ORIGINAL ARTICLE

ACTA Vol. 5, No. 4, 2007, 202-212
NEUROPSYCHOLOGICA

TRAUMATIC INJURY TO THE FRONTAL AND TEMPORAL-PARIETAL LOBES OF THE BRAIN AND EXECUTIVE DYSFUNCTION

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Key words: *traumatic brain injuries, executive functions, microgenetic theory*

SUMMARY

Background. *Executive dysfunction is known to be a common sequela of injury to the frontal lobes. However, clinical experience suggests that executive dysfunction may also accompany injury to the temporal and parietal lobes. In addition to focal symptoms, behavioral disorders are often observed in patients after severe traumatic brain injury (TBI), regardless of localization.*

Material and methods*. Forty patients recovering from very severe TBI were examined, including twenty with frontal lobe injuries and 20 with temporal and/or parietal injuries; the groups were matched for age and sex. Apart from the analysis of documentation, the clinical interview and the psychological interview, the patients were also examined with the Clinical Test of Executive Functions – Revised (CTEF-R) and with the Mini-Mental State Examination (MMSE).*

Results. *Executive dysfunction appeared in almost all these patients regardless of the location of the brain damage. Nevertheless, the degree of dysfunction was considerably higher in patients with frontal lobe damage. No essential correlations were found between the level of overall cognitive functioning measure by the MMSE and the level of executive dysfunction.*

Conclusions*. Executive dysfunction is not the result of frontal damage alone, but frontal damage is a predictor of its severity. Microgenetic theory can be used to explain best the results we obtained.*

INTRODUCTION

The so-called "executive functions" consist in the ability to work out new behavioral patterns and ways of thinking in response to changing situations (Goldberg 2001, Pachalska 2002, 2004, 2007), as well as the ability to critically assess the new patterns and adjust them if necessary (Burgess 2003). Thus they are essential to adaptation (Lezak 1995). According to Mally and DeNatale (2001), the executive functions involve:

– creating a finite number of possible alternative behaviors in reaction to a situation that requires some response;
– choosing and initiating behaviors oriented towards implementing the primary goal;
– setting intermediate goals with a view towards their contribution to achieving the primary goal;
– monitoring the adequacy of behavior;
– correcting and modifying particular behaviors when conditions change;
– drawing conclusions from both success and failure for reference in the future;
– maintaining consistency of direction.

Executive functions can be summarized more succinctly, and perhaps more pragmatically as well, by the acronym "3S," which refers to:

– *starting* (initiating action);
– *stopping* (suppressing action);
– *shifting* (changing action in progress when the situation changes or the chosen action is not effective).

Regardless of how they are described in particular, in whole or in part, the executive functions bridge the gap between cognition and behavior, or, in other words, between thinking and acting. These are the processes responsible for the planning, collecting, coordinating, sequencing and monitoring of various different cognitive and motor functions. The executive functions play the main role in initiating one's own "private" action, and also in those activities which require interaction and cooperation with other people. Executive dysfunction thus may involve "thinking without acting," when knowledge of what needs to be done does not produce any organized effort to do anything, or "acting without thinking," when behavior is chaotic and impulsive, not guided by any discernable plan or purpose (Pachalska et al. 2002). The problems resulting from executive dysfunction also make the learning of new behaviors difficult, which often means, among other things, that the patient is not able to take full advantage of rehabilitation (Burgess and Robertson 2002, Pąchalska 2007).

Much of what has been said to this point may remind many readers of the classic clinical picture of "frontal syndrome." Shallice (1988) determined that the frontal lobes are acting in a 'supervisory' way. Pąchalska defines this in a similar way (2007). Equating the executive functions with the functions of

the frontal lobes constitutes the simplifying of the cognitive-behavioural metaprocesses that are the essence of the central executive system. Many neuropsychologists are more accustomed to using the term "frontal syndrome" than "executive dysfunction," while others (Burgess 2003) claim that "frontal syndrome" is now an outdated concept and not very useful in clinical practice. To be sure, the frontal lobes play an important role in those aspects of executive function that involve planning and organizing behavior. Pachalska (2007) points out that while executive dysfunction plays a considerable role in the clinical picture of "frontal syndrome," the term involves more than just those disorders that are clearly caused by damage to the frontal lobes. At the same time, not every patient with problems in the area of executive functions has damaged frontal lobes, and not every patient with frontal lobe damage has clearly identifiable disorders of the executive functions. Since the term "frontal syndrome" in turn also includes particular personality disorders, in addition to the behavioral aspects related to executive dysfunction, it is difficult to justify either using the two terms as synonyms, or discarding one or the other of them. Despite the obvious overlap, each of them has its own domain of reference.

The neuropsychological diagnosis of patients with brain damage, especially but not exclusively when frontal lobe functions are compromised, is not complete without some assessment of the executive functions. This requires that we apply a number of different approaches to testing, however, since patients with frontal lobe damage display different problems in performing their daily life activities and react differently to the instructions they are given than do patients with damage in more posterior areas, i.e. the temporal-parietal area. Executive dysfunction manifests in a wide array of difficulties encountered both in various neuropsychological tests, and in real-life situations. Hence one should take note of the qualitative character of these difficulties, not just the presence or absence of error on a given task (Brown & Pachalska 2003).

The aim of our study, then, was to describe executive dysfunction in a population of brain-damaged patients, hypothesizing a distinction between those who have sustained injury to the frontal lobes, and those whose lesions are more in the temporal and parietal regions. Thus the theoretical aim of the study was to ascertain whether there was any connection between the occurrence and character of executive dysfunctions and the location of the injury in the anterior or posterior areas of the brain. Above all, it is important to determine whether executive dysfunction is connected exclusively with damage to the frontal lobes, which would presumably involve the loss of certain specific frontal lobe functions essential to executive functions, or may also result from a less specific loss of cognitive efficiency (including memory and intelligence deficits), which may deteriorate after damage to the temporal and parietal lobes.

MATERIAL AND METHODS

We studied forty patients with severe, very severe and extremely severe traumatic brain injury (TBI), under treatment at the Neurotraumatology Clinic of the Jagiellonian University's College of Medicine in Cracow, Poland, or under the auspices of the Foundation for Persons with Brain Dysfunctions (also in Cracow) in 2005-2006. Group A included 20 patients with traumatic lesions visible on computer tomography of the brain exclusively in the frontal lobes, while group B was comprised of 20 patients with lesions in the temporal or parietal areas, without any visible damage to the frontal lobes. All these patients were at least 3 months after the initial injury (and thus well after the acute stage), but not more than 3 years. The patients from group B were paired with those from group A according to gender, age (within 2 calendar years) and the severity of the trauma. There were 15 men and 5 women in both examined groups, which gives a proportion of 75% to 25%, which is consistent with the usual frequency of head trauma in males and females. The average age of the patients in group A was 35.2 years (SD = 12.52 years), whereas in group B the average age was 35.1 ± 12.32 years. The slight difference in the average age was not statistically significant, so the groups were homogeneous in this respect.

In TBI, of course, it is notoriously difficult to make the claim that any part of the brain is completely undamaged, but in this population the differences in location of the primary traumatic injuries were very obvious. Patients with lesions in both anterior and posterior areas were not included in the study. Patients under 18 and over 60 years of age were also excluded from the examinations, as were those whose physical or overall mental state would make it hard or impossible for them to perform the tasks involved in our research.

All patients signed a form granting consent to participation in the study, and the research project received the consent of the local bioethics committee.

The independent variables included gender, age, education, the cause of injury (e.g. accident, fall, violent assault, an accident at work), the main pathomechanism of the brain damage (loss of cerebral tissue, hematoma, contusions, etc.) and the severity of the trauma.

Tables 1 and 2 give the essential clinical information concerning the patients from both groups.

In both groups the largest percentage of patients (60%) sustained a TBI as a result of a traffic accident; 20% were injured in a fall from a height, mainly from stairs or from a ladder (in one case this was an unsuccessful suicide attempt). Two persons from group A and one person from group B sustained a TBI at work (in all three cases the accidents took place on a construction site). In group A one person had been mugged, whereas two persons (one in each group) sustained a TBI while engaged in sports activities (in one case while skiing, in the other while climbing in the mountains). There were 9

Table 1. Clinical data for persons from group A

ID	Sex	Age	Education	Cause of TBI	Primary pathomechanism	Severity
1	M	18	primary	traffic accident	brain contusion	very severe
2	M	37	vocational	fall from height	subdural hematoma	severe
3	F	59	secondary	traffic accident	subdural hematoma	very severe
4	M	26	secondary	traffic accident	brain contusion	severe
5	F	34	vocational	work accident	brain contusion	extremely severe
6	M	19	vocational	traffic accident	subdural hematoma	extremely severe
7	M	45	primary	traffic accident	brain contusion	very severe
8	M	32	secondary	traffic accident	epidural hematoma	severe
9	M	21	vocational	traffic accident	brain contusion	very severe
10	F	49	vocational	fall from height	subdural hematoma	severe
11	M	55	secondary	traffic accident	brain contusion	severe
12	M	32	secondary	sports injury	subdural hematoma	very severe
13	M	48	college	traffic accident	subdural hematoma	severe
14	M	22	college	fall from height	brain contusion	extremely severe
15	M	55	primary	assault and battery	brain contusion	extremely severe
16	F	29	secondary	traffic accident	subdural hematoma	severe
17	M	30	primary	fall from height	subdural hematoma	severe
18	M	33	primary	traffic accident	brain contusion	very severe
19	F	32	secondary	work accident	epidural hematoma	severe
20	M	28	college	traffic accident	brain contusion	very severe

Table 2. Clinical data for persons from group B

ID	Sex	Age	Education	Cause of TBI	Primary pathomechanism	Severity
1	M	18	secondary	work accident	brain contusion	severe
2	M	35	vocational	traffic accident	subdural hematoma	very severe
3	F	60	vocational	traffic accident	subdural hematoma	severe
4	M	26	primary	traffic accident	brain contusion	extremely severe
5	F	35	secondary	traffic accident	brain contusion	extremely severe
6	M	18	vocational	traffic accident	subdural hematoma	very severe
7	M	44	secondary	traffic accident	brain contusion	severe
8	M	32	primary	fall from height	epidural hematoma	very severe
9	M	22	vocational	traffic accident	brain contusion	severe
10	F	48	vocational	sports injury	subdural hematoma	severe
11	M	54	college	fall from height	brain contusion	very severe
12	M	32	primary	work accident	subdural hematoma	severe
13	M	47	secondary	traffic accident	subdural hematoma	extremely severe
14	M	23	primary	fall from height	brain contusion	extremely severe
15	M	55	primary	work accident	brain contusion	severe
16	F	30	primary	fall from height	subdural hematoma	severe
17	M	29	college	traffic accident	subdural hematoma	very severe
18	M	32	secondary	traffic accident	brain contusion	severe
19	F	32	college	traffic accident	epidural hematoma	very severe
20	M	29	college	work accident	brain contusion	severe

severe, 7 very severe and 4 extremely severe injuries in the both groups. Some differences were found between the groups in terms of the main pathomechanism, which is largely dependent on the mechanics of the blow that inflicted the injury.

In group A, 8 persons had brain contusions in the frontal lobes, 7 persons had been operated for subdural hematomas, and 1 person had suffered an epidural hematoma; 4 persons had lost cerebral tissue due to an open skull fracture (for example one person was hit in the face by a circular saw, while

in another case, a wooden stake punctured the patient's skull on the upper edge of the eye socket after a fall from the stairs). In group B, however, there were no cases of cerebral tissue defect; 8 persons had been operated for subdural hematomas, and 2 for epidural hematomas, while 10 persons had sustained brain contusions in the dorsal, lateral part of the cerebral cortex.

For purposes of comparison, we used the following methods:

– documentation analysis;
– a clinical interview;
– a psychological interview;
– the Clinical Test of Executive Functions – Revised, developed by M. Pachalska, B.D. MacQueen, and B. Grochmal-Bach;
– the Mini-Mental State Examination (MMSE).

The statistical methods included the F Test (Fisher) to compare variance, the t-Student test to test for significance between paired groups, and regression analysis using the linear Pearson's r to test for correlation.

RESULTS

Tables 3, 4 and 5 summarize the results obtained by the patients we studied on the Clinical Test of Executive Functions - Revised (CTEF-R) presents.

The comparison is shown graphically in Fig. 1.

To sum up, the global results for of all seven tasks and all four accomplishment parameters were better in group B (with temporal and parietal lobe damage) than in group A (with frontal lobe damage), and all these differences were statistically very significant. In no case, however, did the results from group B show no signs of executive dysfunction.

The MMSE was administered to both groups.

The results from the CTEF-R and the MMSE failed to correlate significantly.

Table 3. Average results achieved by the patients from group A on the Clinical Test of Executive Functions – Revised

Task	Independence		Performance time		Efficiency		Prompting	
	x *	SD **	x	SD	x	SD	x	SD
I. Preparing a sandwich	2.1	0.72	2.1	0.97	1.9	0.72	1.9	0,85
II. Fetching water	3.1	0.85	2.4	0.88	2.6	0.88	2.2	0,93
III. Buying a newspapaer	1.9	0.79	2.0	0.79	2.0	0.65	2.0	0,92
IV. Writing a letter	2.1	0.89	1.9	0.75	1.8	0.83	2.0	1,00
V. Addressing an envelope	2.1	0.72	2.2	0.95	2.1	0.72	2.0	0,6
VI. Telephoning	1.5	0.51	1.4	0.50	1.4	0.59	1.3	0,57
VII. Finding a key	1.3	0.47	1.4	0.49	1.3	0.47	1.3	0,47
TOTAL	14.1	2.52	13.3	2.57	13.1	2.35	12.6	3.03

*x – mean
**SD –standard deviation

Table 4. Average results achieved by the patients from group B on the Clinical Test of Executive Functions – Revised

Task	Independence		Performance time		Efficiency		Prompting	
	x *	SD **	x	SD	x	SD	x	SD
I. Preparing a sandwich	3.8	0.79	3.6	0.99	3.3	0.91	3.7	0.81
II. Fetching water	3.8	0.44	3.7	0.57	3.5	0.69	3.5	0.69
III. Buying a newspapaer	3.4	0.94	3.5	0.83	3.3	0.98	3.2	0.89
IV. Writing a letter	3.4	0.88	3.3	0.97	3.3	0.80	2.8	0.55
V. Addressing an envelope	2.9	0.59	3.0	0.94	3.0	0.92	2.9	0.88
VI. Telephoning	3.3	0.91	3.2	0.88	3.5	0.83	2.7	0.86
VII. Finding a key	3.7	0.92	3.6	0.99	3.7	0.92	2.9	1.14
TOTAL	24.1	4.77	23.7	5.05	23.5	4.81	21.5	3.27

*x – mean
**SD –standard deviation

Table 5. Descriptive statistics for both examined groups in respect to global results from the Clinical Test of Executive Functions

Statistic	Group A	Group B
Mean	53.4	92.7
Standard error	1.92	3.69
Median	53	97
Mode	53	97
Standard deviation	8.57	16.49
Sample variance	73.50	271.82
Range	37	68
Minimum	43	36
Maximum	80	104
CI 95%	4.01	7.72

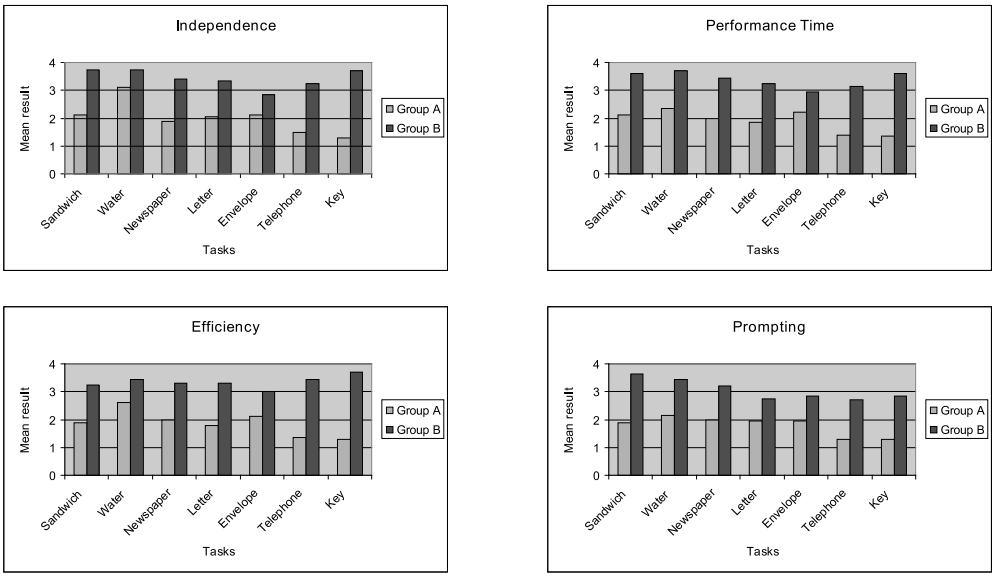

Fig. 1. Results achieved by both study groups in all 7 tests tasks from the CTEF-R in respect to the level of independence, performance time, efficiency, and prompting

Table 6. Statistical significance of differences between the two groups

Parameter	Student's test		F Test	
	t	p	F	P
1 Total score	-10.2457	0.0000***	0.0270	0.0042**
2. Sandwich	-7.9015	0.0000***	0.8633	0.3760NS
3. Water	-5.7704	0.0000***	3.7694	0.0033**
4. Newspaper	-5.8399	0.0000***	0.6547	0.1820NS
5. Letter	-6.9978	0.0000***	1.3443	0.2626NS
6. Envelope	-4.5017	0.0001***	0.6350	0.1653NS
7. Telephone	-12.2540	0.0000***	0.2911	0.0050**
8. Key	-11.3857	0.0000***	0.1193	0.0000***
9. Independence	-9.3572	0.0000***	0.2794	0.0039**
10. Performance time	-7.9598	0.0000***	0.2597	0.0025**
11. Efficiency	-9.8497	0.0000***	0.2391	0.0015**
12. Prompting	-9.9460	0.0000***	0.3748	0.3748NS

* - $p < 0.05$
** - $p<0.01$
*** - $p<0.001$

Table 7. Descriptive statistics for results from the Mini-Mental State Examination (MMSE) obtained from patients from both study groups

Statistic	Group A	Group B
Mean	22.3	13.5
Standard error	0.73	0.61
Median	22	13
Mode	19	17
Standard deviation	3.25	2.72
Sample variance	10.55	7.41
Range	9	10
Minimum	18	9
Maximum	27	19
CI (95.0%)	1.52	1.27

DISCUSSION

Brown's microgenetic theory (2000) can be used to explain the phenomena observed in our study.

According to Brown (2005), the individual's behavior at a given moment evolves from the oldest and deepest parts of the brain (the brainstem) to the youngest and most external (the neocortex). Every behavior has a stratification resulting from the structure of the brain itself. The lower in the nervous system the mental state is formed, the more this state is characterized by simplicity, unity, directness, generality; the higher the state evolves, the more it is specified, complex, unique, indirect.

In microgenetic theory behavior develops in three stages, corresponding to the stages of the brain's development, that is:

– at the level of the brainstem (including the midbrain), both stimulus and response are immediately categorized to one of a very few, very broad categories (e.g. "edible" or "dangerous"). The appearance of the object automatically initiates a stereotyped, largely instinctive reaction suitable for a stimulus of this type.
– at the level of the limbic system, the emotions are dominant (affect, mood). Under the influence of the limbic system one is guided by likes and dislikes, seeking pleasure and avoiding pain. The choice of one from many possible behaviors depends mostly on preferences and will.
– on the level of the neocortex, the brain makes an analysis of the features of the perceived objects and selects a reaction that seems suitable for the individual's best interests or declared values (esthetic, moral etc.).

The choice takes place in a split second on the lowest level, in seconds in the limbic system, but in the domain of the cerebral cortex the process make take a few seconds, minutes, hours, or weeks, months, even years.

Many descending (afferent) neural pathways from cortex to lower structure are inhibitory, while of all the parts of the cerebral cortex the frontal lobes have the richest net of neuronal connections with the subcortical nuclei of the brain and, above all, with the limbic system. That is why the frontal lobes play such an important role in the process of shaping behavior. According to Brown (2005), it is possible to compare this to the way a sculptor creates a work of art by removing what is unnecessary and ugly, leaving only what is beautiful. When the frontal lobes stop acting and are disorganized or weakened, then the behaviors reach the surface from the thalamus and from the limbic system in an "unfinished" state, and are not subject to this "carving." Alternatively, trauma to the lower levels may deprive the individual of the motivation to act.

However, this does not have any influence on the posterior processes, mostly involved with perception, which continue to process information and create a picture of reality (Brown & Pąchalska 2003). This fact explains the phenomenon pointed out by Pąchalska (2007), that in patients with frontal syndrome, in spite of the bizarreness of certain behaviors, premorbid intelligence is often preserved in the patient. In other words, as the wife of one of our patients from group A remarked, "he speaks so wisely and acts so stupidly." Normal cognition is an essential but not sufficient condition for normal behavior (Pachalska et al. 2003).

Thanks to microgenetic theory it is possible to explain why the task "finding the hidden key" is so difficult for patients with frontal lobe damage. In this task, the subject does not have any physical stimulus, but must develop it within her own mind. She must ask herself where it is possible to hide a key, and search there. The lack of motivation caused by frontal lobe damage leads the patient to disorganized, irrational searching, which is quickly abandoned, even though the patient is often able to describe how a logical search should look. The opposite happens in the case of a person with damage to

the posterior lobes. In such a person, the brain forms a picture of the world which is incomplete, distorted, difficult to recognize. In this situation, it is difficult to speak of full awareness, although the individual can behave "normally" within certain boundaries, when the process of shaping behaviors by the frontal lobes has not been disrupted. For this reason the tasks of writing a letter and addressing an envelope was most difficult for patients from group B. The patients often did not realize at all they were not performing this task correctly.

CONCLUSIONS

Executive dysfunction appears in almost all patients after a severe, very severe or extremely severe traumatic brain injury regardless of its location in the brain. These disorders occur on a considerably greater scale, however, in patients with frontal lobe damage than in patients with damage to the temporal and parietal areas of the brain.

There was no significant correlation between the general level of cognitive functioning and the executive dysfunctions in patients with severe traumatic brain injury.

Those tasks which required an effort of imagination on the part of the subject turned out to be most difficult for the patients with frontal lobe damage, while the writing tasks turned out to be most difficult for patients with temporal and parietal lobe damage.

The results we received require confirmation on a larger patient population.

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