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Neurobehavioral consequences of cerebral stroke and its influence on rehabilitation

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Abstract
The consequences of stroke pose serious social and economical problems and are the leading cause of disability and inability to live independently without support as adults in developed countries. As a result of population ageing this number will probably increase steadily in the next decades. At the same time, due to better acute treatment, case fatality rate is declining. The most common neurobehavioral disorders include cognitive impairments and neuropsychiatric syndromes such as depression, apathy and angst/anxiety.

Key words
stroke, psychopathology, cognitive impairments, neurorehabilitation

Introduction

Consequnces of stroke are a serious social and economic problem; they are the main cause of disability and an inability to exist unaided of adults in developed countries [1, 2, 3, 4]. Stroke is the second most frequent cause of death in the world, after cardiovascular diseases [5, 6], and in Poland the third one after cardiovascular diseases and tumors [7]. 60,000 people suffer a stroke in Poland each year. Since the population is aging, this number will probably increase steadily in the next decades. At the same time, case fatality rates are declining due to better acute treatment of stroke [5]. Forecasting and prognosis depend on the localisation and

focus of stroke, comorbidities, patient's prior stroke condition, nursing and rehabilitation quality in the first days of hospitalisation [8, 9].

The signs of stroke, among movement syndromes, sensory syndromes, speech and sight difficulties commonly associated with the celebral event, also include neuropsychological symptoms, whose occurence requires specialist procedures, and their omission may result in no effects in the rehabilitation process. The most common impairments after stroke are neurobehaviorual disorders which include cognitive impairments and neuropsychiatric syndromes (among others depression, apathy, fear [10, 11].

Psychopathological syndromes are common complications after stroke, and they may occur both in the early and late phase of stroke [12, 13, 14, 15].

The occurence of the below given syndromes present in the neurobehavioural syndromes have an impact on the rehabilitation process;

- memory impairments (eg. short and long-term, semantic, episodic),
- attention deficit,
- executive disfunctions,
- perception disturbances,
- restlessness and motor drive lowering,
- abstract thinking impairment,
- judgment impairment.

These symptoms may be accompanied by: aphasia, apraxia, agnosia, alexia, acalculia, anosognosia [3, 16, 17]. The occurence of the symptoms menioned here makes the therapy longer and a late recovery, yet, on the other hand, their presence has raised the awarenes amongst therapists of the quiddity of the neurocognitive dysfunctions and the need for their imrovement. A model of conduct with a stroke patient has been worked out, in which there are diagnostic and treatment procedures and also rehabilitation procedures carried out at the same time. Early rehabilitation after stroke should be carried out as early as possible (1-2 days after the appearance of the condition). The procedures are defined in Helsingborg Declaration, which assumes that all patients in the acute phase of stroke have the right to access the rehabilitation services, without a pre-selection [7]. According to the WHO definition, rehabilitation is a complex set of measures that assist individuals, who experience physical or menatl

disability, which aims to achieve and maintain optimum functioning, participation in the labour market, and civic life [4].

The term neurorehabilitation includes a wide range of revalidation procedures with patients with disabilities of the central nervous system [3]. This definition was popularised in the 80s of the last century when a significant progress took place in the basic and clinical sciences of CNS diseases. The development of modern medical techniques and research progress in neurobiology, neuropsychology are the basis for new strategies, including rehabilitational ones with neurogenic dysfunction patients. The theoretical basis of brain damage patient rehabilitation, regardles of the initial cause, is the brain plasticity.

Neuroplasticity

The term neuroplasticity was introdused into physiology by Polish scientist Jerzy Konorski in his monograph published in 1948. He understood this notion as an ability of neurons to undergo permanent changes during the learning process [18]. Fundamental mechanisms of biological nature have been recognised (relating to structural and functional changes in the CNS) as responsible for lessening the signs of the disease in the process of convalescence and therapy. Spontaneous wearing off of the deficits after some time after a stroke very often arises from the compensational neuroplasticity mechanism [19]. It includes the functional and structural changes, which are the basis for the compensation processes of disorders results. The withdrawal of the diaschisis symptoms, normalisation of biochemical changes (among others completing the missing neurotransmitters), the restoration of blood flow (reperfusion) in the ischemic area (penumbra) are listed among the functional changes [19]. The structural changes include the regeneration of the connections between neurons (synaptogenesis) and the growth of new axons (neurogenesis) [19]. Apart from a spontaneous dynamics of compensational neuroplasticity mechanisms, a process of modification of the condition of the neural network caused by behavioral influences is also triggered [20].

The recovery processes taking place in the brain sped up by the neurological treatment and the functional and structural changes being the effect of the neuropsychological activities interact. They create a possibility for the improvement in the patient's functioning thanks to the restoration and substitution of the disturbed functions, activation of extra

afferentations, rebuilding of functions, reintegration of neural connections, switching on new ways of brain reserves use and optimisation of the neural network activity [20].

The body's reactions to the changes taking place in the environment are modified by the experience acquired throughout personal life. Two processes serve it – learning and memory. Both of the phenomena, as defined by neurobiology, may occur only in the nervous system [21, 22]. Learning is acquiring new information, that is creating an inner representation of experiences in the nervous system; whereas memory means retaining these representations in time with a possibility of using them in the neural processes [22, 23].

The processes of learning and memory are one of the most crucial expressions of the nervous system abilities to undergo plastic structural and functional changes. The effectiveness of these processes decreases with age, yet it does not disapear. Memory may be understood in categories of a specific group of neurons producing impulses according to the same pattern each time when activated. It is created due to a long-term potentiation [LTP]. It is a process, in which each time a given sequence of neurons produces an impulse, the connections between these neurons are strengthened. It is a result of the Hebbian Learning Rule: if an A cell axone constantly takes part in a B cell stimulation creating its activation, it makes a metabolic change in one or in both cells leading to the increase in the activation efficiency of B by A [24, 25]. A range of kinds of memory has been distinguished and it is known that certain brain areas are more important for creation of a given kind of memory, and others less [22, 26]. We may consider memory in relation to the length of period for which memories are stored (immediate, operational, short-term, long-term), what kind of information one must remember (semantic, episodic, procedural), in relation to modality (verbal, visual), the method of information storing and updating, and the level of involvement of awareness at the retrival stage (declarative and nondeclarative) [27, 28]. The above classifications have been created by psychologists [22]. Short-term memory, from the perspective of neurochemists, lasting from a few to several dozen minutes, does not depend on protein syntheses and may be disrupted by electrical shocks. Long-term memory, dependent on protein syntheses, follows short-term memory and is not disturbed by electrical shocks [19].

Due to the brain tissue damage with various factors the motory, sensory and cognitive deficits are created. According to the clinical experience, persons with cognitive impairments are very often more helpless than motor deficit persons. The assumption that the brain of an adult is characterised by a certain degree of plasticity ensuring at least a partial reconstruction of the impaired functions is the basis for realistic therapeutic optimism [29]. Poststroke cognitive impairments may accompany motor deficits, but they are very frequently the leading symptom of post-stroke psychopathological disorders [23, 13, 14].

Poststroke neurobehavioral disorders

The signs of cognitive behavioral disorders resulting from brain dysfunctions are commonly of a chronic character; they may have a sudden onset, or grow slowly in a little specific way [30]. The brain damage consequences, mainly of a focal origin (stroke), present themselves in the following domains [31, 10, 32, 33].

1. Memory disorders (difficulties in remembering, storing and retrieving of information, confabulations).
2. Attention disorders (paying, splitting, selecting, processing speed).
3. Orientation disorders (difficulties in relating to time, space, self--being).
4. Learning, comprehending and judgement disorders (depleted thinking, imprecise thinking, lack of planing and predicting capabilities, disorders in understanding abstract concepts).
5. Emotion control weakening (pathological laughter, cry, extreme emotions).
6. Social competence deficits (recognising emotions, empathy).
7. Apathy and shallowing of emotions (limited experiencing of all emotions).
8. Behavioral initiation disorders (disorders of executive function, planning and decision taking, working memory, thinking flexibility).
9. Situation appropriateness assessment disorders and conduct adequacy disorders (lowering of personal appearance standards, keeping hygiene, sexual behaviour, language).
10. Difficulty to comprehend and formulate messages (aphasia).

11. Visual-spatial processing disorders (difficulties in visual-motor integration, graphomotor skills disorders, constructive skills disorders, praxia, gnosia).

The cognitive behavioral disorders, neuropsychiatric syndromes and neuropsychological disorders [31] have appeared in the DSM-V clasification in a new category under the name "neurocognitive disorders". The new terminology replaces "organic psychic disorders" in the DSM--III-R, and, respectively, "dementia, delirium, amnesia and other cognitive disorders" in the DSM-IV. It is worth considering that the word "organic" has been omitted in the DSM IV classification since it assumes incorrectly that "non-organic" psychic disorders have no biological basis [34]. The neurocognitive disorders include a group of which are clinically manifested in an acquired damage to the cognitive functions. These include [10]:

- delirium,
- deep neurocognitive impairtment syndromes (among others impairments caused by dementia, by a brain injury, by HIV virus infection, by pharmaceutical drugs),

mild cognitive impairments syndromes (other cognitive disturbances).

Acute mental disorder which could appear both in the early and late phase of stroke, being the so-called acute condition in medicine, is delirium [13].

The symptoms of delirium are:

- decrease of attention sustainability in relation to external stimuli, focus of attention continual change with inadequate alternating attention,
- abnormal thinking, disorderly and disconnected speech,
- decrease of a wakefulness level, disorders of sleep-wake schedule,
- perception disorders, false interpretations, illusions, hallucinations,
- motor anxiety or decrease of motor drive,
- time and place disorientation, people's misrecognition,
- memory impairment, inability to learn new material and to retireve past events.

The disorders show soon, usually within a few hours or days; they have a variable intensity in twenty-four hours, with a frequent exacerbation of symptoms at night [30]. There are three types of delirium: hyperactive

delirium (psychotic symptoms where hyperactivity prevails), hypo-active delirium, which may be incorrectly interpreted as depression (sedation prevails), and mixed delirium (hypo- and hyperactive) [35, 10].

Delirium frequently occurs in hospitalised patients and ranges from 10% to 30 in hospitalised patients [30, 13, 36], and from 10% to 48% in patients in the acute phase of stroke [37]. Delirium patients have almost a 5 times higher risk of mortality within 12 months; they require a longer hospitalisation period, and are more likely to be dischared to long-term care institutions. Compared to non-delirious stroke patients, delirious patients have lower quality of life and a higher risk of developing dementia within two years after the stroke [37].

The delirium predisposing factors are: older age, presence of dementia, organic and mental illnesses [36]. There are numerous causes for delirium: infection, high fever, metabolic disorders, liver and kidney failures, endocrine disorders, thiamine deficiency, psychoactive substances poisoning, or caused, by their withdrawal, the substance withdrawals, significant blood loss, post-operative conditions, cardiac arrhythmia and heart failure, malignant hypertension, head injuries, convulsions, adverse drug reactions, focal brain damages [30]. The risk factors for acute phase stroke delirium are: older age, specific post-sroke syndromes (aphasia, neglect syndrome, dysphagia), visual disturbances, elevated cortisol level and the influence of anticholinergics [35].

Delirium, as a medicine acute condition, requires a quick identification of its cause and implementation of management. The course of delirium most frequently is turbulent, but it sometimes happens that it disappears by itself. If untreated, it may lead to death or permanent dementia. Casual treatment generally brings a quick recovery, yet deficits sometimes remain [30, 13].

Dementia is another disorder (associated with stroke) of a global character [5, 38, 39]. Dementia is characterised by a gradaual decline in intellectual functions leading to social and professional disturbances. Memory, orientation, abstract thinking, ability to learn, visual-spatial perception, language functions, constructive praxy, and upper executive functions – such as planning, organisation and action sequencing are impaired in the state of dementia. Unlike delirium patients, dementia patients have retained consciousness until the late stages of the condition [30]. Delirium is most frequently associated with a systemic illness

or drug poisoning, while dementia is caused by a primary degerative or structural brain disease.

Dementia, which develops after a stroke, called poststroke dementia (clinical name) is any kind of dementia appearing after a stroke regardless of its probable cause [5]. Vascular dementia is not synonymous with post-stroke dementia, but merely one of its probable causes and is responsible only for some poststroke dementia cases. According to various authors, the development of poststroke dementia is determined by: age, lower education, diabetes, cardiac arrhythmia, a prior ischemic stroke, aphasia, and a more serious neurological deficit upon hospital admission, lower intelectual capacity prior to a stroke, cerebral atrophy, changes in the white matter [5, 39]. Some poststroke dementia patients are characterised by a gradual development of dementia syndromes, which suggests a degenerative basis of dementia. There ia an increasing number of reports concerning numerous, well-recognized risk factors of atherosclerosis and stroke which are diagnosed also in degerative dementia persons. They include, among others: older age, hypercholesterolemia, arterial hypertension, cigarette smoking, atrial fibrillation, diabetes, the APOE gene polymorphism [39].

The clinical data shows that medium-sized strokes in the caudate mucleus, thalamus, hippocampus, and the Sylvian fissure area on the dominant side worsen cognitive functions in the disease picture [12, 14]. Most authors consider the changes in the white matter, including the paraventricular one, or semioval centres, to be crucial to diagnose vascular dementia [12]. The presence of other changes, such as: numerous micro myocardial infarctions detected in the MRI examination, the hyperintensive foci in the white matter, or the presence of a large (strategic) stroke focus are not considered as typical for the pure cerebrovascular disease, and are not typical of vascular dementia.

The key symptom of dementia (massively important in the rehabilitation aspect) is the short-term and long-term memory impairment. Difficulty reproducing names of three objects after five minutes is a sign of a short-term memory impairment, an inability to learn new material. A patient's difficulty in remembering data of their own past, both the nearest and distant – what they did the previous day, if they remember the dates of historical events and commonly known facts – is a sign of a long-term memory impairment [30].

Deterioration in cognitve functions mostly lessens after a few weeks, yet in some patients the cognitive deficit remains. The present data shows that in 25% to 30% of isschemic stroke patients sudden or late vascular cognitive disorders or vascular dementia develop [2, 6].

Summing up, both vascular changes in the brain and neurodegenerative diseases contribute to poststroke dementia. Their mutual inclinations will be frequently found in clinical use. The published clinical research does not provide encouraging information on possibilities of pharmacological prevention of poststroke dementia [5]. The risk factors control of vascular diseases and secondary preventive medicine of cerebral strokes are the key to the reduction of disorders and poststroke dementia.

In the 70s, for the first time, attention was paid to the fact that post-stroke patients hospitalised in rehabilitation departments suffer more frequent and more severe symptoms of depression than patients after orthopaedic surgeries [14]. Further research has proven that depression is the most common psychiatric complication in post-stroke patients. Depression is common after stoke, with rates, according to various authors, of 29-33% in patients within the first year after stroke [37]. Based on the review of the latest literature [5], approximately a third of stroke patients suffer depression within the first three months after the vascular event. Studies based on the extended observations has demonstrated that poststroke depression (PSD) is frequently a chronic disease with a remission-recurrent pattern [5].

PSD patients have a worse recovery prognosis, a higher risk of cognitive disorders development, which influences participation in rehabilitation in a negative way [6]. According to the biopsychosocial model, PSD is caused by biological factors (base pathophysiology of the brain and vessels), and by psychological ones, as a secondary reaction to physical, cognitive and social consequences of a stroke [6].

Early studies implied that PSD is mainly associated with the left hemisfere lesions, however, a recent review of studies and meta-analysis have found no support for this “location hypothesis” [5]. Other researchers suggest that lesions in fronto-subcortical regions are involved in develpoing PSD. They are often accompanied by impairments in executive functioning. Current interests of researchers in the role of the PSD base mechanisms are focused on the biological markers, e.g. the CRP level, neopterin concentration, hemocysteine level, deficiency of monoamine

neurotrasmitters, folic acid and B12 vitamin deficiency [5]. Other factors which can cause the PSD development include cognitive disorders, physical disability, communication problems, anxiety, lower quality of life, low social support. The study conducted by van Mierlo *et al*. [5] has revealed a wide range of psychological factors worsening depressive disorders including: passive way of coping with stress, hopelessness, nonacceptance, inablity to see advantages – catastrophisation. Risk factors predisposing to PSD are also: prior strokes and depression episodes, female gender, living alone, stressful social factors appearing prior to stroke [14, 26].

Most authors believe that the basic symptoms of PSD are: depressed mood, reduced appetite or weight loss, energy loss, insomnia and social withdrawal. Yet, thought disorder, guiltiness and lowered self-esteem, emotional oversensitivity and daily mood swings appear less often. The PSD occurrence is linked to a higher mortality rate (in the acute phase of stroke) and suicidal thoughts [14].

Since the occurence of PSD symptoms influences the patient's health in a negative way, it is recommended to start antidepressant therapy as soon as possible after the symptoms have been confirmed. So far it has not been established unequivocally which group of antidepressants is more effective in treating PSD.

Anxiety disorders are the second most frequent psychiatric stroke disorder [5]. According to various authors the percentage of patients with anxiety disorders ranges from 18-25% [37]. Research has shown that depression and anxiety are often treated as natural psychological reactions after stroke and require no treatment [40]. Thus, from the perspective of long-term outcome of stroke patients, systematic screening is essential.

The most common clinical syndromes include: anticipatory anxiety, concern, energy loss and increased muscle tone. Anxiety disorders can co-occur with depressive disorders; some authors associate it with stroke cortical lesions. Others prove that anxiety disorders in the acute phase of stroke may precede PSD symptoms later [40]. Anxiety disorders more frequently occur in patients who abused alcohol in the past [14].

Benzodiazepines are the medications most commonly used in treating anxiety disorders, treatment which should be time-limited due to their quick psychic and physical dependence. Similar efficacy to diazepine when treating PSA disorders has been proven with busiprone or with SSRI group antidepressants therapy [14].

Emotional lability is a disorder which involves the lessening of control over emotions, so that an individual suffering from it finds themselves crying uncontrollably at something that is only moderately sad (or not sad at all), or laughing in an uncontrolled way when a situation is not objectively humorous. Pathological crying results in stereotypical outbursts of crying in response to various emotional stimuli, such as joy, excitement, sadness or social situations. A sudden onset of crying or laughing occuring in response to external stimuli (eg. a physician asking about general feeling) or internal stimuli (eg. thinking about something sad or funny) in an inappropriate context [5]. There have been reports of emotional lability following a variety of central nervous system disorders (eg. multiple sclerosis, Parkinson's disease), but the condition most often follows stroke [5]. Research indicates that emotional lability syndromes such as pathological crying or laughter appear in 15% of acute phase stroke patients. According to the recent meta-analysis, among 15 individuals, 1-5 patients experienced emotional lability one month prior stroke [5].

Most researchers consider pathological crying as a sign of serotonin conduct disorders and SSRIs are used in treatment. The literature data suggests that the use of antidepressants reduces the frequency and severity of uncontrolled outbursts of crying or laughter in poststroke emotional lability.

Apathy has been defined as deep disorders of motivational aspect of any conscious goal-oriented behaviour [41]. In the literature descriptions of these symptoms have appeared in the past under different names: the loss of psychic autoactivation (LPA), psychic akinesia, aboulia, akinetic mutism, frontal adynamia, depression, dementia, etc. In 1990 Martin suggested the term *apathy or indifference syndrome* to describe disorders in the field of a broadly understood behavioral sphere, whose cause lies in the primary motivating factor deficit [5, 41]. The term has been already used widely in the subject literature (it was used earlier also in psychiatry to describe negative symptoms in patients with no brain structural lesions, eg. Schizophrenia).

Poststroke apathy occurs in 34.6% of patients within 4 months following stroke [5]. A review of current literature associates motivational deficiency disorder in behaviour with dysregualation of one or a few existing cortical-subcortical functional systems regardless of the fact which

circuit has been damaged. Three circuits, particurally significant in behaviour regulating are anatomically organised to arise and finish in different parts of the prefrontal cortex, excluding from the system the basal ganglia and thalamus. The most important for the motivating factor, thus also for initiating of any goal-oriented and conscious behaviour, is the so-called limbic circuit arising in the cingulate cortex and further including the ventral striatum part, ventral globus pallidus part and also medial dorsal thalamus parts, which give rise to projections back to the frontal part of the cingulate cortex. The limbic structures of the stratium and of the globus pallidus play a particularly important role in the neural system initiating (motivating) behaviour, and here it is important that they receive afferent information from the amygdala (engaged in emotional marking of sensory stimuli) and from the hippocampal formation responsible for, among other things, comparing new stimuli with previous experience and giving rise to projections to the basal ganglia, among other things, engaged in initiation of motor behaviour [5, 41].

Neurorehabilitation of an apathy syndrome patient causes numerous difficulties already at the therapy developing stage in relation to the essence of deficit. Apathy – especially severe – may lead to therapeutic helplessness and – apart from cognitive disorders, depression and pain – is a factor especially hindering a patient's rehabilitation process [41, 42].

From the theoretical point of view, the following can be included in the group of medications positively influencing apathy: monoamines, their precursors and antagonists of receptors for monoamines, and also medications increasing their concentration [42].

Cognitive rehabilitation

The therapy of cognitive functions disorders is based on the general assumption that the improvement of cognitive functions is a condition of reducing general disability following brain damage [33]. Good effect of treating speech disorders has been known for years [17]. The verbal communication improvement is very important, but rehabilitational interventions must be expanded including other areas of cognitive functioning, such as: attention, perception, thinking, constructing, programming, intelectual activity controls. The group of these effects is named cognitive rehabilitation therapy (CRT) and neuropsychologists play the leading role in this therapy. Their role is to establish the existing neurocognitive

deficits and to develop procedures aiming at restitution of functions or compensation-adaptation actions [32, 43].

Modern technical equipment with suitable computer software lends itself to cognitive rehabilitation [9, 21, 29, 44, 45]. It results from the clinical practice perspective that awareness of one's deficits is a crucial factor determining success in rehablitation [16]. Without realising and understanding abnormalities in one's functioning, a patient does not make an effort to correct errors, does not focus on exercise, which usually creates no expectations to reorganise the disabled activity. It can be really easily observed in patients with asonognosia the hemineglect syndrome, sensory aphasia or with the conduct and behaviour programming disorder due to frontal lesions [29].

Summary

Stroke is the second most common cause of death and the leading cause of acquired disability in adults in the developed countries (WHO) [1]. Stroke most often affects the motor functions. The neurocognitive disorders, including neuropsychiatric disorders are common and permanent poststroke consequences. They influence quality of life and long-term prognosis in a big extent. Depression occurs more frequently in poststroke patients than in general population [5]. Anxiety is also common following stroke [5], similarly to apathy syndrome [37]. The origin of these syndromes is unknown, but their prevalence suggests common base mechanisms, that is fluctuasions of neurotransmitters, inflammations or disturbed links with the limbic system. Poststroke cognitve functions disturbances are often overlooked due to the dominant character of the other 'vascular' deficits, such as: hemiparesis, hemianopsia, at least at the initial phase of disability [2, 32, 33]. It results from the clinical experience that cognitive functions disorder patients are more helpless than patients with a motor deficit who, with suitable organisation of life, can become relatively independent and, most of all, they do not lose mental and emotional attention. However, difficulties in verbal communication, reading and writing disorders, inability to remember current information or retrieve past information, difficulties in logical reasoning, inadequate emotional reactions, reduced capacity for higher-level emotions, and limited awareness of one's deficits create considerable disruptions in social functioning and in the circle of

the closest people. The awareness of the cognitive deficits rehabilitation necessity is on the rise among patients, both in the medical society and among patient's families [4, 23]. The improvement of the cognitive functioning is not the only form of psychological aid to a poststroke patient. The professional psychotherapy aimed at support, alleviating pathological emotional behaviour, insight improvement, relaxion training, patient and their relatives' psychoeducation are an essential element of the improvement process [14, 26]. Psychotherapy and cognitive therapy should not be treated just as a luxurious extra to rehabilitation, but as an integral element of comprehensive rehabilitation to improve poststroke patients' quality of life since, quoting Howard A. Rusk *We have learned to add years to human life, so we are responsible for adding life to those years...*

Neurorehabilitation is a complex process requiring comprehensive measures on various levels. Identifying, control and modification of risk factors of neurocognitive impairment patients is crucial [2, 3, 4]. It is important to introduce pharmacological and non-pharmacological forms of treatment as soon as possible. Further research on neurophysiological processes undergoing in brain injury patients and analysis of the effectiveness of employed therapeutic strategies are needed.

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