RESEARCH ARTICLE

ACTA Vol. 22, No. 3, 2024, 435-454
NEUROPSYCHOLOGICA

Received: 12.04.2024
Accepted: 02.09.2024

A – Study Design
B – Data Collection
C – Statistical Analysis
D – Data Interpretation
E – Manuscript Preparation
F – Literature Search
G – Funds Collection

AN EVALUATION OF FRONTAL SYNDROME 20 YEARS AFTER TRAUMATIC INJURY TO THE FRONTAL LOBES OF THE RIGHT AND LEFT CEREBRAL HEMISPHERES

Rafał Morga[1[A,B,C,D,E,F]]
Jolanta Góral-Półrola[2[A,B,C,D]]
Małgorzata Goździewska[3[A,B,C,D]]
Agnieszka Buczaj[4[A,B,C,D]]

[1] Prof. B. Frańczuk Orthopedic and Rehabilitation Hospital in Lesser Poland, Kraków, Poland
[2] The Old Polish Academy of Applied Sciences in Kielce, Poland
[3] Department of Medical Anthropology, Institute of Rural Health, Lublin, Poland
[4] Departament of Technology Fundamentals, University of Life Sciences, Lublin, Poland

SUMMARY

Background: Traumatic brain injury (TBI), particularly damage to the frontal lobes, often produces a specific configuration of behavioral and personality changes known as frontal syndrome. Recent data, however, indicate that patients, depending on whether the damage has occurred in the left or right hemisphere of the brain, manifest one of two profiles of this syndrome in which negative or positive behavioral disturbances predominate. The purpose of this study was to evaluate the differences in frontal syndrome profiles present in patients 20 years after injury to the frontal lobes of the right and left hemispheres.

Material/ Methods: The study included a total of 360 patients with brain injury confirmed by neuroimaging studies, rehabilitated at the Reintegration and Training Center of the Polish Neuropsychological Society. The subjects were matched by age and gender into 2 experimental groups: Group A, included 180 patients (including 90 men and 90 women) with post-traumatic frontal lobe damage in the left hemisphere of the brain, and Group B, which included 180 patients (including 90 men and 90 women) with post-traumatic frontal lobe damage in the right hemisphere of the brain. The study employed documentation analysis, clinical interview and the Frontal Syndrome Behavior Questionnaire (FBInv). In accordance with the guidelines of this questionnaire, assessments were made based on an interview with the patient's caregiver in the absence of the patient. The study was conducted twice: two years (baseline) and about 20 years (follow up) after the brain injury.

Results: It was found that thepatients with left frontal lobe damage were characterised by significantly higher levels of so-called negative behavioural disorders overall and in the dimensions of apathy, passivity, indifference, concreteness, disorganisation, logopenia, apraxia of speech and perseveration, as well as significantly lower levels of loss of insight. While the patients with right frontal lobe damage were characterised by significantly higher levels of so-called positive conduct disorder overall and in the dimensions of hypersensitivity and irritability, excessive cheerfulness, unreasonable behaviour, inappropriateness and an absence of polite behaviour, aggressiveness, hyper- orality, hyper-sexuality, compulsivity, urinary and faecal incontinence and alien handedness. In addition, it should be noted that most of the recorded effects were found to be strong ($\eta 2 > 0.14$). The results indicated that 20 years after the brain injury, in terms of positive behavioural disorders overall, the group of patients with right frontal lobe damage showed a stronger decrease in disorders ($\eta 2 = 0.64$) over time when compared to patients with left frontal lobe damage ($\eta 2 = 0.14$).

Conclusions: We found that in the patients 20 years after post-traumatic damage to the frontal lobes is still manifesting frontal syndrome, however its profile is dependent on whether the damage occurred in the left frontal lobe or the right frontal lobe. The Frontal Syndrome Behavior Questionnaire can be used in the differential diagnosis of frontal syndrome after damage to the frontal lobes of the right and left hemispheres of the brain.

Keywords: conduct disorder, apathy, disinhibition, Frontal Syndrome Inventory, carers

INTRODUCTION

Traumatic brain injury (TBI) occurs as a result of sudden mechanical energy acting on the skull with a force exceeding the adaptive capacity of the skull and meninges. The force transmitted to the head or body causes damage and neuropathological dysfunction (Absher and Cummings 1995; Walsh 2000; et al. 2000, 2003, 2007; Fleminger and Ponsford 2005). This is followed by changes in the functioning of the central nervous system, resulting either from damage to brain structures or from functional disorders associated with disruption of the normal chemical and electrical processes of neurons in the brain (Pąchalska, Kaczmarek and Kropotov 2021).

Traumatic brain injury is classified as mild, moderate, and severe based on the Glasgow coma scale (GCS). Traumatic brain injury patients with a GCS of 13 to 15 are classified to be mild, which includes the majority of the patients examined here. Patients with a GCS of nine to 12 are considered to have a moderate traumatic brain injury, while patients with a GCS below eight are classified as having a severe traumatic brain injury. This activity reviews the workup of diffuse axonal injury and the role of health professionals working together in managing this condition (Mesfin et al. 2023).

Traumatic brain injury is a major source of health loss and disability worldwide. Globally, the annual number of TBI cases is estimated at 27 to 69 million.[1] Many trauma survivors live with significant disability, which constitutes a major socioeconomic burden (Fleminger and Ponsford 2005; Theadom et al. 2024). The pathophysiology of TBI includes primary and secondary sequelae of brain injury:

1. Primary sequelae of TBI refer to the immediate and direct consequences of the injury to the brain. These include brain contusions, axonal damage (DAI).
   - Brain contusions: These are bruises on the brain tissue caused by impact. They can lead to swelling, bleeding and increased intracranial pressure, potentially causing further damage to brain structures (Pachalska 2007).
   - Intracerebral and extracerebral hemorrhages: intracerebral hemorrhages occur in the brain tissue itself and can cause significant damage depending on the location and size of the bleeding and extracerebral hemorrhages; these include subdural, epidural and subarachnoid hemorrhages, which occur in the spaces around the brain. They can compress brain tissue and interfere with normal brain function (Tenny and Thorel 2024);
   - Diffuse axonal injury (DAI) is a type of traumatic brain injury (TBI) that results from a blunt injury to the brain.This involves extensive damage to the white matter pathways of the brain due to the sheer forces exacted during injury. DAI can interfere with communication between different parts of the brain and is often associated with severe and long-term impairments (Mesfin et al. 2023).[2]

[1] Traumatic brain injury: Epidemiology, classification, and pathophysiology - UpToDate

[2] Primary injuries can lead to secondary damage, including inflammation, oxidative stress and disruption of the blood-brain barrier, which can exacerbate the initial injury.

2. *Secondary sequelae of TBI* refer to the long-term consequences that may occur after the initial injury.[3] They generally result from a cascade of molecular mechanisms of damage that are initiated at the time of the initial injury and last for hours or days. It is likely that secondary brain damage can be exacerbated by modifiable systemic events such as reduced cerebral perfusion pressure (CPP), hypoxia, fever and seizures. Pachalska (2007) has stated that these consequences might affect different aspects of a person's health and functioning, and has divided them into.
   - *Cognitive impairment:* difficulties with memory, attention and executive functions. These cognitive deficits can significantly affect daily life and work.
   - *Neurobehavioral changes*: TBI can lead to changes in behavior and personality, including increased irritability, aggression, depression and anxiety (see also Kertesz et al. 2007).
   - *Neuroendocrine disorders*: TBI can interfere with the normal functioning of the endocrine system, leading to conditions such as hypopituitarism, which can cause symptoms such as fatigue, depression and cognitive impairment.
   - *Vision disorders:* including double vision, blurred vision and visual processing difficulties, are common after TBI.
   - *Sleep disorders*, which can exacerbate other symptoms and make recovery more difficult.
   - *Post-traumatic epilepsy:*some people may develop epilepsy after TBI, which can further complicate their recovery and quality of life.
   - *Chronic pain and headaches* and other types of chronic pain are common after TBI.
   - *Neuroinflammation and oxidative stress:*these processes can continue long after the initial injury, contributing to ongoing neuronal damage and impaired function.

The frontal lobes, due to their location in the skull, are often exposed to impact during an accident, such as from a collision, forward fall, etc. Thus, these are events that lead to what is known as closed cranial trauma, i.e., brain damage caused by physical forces without compromising the structural integrity of the skull. Traumatic damage to the frontal lobes of the brain often causes a specific configuration of changes in behavior and personality called frontal syndrome (Brown 1987; Kaczmarek 1993; Vilkki 1995; Absher and Cummings 1995; Walsh 2000; Kertesz et al. 2003, 2007; Kolb and Whishaw 2003; Pąchalska 2007).

[3] Chronic TBI, which might developed after injury, represents a spectrum of disorders associated with long-term consequences of single or repetitive TBI and includes chronic traumatic encephalopathy (CTE), chronic postconcussion syndrome, and chronic neurocognitive impairment. CTE is the most severe chronic TBI and represents the neurologic consequences of repetitive mild TBI. It is particularly noted among boxers and football players. CTE presents with behavioral, cognitive, and motor symptoms, and can only be definitively diagnosed postmortem.(Jordan et al. 2014).

=According to many, this damage is the cause of behavioral regulation disorders, but determining the essence of these disorders is by no means an easy task (Kaczmarek 1993; Geyer et al. 2002; Kertesz et al. 1997). The subject literature distinguishes two approaches to the issue of "behavior regulation":

- the frontal lobes are responsible for inhibiting undesirable behavior by rejecting, among the numerous possible behavioral choices in a given situation, those options that contradict moral, religious, cultural values, etc. (Kaczmarek 1993);
- frontal lobes are responsible for forming and activating action programs that determine human behavior (Brown 1987; Vilkki 1995).

These approaches are not necessarily mutually exclusive, for due to the complexity of both the neuroanatomical structures in question and the behaviors being regulated, it seems unlikely that a single, relatively simple mechanism is involved (Fuster 2000; Pąchalska et al. 2000; 2021; Absher and Cummings 1995). Evidence of brain damage is detected by laboratory and neuroimaging studies and appropriate therapy is instituted, However, there are clinical signs that may be delayed such as, behavioral disorders.

Diagnosing behavioral disorders in patients with frontal lobe injuries can be complex, as the frontal lobes are central to various cognitive and behavioral functions (Kaczmarek 1993; Vilkki 1995; Absher and Cummings 1995; Walsh 2000; Kertesz et al. 2003, 2007). However, recent studies using new neuroimaging methods indicate that depending on whether the traumatic brain injury has affected the left or right frontal lobe, frontal syndrome can manifest itself in different behavioral disorders (see Table 1).

Patients with post-traumatic frontal lobe damage may developed frontal syndrome (Absher and Cummings 1995; Kertesz et al. 2003, 2007). Frontal syndrome is defined as a syndrome of behavioral disorders associated with damage to the frontal lobe cortex of the brain. It may be accompanied by symptoms unrelated to cortical damage and not part of the clinical picture of the syndrome,

Table 1. Side of frontal lobe damage vs. description of type of disorder

The side of brain damage	Type of disorder	Description of the type of disorder
Left frontal lobe	Language disorders	Aphasia, specifically Broca's aphasia, which affects speech production.
	Cognitive symptoms	Problems with planning, organization and execution of tasks.
	Mood changes	Depression and apathy are more common.
Right frontal lobe	Behavioral disorders	Patients may exhibit disinhibition, impulsivity, and socially maladjusted behaviors.
	Emotional symptoms	There may be a lack of empathy, emotional blunting or euphoria.
	Spatial awareness	Problems with spatial orientation and neglect of the left side of space.

Source: study by M. Pąchalska, based on Brown 1987; Kaczmarek 1993; Vilkki 1995; Absher and Cummings 1995; Walsh 2000; Kertesz et al. 2003, 2007; Kolb and Whishaw 2003; Pąchalska 2007; Theadom et al. 2024)

such as those associated with an increase in intracranial pressure or damage to other parts of the brain by a proliferative process, or following a brain injury (Kertesz et al. 1997; Walsh 2000; Kolb and Whishaw 2003; Pąchalska 2007). Various factors determine how a patient's frontal syndrome picture will develop, among them being: (1) the patient's pre-disease (pre-accident) personality (resulting, incidentally, to some extent from the peculiar organization of the brain), (2) the location and extent of the damage, (3) the type (nature) of the damage, which may result most generally from an ischemic or hemorrhagic stroke, a neoplastic process, a traumatic brain injury or a degenerative process (Prigatano 2009).

The first description of this syndrome can be found in articles describing the famous case of the American engineer Phineas Gage, who had his skull punctured by an iron rod due to a serious accident in 1848 (Macmillan 1986; Pachalska 2007). Gage survived the accident, but his behavior and character were so radically altered that he could no longer function in society, at work and at home. The case drew the attention of specialists, and today the "Gage case" can be considered the beginning of neuropsychology (Herzyk 2000; Pachalska 2007). Moreover (as indicated by the works cited from a very rich subject literature), this case is often referred to in order toclarify important issues related to frontal lobe functions and the effects of their damage (Pachalska 2007; 2008; Damasio 1999; Geyer et al. 2002),

In the neuropsychological literature, another interesting case study of a patient with frontal lobe syndrome was presented (Pachalska et al. 2014). The patient called the Polish Phineas Gage (PG, age 27, owner/operator of a construction company) suffered a severe head injury after a fall from scaffolding at a construction site. Two wooden pegs were broken off and driven deep-into his cranium, from left to right. However, the patient not only survived the injury, he did not even lose consciousness after the accident. The damage to the brain affected primarily the frontal lobes. The upper peg penetrated the right frontal lobe. In the years that followed neurosurgery, the authors observed a pattern of behavioral disorders consistent with frontal lobe syndrome, similar to the famous 19th-century case of Phineas Gage. These symptoms make it impossible for the patient to adapt to social and cultural life. The authors describe the profile of negative and positive behavioral disturbances in PG patient, and stated that these behaviors developed over time into orbitofrontal syndrome. Microgenetic theory is used to interpret the formation of the symptom.

Given the nature of the neurobehavioral abnormalities observed in Gage (Macmillan 1986), the Polish Phineas Gage (Pachalska et al. 2014), and many other patients with damage to the prefrontal area of the brain in particular, and the fact that this area, which is phylogenetically and ontogenetically the youngest part of the human brain, comprises only 3.1% of the new cortex in cats, 13% in larger monkeys, and as much as 24% in humans (Kaczmarek 1987), it is difficult to avoid the conclusion that the frontal lobes play a key role in those aspects of behavior that specifically characterize human behavior in society, as well asin culture (Pachalska 2007).

Lebrun (1995) states that the most characteristic clinical signs of frontal syndrome are:

- *neuropsychological abnormalities*, including: apraxia, perseveration, ease of distraction, lack of regularity in the process of looking at a picture with a tendency to focus attention on a relatively small area, difficulty in reproducing previously learned information as a result of learning new information, disruption of access to information stored in memory,
- *characteristic speech disorders of a different nature* depending on the location of the damage in the right or left hemisphere of the brain: with damage to the right frontal lobe, aprosodia predominates, frequent use of stereotyped utterances, literal interpretation of metaphors, proverbs, etc., inability to identify or interpret similarities and differences,formulate inductive generalizations, etc., while with damage to the left frontal lobe – there is a non-fluency of speech and apraxia of speech;
- *emotion and mood disorders, including:* apathy, indifference, emotional lability, excessive cheerfulness, shallowness of emotions, mood swings, possibly catastrophic reactions, irritability, quarrelsomeness, a tendency to offend,
- *neurobehavioral disorders,* including: unpredictability, tendencies to make inappropriate jokes, unreasonable behavior or inhibition, possible exhibitionist tendencies, impulsive behavior, feeling excessive pleasure from taking risks, lack of reason.

Tests targeting single neuropsychological functions, although indicative of specific neurobehavioral disorders, do not allow a detailed description of the frontal syndrome picture. This is because these tests do not always detect specific disorders (lack of sensitivity) and do not exclude non-specific disorders (lack of specificity) in the behavior of patients with frontal syndrome. Moreover, in recent years, many researchers have pointed out that there is no significant correlation between the results of so-called laboratory tests and the actual behavior of the patient at home (Geyer et al. 2002). In the case of frontal syndrome, this poses a significant problem, as complex behavior is primarily involved. A good tool here, as previous studies have shown (cf. Pąchalska, Kaczmarek and Kropotov 2014), would be the Frontal Syndrome Behavioral Inventory (FBInv) Questionnaire, which was developed by A. Kertesz et al. (1997) and adapted to Polish conditions by Pąchalska and MacQueen (2002).

The purpose of this study was to evaluate the differences in frontal syndrome profiles occurring in patients 20 years after injury to the frontal lobes of the right and left hemispheres of the brain using the Frontal Behavioral Inventory (FBInv) Questionnaire.

MATERIAL AND METHOD

The study included a total of 360 patients following brain injury caused by a car accident confirmed by neuroimaging studies, rehabilitated at the Reintegration and Training Center of the Polish Neuropsychological Society. The sub-

jects were matched by a pairing method (by age and gender) into 2 experimental groups: Group A, comprised 180 patients, including 90 men (50% of the group) and 90 women (50% of the group) with post-traumatic frontal lobe damage in the left hemisphere of the brain, and Group B, which comprised 180 patients, including 90 men (50% of the group) and 90 women (50% of the group) with post-traumatic frontal lobe damage in the right hemisphere of the brain.

As a measure of the depth of brain injury, we used:

- the depth of coma, usually measured immediately after the injury using the Glasgow Coma Scale (GCS);
- the duration of unconsciousness (the period during which the patient's GCS scores were below 9);
- the duration of post-traumatic amnesia (PTA), i.e., the period from the injury to the patient's recovery of short-term memory continuity;
- the presence of neurological symptoms, such as hemiparesis.

INCLUSION AND EXCLUSION CRITERIA

In accordance with the protocol of the EuroacademiaMultidisciplinariaNeurotraumatiologica - EMN (cf. Moskała 2023), the study included: people without mental illness or comprehension disorders [screening to exclude dementia and comprehension disorders]. Subjects with violent trauma [mainly a traffic accident] with MRI-confirmed damage to the frontal lobes: primary [as a result of the accident], secondary [due to a hematoma in the frontal area]. Subjects who were in a prolonged coma and whose post-traumatic amnesia was longer than 4 weeks, All patients and their families consented to participate in the study.

Subjects with a history of: previous head injury, stroke, psychiatric illness, mental retardation, who suffered from post-traumatic psychosis (Capgras syndrome, Fregoli syndrome, post-traumatic paraschizophrenia), who were under 18 years of age and over retirement age, manifested dementia (including post-traumatic dementia) were excluded: subjects with an MMSE test score of less than 19 points [the limit between mild and moderate dementia] were excluded.

Patient demographics are shown in Table 2, which shows that the study groups do not differ significantly in either age, education, time duration since injury or illness, or average rehabilitation period. Patients in Group A and Group B have similar mean ages; 38.84 and 39.29 years, respectively. Noteworthy is the fact that all patients in study 1, that is, at the time of the traumatic brain injury, were teenagers, and that in study 2, 20 years after the injury (follow-up), they are at the so-called "working age".

Table 2. Demographics of patients in the study groups

Lp.	Features	A	B
1.	Age	38.84 7.1	39.29 8.1
2.	Education (in years)*	3.1	6.1 1.8
3.	Time duration since injury (average, range in years)	19.8 2.4	20.1 3.1
4	Outpatient rehabilitation period (in years)	4.3 3.1	1.8

Table 3. Frontal Syndrome Assessment Scale

Number of points	Degree of disturbance
49 – 72	Deep degree of disorder, problems occur constantly
25 – 48	Moderate degree of disorder, problems occur fairly often
1 – 24	Slight degree of disturbance, problems occur episodically
0	Lack of the syndrome

Method

The Frontal Behavioral Inventory (FBInv) Questionnaire, which was developed by A. Kertesz et al. (1997; 2000) in its authorized Polish version by Pąchalska and MacQueen (2002), was employed in the study. The FBInv test contains questions targeting all axial symptoms of frontal syndrome and is therefore used in the clinical practice of patients with post-traumatic brain injury to diagnose frontal syndrome (cf. Pąchalska 2007).The evaluation of the frontal syndrom is carried out according to the scale presented in Table 3.

Study protocol

The study was conducted twice: the first time two years (baseline) and the second time about 20 years (follow-up) after the brain injury itself. According to the guidelines of this questionnaire, assessments were made based on an interview with the patient's caregiver in the patient's absence. The caregiver was asked 24 targeted questions about the patient's post-injury (or in the case of FTD patients, post-illness) behavior. The investigator emphasized that he was mainly interested in those changes that occurred during this period of time. The results were recorded in 24 categories: 12 of these categories relate to so-called "negative"behavioral disorders, namely: (1), apathy, (2) passivity, (3) indifference, emotional shallowness, (4) stubbornness and rigidity of thought, (5) concreteness, (6) neglect of appearance, (7) disorganization, (8) lack of attention, (9) loss of insight, (10) logopenia, (11) apraxia of speech, (12) perseveration, and 12 of these categories relate to so-called"positive"behavioral disorders, namely (1) hypersensitivity and irritability, (2) excessive cheerfulness, (3) unreasonable behavior, (4) inappropriateness and lack of social politeness in behavior, (5) impulsivity, (6) restlessness, (7) aggressiveness, (8) hyper-orality, (9) hyper-sexuality, (10) compulsivity, (11) urinary and/or fecal incontinence, (12) foreign hand.

The caregiver of a person with post-traumatic brain injury was tasked with answering the question in each of the categories listed above. In case of difficulties in understanding the question, the examiner clarified the meaning of the question, gave examples, supplemented the information, etc., and noted down the answers, comments, explanations and additions of the subject. Scores were given using the method of "competent judges" (neurologist, neuropsychologist, neuropsychiatrist and neurolinguist) according to a three-point scale:

- 0. = no pathological behavior in a given area;
- 1. = mild degree of disorder, problems occur episodically;
- 2. = moderate degree of disorder, problems occur relatively frequently;
- 3. = deep degree of disorder, problems occur very often.

RESULTS

The distribution of the ratings of the caregivers of the surveyed patients in the two groups A and B in terms of the total number of points given to the patient two years after the brain injury (baseline) and 20 years afterwards(follow-up) in each category of the FBInv Questionnaire is illustrated in Table 4.

Differences in the profile of behavioural disorders occurring in patients with left and right frontal lobe damage

The results for the main effect of the study groups were analysed first. The results obtained indicated that the differences in the profile of behavioural disorders were:

- patients with left frontal lobe damage (Group A) were characterised by significantly higher levels of so-called negative behavioural disorders overall and in the dimensions of apathy, passivity, indifference, concreteness, disorganisation, logopenia, apraxia of speech and perseveration, as well as significantly lower levels of loss of insight.
- patients with right frontal lobe damage (Group B) were characterised by significantly higher levels of so-called positive conduct disorder overall and in the dimensions of hypersensitivity and irritability, excessive cheerfulness, unreasonable behaviour, inappropriateness and a lack of social politeness, aggressiveness, hyper-orality, hyper-sexuality compulsivity, urinary and faecal incontinence and alien handedness. In addition, it should be noted that most of the recorded effects were found to be strong ($\eta 2 > 0.14$).

The results for the main measurement effect were then analysed. The results indicated that patients in the first measurement were characterised by significantly higher levels of negative conduct disorder overall and in the dimensions of stubbornness and rigidity of thinking, concreteness, neglect of appearance, disorganisation, lack of attention and loss of insight; positive conduct disorders overall and on the dimensions of impulsivity, restlessness, hyper-orality, hypersexuality, compulsivity and urinary and faecal incontinence, as well as significantly lower severity of negative conduct disorders on the dimensions of apathy and passivity and positive conduct disorders on the dimensions of hypersensitivity and irritability, excessive merriment, unreasonable behaviour, inappropriateness and lack of culture in behaviour and aggressiveness. In addition, it should be noted that most of the recorded effects were found to be strong ($\eta 2 > 0.14$).

Changes in behavioural disorders 20 years after brain injury for individual characteristics

In the last step, the results for the interaction effect of group and measurement were analysed. The results indicated that 20 years after the brain injury, in terms of positive behavioural disorders overall, the group of patients with right frontal lobe damage showed a stronger decrease in disorders ($\eta 2 = 0.64$) with time when compared to patients with left frontal lobe damage ($\eta 2 = 0.14$).

Table 4. Results of a two-factor analysis of variance in a mixed design testing the relationship between damage side and elapsed time and scores in each category of the FBInv Questionnaire (N = 360)

	Examination 1				Examination 2									
	Injuries to the left frontal lobe		Injuries to the right frontal lobe		Injuries to the left frontal lobe		Injuries to the right frontal lobe		Main effect of groups		Main effect of measurement		Group and measurement interaction effect	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*	η^2	*F*	η^2	*F*	η^2
Apathy	2.31	0.62	0.91	0.84	2.68	0.47	0.81	0.49	**1345.69*****	0.79	**8.96****	0.02	**24.52*****	0.06
Aspontaneity	2.39	0.59	1.28	0.47	2.73	0.44	1.33	0.50	**1058.86*****	0.75	**28.42*****	0.07	**14.50*****	0.04
Indifference, Emotionalflatness	2.11	0.64	1.33	0.49	2.44	0.50	1.30	0.49	**567.13*****	0.61	**15.88*****	0.04	**21.94*****	0.06
Inflexibility	2.52	0.50	2.42	0.52	1.51	0.50	1.57	0.51	0.10	<0.01	**517.17*****	0.59	3.17	0.01
Concreteness	2.59	0.49	2.40	0.51	1.41	0.49	1.26	0.46	**21.03*****	0.06	**1126.68*****	0.76	0.51	<0.01
Personal Neglect	2.62	0.49	2.31	0.50	1.31	0.60	1.48	0.50	2.20	0.01	**681.11*****	0.66	**32.93*****	0.08
Disorganization	2.74	0.46	2.31	0.49	1.40	0.49	1.50	0.50	**16.42*****	0.04	**828.24*****	0.70	**44.42*****	0.11
Inattention	2.88	0.30	2.89	0.31	1.31	0.48	1.41	0.51	2.32	0.01	**2302.51*****	0.87	2.43	0.01
Loss of Insight	2.30	0.78	2.87	0.35	1.68	0.47	1.79	0.59	**54.16*****	0.13	**484.22*****	0.57	**36.49*****	0.09
Logopenia	2.72	0.46	0.81	0.44	2.68	0.47	0.83	0.44	**2450.04*****	0.87	0.01	<0.01	0.74	<0.01
Verbal apraxia	2.78	0.40	1.32	0.50	2.81	0.40	1.31	0.50	**1511.20*****	0.81	0.04	<0.01	0.15	<0.01
Perseveration	2.21	0.75	1.67	0.48	2.24	0.43	1.68	0.50	**179.18*****	0.33	0.36	<0.01	0.22	<0.01
So-callednegative behavioral disorders	2.51	0.19	1.88	0.18	2.02	0.13	1.36	0.13	**2971.04*****	0.89	**1882.88*****	0.84	1.61	<0.01
Irritability	1.53	0.50	2.57	0.50	1.47	0.50	2.81	0.41	**1040.60*****	0.74	**10.21****	0.03	**16.12*****	0.04
Excessive Jocularity	0.78	0.48	2.52	0.53	0.79	0.49	2.79	0.42	**2365.52*****	0.87	**15.62*****	0.04	**16.95*****	0.05
Poor Judgement	1.40	0.91	2.21	0.43	1.68	0.47	2.42	0.51	**233.44*****	0.39	**38.11*****	0.10	0.71	<0.01
Inappropriateness	1.13	0.81	2.42	0.52	1.11	0.66	2.68	0.48	**980.39*****	0.73	**7.85****	0.02	**7.21****	0.02
Impulsivity	2.08	0.83	2.43	0.52	1.32	0.47	1.08	0.82	0.94	<0.01	**414.29*****	0.54	**29.65*****	0.08
Restlessness	2.44	0.49	2.51	0.50	1.39	0.49	1.31	0.50	0.09	<0.01	**911.71*****	0.72	**6.65***	0.02
Aggression	0.71	0.57	2.52	0.50	1.22	0.46	2.92	0.29	**3457.71*****	0.91	**142.73*****	0.29	2.26	0.01
Hyperorality	0.43	0.49	2.33	0.49	0.41	0.49	1.37	0.48	**1482.53*****	0.81	**175.65*****	0.33	**184.01*****	0.34
Hypersexuality	0.89	0.72	2.10	0.80	0.87	0.33	1.53	0.50	**423.87*****	0.54	**41.29*****	0.10	**34.00*****	0.09
Utilization Behavior	1.17	0.57	2.32	0.49	0.71	0.48	1.41	0.51	**484.43*****	0.58	**393.96*****	0.52	**34.62*****	0.09
Incontinence	1.22	0.62	2.68	0.48	1.23	0.52	1.29	0.48	**460.42*****	0.56	**255.84*****	0.42	**277.25*****	0.44
Alien Hand	0.19	0.40	0.29	0.47	0.22	0.41	0.30	0.47	**5.44***	0.01	0.18	<0.01	0.04	<0.01
So-called positive behavioral disorders	1.16	0.18	2.24	0.18	1.03	0.13	1.83	0.15	**5944.44*****	0.94	**544.86*****	0.60	**158.60*****	0.31

p*< 0.050; ** *p*< 0.010; * *p*< 0.001

That in terms of apathy, there was a stronger increase (η2 = 0.08) over time in the left frontal lobe lesion group when compared to patients with right frontal lobe lesions (η2< 0.01); that in terms of passivity, there was a stronger increase (η2 = 0.10) over time in the left frontal lobe lesion group compared to patients with right frontal lobe lesions (η2< 0.01); that in terms of indifference, patients with left frontal lobe damage showed a stronger increase (η2 = 0.10) over time compared to patients with right frontal lobe damage (η2< 0.01).

In terms of personal neglect, a stronger decrease (η2 = 0.59) over time was noted in the group of patients with left frontal lobe damage compared to patients with right frontal lobe damage (η2= 0.37). In terms of disorganisation, patients with left frontal lobe damage showed a stronger decrease (η2 = 0.64) over time when compared to patients with right frontal lobe damage (η2= 0.41); that in terms of loss of insight, patients with right frontal lobe damage showed a stronger decrease (η2 = 0.52) over time compared to patients with left frontal lobe damage (η2= 0.26);

That in terms of hypervigilance and irritability, there was a stronger increase (η2 = 0.07) over time in the group of patients with right frontal lobe damage compared to patients with left frontal lobe damage (η2< 0.01); that in terms of excessive cheerfulness, patients with right frontal lobe damage showed a stronger increase (η2 = 0.08) over time compared to patients with left frontal lobe damage (η2< 0.01); that in terms of inappropriateness, patients with right frontal lobe damage showed a stronger increase (η2 = 0.04) over time compared to patients with left frontal lobe damage (η2< 0.01).

In terms of impulsivity, there was a stronger decrease (η2 = 0.48) over time in the right frontal lobe-impaired group compared to the left frontal lobe-impaired patients (η2= 0.24); in terms of anxiety, there was a stronger decrease (η2 = 0.60) over time in the right frontal lobe-impaired group when compared to the left frontal lobe-impaired patients (η2= 0.52); that, in terms of hyper-sexuality, patients with right frontal lobe damage showed a stronger decline (η2 = 0.50) over time compared to patients with left frontal lobe damage (η2< 0.01); that, in terms of hyper-orality, patients with right frontal lobe damage showed a stronger decrease (η2 = 0.17) over time compared to patients with left frontal lobe damage (η2< 0.01); that, in terms of compulsivity, patients with right frontal lobe damage showed a stronger decrease (η2 = 0.48) over time compared to patients with left frontal lobe damage (η2 = 0.21); as well as in terms of urinary and faecal incontinence patients with right frontal lobe damage showed a stronger decrease (η2 = 0.60) over time when compared to patients with left frontal lobe damage (η2< 0.01).

The analysis of the results leads us to conclude that with regards to both two years (baseline) and 20 years (follow-up) after brain injury, different frontal syndrome profiles are found depending on whether the left or right frontal lobe of the brain was damaged. It is noteworthy that the alien hand phenomenon in both study one (baseline) and study two (follow-up) is found in only a small number of subjects from both groups.

DISCUSSION

Our studies have shown that brain injury and especially damage to the frontal lobes lead in many to the development of a variety of behavioural disorders. In many of these people, two years after the brain injury, the configuration of these changes can be diagnosed as frontal syndrome. This syndrome still persists 20 years after the brain injury.It was found that thepatients with left frontal lobe damage were characterised by significantly higher levels of so-called negative behavioural disorders overall and in the dimensions of apathy, passivity, indifference, concreteness, disorganisation, logopenia, apraxia of speech and perseveration, as well as significantly lower levels of loss of insight. While the patients with right frontal lobe damage were characterised by significantly higher levels of so-called positive conduct disorder overall and in the dimensions of hypersensitivity and irritability, excessive cheerfulness, unreasonable behaviour, inappropriateness and lack of culture, aggressiveness,hyper-orality, hyper-sexuality, compulsivity, urinary and faecal incontinence and alien handedness. In addition, it should be noted that most of the recorded effects were found to be strong ($\eta 2 > 0.14$). The results indicated that 20 years after the brain injury, in terms of positive behavioural disorders overall, the group of patients with right frontal lobe damage showed a stronger decrease in disorders ($\eta 2 = 0.64$) over time when compared to patients with left frontal lobe damage ($\eta 2 = 0.14$).

It is difficult to compare our results with other data from the subject literature, as the authors do not differentiate behavioural disorders according to the side of brain damage. Most of the current literature has found that cognitive behavioral and emotional deficit following TBI occurs within the first six months whereas after 1–2 years the condition becomes stable. Identifying the risk factors for poor outcome is the first step to reduce the sequelae. Patients with TBI have an adjusted relative risk of developing any NPS several-fold higher than in the general population after six months of moderate–severe TBI. All NPS features of an individual's life, including social, working, and familiar relationships, may be affected by the injury, with negative consequences on quality of life (Torregrossa et al. 2923).

Some long-term studies, unfortunately often weakened by low follow-up rates, show surprisingly good results over time (Fleminger and Ponsford 2005). It has been widely assumed that most of the recovery following severe traumatic brain injury (TBI) occurs within the first 6 months, and that virtually all of the recovery occurs within the first 1-2 years post-injury. In an effort to evaluate the long-term recovery of patients who had sustained severe TBI, we interviewed the relatives and significant others of 20 patients who had sustained TBI at least 5 years earlier, using a modified version of the Portland Adaptability Inventory. Retrospective ratings were collected to evaluate the patients' psychosocial, cognitive, physical, and emotional status prior to their injury, and at 1, 2, 5, and an average of 10.3 years post-injury. The results indicated that TBI patients exhibit significant improvements in their social, cognitive, physical, and emotional functioning after 2

years post-injury regardless of the severity of their initial brain trauma. These data suggest that patients who sustain severe TBI continue to make gradual improvements in their functioning for at least 10 years post-injury. These findings contradict the widely held assumption that the recovery process ends after 1 or 2 years post-injury (Sbordone et al. 1995).

In other longitudinal studies, forty-four survivors who acquired moderate and severe TBI during 1995–1996 were followed 10 and 20 years postinjury. The outcomes were Glasgow Outcome Scale Extended (GOSE), Community Integration Questionnaire (CIQ), and SF-36 questionnaire (SF-36). Multiple regressions were performed to examine the relationship between follow-up measurements, controlling for baseline demographics and injury severity.There were no significant differences in baseline age and civil status between moderate and severe TBI, but patients with severe injury had significantly lower employment rates ($p = 0.05$). The mean age at 20-years follow-up was 50.8 (*SD* 11.4) years, and 73% were males. Most patients showed good recovery (52%) or moderate disability (43%). Disability levels remained stable between and within severity groups from 10 to 20 years. Community integration including social integration improved from 10 to 20 years ($p = 0.01$ and $p = 0.005$, respectively). HRQL remained stable, except for the subscales of Bodily Pain and Role Emotional ($p = 0.02$ and $p = 0.06$). Depression at 10 years and females were associated with poorer mental health, while productive activity at 10 years indicated better physical and mental health at 20 years postinjury, respectively. Functional limitations persist even decades after moderate and severe TBI, with poorer prognosis for females and persons who were depressed at the 10-year follow-up. Highlighted should be the development and evaluation of targeted long-term follow-up programs and access to rehabilitation services for these groups. Improved community integration despite stable functional limitations draws attention to long-term adaptation to adversity and illness (Andelic et al. 2018).

It should be mention that recently TBI has been considered as a disease process instead of an isolated event, with acute and chronic consequences (Masel B.E., DeWitt 2010). Following the injury, neuropsychiatric disturbances (NPS) can occur as primary psychiatric complications or could be an exacerbation of pre-existing compensated conditions. Lauterbach et al. have showed that NPS were higher in patients with a pre-injury history (83.2%) than those without (63.6%). Moreover, 59% showed one or more Axis I disorder before TBI and substance-use was the most common pre-injury disorder (38.5%), whereas 56.5% were diagnosed as a new diagnostic class (Lauterbach et al. 2015). In all probability, premorbid factors of the psychological status, personality, contingencies, environmental reinforcements play an important role in determining the clinical picture of the “frontal personality” (Passingham et al. 2010). The symptoms could appear acutely or develop more gradually and insidiously influencing the grade of disability. Patients with TBI have an adjusted relative risk of developing any NPS several-fold higher than in the general population after six months of moderate-to-severe TBI (Whelan-Goodinson et al.2009). A review by Babbage et al.

(2011) reported a rate of 39% of impairment in emotion recognition and reduced levels of empathy as well as impairment of the so-called theory of mind (ToM) (Premack and Woodruff 1978), with a detrimental effect on the life satisfaction of their relatives and caregivers (Martín-Rodríguez et al. 2010).

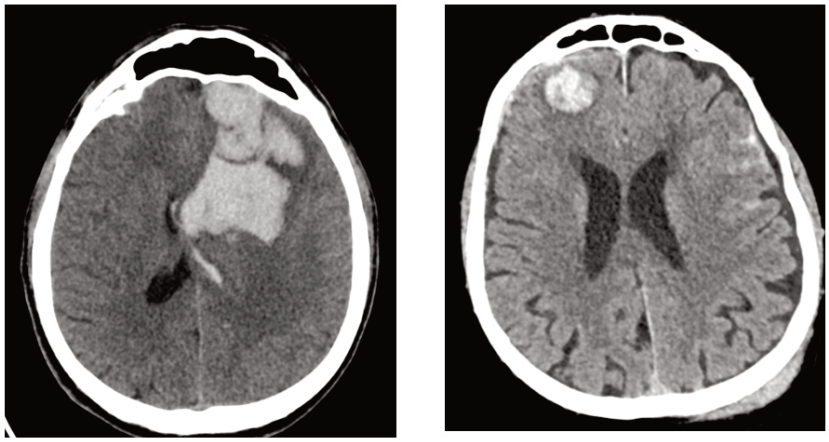
The most common psychiatric complication associated with TBI is depression, with prevalence rates ranging between 6% and 77%, while 2–50% of TBI patients are affectedby generalized anxiety disorders (Osborn et al. 2016). It appears that rates of mood and anxiety disorders increase throughout the first year and that anxiety disorders emerges earlier post-injury than mood disorders (Gould et al. 2011; Koponen et al. 2011; Diaz et al. 2012; Ganau et al. 2018;). A peak of NPS was recorded in the first year, with a subsequent significant declineover time in anxiety disorders by 27% with each year post-injury, although mood and substance-use disorder rates persisted steadily (Lauterbach et al. 2015).

Factors associated with depressive disorders in TBI patients include age, lifestyle, being a young adult, premorbid substance misuse, especially in male patients (Ashman et al. 2004). Other risk factors are lower education levels (Whelan-Goodinson et al. 2010), previous psychiatric symptoms, including anxiety, intellectual deficits and left prefrontal cortical lesions (Leopold et al. 2011). Some studies reported a frequency of NPS of 77% in males and of 71.4% in females ($\chi^2 = 0.46$, df = 1, $p = 0.50$). Depression in TBI individuals has been linked to decreased community integration, overall functioning, a decrease in quality of life, aggression, poor recovery, and higher rates of suicidal ideation and suicide attempts. Likewise, anxiety disorders in TBI patients are associated with poor social interpersonal functioning, a decline in independent living, and acts as a positive predictive factor for the development of depression in TBI individuals. Moreover, delirium (Ganau, Lavinioand Prisco 2018) and status epilepticus (Prisco et al. 2020) in neurointensive care unit are very well-known risk factors for developing behavioral disorders at long term follow-up.

In the case of a patient with behavioural disorders, however, it is worth emphasising that the results obtained may be influenced by the location of the injury within the frontal lobe itself and the side of the injury (left or right). Also important are the extent of the brain damage after the injury, including the presence of a brain haematoma, the size of the haematoma and the site of its formation (cf. Fig. 1), as well as the associated neurosurgical intervention and the associated damage to the patterns of neuronal connections responsible for behaviour.

Our results are consistent with previous observations that the clinical picture of the frontal syndrome of a patient after a left frontal lobe injury differs from that of a patient after a right frontal lobe injury (Pąchalska 2003). These findings are of applied importance as they imply the need for individualised, tailored rehabilitation for such patients.

It should be emphasized that the profile of behavioral disorders is also influenced by the self-system of the patient after brain injury. Needless to say, it is individualized, different in each person. The proper functioning of the self system depends on the integration and interaction of all types of self. Therefore, it is pro-



A) B)

Fig. 1. Cerebral haematomas:
(A) CT scan of the head in axial projection. A widespread hematoma of the frontal lobe of the left cerebral hemisphere (dimensions 61 mm x 35 mm x 46 mm) is seen compressing and displacing the ventricular system of the brain to the right side.
(B) CT scan of the head in axial projection. Visible hematoma of the frontal lobe of the right cerebral hemisphere measuring 18 mm x 24 mm x 17 mm)/. Also visible is blood in the subarachnoid space around the frontal lobe of the right cerebral hemisphere and in the cerebral grooves of the temporal lobe of the left cerebral hemisphere.
Source: clinical material by R. Morga

posed to integrate the concept of self including individual (objective and subjective) and social (collective and cultural) (Pąchalska 2019). This concept, however, should include the minimal (working) and longitudinal (autobiographical) self, which is the basis for the formation of the self system.

The self system requires the nesting of the minimal (working) and longitudinal (autobiographical) Self, and a change in understanding of the concepts of individual and social Self in terms of thought process (cf. Fig.1).Therefore:

1. The individual self includes:
   - *the objective self*, understood as the organism, the body together with the states and processes occurring within it. The objective self has consciousness, but it lacks self-awareness and meta-consciousness (awareness of mental operations on its own subject). The subject does not express their own thoughts but acts according to ready-made schemes: they are not the author of themselves. As soon as you realize the existence of the outside world, your objective self also becomes the object of perception. This process enables the subjective self to be formed;
   - *the subjective self*, having consciousness, self-awareness and meta-consciousness, enabling one to know oneself and act in accordance with one's own needs and values as well as the requirements of the environment. In Obuchowski's view (op. cit.), the subjective man is the Author of Himselves,

who has a sense of separateness, autonomy, has insight (introspection), the possibility of self-assessment and self-control and creativity. The subjective self conditions the appearance of individual identity.

2. The social self, includes:
   - *the relational self,* understood as an image and description of the Me – You (interactions), from an individual and social perspective taking into account relationships with other important people and social groups around which, social identity develops.
   - *the cultural self*, understood as an image and description of the Us – Them, from an individual and social perspective including nesting in the culture or subculture of a given social group around which cultural identity develops.

Summing up, the modified processual approach to the self-system takes into account the concept of nesting of the minimal (working) and longitudinal (autobiographical) self in the individual and social self in the processual approach, and creates the basis for the development of the self system. It also allows for a better explanation of the disintegration or loss of this system in those with various kinds of brain damage. It also allows for more effective rehabilitation interactions to be offered to such people (see also Prigatano 2012)

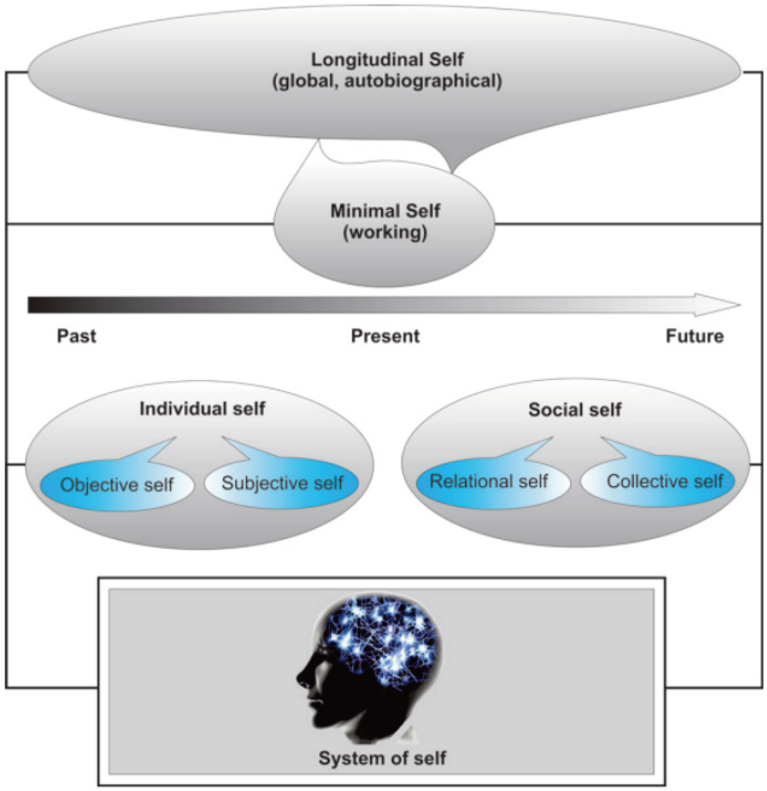


Fig. 2. Modified, process model of the self system
Source: M. Pąchalska (2019), with permision

It is known from clinical observation that there are changes in the behaviour of the trauma patient as rehabilitation progresses. Our study confirmed that longitudinal studies of these changes are appropriate. Our results allow us to conclude that the Polish version of the FBInv questionnaire (Pąchalska and MacQueen 2002) provides a reliable and useful picture of the entire behavioural disorders of the head trauma patient, who, as is well known, often shows greater disturbances in social functioning, including family life, than their clinical status implies.

Planning long-term individualized support for patients with a variety of brain injuries is a challenge in modern medicine (cf. Morga et al. 2023). This includes patients with frontal lobe brain injuries. Ultimately, it is the injured person who must cope with the aftermath of the brain injury throughout his or her life. It is important to understand their perspective.

CONCLUSIONS

We found that in the patients 20 years after post-traumatic damage to the frontal lobes are still manifesting frontal syndrome, however the profile is dependent on whether the damage occurred in the left frontal lobe damage or right frontal lobe damage, The Frontal Syndrome Behavior Questionnaire can be used in the differential diagnosis of frontal syndrome after damage to the frontal lobes of the right and left hemispheres of the brain.

REFERENCES

Absher, J. R., Cummings, J. L. (1995). Neurobehavioral examination of frontal lobe functions. *Aphasiology, 9*(2), 181-192. doi: 10.1080/02687039508248705.

Andelic, N., Howe, E. I., Hellstrøm, T., Sanchez, M. F., Lu, J., Løvstad, M., Røe, C. (2018). Disability and quality of life 20 years after traumatic brain injury. *Brain and Behavior, 8*(7), e01018. doi: 10.1002/brb3.1018.

Ashman, T. A., Spielman, L. A., Hibbard, M. R., Silver, J. M., Chandna, T., Gordon, W. A. (2004). Psychiatric challenges in the first 6 years after traumatic brain injury: cross-sequential analyses of Axis I disorders. *Archives of Physical Medicine and Rehabilitation, 85,* 36-42. doi: 10.1016/j.apmr.2003.08.117.

Babbage, D. R., Yim, J., Zupan, B., Neumann, D., Tomita, M. R., Willer, B. (2011). Meta-analysis of facial affect recognition difficulties after traumatic brain injury. *Neuropsychology, 25*(3), 277-285. doi: 10.1037/a0021908.

Brown, J. W. (1987). The microstructure of action. In: E. Perecman (Ed.), *The frontal lobes revisited.* (pp. 251-272). New York: IRBN Press.

Damasio, A. R. (1999). *Błąd Kartezjusza. Emocje, rozum i ludzki mózg*. Tłum. M. Karpiński. Poznań: Dom Wydawniczy Rebis.

Diaz, A. P., Schwarzbold, M. L., Thais, M. E., Hohl, A., Bertotti, M. M., Schmoeller, R., ... Walz, R. (2012). Psychiatric disorders and health-related quality of life after severe traumatic brain injury: a prospective study. *Journal of Neurotrauma*, *29*(6), 1029-1037. doi: 10.1089/neu.2011.2089.

Fleminger, S., Ponsford, J. (2005). Long term outcome after traumatic brain injury. *BMJ, 331*(7530), 1419. doi: 10.1136/bmj.331.7530.1419.

Fuster, J. M. (2000). Executive frontal functions. *Experimental Brain Research, 133,* 66–70. doi: 10.1007/s002210000401.

Ganau, M., Lavinio, A., Prisco, L. (2018). Delirium and agitation in traumatic brain injury patients: an update on pathological hypotheses and treatment options. *Minerva Anestesiologica, 84*(5), 632–640. doi: 10.23736/S0375-9393.18.12294-2.

Geyer, B., Friedman, C., Pogge, D., Huron, R. (2002). The convergent validity of measures of attention. *Journal of the International Neuropsychological Society, 8*(2), 310.

Gould, K. R., Ponsford, J. L., Johnston, L., Schönberger, M. (2011). The nature, frequency and course of psychiatric disorders in the first year after traumatic brain injury: a prospective study. *Psychological Medicine, 41*(10), 2099-2109. doi: 10.1017/S003329171100033X.

Herzyk, A. (2000). *Mózg – emocje – uczucia. Analiza neuropsychologiczna.* Lublin: Wydawnictwo UMCS.

Jordan, B. D. (2014). Chronic traumatic encephalopathy and other long-term sequelae. *Continuum: Lifelong Learning in Neurology, 20*(6), 1588-1604. doi: 10.1212/01.CON.0000458972.94013.e1.

Kaczmarek, B. L. J. (1987). Regulatory function of the frontal lobes. A neurolinguistic perspective. In: E. Perecman (Ed.), *The frontal lobes revisited*. (pp. 225-240). NewYork: IRBN Press.

Kaczmarek, B. L. J. (1993). Neurolinguistic aspects of crime-related frontal lobe deficits. In: H. V. Hall, R. J. Sbordone (eds.), *Disorders of Executive Functions: Civil and Criminal Law Applications* (p. 79–105). Winter Park, FL: PMD Publishers.

Kaczmarek, B. L. J. (1993). *Płaty czołowe a język i zachowanie człowieka*. Lubin: Wydawnictwo Popularnonaukowe "Linea".

Kertesz, A., Blair, M., McMonagle, P., Munoz, D. G. (2007). The diagnosis and course of frontotemporal dementia. *Alzheimer Disease & Associated Disorders, 21*(2), 155-163. doi: 10.1097/WAD.0b013e31806547eb.

Kertesz, A., Davidson, W., Fox, H. (1997). Frontal behavioral inventory: diagnostic criteria for frontal lobe dementia. *Canadian Journal of Neurological Sciences, 24*(1), 29-36. doi: 10.1017/S0317167100021053.

Kertesz, A., Davidson, W., McCabe, P., Munoz, D. (2003). Behavioral quantitation is more sensitive than cognitive testing in frontotemporal dementia. *Alzheimer Disease & Associated Disorders*, *17*(4), 223-229.

Kertesz, A., Nadkarni, N., Davidson, W., Thomas, A. W. (2000). The Frontal Behavioral Inventory in the differential diagnosis of frontotemporal dementia. *Journal of the International Neuropsychological Society, 6*(4), 460-468. doi:10.1017/S1355617700644041.

Kolb, B., Whishaw, I. Q. (2003). *Fundamentals of Human Neuropsychology.* New York: V. Worth Publishers.

Koponen, S., Taiminen, T., Hiekkanen, H., Tenovuo, O. (2011). Axis I and II psychiatric disorders in patients with traumatic brain injury: a 12-month follow-up study. *Brain Injury, 25*(11), 1029-1034. doi: 10.3109/02699052.2011.607783.

Lauterbach, M. D., Notarangelo, P. L., Nichols, S. J., Lane, K. S., Koliatsos, V. E. (2015). Diagnostic and treatment challenges in traumatic brain injury patients with severe neuropsychiatric symptoms: insights into psychiatric practice. *Neuropsychiatric disease and treatment, 11,* 1601-1607. doi: 10.2147/NDT.S80457.

Lebrun, Y. (1995). Luria's notion of '(frontal) dynamic aphasia'. *Aphasiology, 9*(2), 171-180. doi: 10.1080/02687039508248704.

Leopold, A., Krueger, F., Dal Monte, O., Pardini, M., Pulaski, S. J., Solomon, J., Grafman, J. (2012). Damage to the left ventromedial prefrontal cortex impacts affective theory of mind. *Social Cognitive and Affective Neuroscience, 7*(8), 871-880. doi: 10.1093/scan/nsr071.

Macmillan, M. B. (1986). A wonderful journey through skull and brains: The travels of Mr. Gage's tamping iron. *Brain and Cognition, 5*(1), 67-107. doi: 10.1016/0278-2626(86)90062-X.

Martín-Rodríguez, J. F., León-Carrión, J. (2010). Theory of mind deficits in patients with acquired brain injury: A quantitative review. *Neuropsychologia, 48*(5), 1181-1191. doi: 10.1016/j.neuropsychologia.2010.02.009.

Masel, B. E., DeWitt, D. S. (2010). Traumatic brain injury: a disease process, not an event. *Journal of Neurotrauma, 27*(8), 1529-1540. doi: 10.1089/neu.2010.1358.

Mesfin, F. B., Gupta, N., Taylor, R. S. (2017). *Diffuse axonal injury.* Treasure Island (FL): StatPearls Publishing (Internet). PMID: 28846342.

Morga, R., Goral-Polrola, J., Gozdziewska, M., Krupa, K., Pachalska, M. (2023). A new form of neurotherapy for a patient with anxiety disorder and anomic aphasia after neurosurgery for a

ruptured brain aneurysm post-COVID-19. *Annals of Agricultural and Environmental Medicine, 30*(2), 331-341. doi:10.26444/aaem/167370.

Osborn, A. J., Mathias, J. L., Fairweather-Schmidt, A. K. (2016). Prevalence of anxiety following adult traumatic brain injury: A meta-analysis comparing measures, samples and postinjury intervals. *Neuropsychology, 30*(2), 247–261. doi: 10.1037/neu0000221.

Pąchalska M. (2003) Neuropsychological Rehabilitation for Post-Traumatic Frontal Syndrome in Patients Recovering from Prolonged Coma: A preliminary report. *Acta Neuropsychologica*. 1(2):194-227

Pąchalska, M. (2007). *Neuropsychologia kliniczna. Urazy mózgu,* t.1. Warszawa: Wydawnictwo Naukowe PWN.

Pąchalska, M. (2008). *Rehabilitacja neuropsychologiczna. Procesy poznawcze i emocjonalne.* Lublin: Wydawnictwo UMCS.Pąchalska M. (2003) Neuropsychological Rehabilitation for Post-Traumatic Frontal Syndrome in Patients Recovering from Prolonged Coma: A preliminary report. *Acta Neuropsychologica*. 1(2):194-227

Pąchalska, M. (2019). Integrated self system: a microgenetic approach. *Acta Neuropsychologica, 17*(4), 349-393. doi: 10.5604/01.3001.0013.6198.

Pąchalska, M., Kaczmarek, B. L. J., Kropotov, J. D. (2014). *Neuropsychologia kliniczna: od teorii do praktyki.* [ang. Clinical neuropsychology: from theory to practice]. Warszawa: WN PWN. ISBN 978-83-01-17841-3.

Pąchalska, M., Kaczmarek, B. L. J., Kropotov, J. D. (2021). *Ja utracone i odzyskane.* Kraków: Oficyna Wydawnicza Impuls.

Pąchalska M., MacQueen B.D. (2002) Kwestionariusz Zachowań Osoby z Zespołem Czołowym – autoryzowana polska wersja "Frontal Behavioural Inventory" A. Kertesza i wsp. 1998: Ortopedia Traumatologia Rehabilitacja 4(1): 81-87..

Pąchalska, M., Talar, J., Baranowski, P., MacQueen, B. (2000). Rehabilitacja funkcji wykonawczych u chorych po zamkniętych urazach czaszki. *Ortopedia Traumatologia Rehabilitacja, 2*(3), 77-87.

Passingham, R. E., Bengtsson, S. L., Lau, H. C. (2010). Medial frontal cortex: from self-generated action to reflection on one's own performance. *Trends in Cognitive Sciences, 14*(1), 16-21. doi: 10.1016/j.tics.2009.11.001.

Premack, D., Woodruff, G. (1978). Does the chimpanzee have a theory of mind? *Behavioral and Brain Sciences, 1*(4), 515-526. doi: 10.1017/S0140525X00076512.

Prigatano, G. P. (2009). *Rehabilitacja Neuropsychologiczna.* Tłum. Łucja Domańska. Warszawa: WN PWN.

Prigatano, G. P. (2012). Anosognosia, denial, and other disorders of phenomenological experience. *Acta Neuropsychologica, 10*(3), 371–384. doi: 10.5604/17307503.1023676.

Prisco, L., Ganau, M., Aurangzeb, S., Moswela, O., Hallett, C., Raby, S., ... Sen, A. (2020). A pragmatic approach to intravenous anaesthetics and electroencephalographic endpoints for the treatment of refractory and super-refractory status epilepticus in critical care. *Seizure, 75,* 153-164. doi: 10.1016/j.seizure.2019.09.011.

Sbordone, R. J., Liter, J. C., Pettler-Jennings, P. (1995). Recovery of function following severe traumatic brain injury: a retrospective 10-year follow-up. *Brain Injury, 9*(3), 285-299. doi: 10.3109/02699059509008199.

Tenny, S., Thorell, W. (2024). *Intracranial Hemorrhage*. Treasure Island (FL): StatPearls Publishing (Internet). PMID: 29262016.

Theadom, A., Jones, K., Starkey, N., Barker-Collo, S., Ameratunga, S., Faulkner, J., ... Feigin, V. (2024). Symptoms and Engagement in Anti-social Behavior 10 Years After Mild Traumatic Brain Injury Within a Community Civilian Sample: A Prospective Cohort Study With Age-Sex Matched Control Group. *Archives of Physical Medicine and Rehabilitation, 105*(2), 295-302. doi: 10.1016/j.apmr.2023.07.016.

Torregrossa, W., Raciti, L., Rifici, C., Rizzo, G., Raciti, G., Casella, C., ... Calabrò, R. S. (2023). Behavioral and psychiatric symptoms in patients with severe traumatic brain injury: a comprehensive overview. *Biomedicines, 11*(5), 1449. doi: 10.3390/biomedicines11051449.

Vilkki, J. (1995). Neuropsychology of mental programming: an approach for the evaluation of frontal

lobe dysfunction. *Applied Neuropsychology, 2*(3-4), 93-106. doi: 10.1080/09084282.1995.9645346.
Walsh, K. (2000). *Neuropsychologia kliniczna.* Polish edition (ed.), D. Kądzielawa. Warszawa: Wydawnictwo Naukowe PWN.
Whelan-Goodinson, R., Ponsford, J. L., Schönberger, M., Johnston, L. (2010). Predictors of psychiatric disorders following traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 25*(5), 320-329. doi: 10.1097/HTR.0b013e3181c8f8e7.
Whelan-Goodinson, R., Ponsford, J., Johnston, L., Grant, F. (2009). Psychiatric disorders following traumatic brain injury: their nature and frequency. *The Journal of Head Trauma Rehabilitation, 24*(5), 324-332. doi: 10.1097/HTR.0b013e3181a712aa.

Address for correspondence:
Rafał Morga
Prof. B. FrańczukOrthopedic and Rehabilitation Hospital
in Lesser Poland, Modrzewiowa
30-224 Kraków, Poland
rafalmor@wp.pl