

Self-assessment of executive function and lateralization of brain pathology: What does the DEX-S profile show?

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Summary

The DEX-S Questionnaire is a tool often used in the self-report of executive difficulties. Numerous data demonstrate that the result of the DEX-S do not differentiate between healthy and clinical groups or people with different characteristics of brain pathology. Limited research taking into account lateralization of damage also did not provide the conclusive data. There were no relationships between the DEX-S result and the results of tasks evaluating cognitive function, including executive functions. There is an ongoing discussion on the clinical and ecological value of the DEX-S.

Aim. In the face of inconclusive data, the own study was undertaken. The objective was: (1) to compare the overall result and the DEX-S profile of healthy people and people with brain pathology including the lateralization of brain pathology; and (2) determining the relationships between the result of the DEX-S and the level of selected cognitive competences. 115 people were enrolled in the study, including people without brain pathology (C; $N = 74$), people with damage to the left hemisphere (LH; $N = 6$), people with damage to the right hemisphere (RH, $N = 12$) and people with damage to both hemispheres (BH, $N = 23$).

Method. In the research the DEX-S, WAIS subtests: Vocabulary, Digit span forward and backwards, the MoCA test and the Affect Scale from the ProCog Questionnaire were applied.

Results. The DEX-S overall result did not differentiate the groups. However, the RH and BH groups obtained the highest average scores and the LP group – the lowest. There were intergroup differences in the results of only a few DEX-S items. Patients with right and both hemispheres pathology reported a significantly higher level of difficulties in attention, greater susceptibility to distractors, deficits in planning, sequential operation and problem solving. The RH group performed poorer in tasks involving cognitive functions in comparison to other patients. All clinical groups differed from healthy persons in terms of results of tests/tasks evaluating selected cognitive functions. There were positive correlations between the DEX-S score and sense of anxiety, no association with age, and incidental correlations with the results of cognitive tasks in each research group.

Conclusions. The results suggest that mechanisms of sense of executive deficits depend on lateralization of pathology. The higher sense of executive deficits in people with right

hemisphere pathology may be due to the efficiency of delayed memory, and may reflect an adequate self-assessment of own competence. The low DEX-S result of the group with the left hemisphere pathology may result from reduced, despite the absence of aphasia, language/semantic skills and not from the lack of insight into executive deficits.

Key words: Dysexecutive Questionnaire – Self (DEX-S), self-assessment of executive functions, lateralization of brain damage.

Introduction

The term “executive functions” (EF) defines processes for planning, controlling and correcting the behavior [1]. The models proposed by the authors [1–6] accentuate different components of EF, but researchers agree that EF are essential for proper cognitive, emotional and behavioral functioning. Executive functions deficits (EFD) manifest themselves in the form of disorganized behavior in different domains of human functioning, including the disruption of self-awareness, interpersonal communication, professional activity, and everyday life [7, 8]. Neural basis of EF is a complex cortical-subcortical network, including, among others, prefrontal cortex (dorsolateral – DLPFC, orbitofrontal – OFC, ventromedial prefrontal cortex – vmPFC) [9, 10], the anterior cingulate cortex (ACC) [11], thalamus, basal ganglia [12], and the cerebellum [13, 14]. Because of extensive neuronal loop, EFD are recognized not only in patients with the frontal and frontal-subcortical pathology [15, 16] but also with the pathology covering the areas posterior to the central sulcus [17] and in many other clinical syndromes of different etiology and characteristic of brain dysfunction [18].

Because EFD are a common consequence of brain damage which interferes with the self-reliance of patients, standardized psychological instruments to objective measure and self-assessment of EF are being improved. These tools, including self-report tools, should have a functional and ecological value which is capable of predicting whether and to what extent/which domains of cognition and behavior reveal executive deficits and disruptions of insight. The data show that some patients with EFD perform routine tasks involving EF just like healthy people, but experience many difficulties in everyday life [6, 19]. This is due to the low ecological value of some techniques (e.g., discussion on the WCST – Wisconsin Card Sorting Test) [4, 6, 20] because they do not reflect natural conditions. A similar discussion also applies to methods that require self-assessment of EF [21]. Among them the most well-known tool is the DEX (*Dysexecutive Questionnaire*) [4, 22], which is an element of the BADS (*Behavioral Assessment of the Dysexecutive Syndrome*) [4, 22]. It is believed that the DEX is sensitive to EFD and it also has ecological value [23, 24]. There are at least 2 versions (DEX-Self/S and DEX-Other/O), and sometimes 3 versions are proposed (DEX-S, DEX-O, DEX-Clinician/C) [25]. In the Self version, the examined person is asked to self-assess the severity of difficulties in performing activities involving EF, while the DEX-O/C version is filled in by a close person/clinician. Information on the level of insight into own executive deficits can be obtained by comparing self-reports and data from methods that objectively assess EF (tests, observation of behavior and/or assessments made by loved ones – so-called index of insight) [23].

Originally the DEX and its variants were mainly used in the diagnosis of patients with the frontal lobes pathology [22], however, due to the frequent occurrence of EFD in other clinical groups, its use has gone beyond the traditional range of applications. Despite the popularity of this tool, its psychometric value is debated. Part of the data showed that the overall result of the DEX-S correlates with the results of the executive tests (in groups with pathology of the prefrontal area) [23], other data do not confirm such a relationship [26]. According to Chan [24], the results obtained in the DEX-S can reflect not only behavioral symptoms typical for EFD but also the symptoms associated (or not) with EFD. This explains the lack of difference in the result of healthy people and people with brain damage and EFD symptoms. Another reason is the multi-variable conditioning of sense of EFD in both clinical and non-clinical groups: age, intensity of positive or negative affect, level of education [27].

Although EFD grows with age [28], paradoxically, the older age promotes better judgment of one's executive functions together with a higher level of positive affect, cognitive functions, language competences, female gender, and a higher sense of health. Sense of executive difficulties in young people results from the higher intensity of negative affect, which decreases with age [29]. However, the latest research shows that a healthy person reporting various types of cognitive complaints (subjective cognitive decline –SCD) perform executive tasks at a lower level. This may suggest that a sense of executive difficulties and the actual poor EF capabilities underlie the overall sense of cognitive deficits [30].

People with brain pathology often believe that they have lower executive efficiency. As mentioned above, various reports do not show differences in the DEX-S between healthy people and people with brain pathology [31], therefore work on improving the accuracy and reliability of the questionnaire in the form of factor analyzes [32, 33] and modification of the classic version of the DEX is being continued [34, 35]. A 4-factor structure of the DEX (emotional and personality, motivation, behavioral, and cognitive disorders) is recommended [36]. Other works suggest 1-factor [37], 3-factor (self-regulation, metacognition, cognition) [33] or 4-factor (inhibition, intentionality, social behavior, and abstract thinking) structure, referring to the proposal of Burgess et al. [23] (inhibition, intentionality, executive memory, positive and negative affect). However, the data concerning the possibility of differentiation and elucidation of the structure and mechanisms of a sense of EFD are still not consistent. Some authors indicate that the DEX-S result/profile does not differentiate between people with a different localization of pathology within the prefrontal area [38], or that some items have a weaker diagnostic value [39]. Other studies show characteristic profiles of complaints which are different for pathology in the orbitofrontal area (OFC), dorsolateral prefrontal cortex (DLPFC) and anterior cingulate cortex (ACC) [39]. Few analyses consider the links between the lateralization of pathology (including not only the frontal lobes) and the DEX-S result/profile. Slachevsky et al. [39], analyzing the data of patients with damage to the right, left or both frontal lobes, showed that patients with bilateral pathology reported significantly more severe complaints, while people with damage to the left and right frontal lobe did not differ in this scope. Van Rijsbergen et al. [40, 41], on the basis of a review of research, indicate that the localization and lateralization of stroke

do not differentiate types of cognitive complaints, including EFD, and differences were related only to the severity of complaints of people after stroke and healthy people. In conclusion, the DEX-S utility assessment in clinical diagnosis is not conclusive, and the research we have undertaken is an attempt to engage in discussion.

Material and method

In reference to literature data, the purpose of our research was: (a) to compare the DEX-S scores obtained by healthy people and people with lateralized pathology of the brain; and (b) to determine the relationship between the DEX-S result and the selected cognitive, emotional, and age variables. The study was approved by the local Ethical Committee (1/2016). All participants provided written informed consent to participate in the study. People with aphasia, hemianopsia and other deficits which make completing the questionnaire impossible, as well as people addicted to alcohol and/or other psychoactive substances and those with the history of mental, neurological or somatic illness, were excluded from the research. Patients were examined by a clinical psychologist in the second week of their stay in the department of neurology. The examination of healthy subjects (volunteers) was carried out by a clinical psychologist at a scientific institution. After considering the inclusion and exclusion criteria, the statistical analysis used data from 115 people, including 58 women (50.4%) and 57 men (49.6%). All persons were right-handed, for all participants Polish was the first language. 11.9% of the participants had primary education, 58% – secondary education, 30.1% – higher education. The subjects did not differ in age (Table 1).

On the basis of the interview, questionnaires and, in the case of people with brain pathology, medical history (including neuroimaging data, i.e., CT – computed tomography and/or MRI – magnetic resonance imaging) 4 groups were identified: persons without a neurological history (control group/C = 74), persons with pathology in the left hemisphere (LH, $N = 6$), right hemisphere (RH, $N = 12$) and both hemispheres (BH, $N = 23$). The etiology of damage was varied – mainly vascular diseases; cranio-cerebral trauma and multiple sclerosis occurred incidentally. Patients whose neuroimaging studies showed structural changes in both hemispheres (87% of patients – vascular etiology; 8.7% – MS; 4.4% – head injury) were qualified to the BH group, while patients with changes limited to one hemisphere were qualified to the LP group (100% of patients – vascular etiology) and the PP group (92% of patients – vascular etiology; 1 person after cranio-cerebral injury), respectively. Due to the size of the lesions, which typically include the frontal, parietal and temporal lobes, the criterion of localization of pathology in the “anterior – posterior area” was not applied.

The following methods were used:

1. The Montreal Cognitive Assessment Scale (MoCa), designed to assess the overall level of cognitive function (max. score 30 points), which includes tasks/subcategories concerning short-term memory, visual-spatial, executive and linguistic functions, verbal fluency, attention, naming, abstraction, and allopsychic orientation [42]. The analysis included both the total score and subscales scores.

2. Affect Scale (AFF) from the ProCog Questionnaire (Patient Reported Outcomes in Cognitive Impairment) to assess the severity of anxiety associated with a sense of cognitive difficulties. The examined person has to choose a response on a Likert scale; lower score means no anxiety, while a higher score – higher level of anxiety associated with a sense of cognitive difficulties (from 0 points/never to 4 points/always). The results on this scale range from 0 points (no anxiety) to 44 points (high level of anxiety) [43].
3. The DEX-S (Dysexecutive Questionnaire/Self) to self-assessment of the intensity of executive difficulties [22]. An experimental version using back translation was used due to the lack of Polish adaptation.
4. The WAIS-R PL subtests: Vocabulary (to assess language competences/semantic knowledge), Digit span forward and backwards – to assess verbal-auditory memory, working memory, cognitive flexibility, and attention [44].

The study involved 2 meetings to minimize factors (e.g., fatigue or anxiety) that could affect the test results; the DEX-S was presented at the end. Data were analyzed using SPSS IMAGO v 22. Because the variables had a normal distribution (the Shapiro-Wilk test), intergroup and correlation analyzes were performed using parametric tests. The significance level was set at $p \leq 0.05$.

Results

The characteristics of the results obtained by the subjects are shown in Table 1. In all tests/cognitive tasks, healthy individuals obtained significantly higher results compared to RH, LH and BH groups. In spite of the similarity of results of clinical groups, patients from the RH group obtained the lowest results (except for the Vocabulary – the lowest result in the LH group). The highest (medium) level of anxiety was observed in the RH and BH groups (although the results of the AFF were within the limits of the low – average results), the lowest level of anxiety was in the C and LH group.

Table 1. Group characteristics (mean *M*, standard deviation *SD*, comparisons between groups ANOVA and post-hoc)

Variables	M (SD) BH (N = 23)	M (SD) LH (N = 6)	M (SD) RH (N = 12)	M (SD) C (N = 74)	F (p)	Post-hoc comparisons (Tukey's test)
Age	62.7 (15.39)	61.8 (7.52)	64.7 (8.8)	59.8 (9.06)	2.1 (0.08)	
Vocabulary WAIS-PL	32.9 (17.5)	20.2 (12.8)	24.0 (10.7)	48.1 (11.5)	25.22*** (0.001)	BH = LH = RH < C (0.000)
Digit span forward WAIS PL	5.4 (1.9)	5.3 (1.9)	4.9 (0.79)	6.7 (2.0)	7.05*** (0.001)	BH = LH = RH BH < C (0.012) LH = C RH < C (0.012)

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Digit span backwards WAIS PL	4.0 (2.57)	4.0 (1.41)	3.75 (1.14)	6.5 (2.38)	11.17*** (0.001)	BH = RH < C (0.003) LH = C
Moca – total score	22.9 (4.4)	22.3 (3.8)	21.83 (2.69)	27.8 (2.59)	24.37*** (0.001)	BH = LH = RH BH < C (0.001) LH < C (0.002) RH < C (0.001)
AFF	15.1 (9.34)	6.67 (10.9)	17.25 (12.49)	7.8 (8.16)	6.92*** (0.000)	BH = RH > LH (0.008) BH > C (0.007) BH > C (0.004)

*** $p \leq 0.001$

To compare the results of the DEX-S items in 4 groups, the test of significance of differences for $k > 2$ groups (parametric ANOVA). The data are shown in Table 2 and Figure 1.

Table 2. The DEX-S – response profile in 4 groups (mean *M*, standard deviation *SD* and comparisons between groups – ANOVA and post-hoc)

Items Range of points 0–4	Function ¹	M (SD) BH (N = 23)	M (SD) LH (N = 6)	M (SD) RH (N = 12)	M (SD) C (N = 74)	F (p)	Post-hoc comparisons (Tukey's test)
I have problems in understanding what other people mean unless they keep things simple and straightforward.	abstract thinking	1.17 (1.19)	0.67 (0.82)	1.67 (1.43)	0.97 (0.90)	1.95 (0.13)	-
I act without thinking, doing the first thing that comes to mind	impulsiveness	1.09 (1.08)	1.17 (1.60)	0.75 (0.87)	1.15 (0.89)	0.62 (0.60)	-
I sometimes talk about events or details that never actually happened but I believe did happen.	confabulation	0.83 (1.15)	0.17 (0.41)	0.42 (0.67)	0.81 (0.83)	1.82 (0.15)	-
I have difficulty thinking ahead or planning for the future.	planning deficits	1.30 (1.18)	0.83 (0.98)	1.67 (1.30)	1.12 (1.11)	1.12 (0.34)	-
I sometimes get overexcited about things and can be a bit over the top at these times.	euphoria	1.30 (1.39)	1.50 (0.84)	1.75 (1.36)	0.9 (1.00)	2.66 [*] (0.05)	LH = BH = RH RH > C (0.04)

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I get events mixed up with each other and get confused about the correct order of events	deficit in determining the sequence of events	1.0 (1.0)	0.5 (0.84)	1.25 (1.29)	0.95 (1.02)	0.72 (0.54)	-
I have difficulty realizing the extent of my problems and am unrealistic about the future.	lack of insight	1.17 (1.30)	0.33 (0.82)	1.08 (1.56)	1.13 (1.09)	0.89 (0.45)	-
I seem lethargic and unenthusiastic about things.	apathy	1.78 (1.17)	0.5 (0.84)	1.75 (1.05)	0.86 (1.00)	4.33** (0.006)	BH > LH (0.05) BH > C (0.03) BH = RH LH = C
I do or say embarrassing things when in company of others.	lack of inhibition in social relations	0.83 (1.11)	0.17 (0.41)	0.75 (1.05)	0.88 (1.04)	0.91 (0.44)	-
I really want to do something one minute but couldn't care less about it the next.	variability of motivation	0.90 (1.12)	0.67 (0.82)	0.75 (1.05)	1.30 (1.08)	2.0 (0.12)	-
I have difficulty showing emotion.	poor affection	1.22 (1.32)	0.33 (0.82)	0.83 (1.27)	1.06 (1.18)	0.99 (0.39)	-
I lose my temper at the slightest thing.	aggression	0.91 (0.85)	1.50 (1.05)	1.25 (0.96)	1.16 (1.22)	0.55 (0.65)	-
I seem unconcerned about how I should behave in certain situations.	disinterest	1.13 (1.25)	0.17 (0.41)	0.67 (0.89)	1.00 (1.13)	1.53 (0.21)	-
I find it hard to stop repeating, saying or doing things once started.	perseveration	0.74 (1.05)	0.17 (0.41)	0.67 (0.98)	1.12 (1.25)	1.93 (0.13)	-
I tend to be very restless, and I can't sit still for any length of time.	anxiety – hyperkinesia	1.04 (1.4)	0.83 (0.98)	0.58 (1.16)	0.95 (0.92)	0.52 (0.67)	-
I find it difficult to stop doing something even if I know I shouldn't do it	inhibition deficit	0.83 (1.07)	0.33 (0.52)	0.58 (0.99)	0.95 (0.99)	1.09 (0.36)	-
I will say one thing but will do something different.	dissociation between knowing and doing	0.87 (1.29)	0.17 (0.41)	0.92 (0.79)	0.86 (0.94)	0.96 (0.41)	-

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I find it difficult to keep my mind on something and am easily distracted.	susceptibility to distraction and attention disorders	0.91 (1.12)	1.33 (1.03)	1.83 (1.69)	0.77 (0.66)	4.49** (0.005)	BH = LH BH = C LH = RH LH = C BH < RH (0.04) RH > C (0.003)
I have trouble making decisions or deciding what I want to do.	problem solving deficit	1.13 (1.14)	1.33 (1.03)	1.67 (1.83)	1.21 (0.83)	0.79 (0.49)	-
I am unaware of, or unconcerned about, how others feel about my behavior.	lack of interest in social roles	0.61 (0.98)	0.50 (0.84)	1.17 (1.53)	0.21 (0.44)	6.39*** (0.001)	BH = LH = RH RH > C (0.001) C = BH C = LH
DEX total score		21.6 (15.70)	13.33 (13.10)	21.83 (16.64)	19.40 (12.01)	0.73 (0.54)	-

¹ [based on 29, 41]; * $p \leq 0.01$; ** $p \leq 0.01$; *** $p \leq 0.001$

Groups do not differ significantly in the DEX-S total score which is below the average score of the test (i.e., less than 40 points). The highest average score was obtained by the RH group (27.3% of the possible result) and the BH group (27%), slightly lower – by the C group (24%), the lowest – by the LH group (16.6%). Sig-

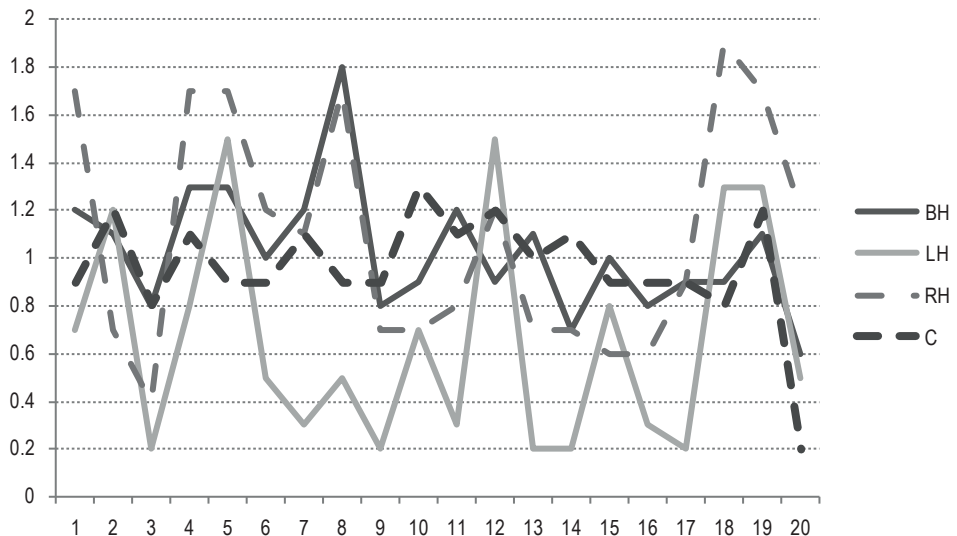


Figure. 1. DEX-S profile responses in 4 groups

nificant differences between groups were revealed only in the case of a few items (Figure 1, Table 2). Compared to the other groups, the RH (sometimes BH) group had a greater sense of euphoria, apathy, attention deficit, susceptibility to distraction, and declared less interest in social situations. The LH group pointed mainly to the presence of impulsivity and aggression, the BH group – to apathy, while healthy individuals emphasized the sense of perseverance, impulsivity, inability to inhibition, motivational fluctuations, and tendency to confabulate.

The second aim of our study was to analyze the correlation between the DEX-S results and the other variables in each group. Correlation results are presented in Table 3.

Table 3. Correlations between the DEX S result and other variables (Pearson's r)

Variables x DEX S	BH group r (p)	LH group r (p)	RH group r (p)	C group r (p)
WAIS-Vocabulary	0.14 ns	0.70 [†] (0.06)	0.35 ns	-0.05 ns
WAIS-Digit span forward	-0.14 ns	-0.13 ns	0.26 ns	-0.03 ns
WAIS-Digit span backwards	-0.31 ns	0.22 ns	-0.29 ns	-0.06 ns
MoCa total score	0.21 ns	0.42 ns	0.32 ns (MoCA subscale – Delayed recall) 0.73** (0.003)	-0.06 ns
Age	-0.13 ns	-0.60 ns	-0.28 ns	-0.001 ns
AFF	0.52** (0.002)	0.86** (0.01)	0.73** (0.004)	0.65*** (0.001)

[†] – statistical trend; ** $p \leq 0.01$; *** $p \leq 0.001$.

There was no correlation between, consecutively, age, Digit span forward and backwards score, the MoCA total score and subscales scores, and the DEX-S in groups. However, there were positive correlations between the higher level of anxiety (AFF) and the higher DEX-S score in each group. It was found that the higher the score in the MoCA Delayed recall subscale, the higher the sense of executive dysfunction in the RH group, and the better the language efficiency (WAIS Dictionary), the higher the sense of EFD in the LH group (correlation *on the borderline of statistical significance*).

Discussion

The mean DEX-S total scores for healthy and clinical subjects were low and similar to those reported in other studies. The lack of significant difference in the DEX-S between groups [22, 31, 32, 45, 46] was also in line with other authors' data. The low DEX-S scores can be explained by the presence of psychological mechanisms activated in the acute/subacute phase of the illness when the patient is focused on current problems, such as motor or existential ones, rather than on cognitive problems. The confrontation with the difficulties in the distant phase of the illness might have

increased the sense of deficit or, in the absence of insight, reduced it [45]. Other explanation for the low DEX-S performance and lack of difference between the groups was associated with the ambiguity of the phrases used in items. Judgments about the tendency to increased impulsivity or confabulation might have resulted from an individual understanding of these terms [29]. The content of the items is related to behavior that affects most people regardless of the clinical context, and do not have to be (or is incidentally) associated with actual cognitive/executive competences [e.g., 15, 24, 28] but rather with negative emotions [e.g., 32, 33].

Analysis of the DEX-S total score, items scores and correlation analysis showed that the DEX-S result might have reflected the mechanisms of self-assessment of executive functions which are dependent on the nature of the brain pathology/brain state. Patients with right hemisphere pathology, in comparison to other patients, reported a more convincing belief about EFD and also obtained low results in tests. This result is not consistent with numerous studies that have shown a link between the right hemisphere damage and anosognosia, which is the lack (total, partial) of awareness of one's own limitations [47], and, therefore, a mood inadequate to the situation [48]. On the other hand, other reports have shown that these patients experience anxiety or anger, they also experience depression even more often than people with lesions in the left hemisphere [49, 50]. According to Schacter [51], negative emotions might have occurred (as a result of unconscious processes) despite the lack of insight into deficits or anosognosia might have taken the discrete/partial character.

Reporting EFD and anxiety by these patients can be explained by the involvement of the right hemisphere in the construction of Self [52], the organization of executive functions [53], attention [54, 55], regulation of emotions [56]. This hemisphere mediates the recall of autobiographical data, in particular those of emotional nature [57], attribution processes referring to self [52] and the regulation of emotions by selecting different strategies depending on external conditions [57]. This is supported by the right-hemispheric representation of executive processes responsible for the arousal control, the processes of external orientation, and the control and monitoring of metacognitive processes [55]. Dysfunction of this hemisphere disrupts the effective strategies for regulating emotions leading to high levels of anxiety and a sense of cognitive deficits.

Different results were obtained in patients with left hemisphere pathology: the DEX-S scores and items scores were low, similar as the intensity of anxiety. Persons with pathology of this hemisphere often present depressive mood but it usually accompanies aphasia [50], which was an exclusion criterion for participation in our study. Dysfunction of the left hemisphere weakens the processes of inner speech and introspection [58], disturbs the cognitive flexibility and attention to verbal stimuli [53]. Therefore, it is possible that lower efficiency of language/semantic competencies in LH group hampers the DEX-S performance and explains the low score. However, it does not authorize to say that it is an indicator of lack of insight into one's own functioning.

The results of the healthy people also deserve some comments. Similarly to the reports by other authors, the DEX-S total score was similar to scores obtained by clinical groups (here: RH and BH). However, the determinants of the sense of EFD

were different, they included personality variables and other variables uncontrolled in this study [59].

The own research has some limitations. These are as follows: small number of study participants, differential etiology of brain damage and the inability to take into account the localization of pathology in the “anterior-posterior area” of the brain. In subsequent studies it is worth to use other methods of cognitive functions evaluation and self-evaluation of executive functions as well as descriptive versions for relatives. It is also worthwhile to refine the DEX-S factor analysis based on the EF models. Conclusion on the sense of EFD and its adequacy on the basis of the DEX-S score requires particular caution due to multivariable conditions, lack of clear definition and indicators of sense of dysfunction.

Conclusions

1. The DEX-S total score did not differentiate between healthy people and people with different lateralization of brain pathology. Despite this, patients with damage to the right and both hemispheres reported, on average, the highest sense of executive difficulties, healthy people reported lower sense of executive difficulties and patients with the left hemisphere pathology – the lowest. Significant intergroup differences occurred occasionally, i.e., in the case of only a few items.
2. Patients with damage to the RH and BH reported a sense of difficulty in a variety of areas: emotions, attention (susceptibility to distractors), social functioning, abstract thinking, planning, sequential activity, and problem solving. LH results in the subsequent items were similar, except for the sense of impulsivity and aggressiveness.
3. Patients from the clinical groups (LH, RH, BH) obtained significantly lower scores in cognitive assessment tests compared to healthy persons.
4. The higher DEX-S score, irrespective of the group, correlated with higher level of anxiety.
5. Incidental correlations between the DEX-S score and the level of language competences (LH) and delayed recall (RH) were observed. Higher efficiency of these competences favored a higher sense of executive difficulties. It is therefore necessary to consider the different cognitive determinants of the sense of executive difficulties (and/or its components) depending on the clinical state of the patients, including the lateralization of brain pathology.
6. Higher level of the sense of executive deficits in patients with pathology of the right hemisphere associated with cognitive difficulties may reflect the adequacy of insight into current limitations. The low scores obtained in the group with left hemisphere pathology do not suggest the lack of awareness of EFD because they result rather from reduced linguistic/semantic performance impeding the DEX-S performance.
7. The profile analysis creates more possibilities to describe the sense of executive deficits than the DEX-S total score.

References

1. Burgess PW. *Theory and methodology in executive function research*. In: Rabbitt P. ed. *Theory and methodology of frontal and executive function*. Hove, U.K.: Psychology Press; 1997. p. 81–116.
2. Stuss D, Knight R. ed. *Principles of frontal lobe function*. Oxford: Oxford University Press; 2002.
3. Luria AR. *The working brain: An introduction to neuropsychology*, translated by B. Haigh. New York: Basic Books; 1973.
4. Jodzio K. *Neuropsychologia intencjonalnego działania. Koncepcje funkcji wykonawczych*. Warsaw: Scholar Publishing House; 2008.
5. Stuss DT, Alexander MP. *Is there a dysexecutive syndrome?* Philos. Trans. R. Soc. Lond. B. Biol. Sci. 2007; 362(1481): 901–915.
6. Chan RC, Shum D, Touloupoulou T, Chen EY. *Assessment of executive functions: Review of instruments and identification of critical issues*. Arch. Clin. Neuropsychol. 2008; 23(2): 201–216.
7. Jodzio K, Biechowska D, Szurowska E, Gąsecki D. *Profilowa analiza dysfunkcji wykonawczych w diagnostyce neuropsychologicznej osób po udarze mózgu*. Roczniki Psychologiczne. 2012; 15(3): 83–100.
8. Stuss DT. *Functions of the frontal lobes: Relation to executive functions*. J. Int. Neuropsychol. Soc. 2011; 17(5): 759–765.
9. Yuan P, Raz N. *Prefrontal cortex and executive functions in healthy adults: A meta-analysis of structural neuroimaging studies*. Neurosci. Biobehav. Rev. 2014; 42: 180–192.
10. Alvarez JA, Emory E. *Executive function and the frontal lobes: A meta-analytic review*. Neuropsychol. Rev. 2006; 16(1): 17–42.
11. Stuss DT, Levine B. *Adult clinical neuropsychology: Lessons from studies of the frontal lobes*. Annu. Rev. Psychol. 2002; 53: 401–433.
12. Ardila A. *There are two different dysexecutive syndromes*. J. Neurol. Disord. 2013; 1: 1. <http://dx.doi.org/10.4172/2329-6895.1000114>.
13. Bellebaum C, Daum I. *Cerebellar involvement in executive control*. Cerebellum. 2007; 6(3): 184–192.
14. Mak M, Tyburski E, Madany Ł, Sokolowski A, Samochowiec A. *Executive function deficits in patients after cerebellar neurosurgery*. J. Int. Neuropsychol. Soc. 2016; 22(1): 47–57.
15. Koerts J, Beilen van M, Leenders KL, Brouwer WH, Tucha L, Tucha O. *Complaints about impairments in executive functions in Parkinson's disease: The association with neuropsychological assessment*. Parkinsonism Relat. Disord. 2012; 18(2): 194–197.
16. Sitek E, Sołtan W, Wieczorek D, Schinwelski M, Robowski P, Harciarek M et al. *Self-awareness of executive dysfunction in Huntington's disease: Comparison with Parkinson's disease and cervical dystonia*. Psychiatr. Clin. Neurosci. 2013; 67(1): 59–62.
17. Binetti G, Magni E, Padovani A, Cappa SF, Bianchetti A, Trabucchi M. *Executive dysfunction in early Alzheimer's disease*. J. Neurol. Neurosurg. Psychiatry. 1996; 60(1): 91–93.
18. Loschiavo-Alvares FQ, Sediyaama CYN, Vasconcelos AG, Neves F, Corrêa H, Malloy-Diniz LF et al. *Clinical application of DEX-R for patients with bipolar disorder type I and II*. Clinical Neuropsychiatry. 2013; 10(2): 86–94.
19. Shallice T, Burgess PW. *Deficits in strategy application following frontal lobe damage in man*. Brain. 1991; 114(Pt 2): 727–741.

20. Burgess PW, Alderman N, Forbes C, Costello A, Coates LM, Dawson DR et al. *The case for the development and use of “ecologically valid” measure of executive function in experimental and clinical neuropsychology.* J. Int. Neuropsychol. Soc. 2006; 12(2): 194–209.
21. Biederman J, Petty CR, Fried R, Fontanella J, Doyle AE, Seidman LJ et al. *Can self-reported behavioral scales assess executive function deficits? A controlled study of adults with ADHD.* J. Nerv. Ment. Dis. 2007; 195(3): 240–246.
22. Wilson BA, Alderman N, Burgess P, Emslie H, Evans J. *Behavioural assessment of the dysexecutive syndrome.* Bury St Edmunds, England: Thames Valley Test Company; 1996.
23. Burgess PW, Alderman N, Evans J, Emslie H, Wilson BA. *The ecological validity of tests of executive function.* J. Int. Neuropsychol. Soc. 1998; 4(6): 547–558.
24. Chan RC. *Dysexecutive symptoms among a non-clinical sample: A study with the use of the Dysexecutive Questionnaire.* Br. J. Psychol. 2001; 92(Pt 3): 551–565.
25. McGuire BE, Morrison TG, Barker LA, Morton N, McBrinn J, Caldwell S et al. *Impaired self-awareness after traumatic brain injury: Inter-rater reliability and factor structure of the Dysexecutive Questionnaire (DEX) in patients, significant others and clinicians.* Front. Behav. Neurosci. 2014; 8: 352. Doi: 10.3389/fnbeh.2014.00352.
26. Wood RL, Liossi Ch. *The ecological validity of executive tests in a severely brain injured sample.* Arch. Clin. Neuropsychol. 2006; 21(5): 429–437.
27. Azouvi Ph, Vallat-Azouvi C, Millox V, Darnoux E, Ghout I, Azerad S et al. *Ecological validity of the Dysexecutive Questionnaire: Results from the Paris-TBI study.* Neuropsychol. Rehabil. 2015; 25(6): 864–878.
28. Amieva H, Phillips L, Della Sala S. *Behavioral dysexecutive symptoms in normal aging.* Brain Cogn. 2003; 53(2): 129–132.
29. Gerstorff D, Siedlecki KL, Tucker-Drob EM, Salthouse TA. *Executive dysfunctions across adulthood: Measurement properties and correlates of the DEX Self-Report Questionnaire.* Neuropsychol. Dev. Cogn. B. Aging Neuropsychol. Cogn. 2008; 15(4): 424–445.
30. Stenfors CUD, Marklund P, Magnusson Hanson LL, Theorell T, Nilsson L-G. *Subjective cognitive complaints and the role of executive cognitive functioning in the working population: A case-control study.* PLoS ONE. 2013; 8(12): e83351. Doi:10.1371/journal.pone.
31. Liebermann D, Ostendorf F, Kopp UA, Kraft A, Bohner G, Nabavi DG et al. *Subjective cognitive-affective status following thalamic stroke.* J. Neurol. 2013; 260(2): 386–396.
32. Mooney B, Walmsley C, McFarland K. *Factor analysis of the self-report Dysexecutive (DEX-S) Questionnaire.* Appl. Neuropsychol. 2006; 13(1): 12–18.
33. Simblett SK, Bateman A. *Dimensions of the Dysexecutive Questionnaire (DEX) examined using Rasch analysis.* Neuropsychol. Rehabil. 2011; 21(1): 1–25.
34. Chaytor N, Schmitter-Edgecombe M, Burr R. *Improving the ecological validity of executive functioning assessment.* Arch. Clin. Neuropsychol. 2006; 21(3): 217–227.
35. Simblett SK, Ring H, Bateman A. *The Dysexecutive Questionnaire Revised (DEX-R): An extended measure of everyday dysexecutive problems after acquired brain injury.* Neuropsychol. Rehabil. 2017; 27(8): 1124–1141. Doi: 10.1080/09602011.2015.1121880.
36. Stuss DT, Benson D. *The frontal lobes.* New York: Raven Press; 1986.
37. Pedrero-Pérez EJ, Ruiz-Sánchez-de-León JM, Winpenny-Tejedor C. *Dysexecutive Questionnaire (DEX): Unrestricted structural analysis in large clinical and non-clinical samples.* Neuropsychol. Rehabil. 2015; 25(6): 879–894.
38. Bodenburg S, Dopsch N. *The Dysexecutive Questionnaire Advanced: Item and Test Score Characteristics, 4-Factor Solution, and Severity Classification.* J. Nerv. Ment. Dis. 2008; 196(1): 75–78.

39. Slachevsky A, Peña M, Pérez C, Bravo E, Alegria P. *Neuroanatomical basis of behavioral disturbances in patients with prefrontal lesions*. Biol. Res. 2006; 39: 237–250.
40. Rijsbergen van MW, Mark RE, Kort de PL, Sitskoorn MM. *Subjective cognitive complaints after stroke: A systematic review*. J. Stroke Cerebrovasc. Dis. 2014; 23(3): 408–420.
41. Rijsbergen van MW, Mark RE, Kort de PL, Sitskoorn MM. *Prevalence and profile of poststroke subjective cognitive complaints*. J. Stroke Cerebrovasc. Dis. 2015; 24(8): 1823–1831.
42. Nasreddine ZS, Phillips NA, Bédirian V, Charbonneau S, Whitehead V, Collin I et al. *The Montreal Cognitive Assessment, MoCA: A brief screening tool for mild cognitive impairment*. J. Am. Geriatr. Soc. 2005; 53(4): 695–699.
43. Frank LJ, Flynn JA, Kleinman L, Margolis MK, Matza LS, Beck C et al. *Validation of a new symptom impact questionnaire for mild to moderate cognitive impairment*. Int. Psychogeriatr. 2006; 18(1): 135–149.
44. Brzeziński J, Gaul M, Hornowska E, Jaworowska A, Machowski A, Zakrzewska M. *Skala Inteligencji D. Wechslera dla dorosłych. Wersja zrewidowana – renormalizacja WAIS-R (PL)*. Warsaw: Psychological Test Laboratory; 2004.
45. Holm S, Schönberger M, Poulsen I, Caetano C. *Patients' and relatives' experience of difficulties following severe traumatic brain injury: The sub-acute stage*. Neuropsychol. Rehab. 2009; 19(3): 444–460.
46. Chan R, Manly T. *The application of 'dysexecutive syndrome' measures across cultures: Performance and checklist assessment in neurologically healthy and traumatically brain-injured Hong Kong Chinese volunteers*. J. Int. Neuropsychol. Soc. 2002; 8(6): 771–780.
47. McGlynn SM, Schacter DL. *Unawareness of deficits in neuropsychological syndromes*. J. Clin. Exp. Neuropsychol. 1989; 11(2): 143–205.
48. Gainotti G. *Emotional behavior and hemispheric side of lesion*. Cortex. 1972; 8(1): 41–55.
49. Turnbull OH, Evans CE, Owen V. *Negative emotions and anosognosia*. Cortex. 2005; 40(1): 67–75.
50. MacHale SM, O'Rourke SJ, Wardlaw JM, Dennis MS. *Depression and its relation to lesion location after stroke*. J. Neurol. Neurosurg. Psychiatry. 1998; 64: 371–374.
51. Schacter DL. *Toward of cognitive neuropsychology of awareness: Implicit knowledge and anosognosia*. J. Clin. Exp. Neuropsychol. 1990; 12(1): 155–178.
52. Platek SM, Keenan JP, Gallup GG, Mohamed FB. *Where I am? The neurological correlates of self and other*. Cogn. Brain Res. 2004; 19: 114–122.
53. Vallesi A. *Organisation of executive functions: Hemispheric Asymmetries*. J. Cogn. Psychol. 2012; 24(4): 367–386.
54. Greene DJ, Barnea A, Herzberg K, Rassis A, Neta M, Raz A et al. *Measuring attention in the hemispheres: The lateralized attention network test (LANT)*. Brain Cogn. 2008; 66(1): 21–31.
55. Raz A, Buhle J. *Typologies of attentional networks*. Nat. Rev. Neurosci. 2006; 7(5): 367–379.
56. Salas Riquelme CE, Radovic D, Castro O, Turnbull OH. *Internally and externally generated emotions in people with acquired brain injury: Preservation of emotional experience after right hemisphere lesions*. Front. Psychol. 2015; 6: 101. <http://dx.doi.org/10.3389/fpsyg.2015.00101>.
57. Fink GR, Markowitsch HJ, Reinkemeier M, Bruckbauer Th, Kessler J, Heiss WD. *Cerebral Representation of one's own past: Neural networks involved in Autobiographical Memory*. J. Neurosci. 1996; 16(13): 4275–4282.

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58. Morin A, Michaud J. *Self-awareness and the left inferior frontal gyrus: Inner speech use during self-related processing*. Brain Res. Bull. 2007; 74(6): 387–396.
 59. Szepietowska E. *Skargi na własne kompetencje poznawcze: przejawy, uwarunkowania i znaczenie*. Hygeia Public Health. 2016; 51(2): 141–145.

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