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# Subcortical aphasia after ischemic stroke on basal ganglia of the left hemisphere

SUMMARY

We present a case of an 80-year-old woman who had a brain infarction located primarily in basal ganglia in the left hemisphere. The patient presented with substantial motor and sensory aphasia and right-sided weakness secondary to cardiac embolism. The cortical and subcortical-cortical lesions were excluded by neuroimaging (MRI and CT) studies. The clinical picture of this subcortical aphasia is accompanied by dysarthria and hypophonia. The case described corresponds with expected subcortical aphasia symptoms, confirming importance of basal ganglia in language programming processes.

**Key words:** subcortical aphasia, aphasia sine aphasia, dysarthria, hypophonia, subcortical brain structures, basal nuclei, thalamus

## INTRODUCTION

Aphasic language disorders occur not only in cases of cortical damage to the left cerebral hemisphere but also – as research shows – in the pathology of subcortical structures (discussed in issue 58 of *Brain and Language*, for instance) as well as in cases of cortical and subcortical damage to the opposite hemisphere in the form of crossed aphasia (A. Herzyk 2005). Since the phenomenon of aphasia is most frequently connected with disorders of cortical origin, the use of the notion of aphasia with reference to deficiencies of subcortical origin may be considered

as unjustified (D. Kądzielawa, 1998). This uncertainty of nomenclature is reflected by a term used by T. von Stockert (1974) – aphasia without aphasia (*aphasia sine aphasia*). Other scholars have defined the same phenomenon as "pure," modally specific aphasia (H. Goodglass, 1993). The notion of subcortical aphasia (or subcortical aphasias) that takes account of the location of the damage with clinical symptoms of various disorders typical of the classical, cortical aphasias is used in the contemporary literature (B.E. Murdoch, B.-M. Whelan, 2009).

Subcortical aphasia is most frequently associated with damage to such structures as the thalamus, basal nuclei and various parts of the white matter. The picture of language disorders connected with the above-mentioned location is diverse to such a degree that – in the opinion of some scholars – it is difficult to capture in a concrete set of fixed symptoms (S. F. Cappa, 1997; B. E. Murdoch, B.-M. Whelan, 2009). Researchers frequently underline the multifunctionality of particular deep structures of the brain (A. Herzyk, 2000), damage to which manifests itself in not only aphasia, evocatively called a secondary symptom (W. Ambrosius, J. P. Mejnartowicz, W. Kozubski, 2003).

However, in the opinion of most authors, one can distinguish a "subcortical aphasic syndrome" (A. Ozeren, F. Koc, M. Demirkiran et al., 2006) and differentiate it from a "cortical aphasic syndrome" on the basis of such language skills as: naming, verbal expression, comprehension, repetition of lexemes and syntactic structures. What is also essential is the assessment of additional criteria such as: verbal praxis (ability to repeat short linguistic structures up to 10 times per minute), oral praxis and a possible occurrence of hemiplegia or hemiparesis in the right side of the body of right-handed people (M. Pąchalska, 2011).

There are various classifications of cortical aphasias. In a similar fashion, subcortical aphasias are classified in various ways according to different criteria. These classifications most frequently include nosologic, linguistic (functional) or anatomic divisions. Such diversity stems from the above-mentioned complexity of clinical symptoms of aphasia. An element that would help to differentiate them may be, for instance, the type of a nervous system disease in which damage to subcortical brain structures dominates, e.g. Parkinson's, Huntington's, Wilson's diseases or multiple sclerosis. What they have in common is – most of all – a verbal expression disorder in the form of dysarthria (speech disorders), but also other language disorders, inclusive of aphasia (B. E. Murdoch, D. Theodoros 2002; B. E. Murdoch, B.-M. Whelan, 2009).

Another criterion in the classification of subcortical aphasias is a linguistic character of the clinical picture of disorders. It is built on the basis of a comparison to well-known symptoms of cortical or cortical-subcortical aphasias (M. P. Alexander, M. A. Naeser, C. L. Palumbo, 1987). This functional differentiation of aphasias enables us to differentiate subcortical sensory aphasia from

subcortical motor aphasia. The sensory variant of aphasia, referred to as "pure deafness of words" (D. Kądzielawa, 1998), is characterized by deep disorders of comprehension of heard speech and disorders of repetition with a maintained spontaneous verbal expression and the ability to read and write (H. Goodglass, 1993). Research shows considerably diverse symptoms and depths of disorders. A pure form of this aphasia is rare. Usually it occurs with other symptoms (D. Kądzielawa, 1998), for instance, with difficulties in recognition of tunes (sensory amusia) or in singing only (motor amusia).

Subcortical motor aphasia is connected with speech articulation disorders with retained abilities to understand speech, to read and write. Most frequently this disorder coexists with oral apraxia with articulatory dysfunctions although there is a phenomenon of dissociation of these symptoms. It means that there are patients with articulatory disorders with a retained oral praxis as well as with oral apraxia and fluent spontaneous articulation. The latter patients are able to move their tongue and lips, to blow and whistle. When they are asked to do so, these actions are unfeasible for them (D. Kądzielawa, 1998).

Due to the diverse repertoire of language disorders in subcortical aphasia, scholars frequently discuss the problem of correlation between the site where a particular structure is damaged and a language deficiency caused as a result of this damage. The anatomical criterion allows us to differentiate such subcortical aphasias as: thalamic aphasia and capsulostriatal aphasia (anterior, posterior and global).

Thalamic aphasia was described in literature at the very earliest (C. Fischer, 1959) and certain scholars consider it synonymous with all subcortical aphasias (J.-F. Démonet, 1997). Furthermore, language disorders associated with thalamic damage seem more homogenous than disorders observed in capsulostriatal aphasia. Although there is a certain variability in its linguistic profile, some researchers claim that features of thalamic aphasia are so distinct that it may be considered a "new" type of aphasia (B.E. Murdoch, B.-M. Whelan, 2009).

Its sensory variant (cf. transcortical sensory aphasia) includes fluent verbal expression full of elements of the aphasic jargon with accompanying disorders of comprehension and a retained ability to repeat (M. Radanovic, M. Azambuja, L. L. Mansur et al., 2003). The motor variant of thalamic aphasia is characterized by a broad spectrum of production disorders, from aphonia to mutism, with a retained ability to understand and repeat. Coexistence of the above-mentioned symptoms is typical for mixed thalamic aphasia with accompanying verbal paraphasias and disorders of semantic coherence at the discourse level with non-spontaneity of the speech production process (D. Kądzielawa, 1998).

Aphasic disorders are also discussed with reference to basal nuclei damage (B. E. Murdoch, B.-M. Whelan, 2009). There are divergent opinions on its role in

language processes (B. Crosson, 1992, 3) because the profile of language-related damage extends from light to severe disorders related to: listening comprehension, reading, spontaneous speech, repetition, naming, or writing (B. E. Murdoch, B.-M. Whelan, 2009). The clinical picture of disorders testifies to at least a partial importance of these structures in language programming. A well-documented research into correlation of disorders in the area of the striatum and the internal capsule has demonstrated beyond doubt that language deficiencies may occur in association with lesions in this area of the dominant hemisphere. In addition, consequences of basal nuclei damage that are related to language skills seem more permanent than the nature of language disorders that results from damage to the thalamus (C.-W. Wallesch, H. H. Kornhuber, R. J. Brunner et al., 1983).

Despite the obvious diversity of language disorders, certain scholars attempt to define a distinctive pattern of damage, corresponding to anterior non-fluent aphasias and posterior fluent cortical aphasias (B. E. Murdoch, B.-M. Whelan, 2009). Damage to the internal capsule, putamen, anterior upper part of the white matter produces symptoms such as retained comprehension without features of agrammatic speech with a coexisting slowness and subcortical dysarthria. Damage to the internal capsule, putamen and the posterior part of the white matter crossing the auditory temporal isthmus is connected with a coexistence of Wernicke's aphasia. Global aphasia accompanies disorders at the level of the internal capsule, putamen and the anterior-upper and posterior part of the white matter (M.A. Naeser, M.P. Alexander, N. Helm-Estabrooks et al., 1982).

The above-mentioned dichotomous division of capsulostriatal aphasia is not fully confirmed in clinical conditions. The accuracy and usefulness of this division have been questioned because a certain number of cases has been described where patterns of language impairment could not be explained by means of such anterior-posterior dichotomy. For example, patients with lesions in the area of the striatum and the anterior part of the internal capsule, as described by C.-W. Wallesch (C.-W. Wallesch, 1985), showed symptoms of Wernicke's aphasia with semantic and phonemic paraphasias. It is evident, therefore, that the clinical and anatomical picture of capsulostriatal aphasia is too heterogeneous (C. Weiller, K.W. Willmes, W. Reiche, 1993) and does not make it possible to distinguish a homogenous picture of such aphasia (B. E. Murdoch, B.-M. Whelan, 2009).

Both the spectrum of symptoms in subcortical aphasia and the degree of intensity of individual deficiencies are extraordinarily wide. One can even talk about their variance (A. Herzyk, 2000; 2005). This is confirmed by research carried out by A. Duranowska-Serocka on a group of 10 patients who sustained damage to basal nuclei and white matter as a result of an ischemic or hemorrhagic stroke. This research was repeated three times at different intervals from the moment the patients had fallen ill. All those examined who suffered from a post-apoplectic

damage to deep structures had language disorders of varying intensity: from light to severe ones. Moreover, the observed disorders could not be connected with a strictly defined structure or group of anatomic structures (A. Duranowska-Serocka 2000).

## CASE STUDY

M.J., an 80-year-old right-handed woman was admitted to the Medical University Department of Neurology and Vascular Diseases of the Nervous System, located at the Prof. L. Bierkowski Independent Public Health Centre of the Ministry of the Interior in Poznań, due to an ischemic cerebral stroke with a focus in basal ganglia of the left cerebral hemisphere.

Immediately after the admission to hospital, the patient underwent a computed tomography (CT) examination which did not show significant ischemic lesions (CT of the head – as opposed to MRI – usually makes it possible to visualise ischemic lesions in the brain only 12-24 hours after the stroke). An MRI examination performed on the same day demonstrated an ischemic lesion measuring 19.7cm$^3$ in the structures of the basal nuclei and the internal capsule in the left hemisphere. A follow-up CT performed 4 days after the ischemic stroke showed hypodense lesions corresponding to the area of ischemia and a moderate edema around the apoplectic focus.

Immediately after falling ill, along with paralysis of the right upper limb and paresis of the right lower limb, the patient suffered from language disorders characteristic of global aphasia. Central paralysis of the facial nerve was diagnosed, however, no paralysis of the hypoglossal nerve was found.

Initially, the patient did not attempt to communicate verbally. Some improvement occurred in this regard at a later stage, although the patient was incapable of speaking spontaneously or story-telling. When spoken to, she only spoke isolated words with an unclear articulative pattern, with continuous perseverations. She barely retained the ability of spontaneous conversational speech. The patient adequately responded to greetings: *good morning*, when asked: *How are you? – Good; Can you see this picture? – I can*. She was also able to say a few names of her closest family, but she said *yeah yeah* in answer to most questions. Her spontaneous activity was considerably reduced. Automatic speech was also deeply disordered. The patient was unable to use series of numbers, names of days of the week, months or seasons. She made attempts to sing or recite, but only the first few words had a relatively clear phonetic and articulative contour and later her singing and speech became unclear and then completely incomprehensible.

The skill of repetition was also deeply disturbed. The patient was only able to reproduce a few phonetically and phonologically simple lexemes such as: *dom,*

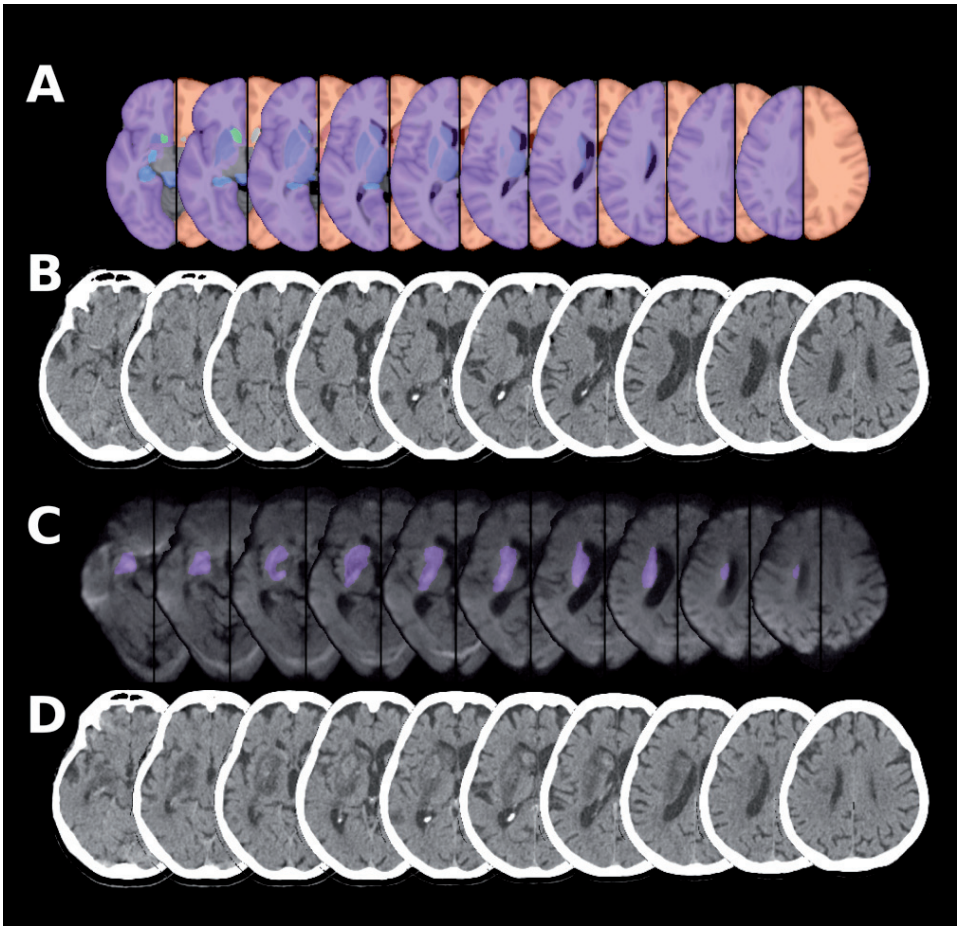


Fig. 1. Visualisation of the location of the ischemic lesion in the patient. The impairment encompasses the anterior and posterior limbs of the internal capsule, the head of caudate nucleus and the putamen on the left side. The images are presented in the neurological convention.

A) MNI brain atlas with a visualised location of the key subcortical structures from the Oxford-Harvard atlas;B) CT of the patient's head performed immediately after the patient's admission to hospital;

C) Diffusion image (DWI) b=1000 in MRI performed during the first 24 hours after the admission;

D) CT of the patient's head during the fourth 24 hours after the admission – evolution of the ischemic lesion can be observed

Images B, C, and D were reduced to the common space of the MNI atlas by means of affine registration.

*tom, bułka, półka, mama, tata, kubek, Maria* [home, book, roll, shelf, mam, dad, mug, Mary]. Phonemic paraphasias were observed when the patient was repeating phonetically difficult words. She also demonstrated lack of verbal fluency, both literal and semantic (E.M. Szepietowska, B. Gawda, 2011) and deep naming disorders as regards names of objects and activities. Occasionally, the patient was able to name them when she was told the first syllable.

Her comprehension of speech was not disturbed. The patient sporadically carried out simple instructions, e.g. she raised her hand. However, she did not understand complex instructions, logical and grammatical sentence structures, names of parts of the face and body or names of objects and activities. The patient would make a gesture of indication but it was frequently inappropriate to the instruction. Furthermore, she did not attempt to communicate non-verbally.

Other skills that were disturbed included categorization on the basis of comparison as well as visual analysis and synthesis. During unsuccessful attempts to copy drawings and shapes there occurred perseverances and the patient persistently repeated circular movements. Attempts to write also proved unsuccessful, which testifies to agraphia. The patient did not attempt to read, either (alexia). She did not indicate or read letters, words or sentences shown to her.

The patient suffered from respiratory, phonatory and articulative disorders which testify to subcortical dysarthria. Her speech was characterized by a shortened expiratory phase, it was stiffened, blurred (close to mutter), slow, with a monotonous intonation pattern and lack of accentuation. Her voice was hoarse, adynamic, monotonous, quiet, she was unable to speak louder (hypophonia) and spoke with difficulty. The above-mentioned symptoms were accompanied by disorders in the functions of facial and articulative muscles. Poor facial expression coexisted with tightened and retracted lips, a falling corner of her mouth on the right side and her mandible in a constrained lockjaw position. Incorrect tension and motor activity of the soft palate produced the effect of nasalization. Movements of the tongue and lips were also considerably limited. The patient did not imitate or make any articulative movements when requested verbally.

The above-mentioned symptoms of impairment testify to a deep subcortical (capsulostriatal) mixed aphasia with dysarthria and hypophonia.

## DISCUSSION

The patient's language disorders confirm that deep aphasia may occur as a result of a cerebral infarction with a focus in the basal nuclei. In the opinion of scholars, such damage may lead to global aphasia (B. Okuda, H. Tanaka, H. Tachibana et al., 1994) although its occurrence in a post-apoplectic impairment of subcortical structures is not frequent (A. Ozeren, F. Koc, M. Demirkiran et al.,

2006) and is connected with a disruption of tracts in the perisylvial region in the cortical areas of speech, without their direct impairment. In a group of 10 patients examined by A. Duranowska-Serocka, subcortical global aphasia was diagnosed in three people, two of whom had suffered from an ischemic stroke. The etiology of the stroke is of great importance here because research results confirm a faster remission of lost language functions in the case of hemorrhagic strokes (A. Duranowska-Serocka, 2000).

The language disorders demonstrated by the patient (M. J.) conform to the well-described pattern of disorders characteristic of such deep structures as the internal capsule, head of caudate nucleus and putamen. These are structures that become damaged most frequently as a result of an ischemic stroke. Research on ischemic strokes confirms a link between the quality of language skills and damage to the anterior limb of the internal capsule. These associations are confirmed by cases of impairment of the above-mentioned structure (A. Duranowska-Serocka, P. Nowacki, W. Posio, 2003).

The research also shows a correlation of the impairment of the anterior limb of the internal capsule with a patient's being less active in his/her contacts with the environment (A. Duranowska-Serocka, P. Nowacki, W. Posio 2003). The striatum including the putamen also performs an important role in language processes (B. Crosson, 1992). Patients with such lesions, similarly to the person we examined, are apathetic, uninterested in communicating, non-spontaneous. The impaired areas constitute the motor centre of the limbic system which, according to some authors, also comprises the abdominal striatum with the accumbens nucleus. The dorsal regions of the caudate nucleus and of the putamen are connected with the motor system (O. Narkiewicz, J. Moryś 2003). The most recent research into the function of the basal nuclei shows their connection with broadly understood cognitive processes, including language processes. They perform an essential role in the process of categorization and (unconscious) learning linked with various social functions that encompass stereotypes and prejudices. They are responsible for attention processes. Their various regions are involved in separate types of tasks. The dopaminergic system of rewarding, connected with the abdominal-medial part of the prefrontal lobe and the abdominal part of the striatum, performs a significant function in the assessment of interpersonal social relations (L. F. Koziol, D.E. Budding, 2010: 175).

Impairments to the basal nuclei are also connected with disorders of production of non-verbal signs and recognition of emotional expression. For instance, patients with Huntington's disease, which is linked with an impairment of the caudate nucleus, suffer from disorders of comprehension of non-verbal signals (L. F. Koziol, D. E. Budding, 2010: 179). In our case study the patient did not

attempt to compensate[1] language deficiencies by means of non-verbal communication. Her facial expression was considerably limited. Neuroimaging research shows that the basal nuclei affect our ability to recognize, understand and produce non-verbal signs, not only with regard to emotional prosody or "melody" of speech but also to emotional expression of the face (L. F. Koziol, D. E. Budding, 2010: 179).

As described above, in the case of subcortical aphasia following from damage to deep structures of the brain, it is difficult to indicate a pattern of deficiencies corresponding to impairment of a specific anatomical structure. It is, however, possible to describe certain elements that are common at the level of language functioning, e.g. dissociation between undisturbed language fluency and disturbed speech fluency. This dichotomy was introduced by M.P. Alexander (1992), who claimed that non-fluent speech is marked by disorders of articulation, disturbed melody and hypophonia, whereas non-fluent language production is characterized by a reduced length of phrases and agrammatism. For example, M.P. Alexander (1992) noticed fluent language production in 12 out of 13 patients examined by him, suffering from non-thalamic subcortical lesions. Their speech production featured the use of grammatically complex structures (sentences containing more than 7 words) with characteristically non-fluent speech. Non-fluent speech with hypophonia occurred in the case described by us. However, we did not observe the above-mentioned dissociation because verbal production itself was also considerably limited. This skill is frequently impoverished in subcortical aphasia resulting from lesions in the striatum region. Lexical and semantic processing is commonly disturbed with other variously damaged abilities of language processing, e.g. at the phonological level (B.E. Murdoch, B.-M. Whelan, 2009). Despite a large degree of diversity of deficiencies in the described language profiles of patients who suffered from the striatum damage, there are certain key features that accompany them. Therefore, it is proposed that a striatal aphasia profile, including language production disorders, should be distinguished, which profile is accompanied by a naming deficiency of various gravity (S.M. Mega, M.P. Alexander, 1994). Obviously, the distinctiveness of this profile may vary depending on the scope of damage or time of emergence of a language deficiency (B.E. Murdoch, B.-M. Whelan, 2009). According to other researchers, the predominant feature in capsulostriatal aphasia is - on the contrary - phonetic language disorders while deficiencies at the lexical and semantic level are linked to impairment of the thalamus (D.C. Kuljic-Obradovic, 2003).

[1] There are two ways in which the gestural method of communication in aphasia can be interpreted. Some scholars treat it as a reflection of disorders of verbal communication (M. Cicone, W. Wapner, N. Foldi et al., 1979; D. McNeill, 1992), while others consider it to be a form of compensation for language disorders (P. Lott, 1999).

As demonstrated in the case described by us, subcortical aphasias are frequently accompanied by dysarthria. It is a well-known fact that lesions in subcortical regions may lead to speech production disorders, yet the essence of the role of such structures as the basal nuclei and thalamus in the motor control of speech remains elusive (B.E. Murdoch, B.-M. Whelan, 2009: 237).

In the recent years, several theoretical models have been developed to explain the role of subcortical structures, cerebellum, basal nuclei and thalamus in the mechanism of occurrence of hypokinetic, hyperkinetic and atactic disorders. They were confirmed in experiments with the use of MPTP (1-methyl1-4-phenyl-1,2,3,6-tetrahydropyridine) and functional neuroimaging. There has been a renewed interest in procedures of stereotactic neurosurgery and deep-brain stimulation (DBS) in the treatment of Parkinson's disease and other basal nuclei disorders. These procedures make it possible to examine the relation of specific lesions or stimulations in the globus pallidus, thalamus with motoric functions of speech. Such procedures contributed to a better understanding of the *basal nuclei – thalamus – cortex* circuit and the *cerebellum – thalamus – cortex* circuits. Currently they are considered as consisting of a series of parallel, multisegmental circuits (rings) or loops. New brain examination techniques make it possible to assess separate disorders occurred in various nodes of the following loops: *basal nuclei – thalamus – cortex* and *cerebellum – thalamus – cortex* (inclusive of substantia nigra, globus pallidus, putamen, caudate nucleus, thalamus, subthalamic nuclei and cerebellum).

The link between basal nuclei and language activity is confirmed not only by monolingual aphasias. Language disorders may also appear in a bilingual aphasia, where L1 (the language acquired first) is disturbed in a consistently stronger manner than L2 (D. Adrover-Roig, N. Galparsoro-Izagirre, K. Marcotte et al., 2011). Disorders of automatic speech, which were also noticed in our case study, are also indicated at this stage of subcortical aphasia. In a bilingual aphasia (following damage to basal nuclei), they relate to the language acquired first more frequently and more strongly.

The phenomenon of subcortical aphasia following a capsulostriatal infarction still attracts a lot of interest not only because of the extremely different symptomatology and location but also due to its patomechanism. Some researchers assign corresponding functions that shape our language behavior to specific subcortical structures, basal nuclei and internal capsule (S. M. Mega, M. P. Alexander, 1994; A. R. Damasio, H. Damasio, M. Rizzo et al., 1982). Other theories assume the existence of functional links between subcortical and cortical structures (the so-called functional loops) damage to which causes a dysfunction of the entire ring (D. Kądzielawa, 1998; M. P. Alexander, M. A. Naeser, C. L. Palumbo, 1987). For instance, research done by E.J. Metter proves that impairment of subcortical struc-

tures disturbs language processes by, for instance, affecting the frontal or temporal cortex (E.J. Metter, 1992).

When discussing the patomechanism of subcortical aphasia, it is impossible to omit the phenomenon of diaschisis. It impairs functions of cerebral areas located remotely from the original damage with which areas it is linked by means of neuronal tracts. A result of this impairment may be a dysfunction of cortical areas responsible for language functions (S. Nadeau, B. Crosson, 1997; D. Perani, G. Vallar, S. Cappa et al., 1987).

Recent research suggests that subcortical aphasia may also be caused by a disseminated ischemic damage or a lasting cortical hypoperfusion of the brain (J.Y. Choi, K.H. Lee, D.L. Na et al., 2007; A.E. Hillis, P.B. Barker, R.J. Wityk et al., 2004). In their research involving patients with an acute stroke limited to the subcortical structures, A.E. Hillis and other scholars (2002) described hypoperfusion in all 28 aphasic patients. It was observed in none of the 12 patients without aphasia. In addition, if blood flow could be restored to the cortex within a few days after the incident, reperfusion was connected with an immediate regression of aphasia (A.E. Hillis, A. Kane, E. Tuffiash et al., 2002).

Similar observations were made by other authors. C. Weiller et al. (1993) found characteristic strictures of middle cerebral arteries in patients with capsulostriatal aphasia. M.K. Han and co-authors (2005) observed that all patients with this type of aphasia suffered from strictures of middle cerebral arteries and internal carotid arteries on the symptomatic side.

It should be noticed that the patient described by us was diagnosed with bilateral, critical strictures of internal carotid arteries, which confirms that cerebral hypoperfusion (reduced blood flow) plays a crucial role in etiopathogenesis of such aphasias.

However, there are still doubts regarding a "pure" subcortical location of infarct focuses in the aphasia in question. M.K. Han, D.W. Kang, S.W. Jeong et al. (2005) found that in infarctions in the region of the capsule and striatum in all patients with aphasia classified as subcortical (10 people), one can also notice small ischemic focuses in the cerebral cortex. These focuses were not visible in conventional MRI examinations but they were found in a much more sensitive diffusion MRI (DWI) (M. K. Han, D.W. Kang, S.W. Jeong et al., 2005).

It should be noted, however, that the patient described by us underwent a DWI examination on a high-class 1.5-tesla MRI scanner (Magnetom Avanto 1.5T, Siemens, Germany) and no cortical focuses were observed.

With reference to the above, it is thought that aphasia connected with a subcortical infarction may be – as a matter of fact – caused by small infarctions in the cerebral cortex which are not seen in CT or MRI but are diagnosable in a diffusion MRI or in a delayed classical MRI (M. K. Han, D. W. Kang, H. Bae et al.,

2003; M.K. Han, D.W. Kang, S.W. Jeong et al., 2005), yet the case presented by us contradicts this thesis.

The visualization of the cortical focus may explain a configuration of miscellaneous types of aphasia in patients with the so-called subcortical infarctions, but it does not explain reasons for the occurrence of aphasia in patients without a cortical infarction in a diffusion MRI (B.E. Murdoch, B.-M. Whelan 2009).

Obviously, there still remains an open problem of an interaction of a striatocortical infarction with a cortical hypoperfusion connected with an accompanying (it appears, in all of the described cases [Han 2005; C. Weiller, K.W. Willmes, W. Reiche et al., 1993]) substantial stricture of middle cerebral arteries and/or internal carotid arteries on the side of the damaged cerebral hemisphere or on both sides. Whether such coincidence is necessary to occur in the aphasia in question requires further analysis. One of the research papers testifies to the occurrence of selective impairments of neurons of the cerebral cortex in the course of an extended cortical hypoperfusion (N.A. Lassen, T.S. Losen, K. Hojgaard et al., 1983). However, not all authors confirm the occurrence of such a phenomenon – pointing out to documented cases of subcortical aphasias without impairments of cortical neurons found in PET (positron emission tomography) (E.J. Metter, W.R. Hanson, C.A. Jackson et al., 1990).

Despite the numerous ambiguities connected with pathophysiology and symptomatology of subcortical aphasias, they can be captured in terms of a fixed repertoire of symptoms that co-create a syndrome characterized by a high degree of variance of the clinical picture of such language skills as comprehension, repetition, naming, story-telling, development of conversational speech and reproduction of automatic speech. Furthermore, aphasic deficiencies coexisting in impairments of subcortical structures (including basal nuclei) confirm that these structures (or their parts) have a share in language programming activities. The nature of their share still puzzling. Scholars develop models that could explain the share of subcortical structures (white matter, thalamus, basal nuclei) in language production. Although each model has its limitations, these models help to generate experimental hypotheses which – in a testing process – contribute to a better understanding of language-related functions of deep cerebral structures (B.E. Murdoch, B.-M. Whelan, 2009: 90).

The most recent research contains theories according to which supposed linguistic and cognitive sources of cortical functions are insufficient. Models of a dual linguistic system and social functioning are being proposed. These models contain two components: basic principles of stimuli processing and the executive system of control. The former enables automatic processing, while the latter makes it possible to adapt to new circumstances. Both of these systems cooperate but they have separate neuroanatomical foundations. The system of executive control of stimuli is connected with a procedural system of learning and phylo-

genetically older regions of the brain. The system of executive control over a linguistic and social behavior in a situational context corresponds to regions of the medial temporal lobe and the related structures. Interactions occurring between these systems make it possible to adapt the originally new, the unknown which ultimately assumes features of the known. For the creators of the above-mentioned model, the best example of *the new – the known* arrangement is the traditional dichotomy of *the verbal – the non-verbal* (L.F. Koziol, D.E. Budding, 2010: 182).

Certainly, recognition of the role of processes and functions performed by subcortical brain structures may considerably change the perception of patients and of the clinical picture of their disorders (cf. L.F. Koziol, D.E. Budding, 2010: 180).

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