

Monitoring the effect of cognitive rehabilitation in patients with a residual type of aphasia

(scientific paper)

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Abstract: *Introduction: The main objective of this paper is to analyze the development of cognitive functions and the effect of cognitive rehabilitation on the process of brain ageing in the context of the Mild Cognitive Impairment (MCI).*

Methodology: Research has a character of a quantitative non- randomized intervention study in which the effect of cognitive rehabilitation in the sample (ALL, N = 67), aged from 18 to 70 years, is observed. The research sample was divided into three groups. The NEURO group (n = 38) included patients meeting criteria for a Mild Cognitive Impairment (MCI). The APHASIA group (n = 15) included people with an expressive type of aphasia and a brain damage. The NORM control group (n = 14) included people without a brain damage, but exhibiting a functional cognitive deficit of a mild neurocognitive impairment. A cognitive rehabilitation effect was evaluated with the Addenbrook cognitive test, a revised version, 2010 (ACE-R), and with the overall IQ score of the WAIS III neuropsychological test. A subjective change was determined with the Clinical Global Impression psychiatric scale (CGI).

Results: A statistically significant effect of cognitive rehabilitation with parameters ACE-R, IQ and a verbal fluency test was confirmed in all monitored groups. In the APHASIA clinical sample, no statistically significant effect was found in any researched verbal subtests, except of significant improvements in cognitive flexibility and verbal fluency. A non-cognitive effect, such as changes in perception in terms of reduction of anxiety and depressive symptoms, was found in the NEURO sample. A small number of valid examinations and the need to use a non-parametric test limited the acquired data.

Conclusion: The effect of cognitive rehabilitation on the development of better cognitive functioning of every person was detected and verified. There was no difference of this effect within the examined groups found.

Keywords: *cognitive rehabilitation, aphasia, MCI, ACE-R, CGI, WAIS III.*

1 Introduction

The aim of the study is to analyze the effect of cognitive rehabilitation on the semantic components in patients with residual expressive aphasia who completed the CR program without an accompanying speech and language therapy. The CR effect is also monitored in patients with a cognitive deficit on an organic basis, and in patients with a functional type of cognitive weakening.

1.1 Mild Cognitive Impairment

According to the ICD-10, the Mild Cognitive Impairment (MCI) corresponds to the diagnostic criteria of modest recognition impairment and is accompanied with changes in the size and structure of the brain. Modest recognition impairment may occur as a residual consequence of the brain structure damage due to the trauma, infections, vascular brain strokes, tumours and others. These acute cognitive deficits usually have a good prognosis in comparison with the deficits of gradually expanding cortical atrophy due to neurodegeneration. Apart from a degree of disability and injury mechanism, a young age, premorbid cognitive reserves (education), timeliness and aimed cognitive rehabilitation are important factors for a good prognosis.

1.2 Relations between aphasia and cognitive functions disorders

The language allows us to describe and express ideas, to control thoughts and actions of every person, and is a prerequisite for a variety of cognitive performance. Its demand for symbolism makes it one of the most challenging cognitive activities. One of the most important human abilities is the ability to communicate, or the conscious use of language as a system of signs and symbols in order to realize the communication plan (Hallowell & Chapey, 2008; Lechta, 2009; Šteňová & Ostatníková, 2011; Kopečný, 2014). A complete loss of already acquired communication skills is called aphasia. It is a selective disruption of language modalities and functions, which occurs in a focal brain damage, and has a negative impact on the quality of life of people with aphasia, on their relatives and caregivers; it affects social functioning and pragmatic aspects of communication (Cséfalvay & Košťálová, 2012; Papathanasiou, Coppens & Potagas, 2011).

The beginning of intensive research on the relationship between the language impairment (aphasia) and cognitive disorders dates back to the 80s of the 20th century (Vukovic, Vuksanović & Vukovic, 2008). Early supporters of the presence of non-verbal cognitive impairment in people with aphasia argued that the cognitive impairment was (at least partially) a by-product of the language disorder. This phenomenon

was attributed to weakening of the thinking process (Gianotti, 2014). Nowadays, it is thought that the language disorder is manifested by impaired cognition (Baldo, Dronkers & Wilkins et al., 2005). These claims are supported by models that specify an integral relationship between the language and other domains of the cognitive function in people with or without the language disorder (Murray, 2012).

Aphasia therapy is a long process, and the most effective results can be achieved not only by applying the evidence-based therapy (Evidence-Based Therapy), but also in teamwork which provides a holistic approach. It was proved that non-linguistic impairments of cognitive functions are a big predictor of the success of aphasia therapy (e.g. Cahana-Amitay & Albret 2015; Vallila-Rother & Kiran, 2013; Brownsett & Warren, Geranmayeh et al., 2014). Deficits in some non-linguistic cognitive areas may disrupt not only the recovery process of language skills, but also the process of coping with the disability (Seniów, Litwin & Lesniak, 2009). Overall, an individual profile of non-linguistic damage is observed, which suggests that it is not possible to predict the level of non-linguistic cognitive abilities according to the level of language skills (Helm-Estabrooks, 2002; Seniów, Litwin & Lesniak, 2009; Murray, 2012).

In terms of monitoring the functioning of cognitive functions which can be tested with the use of psychological methods, we can monitor cognitive functioning of speech production as a semantic system as a whole, its word range or the ability to create a concept. In the context of the CHC theory which assumes a cooperation of a wider range of individual functions, the disruption would be manifested in components of crystallized intelligence (the ability to form a concept and knowledge – abilities to communicate previously learned information and the ability to express an opinion fluently), and in components using a long-term memory (GLR). (Flanagan, et al., 2007). It must be mentioned that in terms of a neuroanatomic breakdown of cognitive functions, a verbal production also reflects the quality of the functioning of executive, managerial components which relate to the process of thinking. Impairment at this level may result not only in verbal fluency production, but also in coherence and content-fit.

A key precondition for the functioning of cognitive rehabilitation is based on the principle of brain plasticity (neuroplasticity), defined as a natural ability of the organism to overcome a limitation of genes, and to adapt to a rapidly changing environment. Neuroplasticity is an intrinsic property of the brain, functioning despite various mechanisms related to the age, throughout the entire human life cycle (Pascual-Leone, 2011). This adaptability is based on the principles of using the brain and cognitive reserve, which the organism has built up across its development.

Walker and Hickok (2015) observed the interaction of psycholinguistic components with components of motor control, and on the basis of the results of the interactions they created a new model of speech production in the context of aphasia,

which explained disorders functioning. They pointed out that improvement in one of the components influences an improved performance in the other. Thus, it may be assumed that a cognitive improvement using cognitive rehabilitation will improve the area of expressive speech components as a form of brain adaptation to the disease in the context of linking cognitive structures. This assumption is based on long-term memory components, a better ability to respond quickly and flexibly, and an overall effective functioning of cognitive processes as a whole.

2 Methodology

The work has a character of a quantitative non- randomized intervention study using a control clinical sample. The intervention group (clinical sample) was made up of two groups of patients (ALL, $n = 67$). The first sample were patients who met criteria for the Mild Cognitive Impairment (MCI) and had a cerebral damage, according to the CT, as a result of an acute disease (NEURO, $n = 38$). The second sample included patients with a residual type of aphasia as a result of a brain disease (APHASIA, $n = 14$). The NORM control intervention group ($n = 14$) included people without a cerebral damage, but exhibiting a cognitive deficit of the Mild Cognitive Impairment.

A cognitive rehabilitation effect was evaluated with the Addenbrook cognitive test, a revised version, 2010 (ACE-R), and with the overall IQ score of the WAIS III neuropsychological test. A subjective change of the disease and its improvement was determined with the Clinical Global Impression psychiatric scale (CGI). A level of anxiety and depressive symptoms was measured with the scale of subjective anxiety and depression – HADS (The Hospital Anxiety and Depression Scale).

The results of research were evaluated by statistical analysis and verified by the non-parametric Wilcoxon signed-rank test for paired values at the level of the significant change ($p > 0.05$), and verified by the non-parametric Sign test. Comparison of the effect between samples was verified by the One-way Kruskal-Wallis analysis non-parametric test for independent samples. The calculation was carried out with the STATISTICA Standart 12 GB statistical software.

2.1 Research sample

Based on the statistical comparison of monitored groups (NEURO, APHASIA and NORMAL), it can be stated that samples were age-matching with a similar average (median) length of education, age and problems. There was no significant rate of occurrence of anxiety or depressive symptoms in monitored samples.

Table 1: Input characteristics of the monitored sample (IQ and IVP are presented in IQ score; HADS and MMSE in HS)

	N (men/ women)	Aphasia	Age (min- max)	Education (min- max)	MMSE (min- max)	IQ (min- max)	IVP (min- max)	HADS (min- max)
ALL	67 (31/36)	15	51 (18–70)	2 (0–3)	28 (14–30)	85 (50–113)	91 (50–129)	13 (0–34)
APHASIA	14 (10/4)	14	50.5 (21–70)	2 (0–2)	27.5 (14–30)	84 (55–101)	90 (50–105)	10 (0–24)
NEURO	38 (21/17)	0	48.5 (18–70)	2 (0–3)	27 (18–30)	86.5 (50–113)	91 (56–129)	13 (0–30)
NORM	14 (7/8)	0	55 (20–69)	2 (1–3)	29 (24–30)	85 (68–107)	91 (76–123)	14 (5–34)

With regard to the initialization weakening of cognitive functioning, the input IQ scores correspond to the below-average zone with (IQ 85), according to the median value of all the monitored samples. The verbal comprehension index (VCI), referring to the ability to work with the language (from WAIS III) did not show any significant differences in indication phases of tests.

Patients of the monitored sample met diagnosis criteria based on criteria of the Mild Cognitive Impairment (DSM-5 V), and the description of computed tomography (CT), they had an appropriate medication, including the psychiatric one, before starting the rehabilitation program which was not changed throughout the course. A patient placement to the particular monitored group was performed by an independent, attested neurologist who did not know results of the psychological examination and did not have a prior diagnostic – clinical contact. Residual of aphasia was identified during a psychological examination and reviewed by a post speech and language pathologist on a basis of available medical reports.

The age between 18–70 years was the excluding criterion, MMSE < 20 points for patients without a diagnosed aphasia, sensory disturbance which would make it impossible to complete a program of cognitive rehabilitation, a long-term, pharmaceutical-resistant psychiatric comorbidity; aphasia other than that of an expressive type and a communication disorder on the basis of neurodegeneration (cognitively communication impairment). All monitored patients underwent a cognitive rehabilitation program at the Centre for Cognitive Disorders at the University Hospital in Ostrava. They were enrolled on the rehabilitation program at least three months after the occurrence of an acute illness.

3 Results and discussion

The non-parametric Wilcoxon signed- rank test for paired data was used to test the hypothesis about the significance of changes in cognitive functions in the whole sample (ALL). It verified significant changes in the monitored indexes associated with an effect on the ability to use the language; vocabulary and FLUENCY on the significance level of $p \leq 0.05$. A significant effect in the areas of cognitive functioning in the MMSE, ACE-R and IQ indexes was found in the ALL monitored sample. The monitored effect was found at a highly significant level $p \leq 0.01$. A highly significant effect in the area of non-cognitive changes ($p \leq 0.01$) in the HADS and CGI indexes was found in the ALL monitored sample (see Table 2).

Table 2: CR, ALL effect (Wilcoxon signed-rank test for paired data)

		Test		R-test			Wilcoxon		
ALL	n	Median	min-max	Median	min-max	Σ valid	W	Z-score	value - p
Vocabulary	55	9	2-15	9	2-17	37	200	-2.2856	0.022*
Information	55	9	2-17	10	3.16	37	422.5	1.0711	0.284
Details	55	8	2-14	9	3.16	44	342	-1.7855	0.0734
FLUENCY	59	8	0-14	10	0-14	42	122	-4.12	$p \leq 0.01^*$
MMSE	59	28	14-30	29	19-30	39	184	-2.8747	$p \leq 0.01^*$
ACE-R	59	84	34-97	91	43-100	57	103	-5.7484	$p \leq 0.01^*$
IQ	67	85	50-113	94	52-121	63	261.5	-5.1106	$p \leq 0.01^*$
HADS	54	13	0-34	12	0-31	40	710.5	4,0391	$p \leq 0.01^*$
CGI	63			2	0-4	63	0	-6.9009	$p \leq 0.01^*$

* $p \leq 0.05$

The non-parametric Wilcoxon signed-rank test for paired data was used to test the hypothesis about the significance of changes in cognitive functions in the monitored clinical sample (NEURO). It did not verify any significant changes at the level of importance $p \leq 0.05$ in monitored indexes associated with the effect on the ability to use the language Vocabulary, Information and Similarities. It showed a highly significant change ($p \leq 0.05$) in the FLUENCY index. A highly significant effect in the

areas of cognitive functioning in the MMSE, ACE-R and IQ indexes was found in the monitored NEURO sample. The verified effect was found at a highly significant level $p \leq 0.01$. A highly significant effect in the area of non-cognitive changes ($p \leq 0.01$) in the HADS and CGI indexes was found in the NEURO monitored group (see Table 3).

Table 3: CR, NEURO effect (Wilcoxon signed-rank test for paired data)

		Test		R-test			Wilcoxon		
NEURO	n	Median	min-max	Median	min-max	Σ valid	W	Z-score	value - p
Vocabulary	31	9	2-15	9	2-17	20	54	-1.904	0.057
Information	31	10	2.17	10	3.16	20	149	1.6426	0.101
Details	31	8	2.14	8	3.16	23	88.5	-1.5055	0.131
FLUENCY	33	8	0-14	10	0-14	25	68	-25427	0.011*
MMSE	33	27	18-30	29	22-30	23	42	-2.9198	$p \leq 0.01^*$
ACE-R	33	79	41-97	90.5	63-100	27	84.5	-3.2314	$p \leq 0.01^*$
IQ	38	86.5	50-113	95.5	52-121	36	100.5	-3.6527	$p \leq 0.01^*$
HADS	32	13	0-30	11	0-25	22	219	3.0031	$p \leq 0.01^*$
CGI	36			2	0-4	36	0	-5.2316	$p \leq 0.01^*$

* $p \leq 0.05$

The non-parametric Wilcoxon signed-rank test for paired data was used to test a hypothesis on the significance of changes in the area of cognitive functions in the entire monitored APHASIA sample. It did not verify any significant changes at the level of importance $p \leq 0.05$ in monitored indexes associated with the effect on the ability to use the language Vocabulary, Information and Similarities. (see Table 4).

Given the small number of detected changes (<10), the non-parametric Sign test was used to check the findings (see Table 5). The significance level ($p \leq 0.05$) did not verify a significant improvement in the indexes Dictionary, Information and Similarities in the monitored APHASIA sample. A highly significant change ($p \leq 0.05$) was found in the FLUENCY index. A highly significant effect in the area of cognitive functioning in ACE-R and IQ indexes was detected in the monitored APHASIA sample. The monitored effect was found at a highly significant level $p \leq 0.01$. Given the small number of detected changes (<10), the non-parametric Sign test was used for checking. It did not verify a significant change in the MMSE

index at the level of importance $p \leq 0.05$. A highly significant effect in the area of non-cognitive changes ($p \leq 0.01$) in the CGI index was found in the monitored APHASIA sample. There was no significant change verified at the monitored level of importance in the HADS index.

Table 4: CR, APHASIA effect (Wilcoxon signed-rank test for paired data)

		Test		R-test			Wilcoxon		
APHASIA	n	Median	min-max	Median	min-max	Σ valid	W	Z-score	value - p
Vocabulary	11	8	3.11	8.5	2-12	9	32	1.1255	**
Information	11	9	6.12	9.5	5-12	8	24	0.8402	**
Details	11	8	3.11	9	4-13	11	34.5	0.1334	0.89656
FLUENCY	13	5	0-11	8	1-11	10	0	-2.8031	$p \leq 0.01^*$
MMSE	13	29	14-30	29.5	19-30	9	14.5	-0.9478	**
ACE-R	13	79	34-90	86.5	43-96	12	3	-2.8241	$p \leq 0.01^*$
IQ	14	84	55-101	89	59-109	14	7	-2.8563	$p \leq 0.01^*$
HADS	12	9.5	0-24	11.5	2-26	10	27	-0.51	0.96012
CGI	14			2	1-4	15	0	-3.4078	$p \leq 0.01^*$

* $p \leq 0.05$; ** a small number of valid measurements, the result is not significant at $p \leq 0.05$

Table 5: CR, APHASIA effect (Sign test)

		Test		R-test			Sign test		
APHASIA	n	Median	min-max	Median	min-max	Σ valid	$v < V$ %	Z-score	value - p
Vocabulary	11	8	3.11	8.5	2.12	9	62.5	0.35	0.724
Information	11	9	6.12	9.5	5.12	8	57.1	0	1
Details	11	8	3.11	9	4.13	11	60	0.32	0.752
MMSE	13	29	14-30	29.5	19-30	9	75	1.06	0.289

* $p \leq 0.05$

The non-parametric Wilcoxon signed-rank test for paired data was used to test a hypothesis on the significance of changes in the area of cognitive functions in the monitored NORM sample. It did not verify any significant changes at the level of

importance $p \leq 0.05$ in monitored indexes associated with the effect on the ability to use the language Vocabulary, Information and Similarities and Fluency (see Table 6).

Given the small number of detected changes (< 10) and the insufficient number of valid measurements, the non-parametric Sign test was used to check indexes (see Table 7). The significance level ($p \leq 0.05$) did not verify a significant improvement in the Information, Similarities and Fluency indexes. The non-parametric Sign test verified a highly significant change at the level of importance ($p \leq 0.05$) in the Vocabulary index in the NORM sample. The CR effect may be assumed within this index. A highly significant effect in the area of cognitive functioning in the ACE-R and IQ indexes was detected in the monitored NORM sample. The effect in the ACE-R index was found at a highly significant level $p \leq 0.01$. It did not verify a significant change in the MMSE index at the level of importance $p \leq 0.05$. A highly significant effect in the area of non-cognitive changes ($p \leq 0.01$) in the CGI index was found in the monitored NORM sample. There was no significant change observed at the monitored level of importance in the HADS index, and given the small number of valid measurements (< 10) the finding is not significant at the monitored level of importance $p \leq 0.05$.

Table 6: CR, NORM effect (Wilcoxon signed-rank test for paired data)

		Test		R test			Wilcoxon		
NORM	n	Median	min-max	Median	min-max	Σ valid	W	Z score	value - p
Vocabulary	14	9	6.10	10	6.12	9	5	-2.0732	**
Information	14	9	6.14	11	7.16	10	14	-1.376	0.167
Details	14	8.5	6.11	10	7.12	10	12	-1.579	0.114
FLUENCY	14	10	7.14	12	10.14	7	4.5	-1.6058	**
MMSE	14	29	24-30	30	23-30	8	11	-0.5071	**
ACE R	14	88.5	75-97	94	80-98	10	0	-2.8031	$p \leq 0.01^*$
IQ	14	85	68-107	94.5	77-107	13	10.5	-2.446	0.014*
HADS	10	15	5.34	16	3.31	8	35	2.38	**
CGI	14					12	0	-3.059	$p \leq 0.01^*$

* $p \leq 0.05$; ** a small number of valid measurements, the result is not significant at $p \leq 0.05$

Table 7: CR, NORM effect (Sign test)

		Test		R-test		Sign test		
NORM	n	Median	min-max	Median	min-max	v < V %	Z-score	value - p
Vocabulary	14	9	6.10	10	6.12	88.9	2	0.046*
Information	14	9	6.14	11	7.16	80	1.58	0.114
Details	14	8.5	6.11	10	7.12	81.8	1.81	0.07
FLUENCY	14	10	7.14	12	10.14	85.7	1.51	0.131
MMSE	14	29	24-30	30	23-30	75	1.06	0.289

* $p \leq 0.05$

Using the non-parametric ANOVA KRUSKAL WALLIS test to compare medians of more than two random selections showed no difference in any of the monitored indexes, despite a relatively small number of measurements. The monitored samples (APHASIA, NORM and NEURO) can be considered identical in the range of the CR effect. This finding is verified at the level of $p \leq 0.05$ (see Table 8).

Table 8: CR effect differences in monitored samples. Kruskal-Wallis one-way ANOVA

Statistical significance (Two-tailed)	MMSEE	ACE-R	IQ	CGI	Vocabulary	Information	Details	FLUENCY	HADS
N	38	38	43	43	37	37	37	38	33
Statistical datum of exam	H(2) = 0.592	H(2) = 1.634	H(2) = 0.499	H(2) = 1.003	H(2) = 2.139	H(2) = 1.134	H(2) = 0.106	H(2) = 3.459	H(2) = 1.492
NORM	p = 0.744	p = 0.442	p = 0.779	p = 0.606	p = 0.343	p = 0.567	p = 0.948	p = 0.177	p = 0.474
APHASIA									
NEURO									

$p \leq 0.05$

3.1 Discussion

A statistically significant effect of cognitive rehabilitation on indexes primarily associated with cognitive functioning – the ACE_R and IQ indexes were verified in all monitored groups (see Table 9). An effect in the area of cognitive flexibility (fluency) was found in the whole monitored sample. Fluency in this context is understood as an ability to choose various solutions adaptively as well as an ability to move from

one solution to another fluently. Due to such effective functioning other cognitive functions, which are in the ACE-R and IQ, are used more effectively. The entire monitored sample and a control sample of patients without an identified damage of the cortical brain structure revealed a significant effect in the Vocabulary index, which in the context of using verbal components characterizes a change of abilities to recall and use words. This finding, however, in addition to improving the level of the ability to use words, also corresponds to the ability to recall words from the long-term memory components.

In a clinical sample of patients with a residual type of aphasia, no statistically significant effect in any of the monitored verbal subtests was found, except of a highly significant improvement in cognitive flexibility and verbal fluency. Nevertheless, this improvement rather reflected a more effective use of executive functions, also measured by this test, than the influence of the semantic components improvement. Even if the deficit speech disorders are sorted out, or they completely disappear due to neuroregenerative processes, there is still a significant percentage of patients whose disability persists even in chronic stages (Lendrem & Lincoln, 1985). These chronic lesions are also accompanied by complications of a broader cognitive disorder. Thus, methods of aphasia recovery vary considerably in the spectrum of patients, and the recovery process depends on the level of activation of neuroplasticity processes of the cerebral cortex. Maria Richeter et al. (2008), using a functional magnetic resonance, demonstrated that activation of specific areas of the right hemisphere can be considered a positive sign of aphasia subsiding. According to Hill et al., a bigger blood flow to the brain can give rise to at least a partial recovery of the neurological deficit. Increase in the blood flow to the brain can be caused by a pharmacological intervention but also by the activation of the organism with neurorehabilitation, including activation of cognitive and speech functions.

With regard to the overall activation of the patients, a statistically significant, subjectively experienced positive change due to CR was monitored in the entire sample.

Table 9: Summary of CR effect

	ALL	NEURO	NORM	APHASIA
Vocabulary	$p < 0.01^*$	$p < 0.01^*$	0.021	0.294
Information	0.094	0.387	0.074	0.612
Details	$p < 0.01^*$	$p < 0.01^*$	0.026	0.308
FLUENCY	$p < 0.01^*$	$p < 0.01^*$	0.035**	$p < 0.01^*$
MMSE	$p < 0.01^*$	$p < 0.01^*$	0.093	0.183
ACE-R	$p < 0.01^*$	$p < 0.01^*$	$p < 0.01^*$	$p < 0.01^*$
IQ	$p < 0.01^*$	$p < 0.01^*$	$p < 0.01^*$	$p < 0.01^*$

* $p < 0.01$, $p < 0.05$

3.2 Study limits

Limitations of the study may be influenced by the overall small number of patients in the study, and by using non-parametric tests. The size of the monitored sample and the total number of patients who did not meet required criteria, or who were not enrolled on the research or were expelled from the rehabilitation, corresponded to the current state of knowledge and a similar type of realized studies.

Another limitation of the study may be implementation of a computerized rehabilitation which was individualized on the basis of initial psychological tests and impaired cognitive domains, therefore the same battery of rehabilitation tests was not used for the entire monitored sample.

A limitation of the study may be caused by interpretation of the CT which was interpreted by different radiologists, and insufficient specification of lesions (the size). A sticking point was insufficient identification of cortical lesions incurred. The data were not available. Nor can it be assumed that some patients did not have CNS damage at the microscopic level, which also caused altered cognitive functioning.

Medication was not taken into account in the results of examination. Patients included in the study had a medication which did not change during the rehabilitation program.

In the context of the ongoing treatment in a clinical setting, it was not possible to create fully controlled conditions for monitoring the cognitive rehabilitation effect, and consequently an overall high degree of the individualization setting of the rehabilitation process, due to monitoring conditions of training individualization.

Absence of the speech and language therapy examination is the most disputable limitation within the monitored CR effect in patients with a residual of the language impairment. A diagnosis of an aphasia residual of an expressive type was additional information by an indicating neurologist. A monitored clinical group was created on the basis of this diagnosis.

A limitation of the study may be influenced by the overall small number of patients and a language impairment of the expressive type (APHASIA) and patients without an identified brain damage (NORMAL). Within these samples, no significant changes after the CR application were verified in the use of speech and language. A significant fact limiting the finding was that those areas were not significantly damaged according to the entry examinations (a lower average spectrum according to the Verbal comprehension index of WAIS III). It can be assumed a so-called misleading diagnosis of expressive aphasia.

Even though the sample monitoring the CR effect in language impairments (ALL) was divided into three groups, it showed a small number of valid changes (regarding aphasia it was impossible to complete a monitored subtest in some cases), and examinations and a number of patients in clinical studies (APHASIA; $n = 5$) corresponded

to the usual number of patients with such specific clinical diagnostic examination. The size of the monitored sample and the total number of patients who did not meet required criteria, or who were not enrolled on the research or were expelled from the rehabilitation, corresponded to the current state of knowledge and a similar type of realized studies which also used non-parametric statistical methods.

4 Conclusion

In the context of expressive aphasia, it has been detected and verified that the CR affects a development of cognitive functioning of patients, a highly significant improvement in cognitive flexibility and verbal fluency, nevertheless, without a specific speech and language therapy intervention that complements the treatment of cognitive functions through a non- pharmaceutical way, the treatment is not effective in a predictable range and treatment of these deficits should be supplemented by a specific form of a speech and language therapy intervention. This intervention should be an essential component in acute diseases as well as in cognitive communication disorders in patients with atrophic changes in the context of the Mild Cognitive Impairment and a subsequent development into dementia.

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