SUMMARY

Introduction. "Frontal syndrome," which manifests itself in characteristic personality changes and behavioral disorders, is a frequent after-effect of traumatic brain injury (TBI). There have been no published studies, however, directly indicating the presence of this syndrome in patients aroused from prolonged post-traumatic coma, nor is it known whether or not the duration of the coma influences the course or nature of the syndrome in particular cases. The goal of our study was to fill this gap.

Materials and methods. 38 patients treated in the Rehabilitation Clinic at the Ludwik Rydygier Medical University in Bydgoszcz, Poland, participated in the trial. They were divided into 2 groups: group A included 18 patients aroused from a short-term post-traumatic coma, while group B consisted of 20 patients aroused from a prolonged post-traumatic coma. Frontal lobe lesions were confirmed radiologically in each case. The authorized Polish version of the Frontal Behavioral Inventory (FBInv) by Kertesz et al. was used to measure frontal syndrome.

Results. Symptoms of frontal syndrome as measured by the FBInv occurred in both groups, but the results in group B were significantly worse than in group A, suggesting a more severe frontal syndrome.
Conclusions. Our patients recovering from prolonged post-traumatic coma showed more intense disturbances in particular "frontal syndrome" features. Further research is needed to enlarge the analysis to encompass the possible influence of other important factors on the clinical picture of the patient aroused from prolonged coma.

INTRODUCTION

The loss of consciousness in coma deprives the patient of the ability to perceive herself and her surroundings, regardless of external stimulation (Mumenthaler & Mattle 2001). In clinical practice, coma is diagnosed when the patient does not regain consciousness within 3 days after the adverse event (Pąchalska et al. 2002, Talar 2002).

Consciousness can defined most simply for these purposes neurologically, as the readiness of the central nervous system to receive and process information from the environment. In this binary approach it is one of two possible physiological states of a living being with a developed central nervous system: awake or not awake (Talar 2002:9). When used in psychology and related fields, however, consciousness has a much broader meaning (Grochmal-Bach and Pąchalska 2004), and by implication it can thus occur or function in a variety of distinguishable states: a person who is awake may experience a number of different states of altered consciousness. While the neurological concept of consciousness is to a large extent quantifiable and thus can be operationalized for purposes of statistical analysis, the various psychological definitions of consciousness do not lend themselves easily to quantification. The problem is further complicated by the transitiveness of "psychological" consciousness: from the neurological perspective, one is "conscious" or "unconscious"; from the psychological perspective, one is "conscious of something," or not. Thus a state of neurological unconsciousness is always and by definition pathological, while a healthy person may be and usually is unconscious of many things.

The etymology of the Polish word for "coma", śpiączka, implies a similarity to the state of sleep (śpi means "s/he/it sleeps"). In fact, there is an obvious analogy, consisting in the disconnection of the reception of external stimuli by the cerebral cortex through the brainstem, which modulates the sleep/wake state. During the normal change of physiological state from sleep to wakefulness, a healthy brain goes through several distinct phases, but the transition is usually so rapid that we are not aware of any states intermediate between deep sleep and full wakefulness. In a person aroused from a coma, however, the transition phases are typically prolonged much longer, from hours in the best case to months or even years in the worst; in a "persistent vegetative state," for example, the process of recovering consciousness reaches a certain plateau and proceeds no farther. The curves illustrating the course of this process may be diverse. Regaining "neurological" conscious-
In a patient aroused from a prolonged coma does not ensure that full "psychological awareness" has been regained. Pufal (2004) points out that the mere state of being conscious is a base, forerunner and condition for psychological awareness of self and the environment.

According to Pachalska (2003a, 2004, 2007), the basic pathomechanism of coma consists in:

- a disturbance of the basic physiological rhythm of wakefulness/sleep modulated by the brainstem (which explains why most post-traumatic comas are associated with compressive pressure on the brainstem),
- a state of diminished neural activity in the brain below that of normal sleep (dreams do not appear and sensory stimuli do not produce wakefulness).

If the disturbances of brainstem activity are so serious that the loss of consciousness lasts longer than two weeks, then one may speak of prolonged coma (though the category is admittedly rather inexact and the division at 14 days is rather arbitrary, so that various authorities use different cut-off points). A prolonged coma results in many secondary problems in the central nervous system. A prolonged coma lasting over four weeks is generally thought to have a very unfavourable prognosis, because of the alleged irreversibility of the changes in the brain stem. It does seem clear and even patently obvious that the longer a coma lasts, the greater the possibility that the patient will never be aroused, or even if aroused to neurological consciousness, will never regain full psychological consciousness.

Our own clinical experience (Talar 2002) suggests, however, that this commonly accepted view may be unduly pessimistic. Clinical work done at the Rehabilitation Clinic at Bydgoszcz Medical University from 2000 to 2005 demonstrated in practical terms that it is indeed possible to achieve significant success in the rehabilitation of patients aroused from prolonged coma. The following conditions seem to be essential in order to achieve satisfactory results:

- the earliest possible commencement of procedures aimed at stimulating the central nervous system, especially the sensorimotor systems;
- long-term application of these procedures, even when progress does not seem apparent on the usual scale of several weeks or several months.

Walsh (2000) pointed out that in many patients who have suffered a traumatic brain injury (TBI) and post-traumatic coma, in whom there is complete or nearly complete neurological recovery, some neuropsychological deficiencies still occur. These deficiencies are most often connected with memory and adaptability. In the latter case, other features of "frontal syndrome" may also occur. This is scarcely surprising: the frontal lobes are highly exposed to injury in the case of automobile accidents and falling forward, which accounts for a considerable majority of TBI cases, especially in adults. At the same time, the overall shock to the brain and the contre-coup effect often lead to hematomas in and around the brainstem, producing coma. Later on, then, one of the secondary effects of prolonged coma in and of itself is neuronal loss due to apoptosis and generalized neurodegeneration, producing behav-
ioral and emotional symptoms that in many cases resemble those of fronto-temporal dementia (FTD), especially when there is also primary damage to the frontal lobe.

Subcortical structures of the brain, including the basal ganglia and the limbic system, are equally subject to both the primary effects of the injury itself (the shear effect, for example, often produces subcortical haemorrhages), and the secondary, neurodegenerative effects of prolonged unconsciousness. Thus the whole complex dialectic of affect, mood, personality and cognition (included learned values) that modulates behavior in a sometimes highly unstable manner is almost always destabilized by TBI with prolonged coma. The drive system centered in the brainstem, the affective system centered in the limbic system, and the executive system centered in the frontal lobes are each affected individually, while their interaction with one another, which ultimately produces behavior, can hardly avoid significant changes, usually in the direction of entropy and chaos (Perecman 1987, Fuster 1980, Goldberg 2001). The particular symptoms, however, cannot be ascribed to the loss or dysfunction of a particular cerebral "processor," as in the lesion-based neuropsychology of the last century, but rather to the destabilization of a complex "network of networks," in which the frontal lobes play a major (but hardly exclusive) role (Prigatano 1994, Poon 1992, Kaczmarek 1987).

It would be reasonable, then, to expect that the clinical picture of a patient with frontal damage and prolonged coma resulting from a TBI would resemble in some respects that of patients with other kinds of frontal damage, or that of patients with neurodegenerative changes in the brain, but would be identical to neither, possessing some very specific features. The location of the primary lesion in TBI can be only one of the many factors producing these features; others include the severity of the primary injury, the duration of the coma (which would presumably display some correlation with the secondary effects mentioned above), and the premorbid personality and character of the patient. Brain injury never leaves its imprint on the "tabula rasa" of the healthy brain, but rather on an individual brain that over the course of life to the moment of the accident had developed its own solutions to various problems, and will continue to do so after the accident, though in dramatically altered ways.

The problems implicitly and explicitly raised in the foregoing brief discussion far exceed the possibilities of a single study. Our aim in the present study was to provide some clinical data related to frontal syndrome in patients aroused from post-traumatic coma, with particular attention to

- **symptomatology.** Much of the neuropsychological literature mentions "frontal syndrome" in TBI patients generally, but without much specific clinical detail that would enable a differential diagnosis of "post-TBI" frontal syndrome, not to mention the problems of patients recovering from prolonged coma.

- **possible correlation** between the length of the coma and the qualitative and quantitative aspects of frontal syndrome.
MATERIAL AND METHODS

Our research involved 38 persons aroused from coma after a TBI, all of whom were patients in the Rehabilitation Clinic of the University Hospital in Bydgoszcz. The duration of coma was used to divide the patients into two groups. Twenty patients who had been in coma less than 2 weeks constituted group A, whereas, group B consisted of 18 patients who were in coma for longer than 1 month. Using these somewhat arbitrary cut-off points we attempted to avoid borderline cases, where a patient in coma for 29 days would belong to group A, while a patient in coma for 31 days would belong to group B, and a patient in coma for 30 days would be hard to classify.

The following exclusion criteria were applied:
- coma lasting longer than 2 weeks but less than one month (see above);
- clinical condition too severe to conduct neuropsychological evaluation;
- a history of previous TBI or other brain damage, psychiatric disease, dementia, or intellectual handicap;
- age under school age (7 years in Poland) or over retirement age (65 years).

In the whole study population, the average age was 25.5 years (SD 8.19 years); the youngest patient was 9 years old, and the oldest was 49. The number of women in the examined population was 13, which constituted 34.2% of the total. The average age of the women in our population was 24.1 ± 6.58 years (range: 11-39 years), while the men were slightly older: 26.2 ± 8.95 years (range: 9-49 years); however, the difference was not statistically significant (p=0.23, n.s.).

The average age of the patients in group A came to 24.4 ± 6.57 years; the youngest patient in the group was 11, and the oldest 39. In the women from group A the average age was 23.0 ± 5.68 years (range 11-29 years); in case of the men, 25.6 ± 7.29 years (range: 12-39 years). This group included 8 women, constituting 44.4% of the group.

The average age of the patients from group B was 26.4 ± 9.49 years; the youngest patient in the group was 9, and the oldest was 49. In the women in group B, the average age was 25.8 ± 8.20 years (range: 17-39 years); in the men, 26.6 ± 10.14 years (range: 9-49 years). There were only 5 women (25%) in group B.

During the trial, the following research tools were used:
1. a structured case history directed to the goal of our research. The following issues were particularly emphasized:
   - neurological disorders and coping mechanisms used by the patient to handle physical and mental limitations resulting from the illness;
   - cognitive disorders and difficulties appearing in everyday behavior;
   - emotional disorders and the patient's personality, with particular consideration of available information concerning the premorbid personality.
2. the Frontal Behavioral Inventory (FBInv), developed by Kertesz et al. (1997, 2000) to differentiate fronto-temporal dementia (FTD) from other kinds of dementia. We used the authorized Polish version of the FBInv (Pachalska & MacQueen 2000), which has been standardized for use in clinical practice with post-traumatic patients in order to diagnose and assess frontal syndrome. The FBInv consists of 24 questions focusing on all axial symptoms of frontal syndrome, as follows:

- apathy;
- aspontaneity;
- indifference;
- emotional flatness;
- inflexibility;
- concreteness;
- personal neglect;
- disorganization;
- inattention;
- loss of insight;
- logopenia;
- verbal apraxia;
- preservation;
- irritability;
- excessive jocularity;
- poor judgment;
- inappropriateness;
- impulsivity;
- restlessness;
- aggressiveness;
- hyperorality;
- hypersexuality;
- utilization behavior;
- incontinence;
- alien hand.

The questionnaire is filled out by a caregiver (usually a family member) with the assistance of the clinician, but not in the presence of the patient. The caregiver is asked to rate the severity and frequency of behavioral changes observed in the patient since the accident. The answers are scored on a scale from zero to three:

0 = lack of pathological behavior in the given parameter;
1 = mild degree of disturbances, problems appear occasionally;
2 = moderate degree of disturbances, problems appear relatively often;
3 = severe degree of disturbances, problems appear very often or incessantly.
RESULTS

A summary of the results obtained by our patients from groups A and B on the FBInv are given in Table 1.

In both of the examined groups we observed higher scores in the areas of indifference, emotional flatness and irritability. The patients from groups A and B (or their caregivers) complained of similar difficulties in understanding of metaphors and abstracts. Inflexibility was slightly lower in group A, and slightly higher in group B. This fact should probably be connected with the great difficulties these patients have in adjusting to changing conditions, first of all because of diminished awareness.

Persistency (aimless repeating of identical behaviors, both motor and verbal) was more often noticed in patients from group B.

The excessive jocularity characteristic of frontal syndrome was more pronounced in patients from group A, when compared to those from group B.

Table 1. Results obtained by patients from both study groups (A and B) in the particular questions of the FBInv

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Mean score</th>
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<tbody>
<tr>
<td></td>
<td>A</td>
</tr>
<tr>
<td>Apathy</td>
<td>2.2</td>
</tr>
<tr>
<td>Aspontaneity</td>
<td>2.4</td>
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<tr>
<td>Indifference, emotional flatness</td>
<td>2.0</td>
</tr>
<tr>
<td>Inflexibility</td>
<td>2.3</td>
</tr>
<tr>
<td>Concreteness</td>
<td>2.7</td>
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<tr>
<td>Personal neglect</td>
<td>2.8</td>
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<tr>
<td>Disorganization</td>
<td>2.9</td>
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<tr>
<td>Inattention</td>
<td>2.9</td>
</tr>
<tr>
<td>Loss of insight</td>
<td>3.0</td>
</tr>
<tr>
<td>Logopenia</td>
<td>2.4</td>
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<tr>
<td>Verbal apraxia</td>
<td>2.5</td>
</tr>
<tr>
<td>Preservation</td>
<td>1.7</td>
</tr>
<tr>
<td>Irritability</td>
<td>1.8</td>
</tr>
<tr>
<td>Excessive jocularity</td>
<td>2.4</td>
</tr>
<tr>
<td>Poor judgment</td>
<td>2.1</td>
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<tr>
<td>Inappropriateness</td>
<td>2.5</td>
</tr>
<tr>
<td>Impulsivity</td>
<td>2.8</td>
</tr>
<tr>
<td>Restlessness</td>
<td>2.9</td>
</tr>
<tr>
<td>Aggressiveness</td>
<td>2.4</td>
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<tr>
<td>Hyperorality</td>
<td>1.7</td>
</tr>
<tr>
<td>Hypersexuality</td>
<td>2.1</td>
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<tr>
<td>Utilization behavior</td>
<td>1.7</td>
</tr>
<tr>
<td>Incontinence</td>
<td>1.1</td>
</tr>
<tr>
<td>Alien hand</td>
<td>0.1</td>
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</tbody>
</table>
Interesting results were observed in the case of hyperorality. In the patients from group B, who had been longer in coma, behaviors of this kind were observed more often than in the patients from group A. It often happens that a patient aroused from a prolonged coma constantly calls for something to drink, and eats everything remotely edible that comes within arm's reach (provided of course that they are able to grab it).

Great intensification of utilization behavior (the compulsion to touch, pick up and manipulate all objects that come within reach) was present in both groups. Moreover, patients aroused from coma usually adhere rigidly to all forms of rules and schedules once accepted, and present almost panic-stricken reactions to disorder in their surroundings, or to interruption of an activity that has already been initiated.

The families of the patients from group A less often complained about incontinence, while such problems were common in patients from group B. One may well wonder to what extent the problems with incontinence result from strictly physiological reasons (i.e. lack of ability to control the sphincters), and to what extent from behavioral factors (i.e. the patient simply does not want to go to the toilet, or cannot stop a started activity in order to satisfy physiological needs, etc.). In the latter case, the incontinence would be connected with disturbances of planning and controlling actions, a function of the frontal lobes.

The percentage differences between the two groups in respect to "alien hand" would be very striking, except that the "law of small numbers" comes into play: overall, there were very few positive responses to this question, much less than in published statistics for FTD (Kertesz et al. 1997). It is noteworthy, however, that in several patients from group B this strange phenomenon was observed in its classic form, when one of the hands (usually the left) seems to the patient not to belong to her, and behaves, as it were, independently of the patient's consciousness (e.g. the patient takes food from a plate with the right hand, and immediately puts the food back on the plate with the left hand).

To sum up, Fig. 1 presents the average number of points obtained by patients from both groups (A and B) in particular categories. The scale on the

![Graph]

Fig. 1. Results obtained by patients from groups A and B from the FBInv in the main categories of emotional, cognitive, behavioral and neurological disorders.
y axis was created from the number of features (24) multiplied by the maximum score (3 pts).

Fig. 1 shows clearly that the differences in neurological disorders are the most pronounced between groups, while cognitive disorders differ the least.

**DISCUSSION**

Our results suggest that the duration of coma is not indifferent for the profile of disturbances defined as "frontal syndrome." How can we explain the data? As the duration of coma results rather from the extent of the brainstem contusion than from damage to the frontal lobes themselves, the obtained results seem to be hard to explain, if one assumes that frontal syndrome is a direct result of frontal lobe damage. If that were the case, we would expect to find the factors of location and severity of injury to be more decisive for frontal syndrome than duration of coma. Beyond a general tendency to greater intensity of symptoms with the prolongation of coma, we may also observe that the differences were the most pronounced in the case of the neurological disorders, and the least pronounced in the case of cognitive disorders. The disturbances here called "neurological" take place beneath the threshold of consciousness, and are thus connected with the lower levels of the central nervous system (CNS), while cognitive functions appear in the higher layers, primarily though not exclusively in the cortex. Coma results from brainstem contusion, i.e. on the lowest level of the CNS. In that case, we should not be surprised that in patients after prolonged coma, those features of frontal syndrome that reflect disruption of frontal lobe control over the functions of lower structures also intensify.

Many other differences noted in the obtained results may be explained by the fact that patients with post-traumatic frontal syndrome after prolonged coma have to overcome not only the primary effects of frontal lobe damage, but also deeper disorders in consciousness resulting from having been so long in coma. As suggested in the Introduction, the sole fact of regaining consciousness after coma does not determine consciousness in the full sense of the word (cf. Pachalska 2007). Thus the passivity typical for frontal syndrome as such is accompanied by the passivity of a person who does not really know if she is not perhaps dreaming. Once again, it seems clear that the strict separation of phenomena connected exclusively with the "executive" functioning of the frontal lobes (conceived as a discrete neuroanatomical structure) from those putatively connected with motor and affective disorders initiated subcortically is not only impossible, but also pointless.

The results obtained from both groups thus support the conclusion that the duration of coma intensifies disturbances in the scope of respective features of "frontal syndrome," especially the neurological ones.
The Frontal Behavioral Inventory (FBInv), originally prepared in Canada and made available in Poland, seems to be an adequate tool for the examination of symptoms observed in this population of patients.

Further research directed towards a deeper analysis of the possible influence of other important factors on the clinical picture of the patient aroused from prolonged coma seems warranted. The premorbid personality of the patient, which may have a significant impact on the way in which the damaged brain handles the radically changed conditions of life after a TBI and prolonged coma, is surely one of the most significant issues here (cf. Pachalska 2003a, 2003b, 2007). Possible correlations between results obtained from the FBInv and individual psychometric parameters (e.g. intelligence, memory, concentration, etc.) should also be examined. In order to explain the interaction of the frontal lobes with the other brain structures in the process of forming and controlling behavior, one might also wish to compare detailed information on the nature and localization of brain lesions to the frontal syndrome symptoms revealed by the FBInv.

**CONCLUSIONS**

As a result of our research we reached the following conclusions:

1. Almost all the patients from both groups A and B were identified as suffering from "frontal syndrome."
2. Patients from group B (after prolonged coma) suffer from more intense disturbances within particular features of "frontal syndrome" when compared to the patients from group A (less than two weeks of unconsciousness).
3. The most pronounced differences involved neurological disturbances.
4. Further research is needed, directed towards a deeper analysis of the possible influence of other important factors on the clinical picture of the patient aroused from prolonged coma.

**REFERENCES**


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