest	4	4	4	4
Lips: at rest, spread, seal	12	10	10	10
Palate: fluids, maintenance	8	8	8	8
Laryngeal: time of phonation	4	4	4	4
Tongue: at rest, protrusion, elevation, lateral	16	16	16	16
BDAE – short form	Max. score			
Auditory comprehension	63	63	63	63
Understanding written language – silent reading	19	19	19	19
Writing	83	83	83	83
ABA-2	Normal scores			
Limb apraxia and oral apraxia	88–100	100	100	100
Subtests requiring speaking				
FDA-2	Max. score			
Respiration in speech	4	0	3	4
Lips: alternate (repeating "oo ee"), in speech	8	6	6	6
Palate in speech	4	3	3	4
Laryngeal: volume (counting to 5), pitch (singing a scale), in speech	12	6	9	12
Tongue: alternate (repeating "ka la"), in speech	8	4	5	6
Intelligibility: words, sentences, conversation	12	3	6	11
BDAE – short form	Max. score			
Oral expression	89	46	86	89
Understanding written language – reading aloud	21	12	20	21
ABA-2	Normal scores			
Diadochokinetic rate	26+	2	16	24
Increasing word length	0–1 s	5	2	1
Total time of words' pronunciation (s)	–	90	19.7	1188
Latency time for polysyllabic words	0–15 s	70	20	12
Repeated trials	28–30	11	14	27
Level of AOS impairments according to the ABA-2	High: ≥5	14	11	4

While distortions and omissions were relatively the most frequent in initial phase of AOS, along with the improvement of intelligible speech, substitutions and transpositions have become more noticeable. Such fluctuations of articulation characteristics correspond with the description of speech along a continuum of AOS, although – as studies have already emphasized – acoustic differentiation of errors can be difficult [6] (e.g. distortions may be perceived as substitutions, distorted sound substitutions are possible) [1]. Our patient's speech was effortful, slow, and disprosodic (reduced stress pattering and intonation, inappropriate pauses), which is another hallmark of the AOS. However, some prosody abnormalities (e.g. slow pace and syllabication) may be explained in part by compensatory efforts of poor articulation and difficulty with achieving specific movement patterns [1,2,7].

Because some characteristics of AOS overlap with other verbal communication disorders, a misdiagnosis of aphasia (especially motor or conduction aphasia) or dysarthria (e.g. spastic dysarthria) is possible. However, detailed analysis of symptoms in the context of intact communicative abilities and cognitive factors allows to differentiate between these syndromes.

In differential diagnosis of AOS and aphasia focusing on sound abnormalities can be especially confusing. It is because perceptual decisions whether errors are linguistic (phonemic/literal paraphasias) due to aphasia or phonetic (e.g. substitutions, additions, transposition, omissions) due to apraxia may be difficult [1–4,6,8]. Although symptoms such as phoneme prolongation and intersyllabic segmentation are not seen in pure aphasias [15] (e.g. in aphasics vowels duration in multisyllable utterances falls within normal limits). The most informative is a comparison of quality of spoken and written language. Strong arguments against the diagnosis of aphasia in our patient were: good knowledge of the language and fast, precise language processing noted in trials of writing, reading comprehension and auditory comprehension from the early stage of stroke. Further helpful features, that grew in significance when speech output was possible, were: lack of linguistic deficits (e.g. naming deficits or agrammatism), relatively the greatest difficulty in initiation of utterances, unexpectedly severe and permanent dysprosody, worse spontaneous speech than repetition (unlike in conduction aphasia) [1,2], and numerous (more frequent than in aphasias) sound distortions [20]. Patient's remarks about good selections of phonemes but trouble with their execution as well as his attempts to self-correction were also important [2].

Differentiation between AOS and dysarthria can be even more complicated. The complex interactions of the central and

peripheral motor control systems make both these syndromes similar when core AOS symptoms are considered: dysprosody, articulatory distortion, slow speech. However, our patient did not exhibit weakness of articulatory muscles typical in dysarthric speech abnormalities. Slight right-sided asymmetry of lips that can cause occasional air leakage and potentially reduced bilabial consonants and vowels accuracy, did not explain the severity of patient's speech problems and gradual global improvement of articulation. Limitation of impairments to articulation and prosody was another strong differentiating feature, because in dysarthria all speech subsystems (respiration, phonation, resonance, articulation, prosody) are usually impaired. Moreover, the patient had no problem with non-speech motor control (e.g. chewing, swallowing, coughing) which is common in dysarthric patients. The analysis of speech errors provides further important information about the clinical picture of AOS. While in dysarthria articulatory errors are linked to the degree and the location of neuromuscular changes and they are typically consistent and predictable (no matter how well practiced and known the utterances are and whether they refer to speaking, reading aloud or repetition), in AOS errors are highly irregular. They are especially frequent in novel or unpracticed utterances. Perseverations are possible as well [1,2]. Detailed articulation tests confirm that articulation errors occur in different positions across speech productions and various errors are found in subsequent attempts to speak the same words [16].

The differentiation of AOS with similar but more common disorders appears so difficult because of the nature of its symptoms. AOS may vary from a complete inability to speak to slightly slow, relatively fluent speech with infrequent, minor sound distortions [1,2,17,18]. Mild AOS can sometimes be only revealed on particularly challenging words or when the speaker is stressed, tired, agitated on under time pressure [2]. Distinctive symptoms of AOS remain still debatable. According to McNeil [19] sound-level signs that do not occur in any other disorders include only: sound distortions, prolonged segments durations and prolonged intersegments durations. Furthermore, clinical practice demonstrates that not all speech and behavioral characteristics of AOS are observed in all apraxic speakers. Dissimilarity of some acoustic features of speech in apraxia, and diversity of results of brain damage localization studies, has led some authors to consider AOS as heterogenic syndrome. The first suggestion was to make a distinction between left parietal variant of AOS from more typical variant caused by a left frontal lesion. The former type includes such symptoms as visual and auditory groping on initiation and within utterances, numerous off-target approximations of phonemes and occasional syllable segregation [6]. On the other hand, Feiken and Jonkers [15], in accordance to classification of limb apraxias, propose three subtypes of AOS. They include: ideomotor AOS with the most salient initiating problems (visual and audible groping restarts, repetition of initial phonemes, hampered speech, decreased speech rate), kinetic AOS with most salient poorly formed phonemes (distortions, substitutions, poor intelligibility), and ideational AOS with most salient errors in sequencing (interchange of phonemes of syllables in utterances).

All these symptom categories were observed in our patient. However their range and severity in relation to one another changed over time, what may suggest an important role of recovery phase for clinical picture. The greatest functional improvements were noted within the first three months following stroke. During this period of time patient's speech changed from muteness to nearly correctly articulated but dysprosodic utterances. In the following months despite the use of speech in social situations, the symptoms did not resolve completely and one year post-stroke the patient still revealed dysprosody. This is consistent with the data from literature showing that AOS often resolves spontaneously (often rapidly in the acute stroke phase), although for some patients it is a chronic condition [21] with persistent dysprosody as a key symptom [9]. Natural recovery processes, typical for the first weeks and months after stroke, should be enhanced by impairment-based therapy (as in case of our patient). It is believed that rehabilitative intervention may modulate mechanisms of neurofunctional plasticity and positively influence the course of clinical improvement [22,23].

The exact nature of AOS remains unclear. In cognitive terms: lexical processing and phonological assembly are considered to be intact; deficits are assigned to the phase of translation of well-formed phonological specification into commands for speech sounds movements [9]. However, what precisely underlies apraxic speech is not sufficiently understood. Some researches argued that AOS is an effect of impairment in the activation or selection of a generalized motor program and/or in the ability to correctly set the parameters specific to a verbal situation. Others describe AOS as a damage to the mechanism of developing a motor program and of efficiently integrating feedback. AOS is also seen as an impairment of the preprogramming stage or disrupted process of assignation of serial order to multiple programs in a sequence [15].

Accurate differentiation of verbal communication disorders is necessary for devising an appropriate treatment approach. In case of AOS, therapeutic strategies should focus on motor processes and motor relearning. A systematic review of behavioral interventions for AOS suggests that specific therapies, especially articulatory-kinematic and rate/rhythm-based interventions, are efficacious [21]. For patients, who are at high risk for permanent and severe AOS, introducing technology to augment verbal communication, as well as alternative and compensatory communication strategies may be beneficial [2,7].

4. Summary

Despite nearly half a century since the first clinical reports of AOS, this disorder is still poorly understood and rarely diagnosed. Clinical description of AOS symptoms and their dynamics may help to identify AOS and distinguish it from more common speech and language disorders, or neuropsychological disturbances following acquires brain damage. This in turn is the basis for the selection of appropriate rehabilitative instruments.

Conflict of interest

None declared.

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None declared.

Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

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