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USEFULNESS OF SLEEP RECORDS AFTER MILD HEAD TRAUMA TO PREDICT SHIFT WORK EFFECTIVENESS

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Summary

Validity of polysomnography for determining the post-traumatic sequelae was evaluated in 33 male patients after a mild head trauma. The results indicate that shortly after the trauma accompanied by the brain commotion disturbances in sleep architecture can be detected by means of polysomnography. We also demonstrate that polysomnography is a sensitive method of evaluation of early post-traumatic alterations within the CNS. Based on the results of the present study we conclude that the described diagnostic procedure should become a steady element of the clinical evaluation and qualification of patients presenting with subjective symptoms as the sequalae of a mild head trauma.

Introduction

Minor head trauma (MHT) accompanied by brain concussions constitutes, as a clinical syndrome, about 2/3 of all the scull and brain injuries [8]. Most of the patients with the past history of the MHT recover within a few weeks without a need for a specific intervention. A third of the patients, however, develop the subjective, post-traumatic syndrome, and half of them never return to work. One year after the injury, 15% of the patients still complain of symptoms affecting their lifestyle [1]. Probably the pathological process developing after trauma could be considered the reason of complaints of sleep disturbances too. Therefore authors suspect that in early period after MHT patient can develop disturbances in sleep. We did not find any research work analizing a sleep pattern in a group of patients in early period after MHT.

The present paper is a part of the research project carried out at the Polish Air Force Institute of Medicine (PAFIM), the Central Clinical Hospital of the Military Medical University School (CCH) and the Institute of Psychiatry and Neurology (IPN) in Warsaw. The project was devoted to evaluation of the state of the central nervous system in patients who had suffered from MHT. Here, we evaluated the sleep pattern of patients in early period after MHT.

Material

The study was conducted in a group of 40 males aged 19-29 years (mean age 22.5 y) who were admitted to the Dept. of Neurology, PAFIM for the MHT accompanied by the consecutive brain concussion. The diagnosis was based on history, physical examination, and the computerized tomography (CT) results. The protocol of the study was approved by the Ethical Committees of both the PAFIM and the CCH and a written consent was obtained from all the subjects allotted to the study.

Selection of the patients for the study was based on the following criteria: skull and brain injuries with brain commotion experienced for the first time shortly before the study (i.e., 24 - 96 hours before hospitalisation), 19-41 years of age, male sex, and no detectable mental disorders, somatic diseases or other physical abnormalities. In addition, no past history of abnormal EEG recordings and no family history of epilepsy were required.

Seven patients should take medication and were excluded from the survey (two of them because of excessive emotional irritability, one because of serious dizziness and three because of persisted moderate and heavy headache).

Control group consisted of 30 healthy paid male volunteers aged 19-29 years (mean age 22.4y) with normal EEG in wakefulness. All examined persons described their sleep as good. None of them abused alcohol, took daytime naps, or underwent pharmacological treatment for at least four weeks before the sleep examination.

Method.

After admission to the Clinic patients were assessed from the point of view of cause of trauma,

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duration of unconsciousness, appearance and duration of posttraumatic amnesia (PTA).

The injuries were evaluated using the Glasgow Coma Scale (GCS). Accordingly, the total score (ranging from 3 points for the most serious condition to 15 points for the least serious one) obtained after the assessment of the eye-opening capacity and the verbal and motor responses was used to divide the patients into the following 3 groups: a) mild (scaled 13-15), b) moderate (scaled 8-12), and c) heavy head trauma (scaled 3- 7).

During the first 24 hours after the admission, the brain CT with no contrasting medium was performed. Normal images of the brain and the cerebellum with no detectable dislocations in the ventricular system were required for the inclusion of a patient in the study. All the CT images were examined by one and the same physician.

On 3rd to 4th day after trauma, by means of "self-estimation sleep form" patients described their wake/sleep status, replying to 12 questions connected with the pattern of sleep after trauma, day sleepiness and naps. They compared it with the quality of sleep before MHT.

On the 5th to 11th day after the trauma, polygraphic examinations were carried out on the Medelec-2 MC apparatus between 9.00-10.00 p.m. and 5.00-6.00 a.m. Standard technique polygraphic record and the criteria of visual scoring of somnogram stages according to Rechtsschaffen and Kales [2,14] were applied. Sleep was evaluated on the basis of the somnogram of the second night (the first night was considered as adaptation night). The same procedure of sleep examination was applied in control group after a normal activity during a week before the examination.

The data concerning sleep EEG were computer processed, mean values and standards deviations for 17 parameters were calculated. Students t-test was used for the statistical analysis of the results. $P<0.01$ was adopted as significant difference.

Results

Patient's characteristics

According to history data 18 patients (54.5%) suffered injury from fights, nine (27.3%) from car accidents and six (18.2%) from fall. Eleven of them (33.3%) were only confused or lost consciousness for less than dozen or so seconds. Twelve patients (36.4%) lost consciousness for 1 to 20 minutes and ten patients lost consciousness for 30 to 60 minutes.

Retrograde amnesia (RA) occurred in every patient and lasted up to a few minutes after trauma. Posttraumatic amnesia (PTA) appeared in every patient as well, but the time of duration was up to 6 hours. Data concerning RA and PTA have only approximate value, because in 27 cases memory improved before admission to the hospital.

On the admission to the Clinic (first-third day after the trauma) the patients complained of a number of symptoms which disappeared after a few days. The complaints included headaches, dizziness, daytime drowsiness, nausea, and emesis. Headaches of mild and moderate intensity (not requiring any regular medication) as well as irritation and anxiety episodes were the longest lasting symptoms (6 subjects (18.2%) complained of them for 14 days after the trauma).

According to the CGS, 25 (75.8%) patients were scored 15 points and 8 (24.2%) patients were scored 14 points. In the latter group, one point was subtracted from the highest score because the patients were able to open their eyes on demand.

Self-estimation sleep form.

All patients had no sleep disturbances and even naps before head trauma. According to the form results (table I) 22 subjects had different sleep abnormalities day sleepiness and naps after MHT. Five of them (gray mark on the table) had excessive sleep disturbances and woke up with the feeling of fatigue.

Polysomnography

Results of patients' sleep examinations are shown in the table II. They are compared with the results of control group. Statistical analysis revealed a reduction of sleep cycle length ($p=0.001$) in patients. However an increase of cycle number ($p=0.009$) during a whole sleep period in subjects after MHT, caused that total amount of NREM sleep is similar in both groups. Another difference found in NREM sleep between two groups is decrease of stage 2 of sleep in patients ($p=01$). Analysing REM stage we observed shortening of REM sleep latency ($p=0.0001$).

Polygrafic registration of sleep in patients took place between 4 to 11 days after trauma. In 19 of them from 4^{th} to 6^{th} day and in 14 from 8^{th} to 11^{th} day after MHT. Statistical analysis showed no difference between two estimated groups.

As it is demonstrated in table I patients had some complaints connected with disturbances in wake – sleep cycle. Significant sleep differences were found in group of patients who, apart from other complaints, notified feeling of fatigue. However because of a small number of this group we should be careful in making a conclusion.

Discussion

Traumatic injury is mainly caused by displacement of intracranial structures in relation to the skull bones. A shift of these structures towards the trauma site creates a negative pressure which results in the formation of a vacuum on the opposite side of the brain. The vacuum sucks in gas bubbles to the cerebral cortex capillaries and breaks down small blood vessels and nervous tissue (the so-called cavitation phenomenon) [2]. After injury focal and diffuse pathological changes occur. MHT is mainly characterized by diffuse changes like diffuse axonal injury (DAI), diffuse microvascular damage (DMD) and delayed secondary injury (DSI). DSI is caused by an uncontrolled vicious cycle of biochemical events at cellular level set in motion by the trauma. DSI has come to be recognized as a major contributor to the ultimate tissue loss after MHT. The complex of pathological processes lead to necrosis and/or apoptosis of nerve cells [6,9].

Exposure to the linear acceleration forces brings about the most pronounced changes in the deep structures of the brain. Angular acceleration damages mainly the cerebral cortex of, particularly frontal and temporal lobes. - in which centres responsible for human behaviour, memory, cognitive and learning ability are found [18]. Experimental research works revealed that some cerebral structures, located in frontal and temporal lobes as well, are involved in creation of final sleep-wake status picture [10]. Thus behavioral and cognitive abnormalities are often found in patients with MHT.

History of sleep disorders is one of factors affecting a quality of shift work. Traumatic brain injury can affect even temporary, activity of systems responsible for wake-sleep cycle. EEG sleep pattern of comatose patients after head trauma was the evidence of changed function of those systems [8]. Prigantano at al. [12] obtained single polysomnograms in a group of 10 subjects who had complains of disturbed sleep after closed injury. All of these patients had been comatose for at least 24 hours. The head-injured patients had less stage 1 sleep and a greater number of awakenings.

This research work has revealed that even after MHT, in early period after trauma, changes of sleep architecture can be observed. Decrease of stage 2 sleep is difficult to explain. It could be the result of affected function of structures (located in frontal and temporal lobes) involved in NREM sleep generating. In patients with diagnosed Alzheimer Disease (AD) pathological changes are especially found in frontal and temporal lobes. EEG sleep pattern of these patients disclosed poor biological efficiency and disturbances of REM stage [17].

Domżal et al. [3] discovered in their study that patients after MHT had a decrease of spindles activity – EEG elements found in NREM sleep pattern. Diminishing of spindle number results in reduction of stage 2 sleep. System consisting of reticular formation, thalamus and cerebral cortex is responsible for creation of spindles [11,13,16]. That is why affection of connections between these structures is thought as essential factor causing decrease of stage2 sleep.

REM stage changes found in examined group may depend on affected function of centres located in cerebral hemispheres. Neurons generating REM stage are found in structures of brain stem but different cerebral nuclei influence on the final shape of this part of sleep [4,5,15,19].

REM sleep is thought to be responsible for brain metabolic regeneration [7]. Shortening of REM stage latency, duration of sleep cycle, and increase of cycle number could be the result of acceleration of the process.

Conclusions:

1. MHT affects a sleep architecture in early period after trauma.
2. Polysomnography is a sensitive tool in discovering of sleep changes developing after MHT. It could help to improve clinical patient estimation and usefulness to perform shift work.

Table I.

Complaints of patients according the „sleep self – estimation form"

Lp.	Number of patient	Complaints				
		Day sleepiness	Naps	Difficulties in falling asleep	Often Awakenings During night	Feeling of fatigue
1.	1	X	X	X		X
2.	2	X				
3.	3	X	X			
4.	6	X				
5.	9	X				
6.	10	X				
7.	11	X	X	X		
8.	12	X		X	X	
9.	13	X	X	X	X	
10.	16	X	X	X	X	
11.	17	X	X			
12.	19	X	X	X	X	
13.	21	X	X	X	X	X
14.	22				X	
15.	25	X	X			
16.	29	X	X			X
17.	30			X	X	X
18.	32	X				
19.	33				X	
20.	34	X	X			X
21.	35	X	X			
22.	36	X	X	X	X	

Table II

EEG sleep pattern of patients after mild head trauma versus control group.

Parameters of EEG sleep pattern	A control group		Patients		Significance of difference
	Mean value	SD	Mean value	SD	
Record time (min)	474.9	8.6	476.3	2.5	p=0.25
Time of sleep (min)	439.1	25.8	433.4	33.1	p=0.45
Total sleep time (min)	433.2	32.5	426.1	33.7	p=0.40
Wakefulness in sleep (%)	1.4	3.0	1.7	2.1	p=0.66
Stage I (%)	6.4	5.9	6.1	3.8	p=0.82
Stage 2 (%)	55.6	7.2	51.3	5.8	p=0.01
Stage 3 (%)	11.0	3.8	11.7	3.1	p=0.42
Stage 4 (%)	4.4	4.1	5.5	4.6	p=0.28
Stage 3+4 (%)	15.4	6.9	17.3	6.0	p=0.25
Total NREM sleep (%)	77.5	4.7	74.8	4.6	p=0.03
Stage REM (%)	22.5	4.7	25.2	4.6	p=0.03
Sleep latency (min)	35.2	24.0	42.8	32.7	p=0.29
Stage 3,4 latency (min)	13.1	5.6	17.3	17.1	p=0.19
REM stage latency (min)	88.9	40.2	54.1	22.7	p=0.0001
Number of sleep cycles	4.0	1.0	4.7	0.8	p=0.009
Number of awakenings	2.0	2.8	2.6	2.4	p=0.37
Mean length of cycle (min)	108.7	23.5	91.5	13.9	p=0.001
Sleep efficiency (%)	91.3	6.3	89.4	7.0	p=0.27
Sleep maintenance (%)	98.6	3.0	98.3	2.2	p=0.67

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