Electroencephalogram in children with minor traumatic brain injury

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Background: Mild traumatic brain injury (MTBI) is one of the most frequent causes for hospitalisation in childhood. Because of different guidelines in the management the diagnostic approach varies substantially. Apart from neuroimaging studies (CT, MRI, sonography) an electroencephalogram (EEG) is often performed without any evidence-based data supporting its use.

Methods: Retrospective analysis of 150 children with MTBI (age 0–16 years), who were admitted to the Children's Hospital of the University of Saarland from January 2006 to December 2007.

Results: Mean age was 4.3 (SD 3.6) years; 55.3% were boys. The most common mechanisms of injury were: Minor fall <1 m of height (60%) and fall >1.5 m of height (10%). The most common symptoms were: one or more episodes of vomiting (60%), somnolence (26.7%) and headache (12.7%). On 118 patients an EEG was performed; 106 (89.8%) were normal, 11 (9.3%) pathological and 1 (0.9%) invalid because of artefacts. The pathological EEGs showed focal findings with localised slowing in nine cases, spike-wave complexes in one case and general slowing in one case. Of the 11 patients with pathological EEG, two had a CT scan, two a MRI and two had cranial sonography; all the neuro-imaging was normal. None of the children required neurosurgical intervention, had a negative outcome or showed persistent symptoms.

Conclusion: The routine performance of an EEG after MTBI in children is not indicated because in most of the cases it is unrevealing, and may lead to unnecessary diagnostic procedures. Instead, children with MTBI should be closely monitored for possible clinical complications and neurological deterioration.

Key words: children; electroencephalogram (EEG); mild traumatic brain injury.

Introduction

Traumatic brain injury (TBI) from unintentional blunt trauma is a leading cause of death and disability among children and teenagers in western countries.1 The majority (approximately 50–80%) of children presenting to the emergency department with head trauma have minor head trauma,2 including those with a Glasgow coma scale of 13–15. Recent studies have tried to identify children at low or high risk to develop brain injuries after blunt head trauma.3 In addition to imaging studies the use of protein biomarkers for detection of injury or prognostication has emerged as an area of clinical and research interest.4 However, despite these advances, there is valid concern that CT scans are overused and that they may be detecting a number of clinically inconsequential findings that require no intervention.5,6

In addition to imaging studies, clinicians and researchers have used electroencephalogram (EEG) to evaluate changes in the electrical activity of the brain following mild TBI (MTBI). Standard clinical EEG analyses are often provided in acute care facilities to detect the presence of focal or generalised slowing as well as to detect the presence of epileptiform activity related to
brain injury. The aim of this study was to evaluate whether MTBI causes pathological electroencephalographic alterations
1 focal slowing (corresponding to a focal anatomic lesion)
2 generalised slowing or (corresponding to generalised cerebral dysfunction)
3 epileptiform discharges (Appendix Fig. 1) (usually localised or multifocal cerebral dysfunction with occurrence of spike-wave complexes secondary to localised lesions)

Moreover, we examined whether these EEG changes led to further imaging studies (cerebral sonography, computed tomography or MRI), and if subsequent neurosurgical interventions were required.

Materials and Methods

This retrospective study was done in accordance with the policy of the Institutional Review Board and Ethics Committee of the University Hospital of Saarland, Homburg, Germany.

Enrolment criteria

Patients (aged 0–16 years) admitted to our hospital with MTBI were included in this study. One of the problems we faced in this study was the lack of a universal definition for MTBI. Different definitions exist for mild head injury and concussion. MTBI in this study was defined as outlined by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitative Medicine: period of post-traumatic amnesia (PTA) not greater than 24 h, an initial Glasgow Coma Scale (GCS) of 13–15, and the loss of consciousness of less than 30 min. These criteria are in line with the recommended definitions of the Report to the Institute of Medicine: period of post-traumatic amnesia (PTA) not greater than 24 h, an initial Glasgow Coma Scale (GCS) of 13–15, and the loss of consciousness of less than 30 min. These criteria are in line with the recommended definitions of the Report to Congress on Mild Traumatic Brain Injury in the United States of the National Centre for Injury Prevention and Control. Exclusion criteria were moderate and severe head injury requiring intensive care medicine treatment or neurosurgical interventions and inflicted brain injuries.

The different mechanisms of injury were categorised in:

- minor fall (e.g. soccer, ice-skating), fall <1 m of height (e.g. bed, couch, swing, perambulator), fall from 1–1.5 m height (e.g. see-saw, changing table, supermarket trolley, baby’s high chair) fall >1.5 m of height (e.g. horse-riding, slide, climbing pole) with minor head collision and others.

EEG recordings

Routine clinical protocol mandated the realisation of an EEG within 48 h after admission, prior to discharge. The follow-up examinations were reviewed until December 2008.

Twenty silver cup electrodes were placed according to the 10–20 international system. Electrode impedances measured less than 5 kOOhm. An IT med® (IT Medical, Usingen, Germany) model EEG Neurofile NT/XP machine was used to record 12 channels. A high-frequency channel was set at 70 Hz; bipolar longitudinal and transversal montages were used. Each EEG recording lasted 15–20 min. All EEGs were evaluated by the same experienced neuropediatricians (GS and SM), based on conventional EEG criteria. Age-dependent EEG differences were taken into account.

Results

Population characteristics

In this study, 150 patients admitted to the Children’s Hospital of the University of Saarland with a minor head trauma between January 2006 and December 2007, were included. Mean age was 4.3 (SD 3.6) years and 55.3% were boys. Patients’ demographics are detailed in Appendix Figure 2.

Mechanisms of injury and place of accident

The five most common mechanisms of injury were minor fall <1 m of height (60%), fall >1.5 m of height (10%), fall 1–1.5 m of height (7.3%), minor head collision (6%) and others (16.7%). In 60% of the cases, accidents happened at home, in 23.3% during free-time activities in swimming pools, playgrounds and sports, in 12.7% at school or kindergarten and in 2.7% on the road.

Symptoms

The clinical presentation of the paediatric patients was very variable. The most common symptoms were one or more episodes of vomiting (60%), somnolence (26.7%), headache (12.7%) and transient loss of consciousness (9%). Less frequent symptoms were nausea (5.3%), transient visual (4.7%) and speech deficit (1.3%), seizure (4%), vertigo (3.3%), motor deficit (2%) and retrograde amnesia (1.3%). In none of the patients symptoms were persistent.

Apart from the neurological symptoms 34% had frontal or facial haematomata, and 16% temporal or occipital haematomata. In 6.1% of MTBI occurred secondary to syncope. In those cases, an electrocardiogram and an echocardiogram were performed. Ophthalmological or otorhinolaryngological work-up was done because of visual alterations, periorbital soft tissue haematoma, epistaxis or trauma to the ear. In case of lacerations or to detect fractures or an intra-abdominal haemorrhage after fall from significant height (4.8%), trauma service was consulted. In 1.3% of the population, oral and maxillofacial surgery was consulted because of dental trauma.

Most patients stayed in hospital for either 2 (25.3%) or 3 days (65.3%).

EEG

One hundred and eighteen of the 150 patients had an EEG: 106 (89.8%) did not show any abnormalities; in 11 (9.3%), pathological changes were noted; and one EEG could not be formally assessed because of artefacts (0.9%).

In 59%, EEGs were performed approximately 48 h after injury; in 20%, 24 h after injury; 21% were performed between 3 and 6 days after the injury occurred. Of the 106 children with an initially normal EEG, five children had an EEG control that
was done because of seizures, a skull fracture and excessive artefacts in the first EEG. Of the 11 pathological EEGs, two had no control, six had a normal EEG control and three a pathological control. The child with the invalid EEG had a normal EEG control 1 week later.

The pathological EEGs showed in nine cases focal findings with focal slowing (increased underlying Delta wave activity); in one case general slowing (generalized underlying Theta wave activity), and in one patient spike-wave complexes (examples are shown in Fig. 1a–c). The epileptiform discharges we found in one of our patients persisted in the follow-up EEG 4 weeks later. This finding was compatible with benign epilepsy of childhood with central temporal spikes (‘Rolando focus’), unrelated to the MTBI.

**Imaging studies following pathological EEG**

In all patients with initially pathological EEGs imaging studies were recommended, but only two of them had a cCT scan, two a CMRI and two a cerebral sonography; in the others, imaging studies were not performed because of lack of parental consent. Cerebral MRI was preferred to cCT because of the significant radiation exposure caused by computed tomography.

Five children with a normal EEG who developed neurological symptoms (transient paraesthesias, transient visual alterations) during their hospital stay had imaging studies. All imaging studies were normal. None of the children required neurosurgical intervention, and none of them had a negative outcome (i.e. trauma-related persistent symptoms).

**Patient follow-up**

Of the 11 children with a pathological EEG, seven had a normal on follow-up, two a pathological and in two no EEG control was performed. Follow-up period was 12–36 months. None of the children included in this study presented again with trauma related neurological symptoms or sequelae after discharge.

**Discussion**

Electrophysiological techniques are among the most frequently used methods to provide information about the functioning of the human brain.10 These techniques are useful in that they are non-invasive and relatively inexpensive. Until today, clinicians and researchers have used EEG to evaluate changes in the electrical activity of the brain following MTBI. Standard clinical EEG analyses are often provided in acute care facilities to detect the presence of focal or generalised slowing as well as to detect the presence of epileptiform activity related to brain injury.10

In this retrospective study, we demonstrated that routine EEG examination is of little value in children with minor head injury as in most of the cases it is unrevealing, and may lead to unnecessary diagnostic procedures. Moreover, we did not see any association between abnormal EEG findings and clinical symptoms (e.g. period of PTA, initial GCS score, loss of consciousness, and neurological deficit) or prolonged recovery or hospital stay in our study cohort. This is in line with previous studies using standard EEG techniques that have not provided a clear depiction of functional change following MTBI.11 Liguori et al. suggested that EEG findings can play a major role in the diagnostic work-up of children with minor head trauma, specifically in asymptomatic patients with normal EEG it is likely that the CT scan will also be normal.12 Contrary to our results, in the study by Liguori et al. a significant percentage of children with MTBI had pathological EEGs, and all children with abnormal CT findings had pathological EEG studies.12 The most common pathological EEG finding in our study consisted in focal slowing, possibly corresponding to a localised brain ‘injury’. However, the functional EEG changes did not go along with sufficient tissue damage to be detected on imaging studies (sonography, CT, MRI), or to warrant neurological interventions of any kind in our cohort.

Early studies reported higher rates of EEG abnormality in subjects with post-concussion syndrome (PCS);13 however, these studies were often qualitative, had no modern radiologic information, lacked detailed analysis of paroxysmal activity (epileptic spike activity not associated with a major seizure), and included individuals outside the current definition of MTBI.14–16 Other early studies did not demonstrate a higher incidence of abnormalities in the EEGs of MTBI patients than in the general population.17 Similarly, in a sample of 54 primarily MTBI subjects who were symptomatic for PCS at the time of investiga-
tion18 observed no concurrent EEG abnormalities in 24-h ambulatory monitoring alone. However, 9.2% of patients had either specific or non-specific paroxysmal activity. In a more recent study, 12 patients with MTBI underwent a clinical examination within 24 h after injury that included a standard clinical EEG assessment based on ‘current EEG criteria.’ No EEG abnormalities were recorded in these patients. Even in patients with structural lesions no focal changes or generalised slow activity have been found.19 Therefore, LeBlanc’s negative characterisation of standard clinical EEG for the assessment of MTBI, when compared with CT or MRI, may be somewhat warranted.20 Korinthenberg et al. demonstrated that post-traumatic syndrome after minor head injury cannot be predicted by serial EEG examinations in children.21 As a consequence, the authors of this study discourage routine EEG examinations in children with very slight head injury and instead recommend parent and patient counselling.21

The standard clinical techniques currently used in most acute care facilities were initially designed to detect seizure activity or abnormal activity associated with large focal lesions. As such, these techniques may be less useful for the detection of mild diffuse damage believed to occur with MTBI as in our study. However, there is a considerable body of experimental work suggesting that more complex EEG paradigms may one day be used to assess changes in brain function after injury (discriminating functions based on patterns of coherence, phase and amplitude).22 Because standard techniques are often used in hospitals, their lack of sensitivity may mislead some to conclude that EEG is generally insensitive to damage. On the basis of the experimental studies, this does not seem to be the case. With continued progress, newer paradigms may eventually be integrated into a standard battery for assessment, thus supplanting standard clinical techniques. This position is consistent with the American Academy of Neurology and American Clinical Neuropysiology Society Guidelines by Nuwer.23 These guidelines
stated that EEG studies on MTBI have resulted in ‘very interesting changes’; however, they were not recommended at that time as diagnostic procedures for PCS. Using the identical rating procedures as these published guidelines, a more recent medical position paper has suggested a limited ‘positive recommendation’ for the use of quantitative EEG in the assessment of PCS.24

Limitations of our study include the retrospective nature of our study design; moreover, the number of children included in our study was too small (inadequately powered) to definitely rule out a positive correlation between an abnormal EEG and a substantial intracranial pathology, given the overall low incidence of significant intracranial and cerebral tissue damage. Moreover, since only in a small proportion of our patients imaging studies were performed, the occurrence of small intracerebral lesions cannot be ruled out with certainty. However, it is ethically questionable to perform imaging studies in this patient cohort as it would expose the child to substantial radiation (cCT), or would mandate the administration of sedatives (cMRI).

In summary, our data suggest that the routine performance of an EEG after minor head trauma in children is not indicated because in most of the cases it is unrevealing and may lead to unnecessary diagnostic procedures. Of note, none of our patients required neurological interventions. Possibly, the recording of an EEG based on individual findings and patients required neurosurgical interventions. Possibly, the EEG after minor head trauma in children is not indicated or would mandate the administration of sedatives (cMRI).

In the future, adequate EEG techniques and other neurophysiological studies may have the potential to detect pathological changes that may have long-term implications more effectively. Of importance, close clinical and parental observation of the affected child with MTBI may be more effective in defining children at risk of developing severe complications.

References

Appendix

Fig. A1 Pathological EEG results. (a) Focal slowing in right temporal occipital lead. (b) Generalised slowing. (c) Spikes-and-wave complexes ('Rolando-Focus').

Fig. A2 Demographic details.