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REVIEW

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Clinical neurophysiology in childhood headache

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Abstract Several neurophysiological techniques are available for examining children with headache. The choice among them is made according to clinical features.
Electroencephalography (EEG) is commonly performed in headache; abnormalities observed are heterogeneous, and specific patterns have been described in different forms of complicated migraine. Quantitative EEG, brain mapping and spectral analysis are at present useful mainly for research purposes and in migraine diagnosis.
Polysomnography studies are just at the beginning but they have been providing interesting findings. Visual evoked potentials (VEPs) have demonstrated conflicting results: increased amplitudes observed in migraine children need to be replicated and methodological flaws need to be corrected. Event-related potentials (ERPs) have been used to shed light on cognitive processes related to migraine during development of the nervous system.
Electromyography studies have documented increased spontaneous muscle activity in tension-type headache, but further studies are awaited.
Overall, clinical neurophysiology is of primary interest in studying functional mechanisms of headache and migraine symptoms. In clinical practice, these procedures are not essential for diagnosis but are relevant for clarifying specific problems.

Key words Childhood headache · Clinical neurophysiology

Introduction

Examinations by means of clinical neurophysiology techniques in childhood headache have been introduced primarily for diagnostic purposes and subsequently for research on neural mechanisms of the disorder. The distinction between primary headaches and symptomatic forms represented the first issue addressed by electrophysiological studies, followed by the aim of distinguishing migraine from other idiopathic forms; several techniques and methods have been employed with conflicting results as to specificity and reliability of the findings obtained [1, 2]. Electrocephalography (EEG), evoked potentials and electromyography, to a lesser extent, have been increasingly used in childhood headache because they are safe, noninvasive diagnostic tools [3, 4]. Concomitantly, clinical neurophysiology gained further attention over time for research purposes, to explore basic mechanisms of headache and to evaluate treatment effects. In childhood, there is additional interest in investigations performed at the onset of the disorder, to shed light on altered functional pathways in the developing nervous system.

Electroencephalography

In migraine, altered cortical excitability or reactivity has been repeatedly described. Since early studies, electroencephalographic abnormalities in migraine have been considered a common finding, but the analysis of existing data is confusing with an incidence of abnormal EEG results ranging from 44% to 73% in childhood [5]. The main causes leading to such different results were due to the lack of agreement on diagnostic criteria and to an unclear definition of what was considered an EEG abnormality [6–9]. Childhood headache diagnostic criteria were not clearly established and accepted for a long time, leading to significant differences in case definition across clinical samples. Unspecific abnormal interictal EEG findings have been described in 41% of children suffering from migraine, but when a more selective approach has been achieved a notable decrease has been reported. Spikes and delta activity such as altered EEG transients have been observed in 0%–9% of cases of migraine [10–13]. Epileptiform abnormalities were detected in 4% of migraine children once stricter criteria of selection were applied; nevertheless the same rate was noted in healthy controls, leaving open the debate about specificity and reliability of these findings. On the contrary, no significantly altered EEG patterns have been reported in tension-type headache (TTH), although data are still a few to permit a general overview in comparison to migraine children [14, 15]. Despite of conflicting reports on the prevalence and type of EEG abnormalities in migraine, it is important to stress that in clinical samples the occurrence of paroxysmal discharges typical of epileptic syndromes of childhood has been recognized. High rates of epileptic EEG activity have been described in chronic childhood headache: EEG findings of migraine attacks have demonstrated sequences of spikes or sharp waves in different brain regions (central, centrotemporal, occipital, focal and multifocal) resembling the ictal recording of epilepsy. The progression of seizure activity with an increase or decrease of paroxysms and the appearance of fast rhythms is more rarely observed. Unspecific slowing in the theta range at centrotemporal regions is common, and even EEG recordings within normal limits are frequent in patients with migraine with or without aura. Therefore, a wide range of electroencephalographic findings is expected (Table 1). Even if not diagnostic, they may be contributory in deciding whether the neurophysiologic examination should be widened.

During headache-free intervals, the most consistent abnormality reported in children with migraine is a photic driving response at frequencies greater than 20 Hz. This is called the “H response”. Although it has low specificity, it is an important marker of migraine and of cortical hyperreactivity to visual stimuli. Bursts of theta rhytms in centrotemporal regions are other common findings in interictal phase of migraine children.

More definite abnormalities have been described during the aura phase of migraine attacks, with complex symptoms consisting in contralateral theta or delta activity, lasting several hours. Yet prolonged bursts of repetitive, posterior rhythmic sharp waves have been detected in basilar migraine attacks alternating with normal activity [16]. Hemiplegic migraine is a complicated, often familiar form, almost invariably associated with paroxysmal abnormalities. Periodic unilateral sharp-slow waves are detected during the acute phase and possibly persist several days after the attack. Furthermore, paroxysmal lateralizing epileptiform discharges have been described as a peculiar pattern mostly observed in patients with structural CNS pathology, i.e. brain abscess and infarction. This is not the case for hemiplegic migraine which usually has a functional basis with complete recovery after the attack. EEG recording in childhood headache is important especially in migraine associated with epileptic syndromes. Migraine features such as visual disorders resembling the aura phase, headache and autonomic

Table 1 Electroencephalographic findings of childhood headache

Reference	Headache type	Cases, n	Age, years	EEG, %		Spikes or sharp waves, %	Slowing, %
				Normal	Abnormal		
Chen et al. [14]	All	211	3–16	75.4	24.6	22.7	0
Kramer et al. [11]	All	257	3–16	79.4	20.6	12.1	8.6
Aysun, Yetuk [13]	All	49	1.5–18	59.2	40.8	10.2	14.3
DeCarlo et al. [12]	All	412	2–17	86.7	13.3	7.3	4.4
Ziegler, Wong [9]	Migraine	27	4–15	48.1	51.9	33.3	0
Whitehouse et al. [8]	Migraine	28	7–15	25.0	75.0	10.7	25.0
Prensky, Sommer [5]	Migraine	64	NR	26.4	73.4	46.9	27.0
Kinast et al. [25]	Migraine	100	3–15	89.0	11.0	10.0	1.0

NR, not reported

symptoms have also been observed in specific childhood epilepsy syndromes. Late onset idiopathic childhood occipital epilepsy (LIOE) of Gastaut type, rolandic epilepsy, mithochondrial encephalopathy and lactic acidosis (MELAS), and basilar migraine with or without seizures share characteristics of both disorders. Epileptic and epileptiform discharges are frequently described, suggesting a common, altered neurophysiological background [17, 18]. Headache and vomiting have also been observed in idiopathic photosensitive occipital epilepsy, usually after paroxysmal discharges have been elicited by photic stimulation [19].

The pattern of occipital spike wave complexes suppressed by eye opening has been observed in the interictal phase of migraine with aura, in occipital epilepsies (especially LIOE of Gastaut type), and in basilar migraine [20, 21]. This is a well recognized but non-specific pattern that suggests caution before deciding which kind of disorder should be diagnosed. Clinical features should be considered more relevant than EEG findings, including the possibility that a differential diagnosis may not be necessary because migraine and epilepsy may coexist [22]. Occipital spike-wave complexes suppressed by eye opening require a careful approach because it occurs in idiopathic as well as in symptomatic occipital epilepsies. At present, it is far from clear the distinction among these forms in spite of common EEG abnormalities. Magnetic resonance imaging (MRI) investigation is strongly indicated to detect whether structural abnormalities underly such an electroclinical pattern. Symptomatic forms may have no clinical differences from idiopathic ones, so that such an examination cannot be delayed.

Paroxysmal discharges observed in migraine are typical of specific epileptic syndromes of childhood and clinical features are frequently overlapping, but putative mechanisms accounting for these links are still under examination. The high incidence of paroxysmal activity located in parietal and occipital lobes during developmental age is likely to constitute the substrate predisposing children with migraine to seizures (Fig. 1). The epileptogenic abnormalities would be activated by metabolic and blood flow changes unfolding during the attack. The association is strengthened by the higher percentage (than expected by chance) of epileptic children suffering from migraine. The prevalence of headache in benign partial epilepsies is the following: 62% in centrotemporal epilepsy (rolandic), 35% in absence epilepsy and 8% in other partial epilepsies [23–25]. The bidirectional association is therefore based on consistent epidemiological data even if many links are still unclear. The two disorders often appear concomitantly under the form of "current" comorbidity, or following one another in a relative short time interval in "successive" comorbidity with varying association with respect to migraine attacks and seizures. Factors determining the chronological sequence of paroxysmal disturbances are not completely understood. Regarding ictal phase, the progression from migraine to epilepsy was considered the most reliable and it was supported by the fact that seizures following migraine aura were described. Actually, the opposite view, derived from clinical observations and neurophysiological data, prevails. In particular, the association of occipital epilepsy and migraine has been detailed. Occipital seizures of LIOE of Gastaut type usually are followed by headache. The sequence is then reversed because seizures generate a migraine-like attack. In spite of the controversies of the past, in this case there is now a relevant agreement widely accepted and documented.

From a clinical perspective, much concern has been directed to visual symptoms that are present in migraine aura as well in visual seizures of occipital epilepsy, mainly in LIOE of Gastaut type. As in early onset of benign

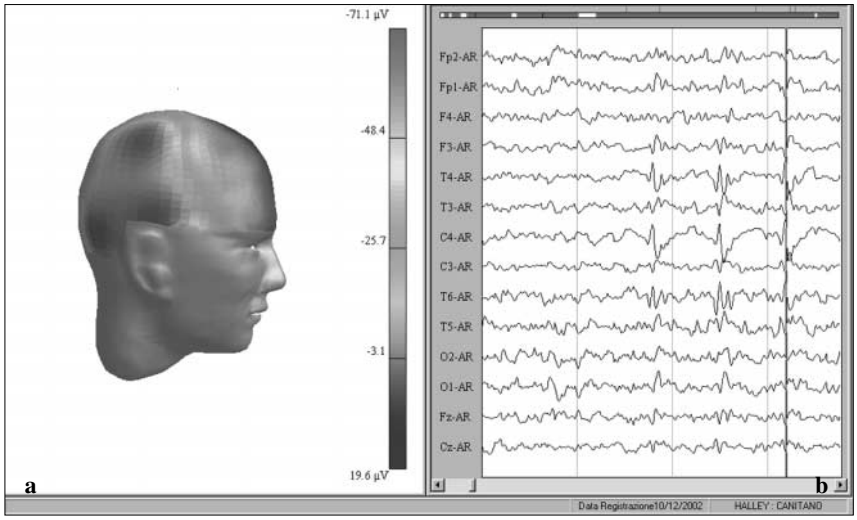

Fig. 1a, b Neurophysiological examinations of a 10-year-old child with migraine and epilepsy. **a** Brain potentials map. Interictal spikes were observed in the right centrotemporal region (*shaded areas*). **b** Electroencephalographic recordings

occipital seizures (EBOS) of panyiotopoulos type, seizures are rarely described and clinical features are distinct. Hallucinations, scotoma, amaurosis and blindness are present in both migraine and epilepsy; often they are not easily distinguished. In spite of this overlapping presentation, a correct diagnosis is recommended for treatment choice. Some guidelines have been proposed recently to differentiate ictal visual symptoms: duration is different, visual aura of migraine is usually longer than visual seizures of LIOE lasting only a few seconds; visual perception disorders have distinct phenomenology (e.g. shape, color, size) when determined by seizures or visual aura. A thorough history is necessary to ascertain the distinct phenomenology [26]. An important shortcoming of this procedure is represented by the limited skill of young children to describe symptoms. A reliable account is rarely obtained and uncertainty often cannot be resolved. Furthermore, EEG abnormalities are not invariably evident and thus do not address the correct diagnosis. Even ictal recordings are normal in as many as 30% of children with occipital epilepsy. On the other hand, occipital abnormalities are reported as more frequently bilateral in migraine and unilateral in epilepsy, eventually providing a useful guide for the differential diagnosis [27].

Quantitative EEG

The aim of reducing the bias of subjective influences in EEG evaluation has been obtained with computerized quantitative analysis performed also in childhood in spite of some limitations related to age. The great variability of EEG patterns and the lack of normative data for age groups represent the major limitations. Studies in childhood migraine have been carried out with interesting but conflicting results. No remarkable differences between migraine with and without aura have been found in most studies during interictal phase even if interesting findings have been reported: interhemispheric asymmetry of alpha rhythm, diffuse or focal slowing, theta activity and increase of fast activities [28]. Spectral analysis and brain mapping have demonstrated a significant variability of spontaneous brain electric activity related to age, duration of illness and interval between attacks; therefore, discordant results from different studies may be the consequence of these uncontrolled variables. More significant EEG changes have been detected during the attacks: unilateral reduction of alpha power on occipital areas contralateral to the affected visual hemifield constituted the relevant finding during the visual aura; concomitantly, a prominent delta activity in frontal areas was also recorded in posterior parietotemporal areas [29]. A similar pattern was observed in childhood migraine attacks without aura, while TTH children did not show any abnormality. The evidence of EEG background modifications in acute phase, with a course resembling that of spreading depression, is at present the more consistent result using quantitative techniques. Persistence of these abnormalities in asympomatic phases would better account for the slowing of alpha rhythm observed in migraine children during headache-free intervals. Therefore when clinical features of headache leave uncertainties for mixed components occurring, brain mapping may be a useful tool to differentiate the type of headache. Routine evaluation with this technique is not warranted as yet; once actual methodological limitations will be improved, quantitative EEG is likely to find wider applications in diagnosis and research of childhood migraine.

Polysomnography

The relationship between headache and sleep is based on clinical observations and on the evidence of common neurochemical and neurophysiological systems implicated. Several sleep disturbances afflict headache patients: delayed sleep onset, frequent awakenings, a number of nocturnal symptoms (e.g. restless legs syndrome, hypnagogic startles and bruxism); increased frequency of symptoms at awakening; and daytime fatigue, somnolence and paralysis. In childhood, an association between parasomnia and headache has been observed. Somnambulism has been documented in high as 20%–30% of migraine children; usually it precedes the occurrence of migraine attacks. The hypothesis is that headache and sleep disorders have a common background, and are the distinct age-related expression of a serotonin system dysfunction [30]. At present, there are no reports of children with narcolepsy suffering from migraine headache, although its increased frequency has been reported in adult narcoleptic patients; thus, it would be of interest to investigate if the association dates back to childhood [31].

Disruption of sleep in the form of excess, insufficient or poor quality sleep could be at the origin of headache and may represent the precipitating circumstance of the attack. Nocturnal headache and morning headache with onset at awakening have to be considered related to sleep stages, they are seen as indicators of the sleep disturbances responsible for the peculiar timing of pain attacks. Sleep fragmentation, insomnia and hypersomnia are the problems related to these types of headache. Furthermore, the degree of deepness of sleep may influence the appearance

Table 2 Sleep disorders observed in childhood headache

Reference	Headache type	Cases, n	Method	Sleep disturbances
Barabas et al. [31]	Migraine Tension-type	222	Clinical	Somnambulism
Bruni et al. [32]	Migraine Tension-type	283	Questionnaire	Parasomnia Awakenings Sleep breathing disorders
Smeyers [33]	Migraine Tension-type	97	Questionnaire	Parasomnia Insomnia Awakenings
Miller et al. [35]	Migraine Tension-type	118	Questionnaire	Parasomnia Sleep anxiety Short sleep

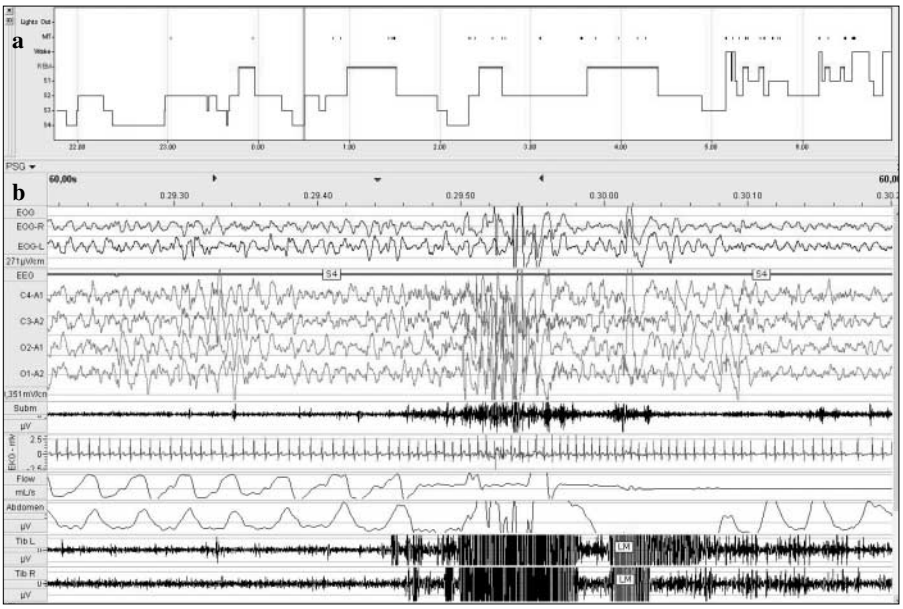

Fig. 2a, b Polysomnography of a child with migraine. **a** Sleep stages **b** Polysomnographic recording

of a migraine attack: slow sleep is thought to release the pain attack [32, 33].

On the other hand, sleep constitutes a powerful resource for terminating a migraine attack or at least for relieving pain and associated autonomic symptoms in both children and adults. The reset of the neuroautonomic system by changing the state of consciousness is the putative mechanism explaining the benefit of spontaneous or induced sleep [34]. Although only a few studies have been conducted on sleep disorders in childhood and adolescent headache (Table 2), this association is suggests further investigations in this field. Indeed, migraine children suffer from sleep anxiety and parasomnia at high rates. The direction of the relationship between sleep and headache is still unclear; nonetheless it can positively address interventions. Modifying sleep habits has decreased headache severity and frequency, and treating migraine improves sleep quality [12]. In a recent study conducted in children with migraine and tension-type headache, these findings have been confirmed: night awakenings, nocturnal symptoms, and daytime sleepiness were observed at high frequency, and no differences were found in the prevalence of the disorders between migraine and tension-type patients [35].

Polysomnographic studies are very limited uncommonly reported. A preliminary study carried out in children with migraine obtained the following findings during two consecutive nights of recording: patients had an increased number of stage shifts and of movement time, and a longer stage I NREM compared to healthy controls; no differences were detected in total sleep time, sleep latency, time in stage II NREM and stage III–IV NREM sleep, and REM latency [36]. Migraine children showed a significant sleep instability with high frequency of stage shiftings and high rate of

movement (Fig. 2). These results need to be confirmed on larger clinical samples, and the recordings should be extended to nocturnal attacks to verify their relationship with REM stage as observed in adults patients. Moreover, high prevalence of sleep-disordered breathing has been observed in migraine children. Snoring and sleep apnea were the main disturbances observed. Morning headache may be caused by sleep apnea, thus careful evaluation of sleep characteristics in this clinical population is warranted [37].

Evoked potentials

Visual evoked potentials

As visual disorders are so frequently encountered in primary headache, mostly during the aura phase, a growing concern has emerged for the functional evaluation of migraine sufferers by means of visual evoked potentials [38]. Only a few studies have been carried out in childhood, and abnormalities during the interictal phase have been described [39–41]. With continuous flicker stimulation, producing steady state visual evoked potential (VEPs), an increased amplitude of F1 component has been observed, associated with spreading of the visual potential to the parietal and frontal regions of the scalp when topographic analysis was performed [42]. Photic driving is dependent on age and on a history of migraine, considering that in childhood it occurs at lower frequencies of stimulation and is less frequent. Amplitude variations of VEPs with flash or pattern reversal stimulation have been reported in migraine; mainly increased amplitudes were detected but lower amplitudes have also been recorded (Table 3). A marked increase of amplitude with flash but not with pattern reversal was the main finding in a group of migraine children during the headache-free interval, indicating abnormal reactivity of the photopic but not scotopic system [43]. The different methods employed are may be responsible for the discordant findings. Also regarding the latency of P100, variable results have been obtained, leaving uncertainties. Studies extended to better defined age and diagnostic groups of children with primary headache may provide more consistent data. Interestingly, an inverse correlation between P100 amplitude and magnesium levels has been reported in children with migraine during the interictal period [44], suggesting that higher VEP amplitude and low magnesium levels are both expressions of neuronal hyperexcitability. Lack of habituation during continuous pattern reversal stimulation has been described in adults but not yet in children [45]. The mechanism for this observation is still undetermined, but it is probably related to the level of arousal and alertness accompanying a dysfunction of the serotonin regulatory role. It is not yet known if the absence of habituation is present also in children at the early stages of the disorder, when development of neurobiological pathways is ongoing and shaping the main neurotransmitter systems.

Brain stem auditory evoked potentials

Brain stem auditory evoked potentials (AEPs) have been studied in migraine. Latencies have been found to be normal during the interictal phase although asymmetries have been noted [46]. In basilar migraine, on the contrary, increased latencies have been reported [47]. However, BAEP results are not clear in migraine, and no studies have been carried out yet in children.

Event related evoked potentials

Changes in cognitive event-related evoked potentials (ERPs) have been demonstrated in both children and adult

Table 3 Visual evoked potentials (VEP) in childhood migraine. All recordings were made in headache-free interval

Reference	Cases, n	Method	Main findings
Wenzel et al. [38]	9	Pattern reversal VEP	P100 latency reduction
Mortimer, Good [40]	28	Pattern reversal and flash VEP (analysis of fast activity)	Increased fast wave amplitude
Brinciotti et al. [27]	43	Pattern reversal and flash VEP	P100 increased amplitude at flash only
Puca, de Tommaso [3]	58[a]	Steady state VEP	Increased photic driving
Genco et al. [42]	40	Steady state VEP	Increased spreading of visual reactivity
Aloisi et al. [44]	20	Pattern reversal VEP	Increased P100 amplitude
Marrelli et al. [41]	34	Pattern reversal VEP (spectral analysis)	Increased P100 amplitude

[a]Children and adults

migraine sufferers, although studies in childhood are a still too few to generalize the data [46]. Increased amplitude of contingent negative variation (CNV) has been detected before the onset of an attack in juvenile migraine [48]; a significant increase of this potential has been reported the day before the attack. On this basis, migraine patients may be distinguished from tension-type headache patients who have an opposite response towards suppression, probably due to an inhibiting effect of chronic pain on slow potentials [49, 50]. CNV amplitude decreases during migraine attacks (a relationship with pain mechanisms has been proposed), while lack of habituation represents the modification described in interictal periods. The event-related potentials have provided discordant results: CNV and P300 amplitudes have opposite courses during a migraine attack, as the former diminishes and the latter increases. Methodological bias and spontaneous fluctuations of electrophysiological activity may be the factors determining such inconsistencies. P300 potential is associated with cognitive information processes including memory, latency depends on cholinergic transmission, and the N2-P3 amplitude depends on the noradrenergic system. An imbalance of the two neurotrasmitter systems is supposed to cause the changes typically seen in migraine. Increased amplitude of N2-P3 during a migraine attack is in fact the remarkable change described in adolescent headache patients [51]. The interictal lack of habituation of cortical processing of visual and auditory information is recognized in migraine. The phenomenon of habituation is supposed to have an adaptive function in normal condition to protect from excessive amount of sustained stimulation. A progressive decline of response to sensory stimulation protects from functional and metabolic overload. Abnormal cortical information processing in migraine with underlying high levels of arousal is likely to interfere with physiological adjustment following repeated sensory stimulation.

Visual and auditory P300 did not lead to habituation in migraine children and adolescents [52]. Evaluation of cortical habituation by means of P300 may have high sensitivity but low specificity to distinguish migraine from TTH children; moreover the process is age-dependent with a positive correlation with P300 latency. Yet, specific cognitive migraine processes may be influenced by brain development and are ongoing until puberty is reached. P300 is promising for future investigations on cortical functions in headache, because memory impairment has been observed in juvenile headache patients [53].

Lack of habituation and increased amplitudes are observed particularly on the day before the attack and therefore are related to the migraine attack-interval cycle as other parameters including EEG background activity in adults as well in children [54]. These changes are dynamic processes unfolding according to the phase of the cycle. The great variability of results reported in evoked potentials research is probably a consequence of this factor. Attempts to establish the period when recordings are performed will eventually make it possible to obtain more homogeneous and comparable data.

Electromyography

Tension-type headache (TTH) has been investigated with electromography (EMG) in order to verify abnormal spontaneous muscle activity expected in this subgroup. Consistent with this hypothesis, an increase of basal values in neck and frontal muscles compared to healthy controls has been reported in children. Furthermore, an increase of basal activity was also recorded in migraine patients but to a lesser extent and did not reach statistically significant values [55]. Higher neck muscle activity was also observed in children with chronic headache [56] during the pain-free period. Patients having chronic disorder showed increased basal activity with respect to a control group. According to these results, children suffering from headache have an excessive level of basal muscle activity, possibly leading to pain and headache. Distinct arousal mechanisms probably are causing the difference between the groups: children with TTH have higher spontaneous muscle activity sustained by upper levels of arousal. Further data are needed to confirm the few findings available to date, including the possibility that prolonged increased basal activity associated with chronic pain is related to the underlying functional peripheral changes such as fatigability.

Conclusions

Clinical neurophysiology is an expanding field in childhood headache. Actual advantages are derived for research aims and for clinical practice at different extents. Forthcoming advanced knowledge is expected to improve classification and clinical management throughout objective parameters. Complex cases with comorbidity and mixed forms of headache, frequently encountered in childhood, remain largely unresolved. Neurophysiological methods may help disentangle the different components leading to headache attacks. A comprehensive evaluation is recommended especially for such conditions and is likely to provide relevant findings for the therapeutic approach. At present, normative studies are awaited for the several techniques described. Moreover methodological discrepancies and diagnostic criteria inconsistencies of the past should be replaced by accurate guidelines.

Electroencephalography and evoked potentials represent a primary source of information about the neural basis of headache. The challenge during development is that maturational processes may be masking or overlapping pathological patterns. The clinical perspective by means of these methods is to make a reliable diagnosis and to place an adequate treatment as early as the disorder appears in order to prevent chronic course and inappropriate therapies.

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