Electroencephalography in Pediatric Epilepsy

JAYA SHANKAR KAUSHIK[1] AND RAJNI FARMANIA[2]

From Departments of Pediatrics; [1]Pt BD Sharma Post Graduate Institute of Medical Sciences, Rohtak, Haryana, and [2]BL Kapur Superspeciality Hospital, Delhi; India.

Correspondence: Dr Jaya Shankar Kaushik, Associate Professor, Department of Pediatrics, Pt BD Sharma Post Graduate Institute of Medical Sciences, Rohtak 124 001, Haryana, India. jayashankarkaushik@gmail.com

Surface electroencephalography (EEG) is a useful electrophysiological investigation for evaluating a paroxysmal event in children. It measures the electro potential difference between two points on the scalp. It is a non-invasive tool that analyzes neuronal maturation and abnormal cortical excitability. EEG helps in differentiating epileptic from non-epileptic clinical event and focal seizures from generalized seizure. This review is to discuss the rational use of interictal scalp EEG in diagnosis of epilepsy and different types of epilepsy syndromes in children. It further highlights its role in febrile seizure, first unprovoked seizure, status epilepticus and unexplained coma.

Keywords: *Diagnosis, EEG, Epileptic syndromes, Guideline, Review, Seizures.*

Electroencephalography (EEG) is a non-invasive, readily available and inexpensive investigation to study the neuronal dysfunction and abnormal cortical excitability in children who present with seizures [1]. Traditional analog EEG machines are being replaced by digital EEG with simultaneous video recording. Surface scalp EEG recording can be conventional short-term recording (30 minutes) or long-term video EEG record (for witnessing and localizing seizure activity).

Sensitivity and specificity of surface scalp EEG to localize the epileptogenic focus depends on factors such as age, type of epilepsy, and nature of EEG recording [2]. Ictal EEG (EEG recording during the seizure) helps to recognize the type of seizure that may not be evident from history and for localizing the epileptogenic zone [3]. Electrocorticography (ECoG) or intracranial EEG is useful for invasive recording of cortical electrical activity by use of electrodes directly on the surface of brain (subdural grids or strips) or deep inside the brain (depth electrodes) [4]. It helps to localize the epileptogenic zone and to map cortical functional areas in drug-resistant epilepsy. Detailed discussion of invasive EEG studies and its role in planning epilepsy surgery is beyond the scope of this review. American Clinical Neurophysiology Society (ACNS) has published technical guidelines for recording digital EEG [5].

PRINCIPLE OF EEG

EEG measures the electropotential difference that arises from the ion trafficking between two points on the scalp. Potential differences between electrodes are amplified and the net signal from each amplifier is displayed on a monitor to provide a graphic record. EEG signals are generated by the summation of excitatory and inhibitory post-synaptic potentials from large, vertically-oriented pyramidal neurons located in layer III, V and VI [6]. These EEG signals are synchronized by subcortical structures like thalamus and brainstem reticular formation. Sleep spindles are considered a result of these thalamocortical phenomena [6]. Large numbers of cortical spikes are not recordable on a routine scalp EEG due to attenuating effect of cerebrospinal fluid, duramater and skull scalp tissue.

TECHNICAL ASPECTS

The surface EEG electrodes made of gold or silver discs (silver chloride) are placed at standard points over scalp with a conductive paste. The International 10-20 system (10 and 20% gap between electrodes) is used for electrode placement [7]. Pediatric EEG routinely requires the placement of 21 electrodes on the scalp with fewer electrodes (minimum of 12 electrodes) in neonates and young infants [8]. EEG can be performed in a laboratory or bedside ambulatory EEG could be used. Additional channels of electrocardiogram (EKG) and respiration are recommended to record physiologic artefacts. EKG during EEG recording helps detect ictal arrhythmia and asystole in children with epilepsy who are prone to sudden unexpected death (SUDEP) [9]. Surface electromyography (EMG) during EEG helps to distinguish epileptic from non-epileptic movements [10]. Electrodes are named according to the underlying area of brain: FP: frontopolar, F: frontal, P: parietal, T: temporal, and O: occipital. Central electrodes are abbreviated as Z

[Fz, Cz, Pz] and referential electrodes include post auricular (A1, A2). The odd numbers (Fp1, F3, P3, C3, T3, T5, T7, O1) depict left side of the hemisphere and even numbers (Fp2, F4, C4, P4, T4, T6, O2) for the right side. These electrodes are either fixed to scalp using conductive paste or electrodes fixed onto a head cap are used.

Patient Preparation

The scalp should be clean and dry. Patient should be instructed to consume antiepileptic drugs (AED) as prescribed. However, AED doses can be reduced or discontinued to facilitate seizure occurrence during long-term video EEG monitoring [11]. Children can have their routine breakfast on the day of appointment. Routine EEG recordings usually lasts for 30 minutes, including hyperventilation for 3 minute and intermittent photic stimulation at 1-30 Hz [5]. Long-term video EEG recordings are particularly useful for pre-surgical evaluation for epilepsy surgery [12]. An ideal EEG should include both awake and sleep record. However, sleep EEG is preferred in younger children considering excessive movement artefacts during the wakeful state. Moreover, sleep EEG provides vital information on maturation of brain [8]. Sleep deprived EEG protocol requires 4-6 hours of sleep deprivation [13]. Children older than 3 years could be kept awake until midnight and woken up at 5:00 AM on the morning of the test. Sleep deprivation is considered to enhance sensitivity of EEG [14]. Triclofos (20 mg/kg/dose), melatonin (2-6 mg/dose) or clonidine (0.05-0.2 mg) can be used for sedation [15]. Intravenous midazolam should not be used to induce sleep due to its suppressive effect on epileptiform discharges. In addition to sleep deprivation, yield of EEG can be increased by repeat recording, prolonging the duration of recording, increasing the number of channels during procedure, simultaneous video recording, and recording both awake and sleep state [16].

Activation Procedure

Infants, young children and children with suspected focal epilepsies require sleep EEG record [17-19]. Sleep EEG is essential for diagnosis of epileptic encephalopathy and continuous spike waves during slow sleep (CSWS) [20]. EEG in awake state is useful to detect generalized epilepsies. Activation procedure include hyperventilation (3 Hz spike wave pattern in absence epilepsies) and intermittent photic stimulation (4-6 Hz generalized epileptiform discharges in Juvenile myoclonic epilepsy). Other activation procedures indicated for specific conditions are: fixation of sensitivity (late onset occipital lobe epilepsy), precipitation by trigger (*e.g.*, video watching) in reflex epilepsies, and suggestion to precipitate paroxysmal non-epileptic events [21]. Reactivity of EEG background is observed by asking the child to close and open the eyes or by touching his various body parts.

EEG REQUISITION

An EEG requisition form from clinician must contain basic demographic profile (name, age, gender, telephone number or email), type of seizure, frequency of seizure, age at onset, indication of EEG, neuroimaging findings, any previous EEG findings, and name of antiepileptic drugs. Neurophysician can decide on the EEG protocol based on clinical diagnosis. For example, in a child with suspected absence epilepsy, one would prefer an awake EEG record with hyperventilation. Sleep deprived sleep EEG record will be considered in a child with suspected focal epilepsy.

EEG INTERPRETATION

ACNS guidelines have outlined five essential components of an EEG report (***Table* I**) [22]. Abnormalities in EEG can be divided into background abnormalities and abnormal epileptiform discharges. Background gives information about the neurologic state of the child. Normal awake record consists of posterior dominant alpha rhythm (8-13 Hz) with reactivity to eye closure. Similarly, sleep background consist of sleep markers of non-REM sleep such as sleep spindles, vertex waves, and K-complexes (***Web Fig.* 1**). Epileptiform discharges have distinct waveforms classified as spikes (<70 ms) or sharp wave (70-200 ms). EEG findings in common self-limited epilepsies and epileptic encephalopathy in children have been summarized in ***Table* II** and ***Table* III**, respectively.

PITFALLS OF EEG

Surface EEG can be normal in few epileptic conditions in children, especially those with remote and deep location of epileptogenic lesion such as interhemispheric area, and mesial and basal cortex [19]. Few genetic types of epilepsy such as benign familial neonatal epilepsy and benign familial infantile epilepsy can have normal interictal EEG. Epileptiform discharges are found in 0-5.6% of normal healthy children and 0.5% of adults without any event of seizure [23]. EEG can be abnormal in approximately 5.7-59% of children with autism spectrum disorder without any clinical seizures [24]. Photic driving response can routinely be found in patients with migraine without any epilepsy [25]. EEG is often reported by neurologist, pediatric neurologist, psychiatrist, neurophysiologist and other physicians with interest in EEG. Hence, there is lot of variability and subjectivity in reporting pediatric EEG. Exclusive training and experience to interpret pediatric EEGs is

TABLE I ESSENTIAL COMPONENT OF AN EEG REPORT (AS PER ACNS GUIDELINES FOR REPORTING EEG).

Parts of report	*Description*	*Sample EEG report*
History	Clinical history, indication, medication, imaging findings	10-year-old boy with multiple episodes of left focal seizure with normal MRI
Technical details	Number of electrodes (21 channel electrodes) Electrode placement technique (10-20 system) use of filters (1.6 Hz-70 Hz) sensitivity (7-10 uV) duration of recording (20-30 minutes) Use of premedication must be documented.	21 channel electrodes were placed using 10-20 system. Record lasted for 30 minutes
	State of the patient: awake, sleep, drowsy or comatose must also be mentioned.	Child was awake and cooperative during the record. HV and PS were performed
EEG description	Background electro cerebral activity Normal awake- as posterior dominant rhythm with reactivity to external stimuli Normal Sleep- presence of sleep markers and preservation of sleep architecture in sleep record	Background consist of 9-10 Hz, 60-80 uV posterior dominant reactive rhythm
	Abnormality in background described in terms of : -Frequency, voltage -Continuous or intermittent -Location (right or left) -Topography (frontal, parietal, temporal or occipital)	There was intermittent delta (2-3 Hz, 60-90uv) slowing in right temporal region
	Epileptiform discharges are described in terms of: -Morphology (spike, sharp, spike wave, polyspike) -Frequency (Hz) -Voltage, location, pattern (run, rhythmic, periodic) -Incidence (rare, intermittent, occasional, frequent, continuous) or preferably quantification and duration of abnormality	There were frequent runs of spike wave discharges (3.5-4.5 Hz, 150-250 uV) arising from right temporal region that lasts for variable duration of 3-5 sec. These discharges occupy almost 60% of EEG epochs. HV and PS did not augment the discharges
Impression	Normal or abnormal; if abnormal: background abnormality and epileptiform discharges	Abnormal EEG record suggestive of right temporal epileptiform discharges with background slowing
Clinical correlation	Focal slowing could indicate underlying structural lesion Lack of organized background could indicate encephalopathy	Possibility of underlying structural lesion needs to be explored.
	Suggestions for sleep record and comparison with previous EEG should be mentioned	

HV: Hyperventilation, PS: Photic stimulation.

essential to understand the normal age-dependent variations and correct characterization of epileptiform discharges. Some of the common errors in pediatric EEG reporting include misinterpreting movement artefacts, high amplitude delta slowing during hyperventilation and normal sleep markers including vertex waves, and K complexes as epileptiform discharges (***Web Fig.* 1**). Other benign epileptiform variants like wicket waves, benign epileptiform transients of sleep (BETS) and rhythmic midtemporal theta bursts of drowsiness (RMTD) can mimic epileptiform discharges to a naïve reader [26]. Awake EEG record can be normal in children with suspected rolandic epilepsy, structural focal epilepsy or CSWS. These abnormalities are detected only on sleep EEG record. Similarly, among those with suspected childhood absence epilepsy and juvenile myoclonic epilepsy, hyperventilation and photic stimulation during awake EEG record is mandatory.

INDICATIONS OF EEG

EEG is an adjunct to clinical evaluation and should be interpreted in clinical context. Indications of using and not using EEG are summarized in (***Box* 1**). Diagnosis of epilepsy should not be reached solely on the basis of EEG

TABLE II CLINICAL AND EEG FINDINGS IN SELF-LIMITED EPILEPTIC SEIZURES AND SYNDROMES

Epilepsy	*Clinical features*	*EEG pattern*
Neonatal onset		
Benign neonatal seizure (Fifth day fit)	Unilateral focal clonic seizure on 4-6th day of life in healthy neonate	Normal or discontinuous background with focal or multifocal abnormalities. Focal, rhythmic theta activity with sharp waves (Theta pointu alternans) can be seen in 60%
Benign familial neonatal seizure	Focal clonic seizure with occasional apneic spells on day-2 or day-3 of life, persist longer in otherwise healthy neonate	Normal background or theta pointu alternans
Infantile onset		
Benign myoclonic epilepsy in infancy (BMEI)	Myoclonic jerk for 1-3 s in developmentally normal child; onset 4 mo to 3 y	Normal background with generalized spike and polyspike discharges
Childhood onset		
Childhood absence epilepsy	Frequent absence seizures in school going child; can be precipitated by hyperventilation	Normal background with 3-4.5 Hz generalized spike wave discharge with frontal dominance
Benign epilepsy with centrotemporal spikes	Seizures during sleep with orofacial motor signs, speech arrest, sialorrhea and unilateral sensory symptoms	Normal sleep background, with sharps/ spike waves in centrotemporal region with a horizontal dipole
Early onset childhood occipital epilepsy (Panayiotopoulos syndrome)	It occurs in sleep with ictal vomiting and autonomic features	Normal background with multifocal, occipital spikes
Late onset childhood occipital epilepsy (Gastaut syndrome)	Visual hallucinations, coloured circular patterns	Runs of rhythmic occipital spikes and sharp waves seen during eye closure called 'fixation off' sensitivity. It disappears when eyes are open and fixating at an object 'fixation on'
Adolescent		
Juvenile absence epilepsy*	Speech and behavioural arrest without aura. May have automatisms	3-4 Hz spike and wave and polyspike wave discharges
Juvenile myoclonic epilepsy*	Myoclonic jerks in morning, generalized or absence seizures	4-6 Hz bilateral polyspike wave and spike slow wave discharges; accentuation by photic stimulation
Nocturnal frontal lobe epilepsy	Focal hypermotor seizures during sleep	Runs of bilateral frontal spike, sharp waves

**May require lifelong antiepileptic medications.*

findings [27]. A wrong diagnosis of epilepsy has widespread social implications apart from side effects of antiepileptic drugs and restriction of physical activities. EEG is often misused in evaluation of a child with abnormal paroxysm to differentiate epileptic from non-epileptic event [28]. Over-interpretation of EEG abnormalities, including focal slowing, generalized and focal epileptiform discharges has often led to syncope being misdiagnosed as epileptic seizures [21]. There is limited role of EEG in children with breath holding spells. Common reasons for misinterpretation of EEG include poor expertise, lack of good quality recording, inappropriate indication, and absence of clinical correlation [27]. Routine surface scalp EEG report should ideally comprise five components: history, technical description, EEG description, impression and clinical correlation [22].

First Unprovoked Seizure

First unprovoked seizure (FUS) is defined as first non-febrile seizure that cannot be explained by an immediate, obvious precipitating cause such as head trauma or intracranial infection. In developing countries including India, focal lesions such as neurocysticercosis (NCC) and tuberculoma are common causes of first unprovoked seizure in children [29]. Thus, in many centers,

TABLE III CLINICAL AND EEG FEATURES OF EPILEPTIC ENCEPHALOPATHY

Age	*Clinical phenotype*	*Interictal EEG Features*	*Background abnormality*	*Epileptiform abnormalities*
Neonatal onset				
Ohtahara syndrome	Tonic spasm	BS in wakefulness and sleep	Bursts of high voltage slow waves 2 to 3 s suppression 3 to 5 s	Intermixed multifocal spikes
EME	Erratic fragmentary myoclonus	BS in sleep, may disappear in awake	Burst 1-3 s Suppression 2-10 s	Multifocal spikes
Infantile onset				
Migratory focal seizures (previously MPSI)	Multifocal clonic autonomic	Migrating epileptiform discharges	Normal to discontinuous	Migrating or multifocal epileptiform discharges
West syndrome	Epileptic spasms in clusters, after waking up from sleep	Hypsarrhythmia Classical or modified	Chaotic, high amplitude, asynchronous slow waves	Multifocal spikes and polyspikes
Dravet syndrome	Recurrent febrile and afebrile seizure Myoclonic, atypical absence and focal seizures	May be normal initially for 1-2 y	Slow diffuse	Generalized, focal or multifocal discharges
Childhood onset				
MAE (Doose syndrome)	Myoclonic- atonic, absences, tonic clonic, tonic	Doose rhythm photosensitive	Normal or doose rhythm (rhythmic parietal theta activity)	Burst of 2-5 Hz generalized spike and polyspike waves
LGS	Tonic seizures, atypical absences, myoclonic seizures	2-2.5 spike wave discharges, PFA	Diffuse slow, absence of sleep markers	2-2.5 spike wave discharges PFA
CSWS	GTCs during sleep, atypical absence, atonic, myoclonic	Frontocentral continuous spike wave	Awake normal Sleep: diffuse slow wave	1.5-2.5 Hz spike wave discharges; activation during sleep; upto 85%
LKS	Seizures in 70% Autistic regression	Temporo-parietal spike wave	Awake normal Sleep: diffuse slow wave	Continuous temporo-parietal spike wave

BS: Burst suppression, EME: early myoclonic epilepsy, MPSI: migrating partial epilepsy of infancy, MAE: Myoclonic astatic epilepsy; LGS: Lennox Gastaut syndrpome, PFA: paroxysmal fast activity; CSWS: continuous spike and wave during sleep; GTC: generalized tonic clonic; LKS: Landau-Kleffner syndrome.

neuroimaging often precedes EEG in evaluation of such children. EEG is recommended as first tier investigation among children with first unprovoked seizure for diagnosis of seizure, epilepsy type and an epileptic syndrome. It may be useful for prediction of long-term outcome or recurrence [30]. Children who have focal epileptiform discharges on EEG have a higher risk for recurrence when compared to those with normal EEG [31]. However, in obvious etiology like neuro-cysticercosis or tuberculoma, EEG should not be routinely requested at outset. EEG may be helpful before withdrawing AED in such patients. Among children with new onset seizures, 18-56% display epileptiform discharges on initial EEG and 15% will never show abnormal findings [32]. EEG done early within first 24 hours of seizure shows background and epileptiform abnormality more frequently [33]. These background abnormalities are often transient and warrant repeat EEG after certain duration to look for persistence of abnormality.

Characterization of Type of Seizure and Syndromic Diagnosis

EEG abnormalities are broadly divided into background abnormalities and abnormal epileptiform waveforms. Background abnormalities include diffuse slowing, asymmetric slowing, discontinuous background and electrodecremental response. Group of disorders with discontinuous EEG background where epileptiform activities contribute to encephalopathy or non-attainment

BOX 1 INDICATIONS OF ELECTROENCEPHALOGRAPHY IN PEDIATRIC EPILEPSY

When to use

- EEG helps in differentiating epileptic from non-epileptic clinical event. Video EEG with capture of ictal event is useful adjunct to support clinical possibility of epileptic event.
- To classify the type of epilepsy into focal or generalized epilepsy and diagnosis of various electro-clinical epilepsy syndrome.
- Video EEG monitoring with spell capture is vital to localize the epileptic focus in case of focal epilepsy.
- To characterize the type of epileptic syndrome based on cluster of clinical seizure semiology, age at onset and EEG findings.
- It helps clinician decide on tapering antiepileptic drugs after a seizure free interval and to predict possible relapse after tapering antiepileptic drug.
- To guide about the etiology in a case of meningo-encephalitis (*e.g.*, periodic lateralized epileptiform discharges in case of herpes simplex encephalitis).
- To diagnose NCSE in case of prolonged coma after status epilepticus or encephalopathy of unknown etiology.
- In children with cognitive or language regression even without seizures, it is indispensable to rule out epileptic encephalopathy like CSWS and LKS.
- To prognosticate an epileptic disorder *e.g.,* Periodic complexes, triphasic waves in a sick patient in ICU is suggestive of poor prognosis. Also, presence of epileptiform discharges predicts seizure recurrence in epilepsy.
- Ancillary test for documentation of brain death.

When not to use

- To exclude a diagnosis of epilepsy; since epilepsy is largely a clinical diagnosis.
- To monitor the progress of epilepsy with EEG.
  (Note: In a children with epilepsy, new onset clinical features like cognitive decline or behavioural issue warrants fresh EEG to rule out NCSE).
- To monitor the efficacy of antiepileptic drugs (AED) in epilepsy except in infantile spasm, LKS, CSWS or absence epilepsy where there could be no change with AED.
  (Note: Valproate and benzodiazepines can decrease the spike burden).
- Intracranial space occupying lesions including stroke without any history of seizures or raised intracranial pressure to form the basis of starting prophylactic AED.
- Clinical history that clearly suggests paroxysmal non epileptic event like shuddering spells, gratification, and syncopal attacks.

CSWS: Continuous spike wave in sleep, LKS: Landau Kleffner syndrome, NCSE: Non convulsive status epilepticus, AED: antiepileptic drug, EEG: Electroencephalography.

of milestones is called epileptic encephalopathy. This includes Early myoclonic encephalopathy, Ohtahara syndrome, West syndrome (***Web Fig.* 2**), Lennox Gestaut syndrome, and Landau Kleffner syndrome. There are signature EEG features to diagnose epileptic encephalopathies as these conditions have urgent treatment implications (***Table* III**). Interictal EEG can be categorized into focal or generalized based on the morphology of epileptiform discharges and organization of background activity. Generalized spike and spike-wave discharges with normal interictal background activity is observed in childhood/juvenile absence epilepsy (CAE/JAE), epilepsy with myoclonic astatic seizures (Doose syndrome), juvenile myoclonic epilepsy (JME) (***Web Fig.* 3**), and epilepsy with eyelid myoclonia (Jeavon syndrome). There are a group of self-limited epilepsies with focal stereotyped spikes wherein the focal spikes can be seen with normal interictal EEG background. This includes Rolandic epilepsy (***Web Fig.* 4**), Panayiotopoulos syndrome and benign occipital epilepsies. Children with structural lesion like Neurocysticercosis, glioma or vascular lesion can also have focal epileptiform discharges (***Web Fig.* 5**). Children with subacute sclerosing panencephalitis can have periodic epileptiform discharges (***Web Fig.* 6**).

Status Epilepticus

EEG is required to rule out nonconvulsive status epilepticus among those with no improvement of altered sensorium following convulsive seizures [34]. EEG is also useful adjunct to monitor seizure activity among

KEY MESSAGES

- A normal EEG does not exclude the diagnosis of epilepsy.
- EEG helps in differentiating epileptic from non-epileptic clinical event, determine seizure type and specific epilepsy syndrome.
- An ideal EEG record must include both awake and sleep state as far as possible.
- Single routine surface EEG can miss majority of focal epilepsies. Surface EEG can be normal in children with remote and deep location of epileptogenic focus.
- EEG report must always be interpreted in clinical context to avoid erroneous clinical diagnosis and management.

those with neuromuscular blockade (which might mask convulsive activity) and high dose suppressive therapy for refractory status epilepticus. Among those with refractory status epilepticus, suppression of epileptiform discharges to achieve burst suppression on EEG is often considered end point for titrating the dose of antiepileptic and anesthetic agents [35].

Comatose Child

Continuous EEG monitoring in intensive care unit (ICU) setting is ideal during management of a child with refractory status epilepticus, heavy sedation, those on neuromuscular blocker, or those being treated with barbiturate for raised intracranial pressure [36]. A comatose child with past history of seizure or seizure like activity requires an EEG to rule out non convulsive status epilepticus. The most common EEG finding in a child with coma is diffuse slowing with reduction in amplitude of waveform. Triphasic waves on EEG could point to ward underlying metabolic disorder. Similarly, periodic lateralized epileptiform discharges (PLED) suggest a focus of irritable cortex seen in space occupying lesion or herpes encephalitis. Serial EEG can help with prognostication. In children with post anoxic coma, burst suppression or isoelectric pattern on EEG is a poor prognostic marker for recovery [37]. EEG monitoring in ICU setting has limited role because of environmental noise and use of sedative drugs.

Febrile Seizure

There is no role of EEG in children with simple febrile seizures [38]. However, EEG is more likely to be abnormal among those with complex febrile seizures, including febrile status epilepticus and focal febrile seizures. Focal epileptiform abnormalities were significantly more frequent among children with complex febrile seizure who subsequently developed epilepsy [39]. However, epileptiform EEG has poor positive predictive value for subsequent development of epilepsy [40], and there is no evidence to support or refute the use of EEG after complex febrile seizure [41]. Hence, there is limited utility of EEG among children with febrile seizures with lack of clinical significance of an abnormal EEG in predicting recurrence or subsequent development of epilepsy.

Discontinuation of AED

Majority of children who are seizure-free for a duration of at least 2 years or more have minimal risk for seizure recurrence [42]. However, the risk of recurrence following withdrawal will depend on type of epilepsy, polytherapy, abnormality on neuroimaging and EEG [43]. Patients with intellectual disability, abnormal neurological examination, older age at onset of seizure, focal seizures and epileptiform abnormalities on EEG have a higher risk of relapse [42]. Abnormal EEG at the time of AED withdrawal has been shown to be associated with a relative risk of recurrence of 1.45 (95% CI 1.18-1.79) [44]. There is an emerging interest on serial EEG monitoring during AED withdrawal to predict risk of recurrence. In a study on 84 children who had seizure recurrence despite normal EEG at the time of drug withdrawal, 24 had abnormal EEG during AED withdrawal [45].

CONCLUSION

A normal interictal EEG does not exclude the diagnosis of epilepsy. EEG is useful to establish the diagnosis of epilepsy, classify the type of epilepsy and to rule out nonconvulsive status epilepticus among children with unexplained coma. EEG is often misused to justify the need for AED among children with clear history of paroxysmal non-epileptic events, headache, simple febrile seizures and head trauma. An abnormal EEG report should always be interpreted in clinical context.

Acknowledgments: Dr Suvasini Sharma and Dr Rachana Dubey Gupta for their suggestions in improving the manuscript; and Prof Kiran Bala and Prof Surekha Dabla, Department of Neurology, PGIMS, Rohtak for their guidance in the manuscript and for providing images of EEG tracings.
Contributors: Both authors contributed to review and synthesis

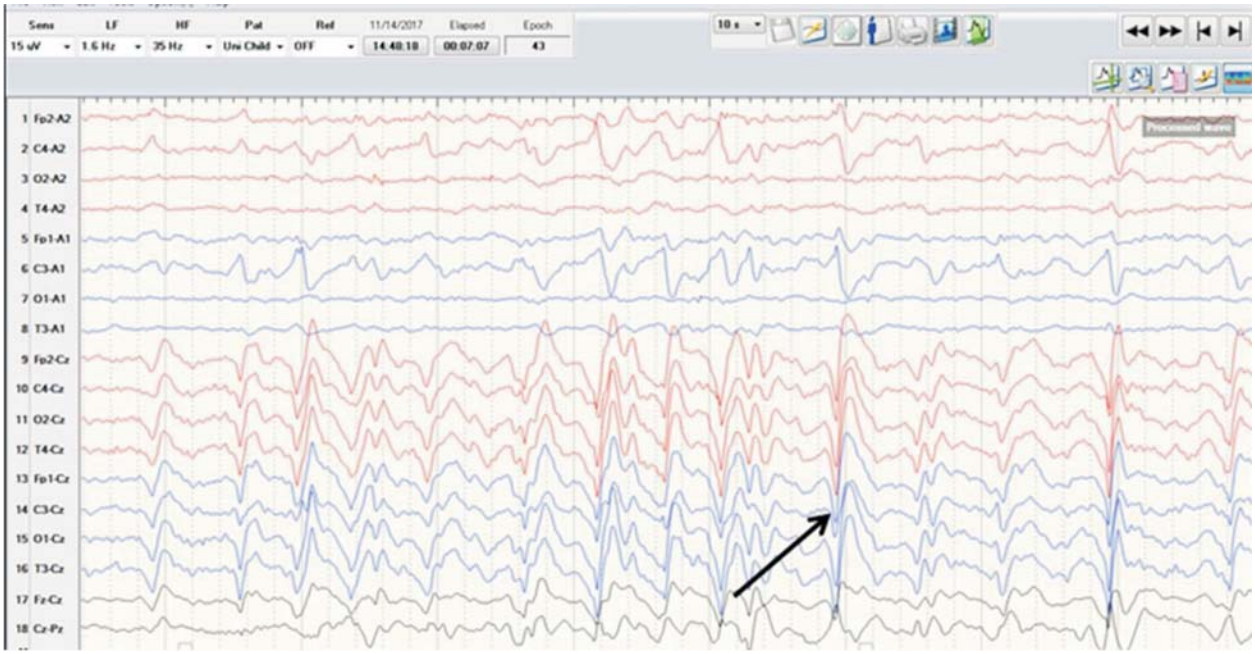
of literature, manuscript writing, and final approval of the version to be published.

Funding: None; *Competing Interest:* None stated.

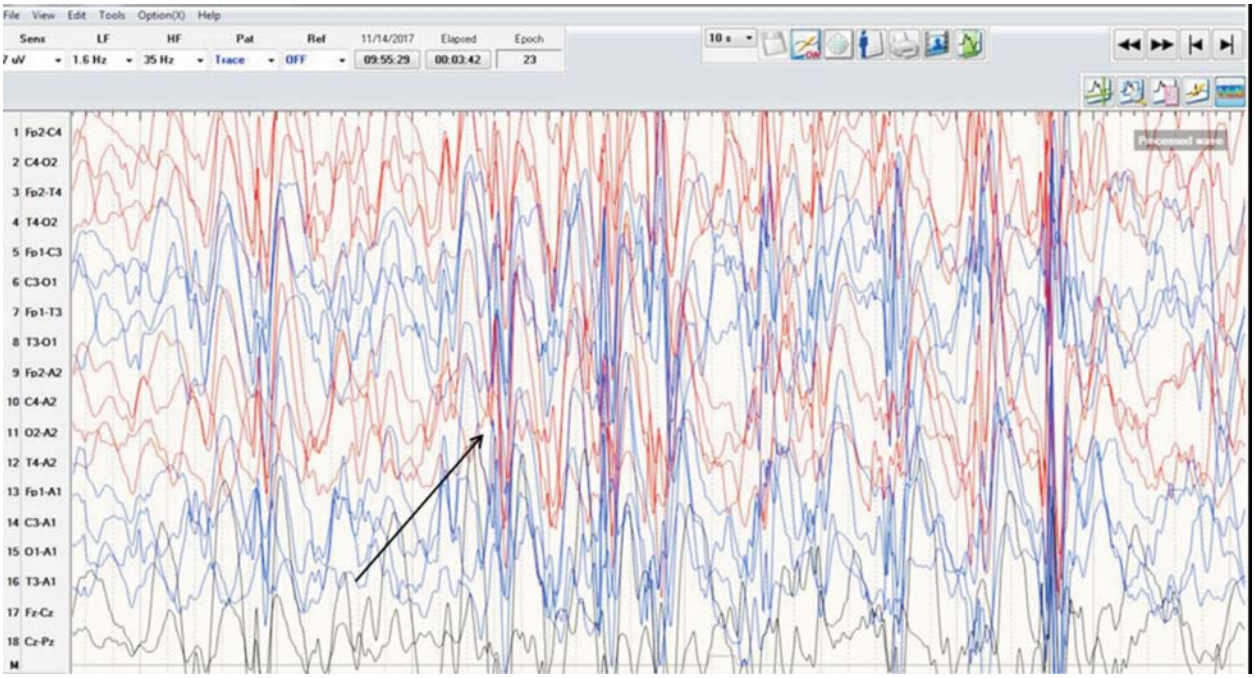
REFERENCES

1. Clancy RR, Bergvist AGC, Dlugos DJ, Nordli DR. Normal pediatric EEG: neonate and children. In: Ebersole JS, editors. Current Practice of Clinical Electro-encephalography. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2014. p. 125-212.
2. Noachtar S, Peters AS. Semiology of epileptic seizures: A critical review. Epilepsy Behav. 2009;15:2-9.
3. Yoshinaga H, Hattori J, Ohta H, Asano T, Ogino T, Kobayashi K, *et al.* Utility of the scalp-recorded ictal EEG in childhood epilepsy. Epilepsia. 2001;42:772-7.
4. Fernández IS, Loddenkemper T. Electrocorticography for seizure foci mapping in epilepsy surgery. J Clin Neurophysiol. 2013;30:554-70.
5. Halford JJ, Sabau D, Drislane FW, Tsuchida TN, Sinha SR. American Clinical Neurophysiology Society Guideline 4: Recording Clinical EEG on Digital Media. J Clin Neurophysiol. 2016;33:317-9.
6. Olejniczak P. Neurophysiologic basis of EEG. J Clin Neurophysiol. 2006;23:186-9.
7. Maus D, Epstein CM, Herman ST. Digital EEG. In: Schomer DL, Lopes Da Silva FH, (editors). Niedermeyer's Electroencephalography: Basic Principles, Clinical Applications, and Related Fields. 5th ed. Philadelphia: Lippincott Williams & Wilkins;. 2005. p. 127-38.
8. Kaminska A, Cheliout-Heraut F, Eisermann M, Touzery de Villepin A, Lamblin MD. EEG in children, in the laboratory or at the patient's bedside. Neurophysiol Clin Clin Neurophysiol. 2015;45:65-74.
9. Bartlam R, Mohanraj R. Ictal bradyarrhythmias and asystole requiring pacemaker implantation: Combined EEG-ECG analysis of 5 cases. Epilepsy Behav. 2016;64:212-5.
10. Hill AT, Briggs BA, Seneviratne U. Simultaneous recording of EEG and electromyographic polygraphy increases the diagnostic yield of video-EEG monitoring. J Clin Neurophysiol. 2014;31:203-7.
11. Rizvi SAA, Hernandez-Ronquillo L, Wu A, Téllez Zenteno JF. Is rapid withdrawal of anti-epileptic drug therapy during video EEG monitoring safe and efficacious? Epilepsy Res. 2014;108:755-64.
12. Kobulashvili T, Höfler J, Dobesberger J, Ernst F, Ryvlin P, Cross JH, *et al.* Current practices in long-term video-EEG monitoring services: A survey among partners of the epilepsy pilot network of reference for refractory epilepsy and epilepsy surgery. Seizure. 2016;38:38-45.
13. DeRoos ST, Chillag KL, Keeler M, Gilbert DL. Effects of sleep deprivation on the pediatric electroencephalogram. Pediatrics. 2009;123:703-8.
14. Giorgi FS, Guida M, Caciagli L, Maestri M, Carnicelli L, Bonanni E, *et al.* What is the role for EEG after sleep deprivation in the diagnosis of epilepsy? Issues, controversies, and future directions. Neurosci Biobehav Rev. 2014;47:533-48.
15. Jain P, Sharma S, Sharma A, Goel S, Jose A, Aneja S. Efficacy and safety of oral triclofos as sedative for children undergoing sleep electroencephalogram: An observational study. J Pediatr Neurosci. 2016;11:105-8.
16. Panayiotopoulos CP. EEG and Brain imaging. In: Panayiotopoulos CP, editors. A clincal guide to epileptic syndromes and their treatment. 2nd ed. London: Springer publication; 2010. p. 147-71.
17. Pillai J, Sperling MR. Interictal EEG and the diagnosis of epilepsy. Epilepsia. 2006;47:14-22.
18. Noachtar S, Rémi J. The role of EEG in epilepsy: A critical review. Epilepsy Behav. 2009;15:22-33.
19. Fowle AJ, Binnie CD. Uses and abuses of the EEG in epilepsy. Epilepsia. 2000;41:S10-18.
20. Gencpinar P, Dundar NO, Tekgul H. Electrical status epilepticus in sleep (ESES)/continuous spikes and waves during slow sleep (CSWS) syndrome in children: An electroclinical evaluation according to the EEG patterns. Epilepsy Behav. 2016;61:107-11.
21. Fattouch J, Casciato S, Lapenta L, Morano A, Fanella M, Lombardi L, *et al.* The spectrum of epileptic syndromes with fixation off sensitivity persisting in adult life. Epilepsia. 2013;54:59-65.
22. Tatum WO, Olga S, Ochoa JG, Munger Clary H, Cheek J, Drislane F, *et al.* American Clinical Neurophysiology Society Guideline 7: Guidelines for EEG Reporting. J Clin Neurophysiol. 2016;33:328-32.
23. So EL. Interictal epileptiform discharges in persons without a history of seizures: What do they mean? J Clin Neurophysiol. 2010;27:229-38.
24. Spence SJ, Schneider MT. The role of epilepsy and epileptiform EEGs in autism spectrum disorders. Pediatr Res. 2009;65:599-606.
25. Fogang Y, Gérard P, De Pasqua V, Pepin JL, Ndiaye M, Magis D, *et al.* Analysis and clinical correlates of 20 Hz photic driving on routine EEG in migraine. Acta Neurol Belg. 2015;115:39-45.
26. Hernandez-Frau PE, Benbadis SR. Pearls and oysters: errors in EEG interpretations: What is misinterpreted besides temporal sharp transients? Neurology. 2011;76: 57-9.
27. Benbadis SR. The tragedy of over-read EEGs and wrong diagnoses of epilepsy. Expert Rev Neurother. 2010;10:343.
28. Laffan A, Langan Y. Understanding EEG indications and reports: A survey of NCHDs. Ir Med J. 2013;106:59-60.
29. Sahu PS, Seepana J, Padela S, Sahu AK, Subbarayudu S, Barua A. Neurocysticercosis in children presenting with afebrile seizure: clinical profile, imaging and serodiagnosis. Rev Inst Med Trop São Paulo. 2014;56: 253-8.
30. Pohlmann-Eden B, Newton M. First seizure: EEG and neuroimaging following an epileptic seizure. Epilepsia. 2008;49(Suppl.1):19-25.
31. Mizorogi S, Kanemura H, Sano F, Sugita K, Aihara M. Risk factors for seizure recurrence in children after first unprovoked seizure. Pediatr Int. 2015;57:665-9.
32. Wirrell EC. Prognostic significance of interictal epileptiform discharges in newly diagnosed seizure disorders. J Clin Neurophysiol. 2010;27:239-48.
33. King MA, Newton MR, Jackson GD, Fitt GJ, Mitchell LA,

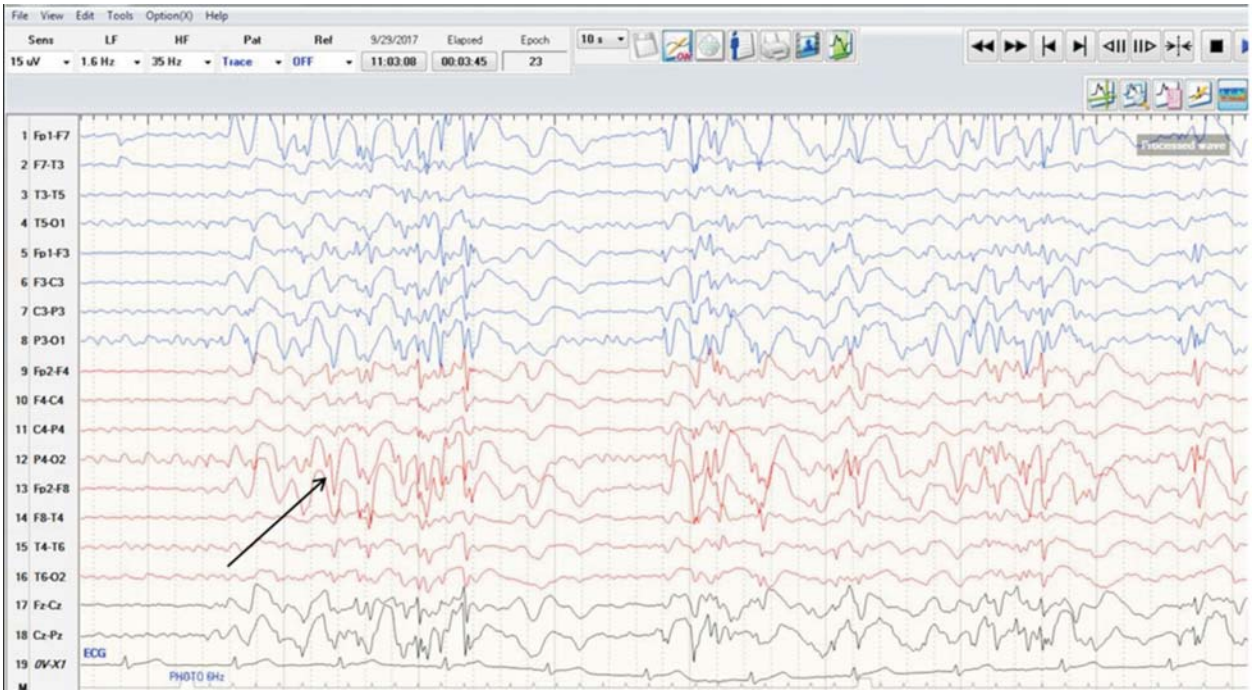
Silvapulle MJ, *et al.* Epileptology of the first-seizure presentation: A clinical, electroencephalographic, and magnetic resonance imaging study of 300 consecutive patients. Lancet. 1998;352:1007-11.
34. Trinka E, Leitinger M. Which EEG patterns in coma are nonconvulsive status epilepticus? Epilepsy Behav. 2015;49:203-22.
35. Bergey GK. Refractory Status Epilepticus: Is EEG burst suppression an appropriate treatment target during drug-induced coma? What is the holy grail? Epilepsy Curr. 2006;6:119-20.
36. Seshia SS, Bingham WT, Kirkham FJ, Sadanand V. Nontraumatic coma in children and adolescents: Diagnosis and management. Neurol Clin. 2011;29:1007-43.
37. Zandbergen EG, de Haan RJ, Stoutenbeek CP, Koelman JH, Hijdra A. Systematic review of early prediction of poor outcome in anoxic-ischaemic coma. Lancet. 1998; 352:1808-12.
38. Subcommittee on Febrile Seizures, American Academy of Pediatrics. Neurodiagnostic evaluation of the child with a simple febrile seizure. Pediatrics. 2011;127:389-94.
39. Kim H, Byun SH, Kim JS, Lim BC, Chae J-H, Choi J, *et al.* Clinical and EEG risk factors for subsequent epilepsy in patients with complex febrile seizures. Epilepsy Res. 2013;105:158-63.
40. Harini C, Nagarajan E, Kimia AA, de Carvalho RM, An S, Bergin AM, *et al.* Utility of initial EEG in first complex febrile seizure. Epilepsy Behav. 2015;52:200-4.
41. Shah PB, James S, Elayaraja S. EEG for children with complex febrile seizures. Cochrane Database Syst Rev. 2015;CD009196.
42. Beghi E, Giussani G, Grosso S, Iudice A, La Neve A, Pisani F, *et al.* Withdrawal of antiepileptic drugs: guidelines of the Italian League Against Epilepsy. Epilepsia. 2013 ;54:2-12.
43. Afshari D, Moradian N. Evaluating the rate of recurrence of epilepsy after therapy discontinuation in 2-year seizure-free epileptic patients. Int J Neurosci. 2012;122:598-601.
44. Berg AT, Shinnar S. Relapse following discontinuation of antiepileptic drugs: A meta-analysis. Neurology. 1994;44:601-8.
45. Verrotti A, Matricardi S, Di Giacomo DL, Rapino D, Chiarelli F, Coppola G. Neuropsychological impairment in children with Rolandic epilepsy and in their siblings. Epilepsy Behav. 2013;28:108-12.



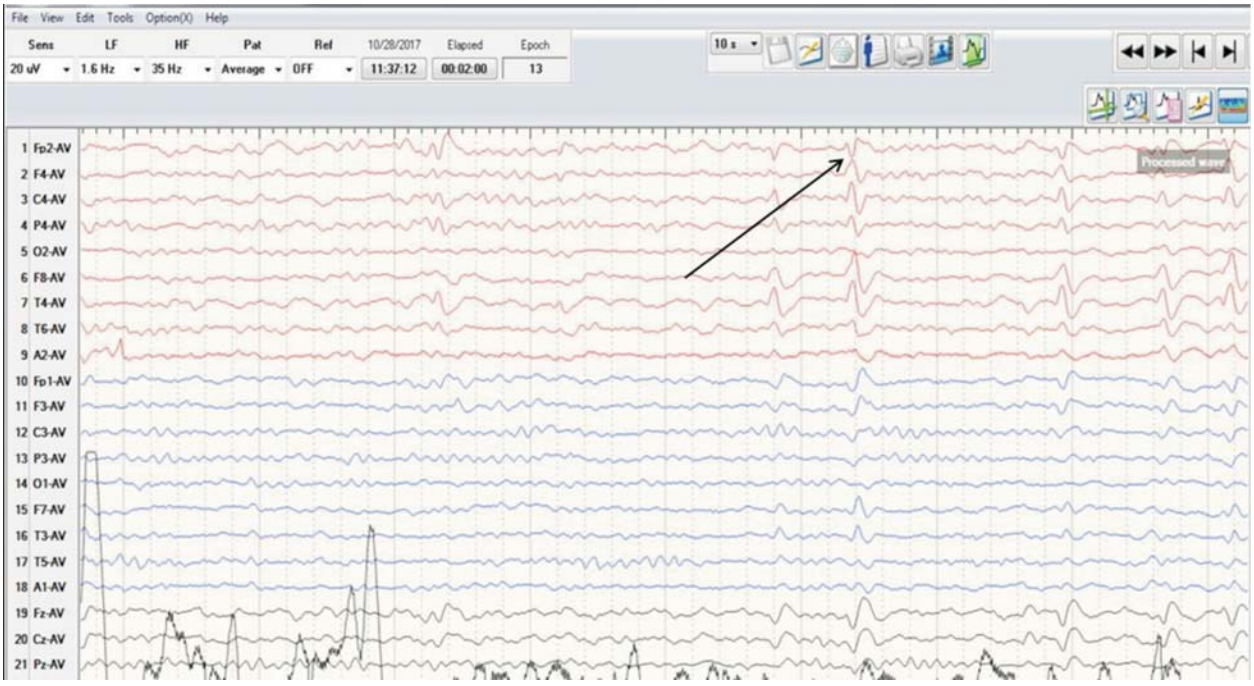
WEB FIG. 1 *EEG (Electroencephalography) showing runs of vertex waves (arrow) observed in normal stage II NREM sleep.*



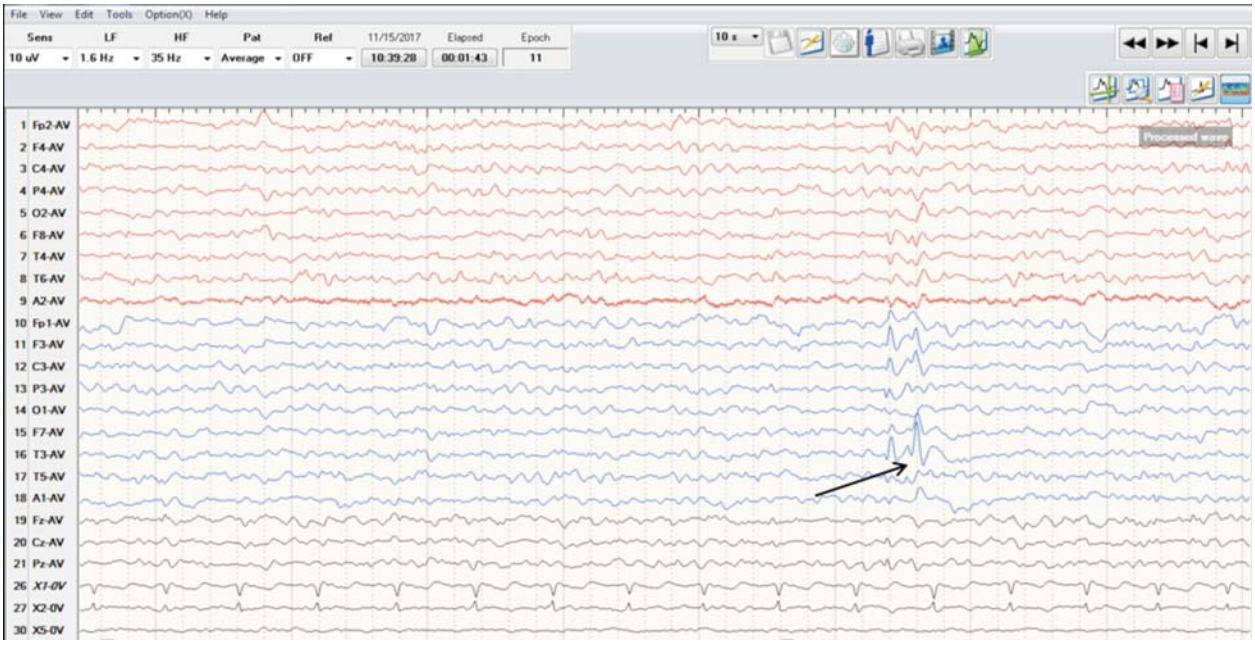
WEB FIG. 2 *EEG (Electroencephalography) showing Classical hysarryhythmia with chaotic background with high voltage epileptiform discharges (arrow).*



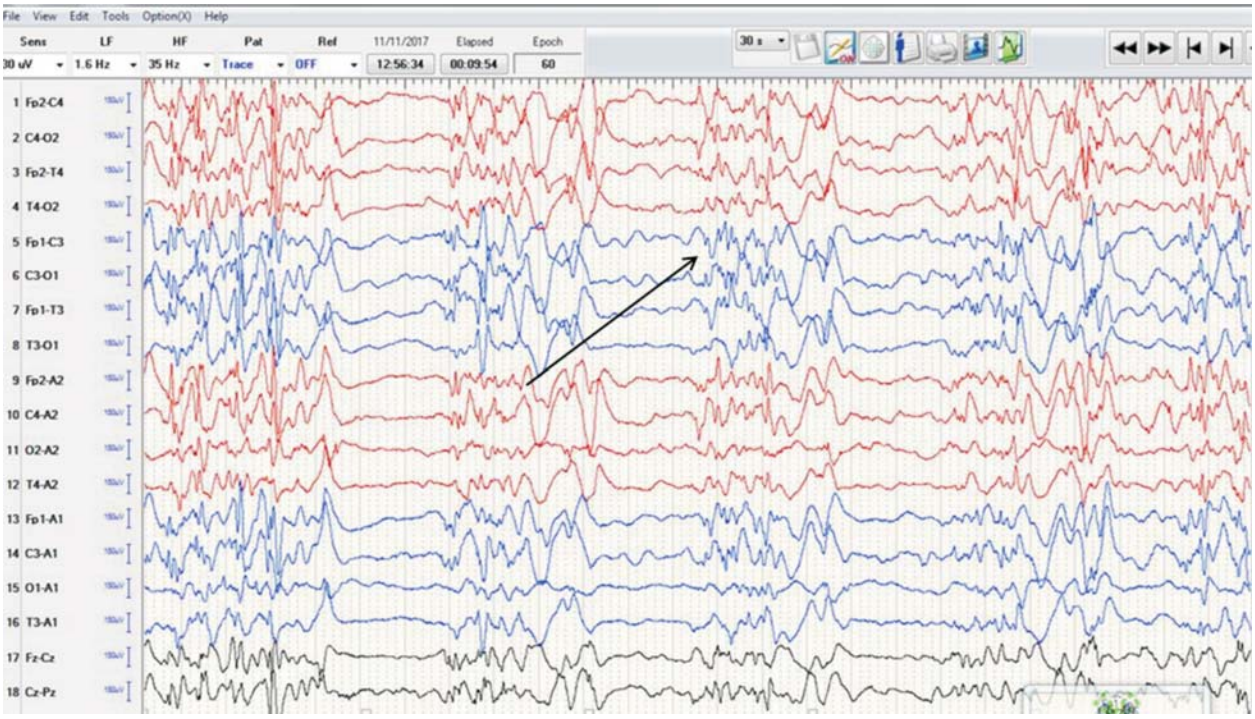
WEB FIG. 3 *EEG (Electroencephalography) showing generalized interictal epileptiform discharges (arrow) (4-4.5 Hz) on photic stimulation in a child with Juvenile myoclonic epilepsy.*



WEB FIG. 4 *EEG (Electroencephalography) showing Rolandic centrotemporal spikes with tangential dipole (arrow).*



WEB FIG. 5 *EEG (Electroencephalography) showing left temporal interictal epileptiform discharges (arrow) in a child with left mesial temporal sclerosis.*



WEB FIG.6 *EEG (Electroencephalography) showing generalized epileptiform discharges (arrow) in periodic interval in subacute sclerosing panencephalitis.*