Evaluation of EEG abnormalities in children with migraine

Ocena nieprawidłowości EEG u dzieci z migreną

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ABSTRACT

Although EEG is not recommended for the clinical diagnosis of recurrent headache in children, but provides a vast potential for exploring the pathophysiology of migraine. **The aim of the paper** was the evaluation of EEG in children with migraine with and without aura. **Material and method.** Our study group consisted of 128 children and adolescents aged 7 - 18 years: 35 with migraine with aura, 45 with migraine without aura and 48 subjects with episodic tension-type headache. Patients with epilepsy or seizures history were excluded. EEG was performed interictally at least 24 hours after the last headache attack. **Results.** EEG was interpreted as abnormal in 62.9% subjects with migraine with aura, 64.4% patients with migraine without aura and in 54.2% children with tension-type headaches. Epileptiform discharges were detected in about 50% of records in each group with recurrent headache. In migraine with aura vs. without aura there was a higher incidence of attenuation of EEG activity and focal slow activity, lower incidence of generalized epileptic activity and generalized slow activity. There was an obvious trend towards a higher incidence of epileptic activity in response to photic stimulation in migraine vs. tension-type headache, particularly in migraine without aura. There were no significant differences in distribution of alpha, beta, theta and delta rhythms of background EEG activity in patients with recurrent headaches. **Conclusions**. The results of our study based on children without seizure history seems to suggest that the increased incidence of EEG abnormalities in childhood migraine did not necessarily point to an epileptic origin. Some differences between interictal EEG patterns could confirm central neuronal hyperexcitability in migraine.

Keywords: migraine, children, electroencephalography

STRESZCZENIE

Chociaż EEG nie jest zalecane jako badanie diagnostyczne napadowych bólów głowy u dzieci, ale może być przydatnym narzędziem w badaniach nad patofizjologią migreny. **Celem pracy** była ocena EEG u dzieci z migreną z aurą i bez aury. **Materiał i metoda.** Naszym badaniem objęliśmy grupę 128 dzieci w wieku 7 – 18 lat: 35 z migreną z aurą, 45 z migreną bez aury i 48 z bólami głowy typu napięciowego. Wykluczono pacjentów z padaczką i napadami padaczkowymi w wywiadzie. EEG wykonywano w okresie bezbólowym, co najmniej 24 godziny po ostatnim ataku. **Wyniki.** Nieprawidłowy zapis EEG stwierdzono u 62,9% dzieci z migreną z aurą, 64,4% z migreną bez aury i 54,2% dzieci z bólami głowy typu napięciowego. Wyładowania padaczkowe znaleziono w około 50% zapisów EEG w każdej z podgrup napadowych bólów głowy. W grupie dzieci z migreną z aurą w porównaniu z migreną bez aury stwierdzono częstsze występowanie zapisów niskonapięciowych i ogniskowej czynności wolnej, natomiast rzadziej występowały uogólnione zmiany napadowe i uogólniona czynność wolna. Zaobserwowano tendencje do częstszego występowania aktywności napadowej jako reakcji na fotostymulację u pacjentów z migreną w porównaniu z bólami głowy typu napięciowego, szczególnie w migrenie bez aury. Nie było istotnych różnic rozkładu fal alfa, beta, hetta i delta w ilościowej ocenie EEG u pacjentów z różnymi typami napadowych bólów głowy. **Wnioski.** Uzyskane przez nas wyniki mogą sugerować, iż duży odsetek nieprawidłowości EEG w migrenie dziecięcej niekoniecznie odzwierciedla pochodzenie padaczkowe. Pewne różnice zapisów EEG w okresie między atakami bólowymi mogą potwierdzać nadpobudliwość neuronalną charakterystyczną dla migreny.

Słowa kluczowe: migrena, dzieci, elektroencefalografia

The non-acute headache diagnosis in children is based essentially on a detailed reports on the patients history and symptoms and on careful clinical examination. The role of electroencephalography (EEG) and the controversies surrounding its attendant use in the evaluation of headaches is being debated. Although early EEG studies of migraine emphasized the frequent abnormal recordings, contemporary reviewers have criticized most of them for various methodological omissions and flaws [1-4]. The only significant abnormality reported was the prominent driving in response to photic stimulation (H response) in migraineurs [1]. Other EEG abnormalities in migraine patients observed during the interictal phase were: interhemispheric asymmetry of alpha rhythm, diffuse or focal slowing and increase of fast activity [5]. Although this is interesting, it is not necessary for the clinical diagnosis of migraine [6]. Accordingly, in guidelines of American Academy of Neurology and Committee of Child Neurology Society [7] and recommendations of EFNS Task Force [8], recording of EEG is not routinely indicated for the diagnosis of non-acute headache in children. However, in many cases, particularly in children with short history of headaches or with the atypical headaches, many physicians find it necessary to supplement the clinical work up of headache patients with several neurophysiological tests.

According to the recent published questionnaire data from Italy, 25 headache specialists, 75% neurologists and 60% of general practitioners recommended EEG examination to some migraine patients seen in the previous 3 months [9].

Although most neurophysiological tests have limited value for headache diagnosis but they have a vast potential for exploring the pathophysiology and the effects of pharmacological treatment [8,10-12]. The current concepts of migraine pathogenesis encompasses a neurovascular hypothesis with centrally generated disruption of trigeminal sensory processing. Several studies have shown that the neuronal structures involved are the brainstem, the cerebral cortex, peripheral and central components of trigeminovascular system [11]. The sequence of activation and the relative roles of these structures are still being debated. Electrophysiological techniques allow the study of some of the structures in vivo and enlarge our knowledge on controversial aspects of migraine pathopysiology such as cortical excitability or central sensitization [12]. Our previous studies have been focused on cortical evoked potentials in children with migraine and seems to confirm an interictal dysfunction in cortical sensory information processing [13,14].

Data from the studies on the use of the EEG in the evaluation of recurrent headaches in children is very difficult to interpret. Methodological problems range from the patient population having mixed types of headaches, ill-defined diagnostic criteria, multiple age groups, lack of control subjects and unclear definitions of EEG abnormalities [3,4,7]. Quantitative frequency analysis of EEG (QEEG) with or without topographic mapping is a more objective method than conventional EEG interpretation. This technique has been used previously in migraine but with contradictory results [1,15].

There are no studies that clearly compare the incidence of EEG abnormalities in migraine vs. non migraine pediatric headache patients [7]. Therefore, in this study we focused on the routine EEG and quantitative EEG analysis in children and adolescents with the migraine with aura and without aura in comparison with episodic tension-type headaches. The aim of our study was the evaluation and comparison of characteristic electrophysiologic features of EEG in children with recurrent headaches without past seizure history.

MATERIAL AND METHODS

The study protocol was approved by the local ethics committee of Medical University in Białystok and fully informed consent was obtained from the parents of all subject studied. The study group consisted of 128 of children and adolescents aged between 7-18 years, all of the patients of the Department of Pediatric Neurology and Rehabilitation, Medical University of Białystok with recurrent headaches. Thirty five of them suffered from migraine with aura (MA), 45 of them suffered from migraine without aura (MO) and 48 subjects have episodic tension-type headache (TTH). Migraine and tension-type headaches were diagnosed based on the criteria of the second edition of International Headache Classification [16]. A neurologist with experience in headache questioned the patients about their clinical features and surveyed clinical neurological conditions, time of onset, frequency of attacks, side of pain, pain intensity and nonheadache symptoms. Subjects were devoid of any other pathology of central nervous system, and/or systemic disease. Patients with epilepsy or seizures history were excluded. None of the patients received prophylactic anti-migraine therapy.

Recording session was performed interictally at least 24 hours after the last headache attack, without assuming any analgesic or any other symptomatic treatment. EEG was performed always at the same time of the day: 9.00 - 12.00 a.m. MEDELEC DG COMPACT 32 apparatus was used for EEG recordings. Electrodes were positioned on FP1, FP2, F7, F3, FZ, F4, F8, T3, C3, CZ, C4, T4, T5, P3 P4, T6, O1, O2 derivations, in accordance with the 10-20 system. The filters were set at 0.3 – 70 Hz. Each recording lasted at least 20 min of EEG spontaneous activity for each subject and included the intermittent photic stimulation (train duration 5sec with interval between trains 2sec, flash rate from 2/sec up to 24/sec.) and hyperventilation for 3 minutes. We interpreted the EEGs visually and qualified as normal or abnormal according to widely accepted criteria [17]. EEG abnormalities included: a. slow activity (generalized or focal), b. attenuation/suppresion of background EEG (decreased amplitude of one type of activity or of all activity) c. focal and generalized increase in EEG activity, d. assymmetry in EEG activity, e. epileptiform discharges (focal spikes, sharp waves, spike-and wave discharges, polyspike complexes, polyspike-and-slow-wave complexes, multiple-sharp-wave complexes, multiple-sharp-and-slow-wave complexes, hypsarrhytmia) .

For quantitative EEG study artifact free epochs were selected. Segments with eye movements, hyperventilation, photic stimulation and other artefacts were excluded. Twenty 2-seconds, segments from frontoparietal (F3-Pz) and parietooccipital (P3-O1) regions were analyzed. Distribution of background activity of alpha, theta, delta and beta rhythms was calculated.

Statistical tests were done using STATISTICA 6.0. The values of parameters within different headache groups were compared by Mann-Whitney U test and Student *t* test for unpaired samples (after checking by ANOVA Kruskal – Wallis analysis). Statistical significance was defined as $p<0.05$. EEG abnormalities were related to age, gender, duration of illness, frequency of headaches, presence of aura, and unilateral location of pain by regression analysis (Spearman rank coefficient).

RESULTS

The mean age of patients when EEG recording was done was 13.06 ± 2.09 years. Female-to-male ratio was 1.3: 1. The demographic and clinical data is presented in table 1. A positive family history of migraine was found in 40% children with migraine, more frequent in migraine without aura. Unilateral pain demonstrated about half of migraineurs.

The distribution of EEG abnormalities is described in table 2. EEG was interpreted as abnormal in 62.9% subjects with migraine with aura, 64.4% patients with migra-

Table I. Demographic and clinical data of the patients

	Migraine with aura (MA)	Migraine without aura (MO)	Migraine (M)	Episodic tension – type headache (TTH)
Number of subjects (n)	35	45	80	48
Age of patients, years (mean ± SD)	14.46 ± 1.93	11.42 ± 2.58	12.67 ± 2.70	13.32 ± 2.86
Gender (female/male)	17f/18m	20f/25m	37f/43m	35f /13m
Duration of illness in months: mean ± SD	21.77 ± 30.04	24.75 ± 22.21	22.90 ± 28.04	15.34 ± 13.63
Unilateral headache location: n (%)	24 (68.6%)	19 (42.2%)	43 (53.8%)	0 (0%)
History of migraine in family: n (%)	10 (28.6%)	22 (48.9%)	32 (40%)	2 (4%)

Table II. Distribution of EEG abnormalities in children with recurrent headaches

EEG findings	Migraine with aura (n=35)	Migraine without aura (n=45)	Migraine N=80)	Episodic tension – type headache (n=48)
Normal (%)	13 (37.1%)	16 (35.6%)	29 (36.3%)	22 (45.8%)
Abnormal (%)	22 (62.9%)	29 (64.4%)	51 (63.7%)	26 (54.2%)
Generalized slow activity (%)	3 (8.6%)	10 (22.2%)	13 (16.3%)	8 (16.7%)
Focal slow activity (%)	7 (20%)	4 (8.8%)	11 (13.8%)	3 (6.3%)
Attenuation (%)	4 (11.4%)*	0	4 (5%)	1 (2.1%)
Fast activity (%)	0	0	0	0
Increase in EEG Activity (%)	0	0	0	0
Interhemispheric Assymetry of alpha rhytm (%)	1 (2.9%)	0	1 (1.3%)	0
Epileptiform discharges (%)	17 (48.6%)	23 (51.1%)	40 (50%)	24 (50%)

* $P<0.05$ vs. migraine without aura (ANOVA Kruskal – Wallis analysis)

ine without aura and in 54.2% children with tension-type headaches. There were no significant differences in the distribution of EEG abnormal patterns according to all migraineurs and tension-type headaches. In comparison of migraine subgroups, the incidence of attenuation of EEG was significantly higher in migraine with aura vs. without aura (11.4% vs. 0%; $P<0.05$). There was an obvious trend, although not statistically significant, towards a higher incidence of focal slow activity in migraine with aura (20% vs. 8.8%) and a higher incidence of generalized slow activity in migraine without aura (22.2% vs. 8.6%). None of the patients demonstrated any fast activity or increase in EEG activity. On the other hand, epileptiform discharges were detected in about 50% of records in each group with recurrent headache.

The distribution of epileptiform discharges according to the different headache types is presented in table 3. The most frequently observed epileptiform discharges consisted of one of the following: focal spikes and sharp waves or generalized epileptic activity. The incidence of focal discharges was similar in all groups: migraine with aura, without aura and tension type headaches. However, the incidence of generalized epileptic activity was significantly lower in migraine with aura vs. migraine without aura (8.6% vs. 26.7%; $P<0.05$). Other epileptiform discharges were rare or not found.

Epileptic activity in response to activation methods shows fig 1. There was an obvious trend, although not statistically significant, towards a higher incidence of epileptic activity in response to photic stimulation in migraine vs tension-type headache (8.8% vs. 2.1%), particularly in migraine without aura (11.1%). The incidence of epileptic activity in response to hiperventilation was similar in migraineurs without aura and tension-type headache sufferers (40% and 37.5% respectively) and lower in migraine with aura (25.7%).

Table 4 presents data of quantitative EEG analysis. There were no significant differences in distribution of alpha, beta, theta and delta rhythms in patients with recurrent headaches.

We analyzed also the correlation between clinical data and EEG findings. There were the negative correlation between EEG abnormalities incidence and the age of patient ($R= - 0.359$; $P<0.001$). We found no significant correlation between the EEG patterns and the gender of patients, aura, disease duration, frequency of attacks, positive history of migraine in family, unilateral location of pain.

Table III. Distribution of epileptiform discharges in EEG of children with recurrent headaches

EEG findings	Migraine with aura (n=35)	Migraine without aura (n=45)	Migraine N=80)	Episodic tension – type headache (n=48)
Focal spikes	0	0	0	0
Focal sharp waves (%)	17 (48.6%)	23 (51.1%)	40 (50%)	23 (47.9%)
Spike-and-wave complexes (%)	0	1 (2.2%)	1 (1.3%)	0
Sharp-and-slow-wave complexes	0	0	0	0
Polyspike complexes	0	0	0	0
Multiple-sharp-wave compexes	0	0	0	0
Generalized epileptic activity (%)	3 (8.6%)*	12 (26.7%)	15 (18.8%)	11 (22.9%)

* $P<0.05$ vs. migraine without aura (ANOVA Kruskal – Wallis analysis)

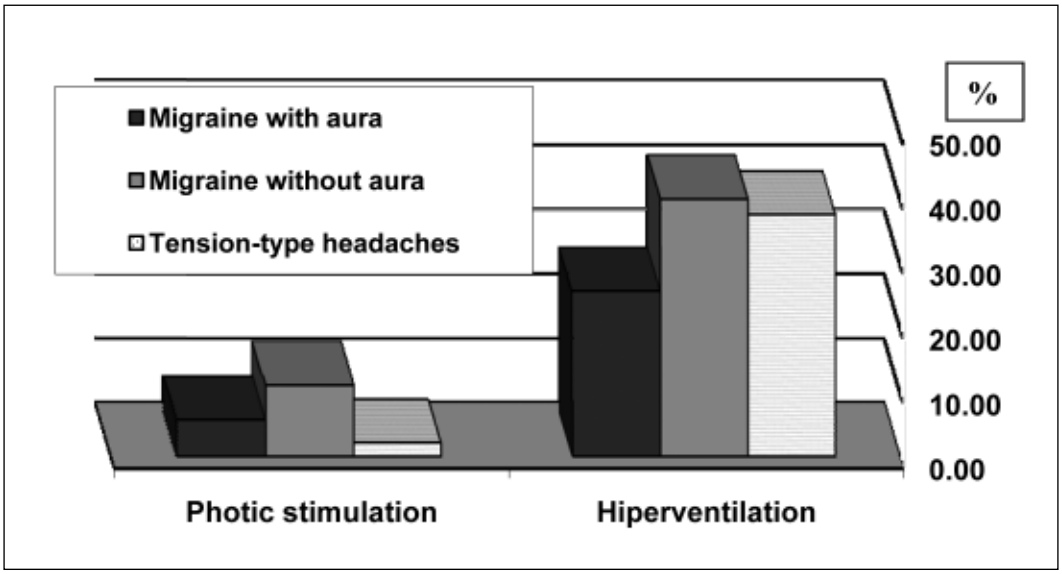

Fig. 1. Epileptic activity during photic stimulation and hyperventilation in children with recurrent headaches (%)

Table IV. Distribution of the background EEG activity in F3-Pz and P3-O1 derivative in children with headaches

	Migraine with aura (n=35)	Migraine without aura (n=45)	Episodic tension – type headache (n=48)
Alpha rhythm (%) in F3-Pz	32.07 ± 13.72	34.13 ± 11.21	34.57 ± 16.25
Alpha rhythm (%) in P3-O1	33.97 ± 12.60	34.063 ± 9.23	37.42 ± 13.76
Theta rhythm (%) in F3-Pz	33.39 ± 8.19	29.53 ± 7.97	20.08 ± 10.99
Theta rhythm (%) in P3-O1	31.75 ± 7.59	30.59 ± 10.24	32.02 ± 7.57
Delta rhythm (%) in F3-Pz	56.44 ± 14.39	57.77 ± 11.47	57.59 ± 11.63
Delta rhythm (%) in P3-O1	52.86 ± 14.11	56.02 ± 14.64	51.75 ± 13.27
Beta rhythm (%) in F3-Pz	8.14 ± 2.69	6.92 ± 1.75	7.09 ± 3.81
Beta rhythm (%) in P3-O1	7.06 ± 2.22	5.46 ± 1.56	7.46 ± 1.58

DISCUSSION

Most of the published studies reported routine EEG data on children with „all types" of recurrent headaches, included tension type-type headaches, migraine, „sinus" headache and so on [3, 18, 19]. A 1960 study of 500 children with recurrent headache reported that 44% of EEGs were abnormal [20]. The percentage of patients with abnormal EEG in other studies varied from 20.6% [3] to 40.8% [19]. Different non specific EEG patterns were recorded: spike discharges, paroxysmal slow wave activity, generalized slowing, and fast activity. Data for the headache subgroups was pooled so that extraction of EEG in patients with migraine vs. all other groups could not be done [7]. Previous studies focused on children with migraine have suggested that the EEG in migraineurs is more likely to be abnormal than in other types of headaches [21,22, 23,24]. The percentage of children with an abnormal EEG was higher and varied from 11% [22] to 51.9%[23], 73.4% [24] and 75% [21]. Nevo et al. analyzed the EEGs of 257 children with headache and found interictal epileptic abnormalities present in 12% cases [25]. They also identified a subgroup of children suffering from migraine with the frequency of specific abnormalities raising to 26.7%. These data correlate cardinally with our results. EEG was interpreted as abnormal in 62.9% subjects with migraine with aura, 64.4% patients with migraine without aura and in 54.2% children with tension-type headaches. Of these 80 children, 16.3% had generalized slow activity, 13.3% had focal slow activity, 5% had attenuation and 50% had epileptiform discharges. Data from literature also demonstrates nonspecific EEG abnormalities in migraineurs. In the retrospective study [24], 64 children with migraine had an EEG with the following features: diffuse slowing (38%), paroxysmal activity (27%) and focal abnormalities (13%). Epileptiform discharges in our patients with migraine were represented most frequent by focal sharp waves and spikes (50%) or generalized epileptic activity (22.9%). Other authors also found high incidence of spike and sharp waves from 10% [22] to 46.9% [24] and slowing of activity from 0% [23] to 27% [24].

The discrepancies between various investigations are probably caused by several factors. Varying diagnostic criteria for migraine were used and none of cited studies [21-24] used the currently accepted IHS criteria. Some authors recognized „seizure headaches" [18] or „seizure-related headaches" [3,26]. Varying criteria of EEG abnormalities were used. For example, specific EEG pattern 14 and 6 cycles per second interpreted as abnormal in 46.4% migraineurs [21] is now considered as a benign variant. Sometimes studies do not define the „typical EEG features" or „typical epileptiform discharge" [18]. Moreover, the authors do not state when the EEG was performed in relation to the epoch of headache although ictal and preictal EEG changes have been reported by some researchers [27]. Important factor is selection of subjects to EEG investigation because of relationship between migraine and epilepsy. Many studies have supported hypothesis of alteration of cortical hyperexcitability as a possible pathological mechanism underlying the onset of migraine and epileptic attacks [11,12,15,28]. Of the 17 patients with paroxysmal EEG, 7 had history of seizures and 4 had a family history of seizures [24]. Therefore we excluded patients with history of epileptic seizures. However, in our study the incidence of epileptiform discharges in migraine was raising to 50%.

We did not find any significant differences in distribution of EEG abnormal patterns according to all migraineurs. However, there was an obvious trend towards a higher incidence of epileptic activity in response to photic stimulation in migraine vs tension-type headache. Some authors reported significantly higher incidence of slowing than the group with other types of headaches [3], but review of available data from literature also do not confirm differences between children with migraine as compared to those with other recurrent headache types [7].

We observed some differences between EEG patterns in migraine with aura and without aura. The higher incidence of attenuation of EEG activity, lower incidence of generalized epileptic activity, trends towards a higher incidence of focal slow activity and lower incidence of generalized slow activity and were found in migraineurs with aura. Piccinelli et al. analyzed the EEGs of 137 children and adolescents with recurrent headache and found specific interictal abnormalities in 11.7% of the whole sample [28]. The distribution of abnormalities across subgroups of patients with headaches in a cited study was significantly different: 5.9% in tension-type headaches, 5.4% in migraine without aura and 43.5% in migraine with aura. The authors suggest the hypothesis of a possible clinical continuum between some types of migraine with aura and epileptic syndromes due to altered neuronal excitability with similar genetic substrates [28]. Channelopathies of voltage-gated Na and K channels might be a link between migraine with aura and epilepsy [11]. This conclusion is also supported by the evidence of proven efficacy of migraine prophylactic drugs, such as calcium channel blockers and anticonvulsants [28].

Intriguing data in our study is represented by the high proportion of epileptic activity in response to photic stimulation and hyperventilation in patients with migraine without aura (11.1%, 5 of 45 and 40%, 18 of 45 respectively) vs. migraine with aura (5.7%, 2 of 35 and 25.7%, 9 of 35 respectively). Another study presented contrary results – high proportion of photosensitivity in patients with migraine with aura (30.4%), while it was not a triggering factor in children with other types of headache [28]. Some authors consider the presence of photoparoxysms in migraine as a negative prognostic factor of epilepsy onset [29].

Quantitative frequency analysis of EEG (QEEG) has been used previously in migraine but with contradictory results. Increased or decreased alpha power, increased asymmetry, increased delta and theta power have been reported interictally in migraine [30,31] Moreover, QEEG power and asymmetry increase 36 hours before the next migraine attack compared with the interictal period [27]. The main spontaneous abnormalities in EEG spectral analysis found during the critical phase of migraine attack were the slowing and asymmetry of the dominant frequency in the alpha range [32]. In our study there were not any sig-

nificant differences in distribution of alpha, beta, theta and delta rhythms in attack-free periods in patients with migraine with aura, without aura and tension-type headaches. Only few studies in literature contained data that compared QEEG in children with migraine with aura and without aura [31,33,34,35]. Valdizan et al found increased theta/alpha ratio in migraine with aura, normal in tension-type headaches [35], other authors did not report any significant differences between interictal EEG in children with migraine with aura and without aura[31,33,34]. However, it is not easy to compare the results from different QEEG studies because different methodological protocols have been applied [15].

CONCLUSIONS

In conclusion, although EEG is not recommended to exclude a structural cause for recurrent headache in children, but presents a vast potential for exploring the pathophysiology of migraine. In our opinion, the results of our study, based on children without seizures history, and data from literature, suggest that the increased incidence of EEG abnormalities in childhood migraine did not necessarily point to an epileptic origin. However, the hypothesis of the link between epilepsy and migraine, particularly with aura, could not be excluded. Some differences between interictal EEG patterns in migraine with aura, migraine without aura and tension-type headaches could suggest central neuronal hyperexcitability in migraine predisposing to the headache attacks.

REFERENCES

[1] Gronseth G.S., Greenberg M.K.: The utility of the electroencephalogram in the evaluation of patients presenting with headache: a review of the literature. Neurology 1995; 45: 1263- 1267.

[2] Sand T.: EEG in migraine: a review of literature. Funct Neurol 1991; 6: 7-22.

[3] Kramer U., Nevo Y., Neufeld M.Y. et al: The value of EEG in children with chronic headaches. Brain Dev 1994; 16: 303-308.

[4] Puca F., de Tommaso M.: Clinical neurophysiology in childhood headache. Cephalalgia 1999; 19: 137-146.

[5] Schoenen J.: Clinical neurophysiology studies in headache: a review of data and pathophysiological hints. Funct Neurol 1992; 40: 191-204.

[6] Evans W.R., Olesen J.: Migraine classification, diagnostic criteria, and testing. Neurology 2003; 60(Suppl.2): S24-S30.

[7] Lewis D.W., Ashwal S., Dahl G. et al.: Practice parameter: Evaluation of children and adolescents with recurrent headaches. Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. Neurology 2002; 59: 490-499.

[8] Sandrini G., Friberg L., Janig W. et al.: Neurophysiological tests and neuroimaging procedures in non acute headache: guidelines and recommendations. Eur J Neurol 2004; 11: 217-224.

[9] Rossi P., Schoenen J., Bolla M. et al.: Implementation and evaluation of existing guidelines on the use of neurophysiological tests in non-acute migraine patients: a questionnaire survey of neurologists and primary care physicians. Eur J Neurol 2009; 16: 937-942.

[10] Magis D., Ambrosini A., Bendtsen L. et al.: Evaluation and proposal for optymalisation of neurophysiological tests in migraine: Part 1 – electrophysiological tests. Cephalalgia 2007; 27: 1323-1338.

[11] Ambrossini A., Schoenen J.: The electrophysiology of migraine. Curr Opin Neurol 2003; 16: 327-331.

[12] Giffin N.J., Kaube H.: The electrophysiology of migraine. Curr Opin Neurol 2002; 15: 303-309.

[13] Boćkowski L., Sobaniec W., Śmigielska-Kuzia J. et al.: The pattern - reversal visual evoked potentials in children with migraine with aura and without aura. Rocz Akad Med. Bialymst 2003; 48: 154 –157.

[14] Boćkowski L., Sobaniec W., Sołowiej E. et al.: Auditory cognitive event-related potentials in migraine with aura and without aura in children and adolescents. (in Polish) Neurol Neurochir Pol 2004; 38: 9-14.

[15] Sand T. Electroencephalography in migraine: a review with focus on quantitative electroencephalography and the migraine vs. epilepsy. Cephalgia 2003; 23(suppl.1): 5-11.

[16] Headache Classification Commitee of the International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. Second edition. Cephalalgia 2004; Suppl.1: 8– 160.

[17] Abou-Khalil B., Misulis K.E. Atlas of EEG & Seizure Semiology. Butterworth Heinnemann, Philadelphia 2006: 99-176.

[18] Chen J.H., Wang P.J., Young C. et al.: Ethiological classification of chronic headache in children and their electroencephalographic features. Acta Paediatr Sin 1994; 35: 565.

[19] Aysun S.: Clinical experience on headache in children: analysis of 92 cases. J Child Neurol 1998; 13: 202-210.

[20] Froelich W.A., Carter C.C., O'Leary J.L. et al.: Headache in childhood. Neurology 1960; 10: 639-642.

[21] Whitehouse D., Pappas J.A., Escala P.H. et al.: Electroencephalographic changes in children with migraine. N. Engl J Med 1967; 276: 23-27.

[22] Kinast M., Lueders H., Rothner A.D. et al.: Benign focal epileptiform discharges in childhood migraine (BFEDC). Neurology 1982; 32: 23 – 27.

[23] Ziegler D.K., Wong G.: Migraine in children: clinical et electroencephalographical study of families: possible relationship to epilepsy. Epilepsia 1967; 8: 171-187.

[24] Prensky A., Sommer D.: Diagnosis and treatment of migraine in children. Neurology 1979; 29: 506-510.

[25] Nevo Y., Kramer U., Rieder-Grosswasser I. et al.: Clinical categorization of 312 children with chronic headache. Brain Dev 1994; 16: 441-444.

[26] Jay G.W.: Epilepsy, migraine, and EEG abnormalities in children: a review and hypothesis. Headache 1982; 2: 110-114.

[27] Bjřrk M.H., Sand T.: Quantitative EEG power and asymmetry increase 36h before a migraine attack. Cephalalgia 2008; 28: 960-968.

[28] Piccinelli P., Borgatti R., Nicoli F. et al.: Relationship between migraine and epilepsy in pediatric age. Headache 2006; 46: 413-421.

[29] Tozzi E., Florio I., Mesturino A. et al.: Hemicrania and photosensitive epilepsy in pediatric age. Clin Ter 1998; 149: 357-560.

[30] Lia C., Carenini L., Degioz C. et al.: Computerized EEG analysis in migraine patients. Ital J Neurol Sci 1995; 16: 249-254.

[31] Genco S., de Tomasso M., Prudenzano A.M.P. et al.: EEG features in juvenile migraine: topographic analysis of of spontaneous and visual evoked brain electrical activity. Cephalalgia 1994; 14: 41-46.

[32] De Tommaso M., Sciruicchio V., Guido M. et al.: EEG spectral analysis in migraine without aura attacks. Cephalalgia 1998; 18: 324-328.

[33] Seri S., Cerquiglini A., Guidetti V.: Computerized EEG topography in childhood migraine between and during attacks. Cephalalgia 1993; 13: 53-56.

[34] Farkas V., Kohleb O., Benninger C. et al.: Comparison of EEG background activity of epileptic children and children with migraine. In: Degen E, Dreifyss FE, eds. Benign localized and generalized epilepsies of early childhood. Oxford: Esevier;1992: 199-205.

[35] Valdizan J.R., Andreu C., Almarcegui C. et al.: Quantitative EEG in children with Headache. Headache 1994; 34: 53-55.

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