POST-STROKE LANGUAGE DISORDERS

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SUMMARY – Post-stroke language disorders are frequent and include aphasia, alexia, agraphia and acalculia. There are different definitions of aphasias, but the most widely accepted neurologic and/or neuropsychological definition is that aphasia is a loss or impairment of verbal communication, which occurs as a consequence of brain dysfunction. It manifests as impairment of almost all verbal abilities, e.g., abnormal verbal expression, difficulties in understanding spoken or written language, repetition, naming, reading and writing. During the history, many classifications of aphasia syndromes were established. For practical use, classification of aphasias according to fluency, comprehension and abilities of naming it seems to be most suitable (nonfluent aphasias: Broca's, transcortical motor, global and mixed transcortical aphasia; fluent aphasias: anomic, conduction, Wernicke's, transcortical sensory, subcortical aphasia). Aphasia is a common consequence of left hemispheric lesion and most common neuropsychological consequence of stroke, with a prevalence of one-third of all stroke patients in acute phase, although there are reports on even higher figures. Many speech impairments have a tendency of spontaneous recovery. Spontaneous recovery is most remarkable in the first three months after stroke onset. Recovery of aphasias caused by ischemic stroke occurs earlier and it is most intensive in the first two weeks. In aphasias caused by hemorrhagic stroke, spontaneous recovery is slower and occurs from the fourth to the eighth week after stroke. The course and outcome of aphasia depend greatly on the type of aphasia. Regardless of the fact that a significant number of aphasias spontaneously improve, it is necessary to start treatment as soon as possible. The writing and reading disorders in stroke patients (alexias and agraphias) are more frequent than verified on routine examination, not only in less developed but also in large neurologic departments. Alexia is an acquired type of sensory aphasia where damage to the brain causes the patient to lose the ability to read. It is also called word blindness, text blindness or visual aphasia. Alexia refers to an acquired inability to read due to brain damage and must be distinguished from dyslexia, a developmental abnormality in which the individual is unable to learn to read, and from illiteracy, which reflects a poor educational background. Most aphasics are also alexic, but alexia may occur in the absence of aphasia and may occasionally be the sole disability resulting from specific brain lesions. There are different classifications of alexias. Traditionally, alexias are divided into three categories: pure alexia with agraphia, pure alexia without agraphia, and alexia associated with aphasia ('aphasic alexia'). Agraphia is defined as disruption of previously intact writing skills by brain damage. Writing involves several elements: language processing, spelling, visual perception, visuospatial orientation for graphic symbols, motor planning, and motor control of writing. A disturbance of any of these processes can impair writing. Agraphia may occur by itself or in association with aphasias, alexia, agnosia and apraxia. Agraphia can also result from 'peripheral' involvement of the motor act of writing. Like alexia, agraphia must be distinguished from illiteracy, where writing skills were never developed. Acalculia is a clinical syndrome of acquired deficits in mathematical calculation, either mentally or with paper and pencil. These language disturbances can be classified

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differently, but there are three principal types of acalculia: acalculia associated with language disturbances, including number paraphasia, number agraphia, or number alexia; acalculia secondary to visuospatial dysfunction with malalignment of numbers and columns, and primary anarithmetria entailing disruption of the computation process.

Kea words: Cognition disorders; Aphasia; Alexia; Agraphia; Acalculia; Mathematics; Stroke

Aphasias

Introduction

There are different definitions of aphasias, but the most widely accepted neurologic and/or neuropsychological definition is that aphasia is a loss or impairment of verbal communication, which occurs as a consequence of brain dysfunction. It manifests in impairment of almost all verbal abilities, i.e. abnormal verbal expression, difficulties in understanding spoken or written language, repetition, naming, reading and writing[1-3].

Diseases affecting blood vessels of the brain are the leading cause of aphasia in about 80% of adults. Ischemic stroke (embolic or thrombotic) as well as hemorrhagic (intracerebral hemorrhage) in the area of anterior cerebral circulation, especially in the territory of the left middle cerebral artery, is a common cause of aphasia syndromes. Temporary aphasia may also be found in patients with transient ischemic attack (TIA); in this case, neurologic deficit as well as disturbance of speech withdraws within 24 h. The main characteristic of aphasia of vascular origin is sudden occurrence and possibility of partial or complete recovery of speech[4-8].

In one of our studies, aphasia was diagnosed in 20.34% of cases and these aphasic disorders were significantly more common in female patients. The most frequent type of aphasia was global (48.51%), followed by Broca's (23.26%) and Wernicke's (8.41%) aphasia. Transcortical sensory, transcortical motor and conductive aphasia were diagnosed in a small number of patients in the acute phase of cerebrovascular incident (CVI). Aphasia was more frequently seen in patients with hemorrhagic stroke (28.14%) compared to those with ischemic stroke (20.58%), but the difference was not statistically significant. In patients with subarachnoid hemorrhage, aphasic disorders were not present at all[9,10]. In the Copenhagen Aphasia Study, the frequencies of different types of aphasia in acute first-ever stroke were as follows: global 32%, Broca's 12%, isolation 2%, transcortical motor 2%, Wernicke's 16%, transcortical sensory 7%, conduction 5% and anomic 25%[5].

Other and less frequent causes of aphasias are head injuries, degenerative diseases and dementias, poisoning, metabolic disorders, infective diseases and demyelination diseases with involvement of the left cerebral hemisphere[4,10-14].

Classification

Both traditional and modern classifications of aphasias are based on elementary clinical characteristics of dichotomies (motor-sensory, expressive-receptive, fluent or nonfluent). The classification based on fluency of spontaneous speech has been widely accepted and enables easy diagnosing the type of aphasia. The main characteristics of nonfluent aphasia include difficulties of articulation, forming short nongrammatical sentences and prosody disorders. On the other hand, fluent spontaneous speech, long grammatically shaped sentences and preserved prosody abilities are basic features of fluent aphasia[1,4,15,16].

During the history, many classifications of aphasia syndromes were established. However, all of these had some limitations, so even today there is no generally accepted classification. Clinical classifications based on fluency of speech, language comprehension and ability to repeat speech seems to be most practical[1,17,18] (Table 1).

Widely in use and still practical is the classification categorizing aphasia syndromes in groups of speech disorders associated with neurologic signs and neuroanatomic localizations of lesions. The main anatomical classification divides aphasias into perisylvian and extrasylvian aphasias, which means that the localization of brain lesion is 'around' or 'away' from sylvian fissure[1,2,18] (Table 2).

Broca's aphasia

Broca's aphasia is also known as motor aphasia, efferent or kinetic aphasia, verbal or syntactic aphasia and expressive aphasia. However, the most frequently

Table 1. Classification of aphasias based on fluency, language understanding and preserved repeated speech

Aphasia	Fluency of speech	Language understanding	Repeated speech
Broca's	Nonfluent	Intact	Disrupted
Transcortical motor	Nonfluent	Intact	Intact
Global	Nonfluent	Disrupted	Disrupted
Mixed transcortical	Nonfluent	Disrupted	Intact
Anomic	Fluent	Intact	Intact
Conductive	Fluent	Intact	Disrupted
Wernicke's	Fluent	Disrupted	Disrupted
Transcortical sensory	Fluent	Disrupted	Intact
Subcortical	Fluent or nonfluent	Variable	Preserved

Table 2. Aphasia syndromes

Syndromes	Fluency of speech	Paraphasias	Repetition	Understanding	Naming	Hemiparesis	Hemisens. disorders
Perisylvian aphasia							
Broca's	Nonfluent	Rare-literal	Disrupted	Preserved	Disrupted	Extensive	Rare
Wernicke's	Fluent	Extensive-mixed	Disrupted	Disrupted	Disrupted	Rare	Occasional
Conductive	Fluent	Extensive-literal	Disrupted	Disrupted	Disrupted	Rare	Extensive
Global	Nonfluent	extensive-mixed	Disrupted	Disrupted	Disrupted	Extensive	Extensive
Extrasylvian aphasia							
Exstrasylvian motor	Nonfluent	Rare	Preserved	Preserved	Disrupted	Occasional	Rare
Aphasia of supplementary motor area	Nonfluent	Rare	Preserved	Preserved	Disrupted	Extensive / crural	Occasional
Extrasylvian sensory	Fluent	Extensive-mixed	Preserved	Disrupted	Disrupted	Occasional	Extensive
Extrasylvian mixed	Nonfluent	Rare	Preserved	Disrupted	Disrupted	Extensive	Extensive
Anomic	Fluent	Rare	Preserved	Preserved	Disrupted	Rare	Rare
Subcortical	Fluent or nonfluent	Extensive	Preserved	Variable	Disrupted	Extensive	Extensive

Fig. 1. Ischemic stroke in frontal lobe (computed tomography) in a patient with Broca's aphasia.

used terms are motor or Broca's aphasia. Lesions that cause Broca's aphasia are located in the lower frontal gyrus, forward towards the motor tract, including premotor and posterior prefrontal regions (Fig. 1).

This type of aphasia is characterized by nonfluent, scarce production of speech, with poor articulation in the form of short sentences with only a few words. Grammatical suffixes are usually not used in words, verbs and adjectives, while the use of nouns remains relatively good. Speech expression is disprosodic because of impairment of rhythm, melody and stress. The way these patients speak sounds similar to telegrams ('telegrammatism').

Auditory understanding is maintained and is usually much better than expressive speech. However, understanding of complex grammatical structures and serial orders is usually impaired. The most frequent difficulties are found in understanding of function words and verbs. Relational words are also difficult to understand, such as above/below, bigger/smaller and sentences expressing communication relations such as 'sister's mother' or 'mother's sister'. Repetition is poor, usually less impaired than spontaneous speech. Difficulties especially occur in repeating complex sentences. The patient simplifies grammar in a sentence. He also shows phonemic distortion and phonemic paraphasia, omitting some phonemes and words. Naming is impaired, as opposed to showing the objects named, which is maintained. Difficulties in naming

the objects are usually the consequence of articulatory disorders, not from the loss of lexical knowledge. In some patients, a combination of lexical and articulatory disorders is possible. Most patients are not able to read aloud or to understand the text they read to themselves. Aphasic writing is also present. Agraphia is manifested in writing large, inappropriately written letters, with literary paragraphy and agrammatism, or 'telegram writing'[1-3,17,18].

The majority of patients with Broca's aphasia have some additional neurologic symptoms such as right side hemiparesis or hemiplegia, ideomotor apraxia of the left arm, and dysarthria.

Wernicke's aphasia

The most frequently used synonym for Wernicke's aphasia is sensory aphasia, and some other names are also used, e.g., acoustic-amnestic aphasia, receptive aphasia and verbal agnosia.

This aphasia is characterized by easy speech production, and is therefore classified as a fluent aphasia with a normal or sometimes above normal speech production. Some patients are so logorrheic that they can be stopped only by energetic reaction of the interlocutor. Therefore, spontaneous speech in this case has well-preserved articulation and prosody. Speech is characterized by long sentences, which seem grammatically correct, but is more or less incomprehensible due to a small or large number of literary and verbal paraphasia and neologisms.

A person with this kind of aphasia has a very poor understanding of the interlocutor and poor repetition. Repetitive speech is generally impaired in proportion with the degree of auditory understanding. In the highest degree of this syndrome, the content of the speech is completely incomprehensible to the interlocutor; then we usually say that the patient neither understands what he said nor is he understood. This is opposed to motor aphasia where the examiner has the impression that the patient understands him, but cannot speak correctly or answer the question properly. Communication of a person with sensory aphasia can be compared to a person who is in a foreign country whose language he does not understand or speak[1-3,18].

Naming (objects and events) is impaired, usually to the degree of anomia, and the patient describes the objects he wants to name. Reading is alexic, and

writing agraphic. His writing has the same features as his spoken language; he uses long sentences which are regularly shaped, but with paraphasia or neologisms.

Unlike motor aphasia, most patients with sensory aphasia have no neurologic symptoms. However, when they are present, it is usually upper quadrantanopsia, or sometimes homonymous hemianopia, hemihyperesthesia, or mild hemiparesis. Wernicke's aphasia is usually caused by a lesion in the dominant temporal

Fig. 2. Ischemic stroke in temporoparietal region (computed tomography) in a patient with Wernicke's aphasia.

lobe, especially in the auditory area in the back upper part of the first temporal gyrus (Fig. 2).

Conductive aphasia

Conductive aphasia is also called afferent or kinesthetic motor aphasia, efferent conductive aphasia and central aphasia. This is a relatively rare type of aphasia, accounting for 5%-10% of cases. It is characterized by easy production of speech with dominant literary paraphasia. Understanding of the interlocutor is relatively good, and so is understanding of the text read, but repetition is outstandingly impaired. Naming is also impaired. Writing is agraphic, and reading alexic, contaminated by paraphasic symptoms[1,17,18].

Fig. 3. Ischemic stroke in parietal lobe (computed tomography) in a patient with conductive aphasia.

This aphasia is clearly different from Wernicke's aphasia because understanding is much better than repetition. It is different from Broca's aphasia because production of spontaneous speech is fluent. Ideomotor apraxia, which includes buccofacial and limb activities, is widespread but not a rule. Neurologic symptoms include a possible, but temporary hemiparesis. Conductive aphasia usually occurs because of a lesion in the back of the perisylvian area of the dominant hemisphere, usually immediately below supramarginal gyrus (Fig. 3).

Global aphasia

This is a relatively frequent aphasia, accounting for 10%-40% of cases. It is also called complete aphasia. Global aphasia is the most serious form of speech disorder. All aspects of speech are impaired, and the patient can usually pronounce just a few words or neologisms. Spontaneous speech is nonfluent, understanding of the interlocutor poor, poor repetition or no repetition at all, and the patient is unable to name objects, read or write (usually complete alexia and agraphia). Speech disorders are usually accompanied by right-sided hemiplegia or hemiparesis, and hemisensory disorders[1].

The degree of verbal dysfunction and localization of causative pathology may vary. It is usually a lesion
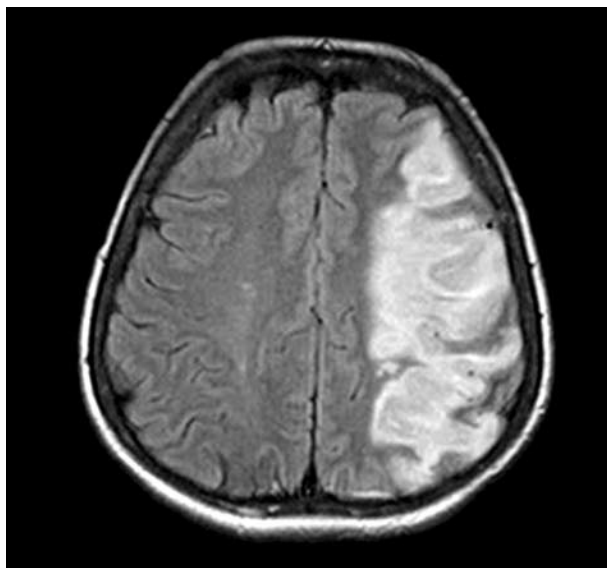
Fig. 4. Ischemic stroke in the middle cerebral artery territory (magnetic resonance) in a patient with global aphasia.

of a large area of the left hemisphere (infarction in irrigation region of the middle cerebral artery) (Fig. 4).

Transcortical motor aphasia

Transcortical motor aphasia or extrasylvian motor aphasia is a nonfluent aphasia which occurs due to damage to the dominant hemisphere outside the speech area or sylvian fissure, which is characterized by a relatively well-preserved ability to repeat. All aphasias that are caused by lesions outside the sylvian fissure are called transcortical. They are usually caused by vascular insufficiency or infarction in the border zone between the middle, anterior and posterior cerebral artery of the left hemisphere. It can also occur as a consequence of tumor, hemorrhage, infection, and in Alzheimer's disease[1,2,17,18].

Transcortical motor aphasia is also called dynamic aphasia. The lesion that causes this type of aphasia may be located in the left hemisphere in front of or behind Broca's area, in the left medial frontal region, also affecting supplementary motor cortex or connections of white matter between these two areas. The main features of this aphasia are difficulties in spontaneous speech production, relatively or well-preserved understanding of speech, unimpaired repetition, impaired naming, impaired reading aloud with good understanding of the text read, and impaired writing.

Transcortical sensory aphasia

This fluent aphasia is characterized by fluent (easily produced) spontaneous speech with paraphasia and echolalia. Echolalia is the basic symptom of this syndrome and that is why it is often misdiagnosed as a psychiatric disease (psychosis).

Understanding of spoken language is considerably impaired, but repetition is intact. Naming, reading and writing are usually considerably impaired. There is variability in reading aloud and impaired understanding of the text read. The combination of neurologic symptoms varies depending on localization and depth of the lesion. The pathologic process that causes this aphasia is usually located in the left parietal and temporal lobe, behind perisylvian area, often in the lower part of the parietal lobe[1,17,18].

Mixed transcortical aphasia

This nonfluent extrasylvian aphasia is also called the syndrome of isolation of speech area. It is very rare compared to other types of aphasia. It is a combination of motor and sensory varieties of extrasylvian aphasia with symptoms of global aphasia, except for preserved ability of repetition of spontaneous speech.

Spontaneous production of speech is impaired (nonfluent) and echolalic. There is poor understanding of spoken language, poor naming, relatively preserved repetition, impaired reading aloud and understanding of the text read, and aphasic writing. Neurologic symptoms are often present, but not constant.

Lesions that cause this aphasia affect frontal and posterior borderline zones (between middle cerebral artery on the one side and anterior or posterior cerebral artery on the other side) of the left hemisphere. Numerous pathologic states accompanied by hypoxia and hypoperfusion of the brain in this region, such as intoxication with carbon-monoxide, acute carotid artery occlusion, acute hypotension, cardiac arrest, etc., may cause mixed transcortical aphasia[1,2,17,18].
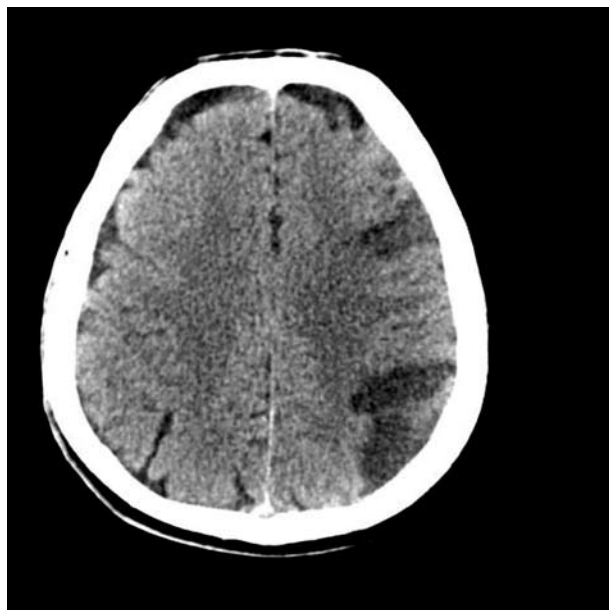
Anomic aphasia

In most aphasias, patients have difficulties in finding words. However, in anomic aphasia, naming is the main and the most common symptom. It is also called nominal aphasia or amnestic aphasia. It is classified as a fluent aphasia with preserved repetitive speech.

Production of spontaneous speech is easy, but the speech is 'empty', with long sentences in which the patient tries to replace the missing words with others (circumlocution). Understanding of spoken language is good, and repetition is good, too. There is some variability in reading aloud, but understanding of the text is intact. Writing can be aphasic. Location of the lesions which cause this syndrome varies, and anomia can be a consequence of a pathologic process located anywhere in the linguistic zone, and in some cases it is even a consequence of processes located in the right hemisphere. If anomic aphasia is combined with alexia and agraphia and Gertsmann's syndrome (right-left disorientation, agnosia of fingers, acalculia and agraphia), the lesion usually affects the dominant angular gyrus (Fig. 5).

Subcortical aphasia

This is one of the recently discovered aphasias. It was possible only after the appearance of computed tomography in the diagnosis of brain damages. Oscillations in symptoms and relatively good outcome with fast recovery of speech impairments are the main features of this aphasia.

The symptoms vary considerably depending on the location of affected subcortical structures. In acute

Fig. 5. Ischemic stroke in posteroparietal region (computed tomography) in a patient with anomic aphasia.

subcortical aphasia, the patient is always mute (without any ability to speak), recovering slowly to hypophonia with slow and poor articulation. Spontaneous speech is contaminated with paraphasias, which disappear when the patient is requested to repeat the sentences spoken by the interlocutor.

Furthermore, other verbal properties are also affected, depending on subcortical pathology. There is a strict tendency of disappearance of verbal disorder and this transience may be accepted as one of the main diagnostic characteristics. However, if the lesion has affected verbal cortical areas, recovery is not complete. Subcortical aphasia is usually caused by ischemia or hemorrhage in the area of irrigation of terminal deep branches of the middle cerebral artery (paraventricular white matter, basal ganglia and thalamus). That is why the symptoms look like transcortical aphasia with good recovery of speech impairments[1,4].

Diagnosis of aphasias

Recognition of aphasic disorders is an important part of neurologic examination. Precise diagnosis of aphasia should be made as soon as possible, which unfortunately is not always the case. One of the reasons is limitation of neuroimaging techniques in all centers and, on the other hand, somewhat deficient knowledge in this field among neurologists and neurosurgeons. Furthermore, not many departments of neurology or neurosurgery have a privilege to have qualified speech therapists. Therefore, early recognition and particularly precise diagnosis of aphasias and early rehabilitation of speech disorders sometimes are missing[1,2,17,18].

Boston test is one of the most precise and widely used aphasia tests in developed diagnostic centers. Boston test examines conversation and fluency of speech, language comprehension, speech expression, repetition, naming, reading and writing. This battery of tests is especially designed for therapy planning and can be useful to monitoring the recovery and efficiency of speech rehabilitation. One of the limitations of the test is the length of examination, which is about three hours. Porchov index of communication skills is a simpler test also having shorter duration (maximum 90 minutes). The test consists of 18 subtests, which are grouped in verbal, graphic and gestual categories.

International test of aphasia (Schuell-Benton) evaluates naming, repetition, fluency of speech, language comprehension, reading, writing and articulation as modalities of speech.

Treatment of aphasias

Treatment of aphasias is multidisciplinary and depends on the symptoms, localization of the brain lesion, etiology and knowledge of the remaining speech and cognitive abilities[1,17,18].

Many speech impairments have a tendency of spontaneous recovery. Recovery of aphasias caused by ischemic stroke occurs sooner, and it is most intensive in the first two weeks. In aphasias caused by hemorrhagic stroke, spontaneous recovery is slower and occurs in the period from the fourth to the eighth week after stroke. The course and the outcome of the aphasia depend greatly on the type of aphasia. Global aphasia has a poor prognosis, unless it is the initial phase of subcortical aphasia. Recovery from Broca's and Wernicke's aphasia varies. Conductive, anomic and transcortical aphasia have a relatively good prognosis. In spontaneous restitution of aphasias, the initial syndromes transform to other clinical forms in 30%-60% of patients[1,6,17,18].

Regardless of the fact that a significant number of aphasias spontaneously improve, it is necessary to start treatment as early as possible. Different rehabilitation procedures have been developed, depending of the type of aphasia.

Our hospital based studies in post stroke aphasias showed best results of speech therapy in patients with Broca's and anomic aphasia, then in those with Wernicke's aphasia, whereas poorest recovery was found in patients with global aphasia. Continuous speech treatment significantly contributed to recovery of post stroke aphasias, regardless of the type of stroke and sex, and showed better results in younger stroke patients[9-21].

The type of aphasia always changed to a less severe form during the first year. Nonfluent aphasia could evolve into fluent aphasia (e.g., global to Wernicke's and Broca's to anomic), whereas fluent aphasia never evolved into nonfluent aphasia. In the Copenhagen study, the following frequencies were found one year after stroke: global 7%, Broca's 13%, isolation 0%, transcortical motor 1%, Wernicke's 5%, transcortical

sensory 0%, conduction 6% and anomic 29%[5]. In our recent study, out of 882 first-ever stroke patients, 192 had aphasia on admission (21.6%), and 61 (43.2%) of them died during hospital stay. Of 131 patients, 74 were contacted one year after stroke onset for new assessment by speech therapist. Twenty of these 74 patients died within one year and two refused assessment. Of the remaining 52 patients, global aphasia was the most frequent type of aphasia on admission (32.6%), followed by Broca's (30.7%) and anomic (23.0%) aphasia. Only 21.8% of 52 aphasic patients were on some kind of speech therapy for some period during the one-year follow up. One year after stroke onset, the most frequent type of aphasia was anomic (38.5%), followed by Broca's (25.0%) and conductive (7.7%) aphasia, and global aphasia in only 3.8%[20,21].

Early treatment of aphasia is important not only because of speech recovery, which is crucial for everyday communication, but also because of the overall rehabilitation of patients with neurologic symptoms accompanying speech impairment. Rehabilitation of motor impairments is more complex and slower in persons with aphasic syndromes, especially if the patient is not treated by parallel rehabilitation of neurologic deficit and speech impairments.

Writing and Reading Disorders

The writing and reading disorders in stroke patients (alexias and agraphias) are more frequent than verified on routine examination, not only in less developed but also in large neurologic departments[8-22]. The reading and writing disorders are aphasic disorders because the comprehension and production of written language are affected more than spoken language modalities[22-25].

There are a variety of theories, which explain the mechanisms involved in reading and comprehension of written language. Reading, of course, requires activation of visual areas in the occipital and temporal lobes, so that the form is perceived, thus revealing that the form is a word. In addition, letters or groups of letters must be recognized and their temporal order ascertained ('orthographic' processing); there must be semantic processing so that the meaning of the word can be derived, and there may be phonological processing so that the sound of the word may be heard within the privacy of one's head. These latter stages of

linguistic analysis involve activation of Wernicke's and Broca's area, and the inferior parietal lobule (IPL), as also demonstrated by functional imaging[26,27]. However, different regions of the brain also interact during various stages of reading, so that in consequence, abnormalities or lesions of different areas can result in different symptoms, such as inability to recognize sentences or long words, whereas the ability to recognize letters and short words remains intact; or inability to derive semantic meaning from words once they are read; or inability to read words, although spelling ability is intact[28]. For example, injuries of the left IPL can disrupt the ability to read and spell. Damage to the adjacent but more anterior-lateral supramarginal gyrus can disrupt the ability to spell by sound and to engage in phonological processing, which is one of the most common correlates of reading disability. Injuries to Wernicke's area can disrupt reading, writing, and all aspects of linguistic comprehension[28]. Lesions to the basal (middle/inferior) temporal lobe can disrupt both reading and naming, a condition referred to as phonological alexia, which is also associated with injuries of the supramarginal gyrus. Furthermore, it has been shown that phonological dyslexia and dysgraphia may be produced by damage to a variety of perisylvian cortical regions, which is consistent with distributed network models of phonologic processing[29].

Alexias

Alexia (from the Greek ά, privative, expressing negation, and λέξις = "word") is an acquired type of sensory aphasia where damage to the brain causes the patient to lose the ability to read. It is also called word blindness, text blindness or visual aphasia. Alexia refers to an acquired inability to read caused by brain damage and must be distinguished from dyslexia, a developmental abnormality in which the individual is unable to learn to read, and from illiteracy, which reflects a poor educational background[23,30].

Most aphasics are also alexic, but alexia may occur in the absence of aphasia and may occasionally be virtually the sole disability resulting from specific brain lesions. There are different classifications of alexias. Traditionally, alexias are divided into three categories: pure alexia with agraphia, pure alexia without agraphia, and alexia associated with aphasia ('aphasic alexia')[31].

More than a century ago, Dejerine described two distinct alexia syndromes: alexia with agraphia and alexia without agraphia. Alexia with agraphia occurs after left-hemisphere parietal damage, whereas alexia without agraphia results from occipital damage together with damage to the splenium of the corpus callosum. Since then, several case reports have supported the clinical and neuropathological patterns of these two alexias. More recently, a third major alexia syndrome has been proposed, based on an anterior lesion in the left hemisphere; it has been called frontal alexia[32-34]. It should be noted that although the symptoms of each of these alexias are relatively clear-cut, associated clinical findings vary considerably depending on the extent of the lesions and involvement of other areas of the cerebrum[34].

Pure alexia with agraphia

Synonyms for this syndrome include parietal-temporal alexia, angular alexia, central alexia, and semantic alexia. The syndrome of alexia with agraphia was described by French physician Dejerine in 1891, where reading and writing are both disrupted with writing impairment usually equal in severity to the alexia, and without significant dysfunction of other language modalities[23,31-34] (Table 3).

Patients display difficulty in comprehending written material that is read silently as well as on reading aloud. Reading of letters and words is impaired, and this difficulty extends to comprehension of numbers and musical notations. The problem with letter identification is not restricted to the visual modality; patients also have problems recognizing words when they are spelled aloud. Parietal-temporal alexia is often associated with fluent paraphasic aphasia[34,35].

Pure alexia without agraphia

In 1892, Dejerine described another acquired alexia syndrome, pure alexia without agraphia. Synonyms for this syndrome include occipital alexia, pure alexia, posterior alexia, pure word blindness, and letter-by-letter alexia. These patients have no gross aphasia, and they can write, either spontaneously or to dictation. The hallmark of this syndrome is the paradoxical inability of the patients to read words they have just written.

Table 3. Features of pure alexia with agraphia

Spontaneous speech	Fluent, often paraphasic
Naming	Often impaired
Auditory comprehension	Intact
Repetition	Intact
Reading	Impaired
Writing	Impaired
Associated signs	Gerstmann's syndrome, right visual field defect
Localization	Left inferior parietal lobule

Alexia without agraphia is easily recognized because it is characterized by a disturbance of reading contrasted with relatively preserved writing skills. Patients typically cannot read what they have just finished writing. The difficulty with letter and word recognition is specific to the visual modality, and patients can spell out aloud and recognize words spelled to them by the examiner.

Letter naming, although initially slow, improves with practice, and the patients often learn to read the individual letters of the word aloud and then decipher the words from their oral spelling[31-35]. The features of alexia without agraphia are shown in Table 4.

Frontal alexia

In patients with frontal alexia, reading comprehension is typically limited to a few single words, usually content words. Reading comprehension of function words such as prepositions and pronouns is impaired. In contrast to their ability to recognize some words, patients are unable to read the individual letters of the word. Spelling words aloud and comprehension

Table 4. Characteristics of pure alexia without agraphia

Spontaneous speech	Normal
Naming	Color-naming difficulty
Auditory comprehension	Intact
Repetition	Intact
Reading	Impaired
Writing	Intact
Associated signs	Right hemianopia; short-term memory loss; occasionally, motor, sensory signs
Localization	Left occipital lobe, splenium, medial temporal lobe

of words that are spelled aloud is also poor. Severe agraphia accompanies alexia, with writing characterized by poorly formed letters, omission of letters, and agrammatical sentences. Frontal alexia is typically associated with nonfluent aphasia. Although these traditional neuroanatomically based distinctions have provided us with better understanding of alexias, they do not fully explain the degree of variability seen in patients with alexia and do not permit subtypes of alexias to be distinguished. Interest has therefore shifted from the anatomical correlates of acquired reading disorders to the neurolinguistic and cognitive mechanisms underlying them. In this approach, various theoretical models of reading have been proposed to account for the performance of normal readers and to identify the components of the normal reading system that are disturbed in the alexia syndromes[34].

Aphasic alexia

Many patients with aphasia have associated alexia. Wernicke's aphasia, for example, frequently affects reading. However, in common usage, the term aphasic alexia refers to alexia with global or Broca's aphasia[25].

Deep alexia

This syndrome evolves in some aphasics with severe reading impairments in which semantically related paralexias are produced in response to written stimuli. The patient may read 'automobile' as 'car' or 'infant' as 'baby'. Such reading is thought to be mediated by the right hemisphere on the basis of iconic recognition[31].

Hemialexia

Alexia may occur with hemispheric lesions that produce profound unilateral neglect. The syndrome usually occurs in patients with right hemispheric lesions and severe hemispatial inattention. The left half of words is ignored so that 'northwest' is read as 'west' or 'basketball' as 'ball'; or the left half may be misjudged, so that 'navigator' is read as 'indicator', 'match' as 'hatch', or 'alligator' as 'narrator'[31,36].

Assessment of Reading

Although brief screening is usually sufficient for identifying the presence of an acquired alexia, detailed assessment is necessary to delineate the precise areas

of breakdown so that an effective treatment program can be developed[22,37]. Thorough evaluation includes assessment of reading comprehension to determine the level at which breakdown occurs, an analysis of single word oral reading, and consideration of associated areas of strengths and deficits including visual skills, writing, naming, and spelling[34].

Reading comprehension and level of breakdown

Reading comprehension tasks provide information about the ability to access semantic meaning from print. All general tests of aphasia have a variety of subtests that assess reading comprehension. These subtests are typically hierarchically organized, beginning with recognition of letters, matching letters written in different forms (e.g., uppercase, lower case, script), and letter naming, then testing progresses to the single word level (e.g., recognizing highly familiar words such as name, name of town, country; matching single words to pictures), the sentence level (e.g., following written commands), and finally to more complex paragraphs (e.g., answering questions about a paragraph that has just been read). However, reading subtests on aphasia batteries such as the Western Aphasia Battery[38] and the Boston Diagnostic Aphasia Examination[39] may have too few items on some tasks and may be insufficient to detect milder problems[40].

A specific test of reading comprehension that has been developed for individuals with aphasia is the Reading Comprehension Battery for Aphasia (2nd edition) (RCBA-2)[41]. The test includes single word comprehension tasks in which a single picture must be matched to one of three words that are orthographically, phonologically, or semantically similar. Sentence comprehension is assessed by having the patient select one of three pictures that correspond to the written sentence. One subtest assesses specifically morphosyntactic reading and another subtest assesses short paragraph comprehension. Longer paragraph comprehension is evaluated with factual and inferential questions[34].

Functional reading of labels and signs is also included. This second edition of the RCBA also includes a lexical decision task in which patients choose a real word from a triad of one real word and two pseudowords. Several tests of reading comprehension provide grade levels and age equivalencies and may be appropriate for individuals with acquired alexia. Such tests include the Gates-MacGinitie Reading Tests[42], the Woodcock Reading Mastery Tests[43], and the Nelson-Denny Reading Test[44].

Treatment

Traditional approaches to the treatment of acquired reading problems typically begin at the level of breakdown, that is, at the grapheme, word, phrase, sentence, or paragraph level, and patients practice tasks that are arranged hierarchically. Some commonly used treatment tasks include letter matching; word-picture matching; word-word matching in which the target may be the category name, an antonym, or a synonym; word-definition matching; phrase or sentence completion; following written commands; and answering yes/no or 'way' questions about a sentence or paragraph. Difficulty is modified by a change in various parameters such as the degree of similarity between the target and distractors; the number of distractors in the field; the frequency, grammatical class, concreteness of the words; or the complexity of the grammatical structure.

Most commercially available workbooks for aphasia have numerous pages of pencil and paper exercises, and more recently a number of computer programs also have been developed that provide practice on these types of reading activities. However, despite their clinical widespread use, these reading tasks and programs for aphasia have not been carefully evaluated and there is little evidence supporting their efficacy.

One randomized clinical trial evaluated the efficacy of computerized treatment in general by using hierarchically organized reading activities consistent with the traditional approach[45]. Fifty-five patients with chronic aphasia were randomly assigned to one of three conditions: computer reading treatment that consisted of visual matching and reading comprehension tasks, computer stimulation such as nonverbal games and cognitive rehabilitation tasks, or no treatment. Patients in the computer groups used computers 3 hours a week for 26 weeks. The results suggested that computerized reading treatment was efficacious with improvements generalizing to noncomputer language performance. It was also shown that these improvements resulted from the language content of the software and not from the stimulation provided by the computer[34].

Agraphias

Agraphia is defined as disruption of previously intact writing skills by brain damage. Writing involves several elements: language processing, spelling, visual perception, visuospatial orientation for graphic symbols, motor planning, and motor control of writing. A disturbance of any of these processes can impair writing. Agraphia may occur by itself or in association with aphasia, alexia, agnosia and apraxia. Agraphia may also result from 'peripheral' involvement of the motor act of writing[24,25]. Like alexia, agraphia must be distinguished from illiteracy, where writing skills were never developed[31].

There are several classifications of agraphia. First, writing disorders can be classified by the underlying cognitive deficits: aphasic agraphia, apraxic agraphia, and spatial agraphia. In addition, 'pure agraphia' indicates the absence of any other language or cognitive disorder. Another way of classifying agraphias is to divide writing into its component psycholinguistic steps and to analyze writing disorders according to the specific step that is disrupted, as in the classification of agraphias, we first distinguish between 'central' agraphia, resulting from disorders of central language processing, *versus* 'peripheral' agraphia, resulting from disorders of the motor aspect of writing. Central agraphias thus affect lexical (word choice), semantic (word meaning), and phonological processes, after which a 'graphemic' (written) version of the word is generated. The peripheral portion of writing involves selection of the proper letter string and the motor output to write it[25].

Table 5. Aphasic agraphias

Agraphia with fluent aphasia
Agraphia with nonfluent aphasia
Alexia with agraphia
Gertsmann's syndrome agraphia
Pure agraphia
Agraphia in confusional states
Deep agraphia
Disconnection agraphia
Praxic agraphia

From practical point of view, agraphias can be divided into two categories: aphasic and nonphasic agraphias. Aphasic agraphias include agraphia with

Table 6. Nonaphasic agraphias

Motor agraphia
Paretic agraphia
Hypokinetic agraphia:
Micrographia with parkinsonism
Hyperkinetic agraphia:
Tremor
Chorea, athetosis, tics
Dystonia (writer's cramp)
Reiterative agraphia
Perseveration
Paligraphia
Echographia
Coprographia
Visuospatial agraphia
Hysterical agraphia

fluent aphasia; agraphia with nonfluent aphasia; alexia with agraphia, Gertsmann's syndrome agraphia; pure agraphia; agraphia in confusional states; deep agraphia; disconnection agraphia; and apraxic agraphia (Table 5). Nonphasic agraphias are motor agraphia, reiterative agraphia, visuospatial agraphia and hysterical agraphia[31] (Table 6).

Aphasic agraphias

For aphasic patients, writing is often the most severely impaired language modality. In aphasic patients, written language typically mirrors spoken language expression, and in some cases the language abnormalities may be more marked in written than spoken language. Thus, in nonfluent aphasias such as Broca's, writing resembles speech: brief, effortful, and lacking in syntax; there is sparse graphic output, with clumsy calligraphy, agrammatism, and poor spelling. Fluent aphasias, especially Werincke's aphasia, also produce fluent errors in writing (have a normal quantity of well-formed letters, but with the lack of substantive words and insertion of literal, verbal, or neologistic paragraphias similar to oral paraphasias), and spelling errors reflect mild deficits[25,31].

Deep agraphia

Deep agraphia parallels deep alexia involving writing rather than reading. These patients have difficulty

in spelling nonwords, deficit in spelling certain classes of words; can write words with concrete, imaginable meanings better than those with abstract meanings and semantic words (nouns and verbs) better than syntactic words (propositions and conjunctions). Errors may involve semantically related words, such as 'chair' for 'desk'. Lesions generally involve the left parietal region, often including the supramarginal gyrus or insula but sparing the angular gyrus[25].

Gerstmann's syndrome and angular gyrus syndrome

In 1924, Josef Gerstmann described a syndrome occurring with discrete left angular gyrus lesions and consisting of a tetrad of clinical findings including agraphia, finger agnosia, inability to distinguish left from right, and acalculia[31]. In 1940, Gerstmann reviewed considerable literature that had evolved concerning the syndrome and concluded that the finding had clinical validity and localizing value[46].

Not all four elements of Gerstmann's syndrome need necessarily occur together; any combination of three items would indicate a left inferior parietal lesion, and other, related deficits including alexia and mild aphasia may be combined[25].

Nonaphasic agraphia

Writing depends on a complex array of motor and visuospatial skills in addition to language abilities.

Motor agraphias. Disruption of any aspect of the motor system, peripheral, corticospinal, extrapyramidal, cerebellar, will produce agraphia, and in each case the muscles, peripheral nerves, or corticospinal tracts produce a clumsy, uncoordinated agraphia secondary to limb paralysis. Micrographia is a common manifestation of parkinsonism and is characterized by progressive diminution in the size of the letters, often accompanied by increased crowding. Action tremors of either the cerebellar or postural type produce disturbances in writing and may make written productions unintelligible. Chorea, athetosis and tics are hyperkinetic movement disorders that influence writing in the same way that they affect other voluntary motor activity. In severe cases, writing is impossible, and even in mild cases the output will be visibly distorted. 'Writer's cramp' is among the best known and most misunderstood of all agraphias. The syndrome of

progressive cramping of the hand and forearm among individuals from professions demanding fine finger movements, including writers, telegraphers, pianists, and violinists, was treated as a neurotic disorder in the past, while today it is well-known focal dystonia.

Reiterative agraphias refer to the abnormal repetition of letters, words, or phrases in writing. Perseveration is a continuation of activity after the appropriate stimulus has stopped. Paligraphia is the rewriting of phrases generated by the patient. Echographia is the rewriting of phrases produced by the examiner.

Visuospatial agraphia is manifested by a tendency to neglect one portion of the writing page, slanting of the lines upward or downward, and abnormal spacing between letters, syllables or words.

Hysterical agraphia. Agraphia may occasionally occur as a hysterical conversion symptom. The agraphia is usually part of monoparesis in which the limb is weak throughout, with slightly diminished tone and normal muscle stretch reflexes. Sensation may or may not be affected[31].

Acalculias

Acalculia is a clinical syndrome of acquired deficits in mathematical calculation, either mentally or with paper and pencil[25,31,47]. These language disturbances can be classified differently, but there are three principal types of acalculia: acalculia associated with language disturbances, including number paraphasia, number agraphia, or number alexia; acalculia secondary to visuospatial dysfunction with misalignment of numbers and columns; and primary anarithmetria entailing disruption of the computation process[31] (Table 7).

Table 7. Classification of acalculias

Aphasia-related acalculias
Number paraphasia
Number alexia
Number agraphia
Visuospatial acalculia
Primary anarithmetria
Symbol agnosia

Aphasia-related disturbances of calculation include paraphasic errors in which the patient makes a verbal paraphasic error, substituting one number for another.

Number alexia and number agraphia may also occur and, in some cases, may be disproportionately greater than letter reading and writing disturbances. Acalculia occurs with nearly all aphasias but is more severe in patients with lesions of the posterior aspect of the left hemisphere involving the parietal cortex[31].

Visuospatial acalculia may occur with lesions of either hemisphere but is most common with right parietal dysfunction. Spacing of multidigit numbers, placeholding values, and column alignment are disrupted.

Primary anarithmetria occurs mainly in the context of Gertsmann's syndrome with lesion in the region of dominant angular gyrus, but it may occasionally be seen as an isolated abnormality with disturbances of the same region. It is an acquired neurologic deficit in mathematical thinking, which does not involve problems in reading and writing of numbers or in spatial arrangement of numbers[31].

Conclusion

Post-stroke language disorders are very complex in their clinical phenomenology and in classifications. Speech disorders of aphasic type, alexia, agraphia and acalculia, in neurology, first of all in the acute phase of stroke, are more frequent than actually verified on routine examination, not only in less developed but also in large neurologic departments. The most important aspect of language disorders is the necessity of testing these functions to detect deficits. Language deficits are important in practical localization of brain lesions and they impair patient ability to function in the world. Regardless of the fact that a significant number of these disorders spontaneously improve, it is necessary to start treatment as early as possible. Early treatment of language disorders is important not only because of speech recovery, which is crucial for everyday communication, but also for overall rehabilitation of patients with neurologic symptoms accompanying speech impairment. Rehabilitation of motor impairments is more complex and slower in persons with these syndromes, especially if the patient is not treated by parallel rehabilitation of neurologic deficit and speech impairments.

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Sažetak

JEZIČNI POREMEĆAJI NAKON MOŽDANOG UDARA

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Jezični poremećaji nakon moždanog udara su česti i uključuju afaziju, aleksiju, agrafiju i akalkuliju. Postoje različite definicije afazija, ali prema najšire prihvaćenoj neurološkoj i/ili neuropsihološkoj definiciji afazija je gubitak ili oštećenje jezične komunikacije koji nastaje kao posljedica moždane disfunkcije. Može se manifestirati oštećenjem skoro svih jezičnih modaliteta – abnormalnošću verbalnog izražavanja, poteškoćama razumijevanja govornog ili pisanog jezika, ponavljanja, imenovanja, čitanja i pisanja. U povijesti razvoja učenja o afazijama predložene su brojne klasifikacije afazičnih poremećaja. Za praktičnu kliničku upotrebu podjela afazija prema fluentnosti, razumijevanju i sposobnosti ponavljanja govora čini se najprikladnijom (nefluentne afazije: Brocina, transkortikalna motorna, globalna i miješana transkortikalna afazija; fluentne afazije: anomična, konduktivna, Wernickeova, transkortikalna senzorna, subkortikalna afazija). Afazija je raširena posljedica lezije lijeve hemisfere i jedna od najraširenijih posljedica moždanog udara, s učestalošću od jedne trećine u akutnoj fazi moždanog udara, a neka izvješća govore i o većoj učestalosti. Mnogi poremećaji govora imaju tendenciju spontanog oporavka. Oporavak afazije uzrokovane ishemijskim udarom javlja se prije, najintezivnije u prva dva tjedna. U slučaju hemoragijskog udara spontani oporavak afazija je sporiji i najuočljiviji je od četvrtog do osmog tjedna od udara. Tijek oporavka umnogome ovisi o vrsti afazije. Međutim, bez obzira na činjenicu da se afazije spontano oporavljaju neophodno je započeti njihovo liječenje što ranije je moguće. Poremećaji pisanja i čitanja u bolesnika s moždanim udarom (aleksije i agrafije) su mnogo češće nego se to utvrdi u rutinskom neurološkom pregledu, i to ne samo u manje razvijenim, nego i u velikim neurološkim odjelima; to su zapravo afazični poremećaji i nerijetko su ove funkcije više oštećene nego govorni jezik. Aleksija je stečeni tip senzorne afazije gdje oštećenje mozga uzrokuje gubitak mogućnosti otprije naučenog čitanja. Naziva se još sljepoća za riječi, sljepoća za tekst ili vizualna afazija. Treba ju razlikovati od disleksije, razvojnog poremećaja gdje osoba nije u stanju naučiti čitati, te od nepismenosti koja odražava siromašnu obrazovnu sredinu. Većina afazičara su aleksični, ali aleksija može biti prisutna i u odsutnosti afazije, te u nekim slučajevima nakon specifičnog oštećenja mozga i kao izolirani poremećaj. Postoje različite klasifikacije aleksija. Tradicionalno, aleksije se dijele u tri kategorije: čista aleksija s agrafijom, čista aleksija bez agrafije i aleksije udružene s afazijama ('afazične aleksije'). Agrafija se definira kao oštećenje ranije naučene vještine pisanja nastalo nakon oštećenja mozga. Pisanje uključuje nekoliko elemenata – jezično procesuiranje, sricanje, vizualnu percepciju, vizualno-prostornu orijentaciju za grafičke simbole, motorno planiranje i motornu kontrolu pisanja. Oštećenje bilo kojega od ovih procesa može oštetiti pisanje. Agrafija se može pojaviti kao izolirani poremećaj ili udružena s afazijom, aleksijom, agnozijom i apraksijom. Može također biti i posljedica zahvaćanja 'perifernog' akta pisanja. Slično aleksiji, agrafija se mora razlikovati od nepismenosti kada vještina pisanja nije ni razvijena. Akalkulija je klinički sindrom stečenog deficita matematičke kalkulacije ili na mentalnom planu ili s 'papirom i olovkom'. Ova jezična oštećenja mogu se različito klasificirati, ali u biti postoje tri glavna tipa akalkulija: akalkulija udružena s jezičnim poremećajima uključujući parafaziju, agrafiju brojeva, aleksiju brojeva; akalkuliju sekundarnu na vizuo-prostornu disfunkciju s nepravilnim položajem brojeva ili stupaca i primarnu anaritmetriju s 'čistim' poremećajem računanja.

Ključne riječi: *Spoznajni poremećaji: Afazija; Aleksija; Agrafija; Akalkulija; Matematika; Moždani udar*