EEG for the Residents in Pediatrics

By

Rajesh Ramachandran Nair
Assistant Professor of Pediatrics
(Pediatric Neurology/Epilepsy clinic)
McMaster Children’s Hospital, Hamilton, Canada

This book is dedicated to the Pediatrics Residents at McMaster whose enthusiasm and curiosity encouraged me a lot.

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What is EEG?

I was always puzzled (or made to) as a medical student (may be still I am puzzled!!!) as to what EKG (ECG when Air India lands in Mumbai) stands for. Is it Electro C(K)ardio Graph, Gram or Graphy? After reading thousands of EEGs over the years, I confess, I had to refer the dictionary for the exact expression of EEG. OK, electroencephalogram is the actual tracing you get if you perform electroencephalography with the electroencephalograph.

It may not be a bad idea to go back to your medical school neurophysiology, which could be potentially boring. Things would be easy if I just mention that EEG represents the summation of the neuronal postsynaptic potentials. The term ‘Brain wave’ is tempting; but may not be viewed as very scientific.

Recording

First I have to introduce a fancy term, ‘10-20 system of international electrode placement’. I am sure you read this term in some EEG reports and wondered what number game it was. Be a proud Canadian! H Jasper at the Montreal Neurological Institute headed the study, which lead to this standard system¹. Holds good even after 50 years! Routine EEG recording is known as scalp EEG recording where you place electrodes over the scalp. You can place electrodes inside the skull and dura matter (intracranial placement of the electrodes). Let’s leave that to hardcore epileptologists and neurophysiologists.

A specific scalp electrode is denoted by an alphabet followed by a number. The alphabet refers to the location (F –frontal, C-central, P- parietal, T- temporal, O- occipital). The number represents the side (odd number for left and even number for right) as well as anteroposterior or superoinferior location (1 is anterior to 3). F₃ represent a left frontal electrode, which is superior (location; not quality) to F₇. The landmarks are bilateral pre-auricular points and, nasion and...
inion (occipital). Electrodes are spaced either 10% or 20% of the total distance between a pair of landmarks. Now you know what is 10-20 system (*diagram 1*).

A channel is the combination of two electrodes. The voltage output of a channel is the difference in the voltage of the two electrodes. F\textsubscript{3} represent an electrode. F\textsubscript{3}-F\textsubscript{7} represents a single channel.

You can make multiple channels by combining two electrodes (e.g. F\textsubscript{1}-F\textsubscript{7}, F\textsubscript{3}-C\textsubscript{3} and so on). Have you seen an EEG reading monitor? On the left side on the screen you can see a column of channels. See the *figure 1*. Here the channels are arranged in a specific order, which is different from *figure 2*. Now you learned what a montage means. Montage is a specific way of arranging channels. Let’s stop here.

By this time the technologist has applied all the electrodes. You have seen the number of electrodes. Regular EEG recording lasts 30 minutes. It is too ambitious to think that every child will enjoy the procedure and wholeheartedly cooperate. Movement can cause significant artifacts interfering with the interpretation of the record. In *figure 3* you can see the ‘movement hurricane’ has completely disrupted the EEG. The poor EEGer cannot generate a satisfactory report based on such a recording. It is difficult to talk about abnormal EEG patterns before we describe what the normal looks like. The normal EEG pattern varies according to the age, sleep-wake state and use of medication. As my aim is not to confuse you, let’s concentrate on the normal EEG pattern of an 8 year old (*figure 1*)

We traditionally classify EEG waves into alpha (8-13 Hz), beta (>13 Hz), theta (4-7 Hz) and delta (1-3 Hz). You do not have to been an EEGer to note that delta waves are slower than alpha waves. After the age of 3 years, the predominant EEG rhythm is alpha, which is maximally expressed over the posterior head regions. Alpha becomes prominent when you close your eyes.
Opening the eyes and visual cues attenuate alpha. Beta waves are seen if the child has received sedatives, benzodiazepines, Phenobarbital or anesthetics. Theta and delta waves are generally abnormal in older children during wakefulness. But when you sleep your rhythm is mixed theta-delta (Figure 4).

All recording are done as per the ‘Minimal standards for electroencephalography in Canada’. Awake EEG is recorded for a minimum of 30 minutes.

**Activation procedures**

1. Hyperventilation- Most of you are aware of an epilepsy syndrome known as ‘childhood absence epilepsy’. In some kids with this type of epilepsy you can precipitate an absence seizure by hyperventilation. Hyperventilation is generally done for a period of 3 minutes.

2. Photic stimulation- A series of light flashes at different frequencies (1- 30 Hz) are shown to the child for 5-10 seconds. It is interesting to note that the occipital electrodes can show the same frequency as the flash frequency during photic stimulation. This is a normal finding (photic driving). You do not have to be reminded that occipital cortex is our visual cortex. Importance of photic stimulation will be discussed later.
Importance of a proper referral form

Before I get into this further, let me ask you a question. How would you feel when you receive an MRI report on your patient as follows? ‘There are multiple white matter hyper-intensities in T2 weighted and FLAIR images. Clinical correlation suggested’. May be the radiologist would have reported ‘this is consistent with CNS vasculitis’ if you had indicated in the MRI request form that the child had joint pain, skin rash, fever, seizures and weakness of one side. The EEGer reads only the EEG and may not know the clinical details.

Recently I read an EEG on a 7-year-old boy. EEG showed sharp waves from the right centro-temporal regions. The natural tendency is to conclude the EEG report, as ‘this is consistent with Benign Epilepsy of Childhood with Centro-Temporal spikes (BECCTS)’. The smart pediatric resident mentioned in the EEG request that ‘child had two episodes of sudden loss of consciousness and fall’. We know this is not a typical symptom of BECCTS (also known as Benign Rolandoic Epilepsy). Hence I concluded the EEG report as ‘the Rolandic epileptiform discharges most likely represent a genetic EEG trait and is less likely to represent the clinical situation’. Child was later diagnosed to have syncope.

Some drugs can significantly affect the EEG pattern. Phenobarbital and benzodiazepines can produce generalized beta activity. A few months back, I had the chance to report the EEG on a 9-year-old girl. She had 4-5 nocturnal focal onset tonic clonic seizures. She was put on antiseizure medication. Two weeks later, she had plenty of seizures and was always drowsy with incoherent speech. EEG showed significant activation of bilateral epileptiform discharges, which were almost continuous. The initial EEG and the history were suggestive of BECCTS (Figure 13A). But the new EEG was completely different (Figure 13B). Patient was started on Carbamazepine
after the first EEG. This information was provided in the referral from. Hence I was able to suggest the possibility of Carbamazepine induced electroclinical worsening in BRE.

In children with cardiac disease, respiratory disorders or vascular malformation of the brain, hyperventilation may be contraindicated. Unless this is specifically mentioned in the referral form, child may be subjected for HV.

Another question! Which of the following two EEG reports would you prefer? The one, which mentions, “There are epileptiform discharges from the left occipital region” or the one, which mentions, “There are epileptiform discharges from the left occipital region. This is consistent with benign epilepsy of childhood with occipital paroxysms BECOP (benign occipital epilepsy)”. If you need to get the second report you should have provided the clinical information (e.g. 7 year old with normal neurodevelopment, 2 seizures recently, history of visual aura and eye twitching followed by vomiting) Figure 7.
Interictal Vs Ictal EEG features: How to interpret the EEG report

Seizure is a paroxysmal event. A standard EEG recording is for 30 minutes. It is unreasonable to hope that the patient will have a seizure during the EEG recording. However, a few patients do develop seizures during EEG recording as they have very frequent seizures even otherwise. EEGer and the technologists are greedy! Hence we try to provoke seizure during EEG recording by hyperventilation or photic stimulation. If a child develops seizure during the EEG recording, we get ‘ictal EEG patterns’ (Figure 9B). Most of the occasions we record only the ‘interictal EEG patterns’. Seizure is abnormal and excessive hypersynchronous neuronal discharge, which occurs, in a paroxysmal fashion. It is reasonable to believe that such a brain is electrically unstable in between seizures. This baseline electrical abnormality can result in ‘interictal epileptiform discharges’. I have seen EEG requests with a note ‘to look for seizure activity’. Yes, if the child develops a seizure during the 30-minute recording, we can capture the electrographic changes during the event. Interictal epileptiform discharges are usually in the form of spikes, spike and waves or sharp waves. They just represent the baseline brain electrical abnormality and not seizure. Spike duration is < 70 ms. Sharp wave is 70-200 ms in duration. The electrophysiological significance is same.

Interictal epileptiform discharges provide us very useful information. Some morphological patterns are specific for certain epilepsy syndromes. Generalized polyspike and wave discharges with normal background activity are seen in primary generalized epilepsy syndromes (e.g. Juvenile Myoclonic Epilepsy JME Figure 6). Diphasic sharp waves from the centrotemporal region are usually typical for BECCTS (Figure 11). If you see interictal epileptiform discharges from one region of the brain, you may want to exclude a structural lesion at that location (if it is not one of the benign localization related epilepsy syndromes like BECCTS and BECOP. Please
don’t be under the impression that interictal abnormalities refer only to epileptiform discharges. Generalized slowing of the background activity can be seen in encephalopathy (Figure 14). Focal slowing of the background activity can be seen in focal structural lesions or in the postictal phase after a focal onset seizure (up to 24-48 hours after a seizure).

Did you have a specific question when you requested the EEG? If the answer is ‘yes’, you will find it easy to interpret the EEG report. Most EEG reports consist of history, body of the report and conclusion/interpretation. Body of the report has description of the awake background activity, activation procedures, epileptiform discharges, and artifacts and sleep features. In a child with developmental delay or encephalopathy, the background activity is usually slow. In the subsequent pages, you shall see more information.
When should I repeat the EEG?

A single EEG recording may not give you answers all the time. In patients with established epilepsy, the rate of finding abnormalities in a single EEG examination is 50-59%. The yield of EEG abnormalities has been reported to increase to 59-82% with repeated EEG examinations. An abnormal EEG in a child with a first unprovoked seizure is associated with a significantly higher recurrence risk, particularly in children with an idiopathic first seizure. All of us know that specific epileptiform discharges in the EEG increase our ability to diagnose definite epilepsy syndromes and prognosticate. There are certain situations in which you would like to repeat an EEG. Pediatricians and Neurologists generally do not treat EEG (we treat patients!). Think about a situation when your treatment is guided by the EEG changes. Such situations do exist. A typical example is treatment of infantile spasm (IS). The therapeutic goal is to control the clinical epileptic spasms as well as hypsarrythmia pattern in EEG. Hence follow up EEG is needed to optimize treatment. Epilepsy is a disorder of brain electric network. As the networks change according to brain maturation, the expression of epilepsy also changes (new seizure types, cognitive changes). An apparently benign epilepsy syndrome can change to a less benign epilepsy syndrome later. If the clinical picture changes, you may want to repeat an EEG to look for altered electroclinical pattern. Some antiepileptic drugs effective in controlling seizures can worsen seizures after variable periods of time. The earliest EEG signature of such a paradoxical AED effect is bilateral and generalized epileptiform discharges. A typical example is carbamazepine in focal onset seizures in children (Figure 13A). Nonconvulsive status epilepticus (NCSE) is being increasingly recognized in children. Even a child with relatively well-controlled seizures on AED can develop NCSE. The reasons could be missing AED or a
part of the natural history of certain epilepsy syndromes like Lennox-Gestaut syndrome. The
diagnosis can be made only by the characteristic EEG changes.

**Use of sleep deprived EEG**

Sleep deprivation can induce seizure in certain epilepsy syndromes (e.g. JME). Interictal
epileptiform discharges can be activated during sleep, especially in benign localization related
epilepsy syndrome (e.g. BECCTS and BECOP). If you suspect an epilepsy syndrome with
activation of epileptiform discharges during sleep and the regular non-sleep EEG does not
provide you with enough information for a proper syndrome classification, you may wish to
order a sleep EEG (*Figure 11*).

Now the question is how can we induce sleep during EEG recording. Some labs use sedative
drugs. Sleep deprived children often fall asleep after hyperventilation in a quite recording room.
Gilbert et al. reported that 57% of sleep deprived, 44% of partially sleep deprived and 21% of
non- sleep deprived children slept during EEG recording. This was an interesting study and the
data were collected from all pediatric EEGs performed during two 2-month periods. During the
first period, all EEGs were performed as ordered, either standard sleep-deprived (SSD) or non-
sleep-deprived (NSD). During the second 2 months, SSD EEGs were performed per routine.
However, non-SSD families were instructed to keep their children awake 2 hours later the night
before the EEG. Those who complied were classified as partially sleep-deprived (PSD). Neither
the presence of sleep nor the use of PSD or SSD protocols increased the odds of epileptiform
EEGs. Authors concluded that sleep deprivation should not be used routinely to increase the
yield of pediatric EEGs. We are probably not interested in this type of situation as our question
is about the benefit of getting a sleep deprived EEG if the first regular awake recording was not
informative. The above-mentioned study did not look in to the utility of ordering a sleep
deprived EEG after a regular awake EEG recording. In an exciting study by Carpay et al, 177 children with one or more recent afebrile seizure with no epileptiform abnormalities (EA) in the first regular EEG underwent sleep deprived EEG. 34.5% showed EA in the repeat EEG.

All of us are aware of infantile spasm (IS), an epileptic encephalopathy affecting infants. The typical interictal EEG pattern is known as hypsarrhythmia (Figure 16). Presence of hypsarrhythmia (HA) is not a must for diagnosing infantile spasm. But in doubtful situations presence of HA clinches the diagnosis of IS. HA pattern may be visible only during the sleep stage. Hence it is important to obtain a sleep EEG if you want demonstrate HA either for the diagnosis of IS or judging the treatment effect.

Landau-Kleffner Syndrome (LKS) is another epileptic encephalopathy affecting young children. A previously normal young child develops acquired auditory agnosia (difficulty to understand spoken language) followed by cognitive regression after experiencing a few focal onset seizures. In some children, no one might have even witnessed a seizure! Sleep EEG shows almost continuous activation of spike and wave discharges or sharp waves, which is almost diagnostic (Figure 10).
**What the EEG can and can't tell you?**

If you want to remember a single point from this book, let it be the following. A normal EEG does not exclude the diagnosis of epilepsy or seizures. In the previous section, I mentioned that in patients with established epilepsy, the rate of finding abnormalities in a single EEG examination is 50-59%. That means single EEG may not show EA even in established epilepsy. Let’s be positive and come back to this after discussing what the EEG can tell us.

Background activity gives you immense information on the base line cerebral neurophysiological function. In children with static (cerebral palsy) or progressive encephalopathy (neurodegenerative disorders), the background activity is usually slow. It could be generalized theta or delta slowing. Focal EEG slowing occurs in focal cerebral structural or functional lesions. Stroke (acute or old), space-occupying lesions (tumors, abscess or cyst), focal encephalitis (viral, immunological or acute disseminated encephalomyelitis) and post-ictal state after a focal onset seizure can result in focal EEG slowing. However, EEG may not be able to give a clue to the underlying focal pathology. Pure white matter lesions are less likely to cause significant EEG slowing (e.g. multiple sclerosis). Focal EEG slowing associated with EA in the same region suggest a presence of a focal epileptogenic structural abnormality rather than benign localization related epilepsy of childhood. MRI Brain is ordered in such situations.

A specific EEG abnormality can be seen in certain acute focal cerebral dysfunction. Examples are stroke and focal encephalitis (e.g. Herpes Simplex Encephalitis HSE). The pattern is known as periodic lateralized epileptiform discharges (PLEDs *Figure 15*). Focal sharp and slow wave complexes (with or without spikes) recur in a periodic or quasiperiodic fashion (1-4 seconds interval). If a child with fever, focal onset seizures and alteration in consciousness has PLEDs in EEG, your first differential diagnosis has to be HSE. Unfortunately PLEDs usually appear 2-3
days after the symptom onset. Absence of PLEDs in EEG does not exclude the possibility of HSE.

All of us are curious to know whether EEG can differentiate recurrent pseudoseizures from true epileptic seizures. A normal EEG can occur in both situations. If the EEG shows specific EAs suggestive of an epileptic syndrome, your question is answered. However if the clinical presentation is not typical of the epileptic syndrome suggested by EEG, you are in trouble. The EEG can help classify episodes as epileptic seizures but only if the eyewitness account is very suspicious of epileptic seizures. Benign Rolandic epileptiform discharges can occur as a genetic trait in up to 2% of normal school aged children who never had seizures. Electro-clinical correlation is the key factor. Up to 3.5% of children who never had seizures and otherwise normal can have sporadic EAs in EEG.

In my opinion, the beauty (use!) of EEG is in identifying the specific epileptic syndrome. Identification of a specific epileptic syndrome is crucial for antiepileptic drug selection, deciding on neuroimaging (Figure 9A and 12) and prognostication. Appendix 1 mentions the specific EEG patterns in common epilepsy syndromes.

Specific EAs can be the first clue to an underlying disorder. Occipital EAs can happen in idiopathic (e.g. BECOP) and symptomatic localization related epilepsy of occipital origin (e.g. occipital cortical dysplasia). This could happen in celiac disease as well. It is not rare to get neurology consult request for ‘child has migraine and EEG shows occipital EAs’. Why did the pediatrician order an EEG on a child with suspected migraine? Let’s assume that the child had visual aura. May be the pediatrician was not sure whether the visual aura was migraine aura or aura of occipital seizure. Colored visual aura is more frequent with occipital seizure. Occipital EAs are not very rare in migraine. We know that migraine is a paroxysmal disorder associated
with increased cortical excitability\textsuperscript{12}. Antiepileptic drugs are used for continuous prophylaxis in migraine with success. Some epileptic syndromes like BECOP and JME are associated with increased prevalence of migraine. However isolated EAs in the EEG of a child with migraine does not change the diagnosis.

You will find many EEG reports mentioning ‘there was photoparoxysmal response (PPR)’. Method of photic stimulation (PS) was mentioned in the initial section of this book. In certain epilepsy syndromes like juvenile absence epilepsy, juvenile myoclonic epilepsy, epilepsy with grandmal seizures on awakening, idiopathic photosensitive epilepsy and rarely occipital epilepsy EAs can be produced by PS. This is known as PPR. Reproducing the EAs by repeating the PS at the same flash frequency will confirm PPR. Thus presence of PPR helps in identifying the correct epileptic syndrome in the right clinical context (Figure 17). Presence of PPR is not an evidence of AED failure in a child with good seizure control. Presence of isolated PPR in the EEG does not necessarily confirm the diagnosis of epileptic seizure. The likelihood of provoking a positive PPR response in a normal individual is about 1 in 4,000. The concordance rate in twins can be about 100\%\textsuperscript{13}.

EEG must always be put in context with the history, physical examination, and neuroimaging findings to ensure that a proper diagnosis is reached.
**Is EEG needed after first unprovoked seizure?**

The practice parameter: ‘Evaluating a first nonfebrile seizure in children’ by the American Academy of Neurology, the Child Neurology Society, and the American Epilepsy Society stated that the EEG is recommended as part of the neurodiagnostic evaluation of the child with an apparent first unprovoked seizure (standard). The same document mentioned that the majority of evidence confirms that an EEG helps in determination of seizure type, epilepsy syndrome, and risk for recurrence, and therefore may affect further management decisions. The EEG may not be important when the etiology is already established (children with brain tumors or other structural disease). This is an unlikely situation in majority of children who develop first unprovoked seizure. King et al studied 300 patients (children and adults) who experienced first unprovoked seizure. Epileptiform abnormalities were seen in 43%. Clinical classification of seizure changed from partial to generalized in many patients after EEG. EEG information helped the epileptic syndrome identification in 30%. This affects clinical decisions like selection of AED and neuroimaging. Some argue that even if the EEG showed epileptiform abnormalities after the first unprovoked seizure, they may not treat the child with AED. Management also includes an attempt for proper syndromic classification and predicting the chance of recurrence of seizure for which EEG is essential. After all, EEG is a non-invasive investigation. If you can demonstrate evidence of a benign localization related epilepsy syndrome of childhood or idiopathic generalized epilepsy syndrome in the EEG, you may not want to order MRI in that child.

**When to order an EEG?**

Let’s start with febrile seizures (FS). There is consensus that EEG is not needed after simple FS. But the issue is not resolved with complex FS. Yucel et al evaluated 159 children who were being treated for CFS and who had no previously known neurological disorder. EEG was
performed on all patients. EEG abnormality was found in 71 cases; 51 of these were diagnosed with epilepsy during follow up. Six of the 16 cases whose EEGs were abnormal between days 2 and 6 were diagnosed with epilepsy. Twenty of the 30 cases whose EEGs were abnormal between days 7 and 10 were diagnosed with epilepsy. All 25 cases that had abnormal EEGs after day 11 were diagnosed with epilepsy. The results suggested that if neurological examination of CFS patients is normal after their clinical status has stabilized, EEG should be performed after 7 days at the earliest, however for the most accurate diagnosis EEG should be performed 10 days after CFS\textsuperscript{15}. Bottom line is ‘early EAs in the EEG after CFS does not predict future epilepsy’.

The yield of abnormalities of an early postictal EEG in children with CFS is low and similar to the reported rate of abnormalities in children with SFS. The routine practice of obtaining an early EEG in neurologically normal children with CFS is not justified\textsuperscript{16}.

It is not clear when to order an EEG after the first unprovoked seizure in children. If your clinical question is whether the event was indeed a seizure or not, whether the EAs are focal or generalized or whether the child has a specific epilepsy syndrome; the timing of the EEG does not matter. It is unclear whether the presence of EAs in an early post-ictal EEG or in late post-ictal EEG is a better predictor of recurrence of epileptic seizure. The practice parameter mentioned that it was not clear what the optimal timing should be for obtaining an EEG. The practice parameter a cautionary note that although an EEG done within 24 hours of the seizure is most likely to show abnormalities, physicians should be aware that some abnormalities such as postictal slowing, that can be seen on EEG done within 24 to 48 hours of a seizure, may be transient and must be interpreted with caution\textsuperscript{14}.

Some neurologists order an EEG before taking the decision to taper off AED in a child with epilepsy after variable seizure-free period (1-2 years). It is not clear whether the presence of EAs
increase the risk of seizure recurrence after AED tapering in all types of epileptic syndromes. Some studies suggest increased risk of seizure recurrence when the EEG shows EAs\textsuperscript{17} but there is no consensus\textsuperscript{18}. Presence of EAs in the EEG may not predict the risk of seizure recurrence in localization related epilepsies but may be associated with increased seizure recurrence in idiopathic generalized epilepsies\textsuperscript{13}. Clinician takes into consideration the natural history of the specific epilepsy syndrome, the neurodevelopmental status and the neuroimaging findings to decide on AED tapering and the decision is not primarily based on EEG.
**EEG in the pediatric critical care unit**

There are two situations where you would like to have EEG in a child admitted in PCCU.

**Diagnostic EEG:** We have already discussed the use of EEG in HSE. Nonconvulsive status epilepticus (NCSE) denotes nearly continuous electrographic seizures lasting >30 minutes without convulsive activity, manifesting as altered mental status or coma\(^{19}\). You are experiencing deja vu! Child with unexplained coma or altered mental status is not an uncommon inmate of PCCU. NCSE occurs in children with and without preexisting epilepsy or previous clinical seizures. Although intractable epilepsy is the most common etiology, NCSE also occurs in children with structural and toxic-metabolic-infectious conditions\(^ {20}\). 23-34% of children who underwent long-term electroencephalogram monitoring in pediatric intensive care units or emergency departments had NCSE\(^ {20}\). The only way to diagnose this condition is by doing an EEG (prolonged EEG is better). Sedative and hypnotic drugs can cause generalized beta activity in the EEG. In suspected intoxication with these drugs, EEG could be helpful.

**Therapeutic monitoring:** Refractory status epilepticus (SE) is treated with midazolam, pentobarbital/thiopentone or propofol infusion. As the child will be on mechanical ventilation and paralytic drugs, the only way to titrate the dose of these drugs by looking at the electrographic changes. The therapeutic end point is suppression of all electrographic seizures (midazolam) or burst-suppression pattern (barbiturate) *Figure 18*. 
Video EEG

Many academic pediatric hospitals have video EEG (VEEG) facility. During EEG recording a synchronized video recording of the patient is undertaken. Video will capture the clinical event and the EEG shall record the ictal changes. There are several situations in which VEEG is performed.

1. Clinician is not sure whether a clinical event is an epileptic seizure or a non-epileptic event. Caregiver confirms the typical clinical event and the EEGer looks for electrographic changes during the event. A typical example is ‘staring’ in a developmentally delayed child. Another one would be pseudoseizures.

2. As part of pre-surgical work up. The focus is to demonstrate specific focal onset of seizure to identify the focal epileptogenic zone.

3. To quantify the number of seizures in a day.

The recording time (hours to days) is decided by the clinical question. If we are investigating regular nocturnal event (sleep disorder vs nocturnal seizure) a single over night VEEG is sufficient. For presurgical work up several habitual (3-5) seizures need to be recorded to identify the consistent focal seizure origin.
Illustrations

Diagram 1: 10-20 system of international electrode placement

Nasion

Inion

Left

Right
Figure-1: Normal Alpha rhythm is seen in the posterior head region of an 8 year old in quiet wakefulness.
Figure 2: Note the change in Montage. This is coronal montage
Figure 3:

Movement artifacts
Figure 4: Normal sleep record showing V waves and sleep spindles
**Figure 5: Ictal or interictal**

Generalized three per second spike and wave discharges in Childhood Absence Epilepsy. Time between two vertical lines is one second.
Figure 6: Interictal

Generalized polyspike and waves in Juvenile Myoclonic Epilepsy
**Figure 7: Interictal**

Left occipital (P3-O1 and T5-O1) sharp waves in a 7-year-old girl with complex partial seizures. She presented with visual aura, eye deviation, unresponsiveness and right arm clonic movements. She has benign epilepsy of childhood with occipital paroxysms (benign occipital epilepsy)
Figure 8: Interictal

14 year old boy with nocturnal focal onset seizure with secondary generalization. Father had similar seizures since young adulthood. MRI brain was normal. EEG showed bifrontal sharp waves (idiopathic localization related epilepsy syndrome-autosomal dominant nocturnal frontal lobe epilepsy).
Figure 9A: Interictal

Sharp waves in the right temporal region (T4) during sleep EEG in an 11-year-old boy with recurrent complex partial seizures (localization related epilepsy of right temporal origin due to right temporal cavernoma)
**Figure 9B: Ictal**

EEG during a clinical seizure in this 11-year-old boy. The seizure starts in the right temporal region (F8, T4, T6)
Figure 10A: Interictal

5-year-old girl who had developmental regression after focal onset nocturnal seizure since the age of four years. Awake EEG showed sharp waves in the left central (C3), left temporal (T3) and right central (C4) region. See the sleep record.
Figure 10B: Interictal

5-year-old girl who had developmental regression after focal onset nocturnal seizure since the age of four years. Sleep EEG recording showed almost continuous sharp waves from bilateral centrotemporal region. This clinched the diagnosis of Landau-Kleffner syndrome.
Figure 11A: Interictal

7 year old boy had 2 nocturnal focal onset seizures (right upper limb tonic clonic seizure). He made gurgling sound during the seizure. Seizure lasted 4 minutes. He was neurologically and developmentally normal. Awake EEG showed an isolated sharp wave in the left centrotemporal region. Look at the sleep record.
Figure 11B: Interictal

7 year old boy had 2 nocturnal focal onset seizures (right upper limb tonic clonic seizure). He made gurgling sound during the seizure. Seizure lasted 4 minutes. He was neurologically and developmentally normal. Sleep EEG showed frequent sharp waves from the left centrotemporal region. The diagnosis is benign epilepsy of childhood with centrotemporal spikes (benign Rolandic epilepsy)
Figure 12: Interictal

3 year old girl with recent onset focal onset seizure. EEG showed left hemispheric spike and wave discharges. MRI revealed left parietal cortical dysplasia.
Figure 13A: Interictal

9-year-old girl with history of 4-5 nocturnal focal onset tonic clonic seizures. EEG before treatment shows bilateral centrottemporal epileptiform discharges.
Figure 13B: Interictal

Two weeks after starting Carbamazepine, she had plenty of seizures and was always drowsy with incoherent speech. EEG showed significant activation of bilateral epileptiform discharges, which were almost continuous.
Figure 14: Background abnormality

11 year old boy was admitted with history of altered level of consciousness. He had hepatic encephalopathy. EEG showed diffuse high voltage theta and delta slowing.
Figure 15: Interictal

6 year old boy developed focal onset seizures, fever and altered level of consciousness. EEG showed sharpwave complexes from the left temporal region at 1-1.5 second periodicity (PLEDs-Periodic Lateralized Epileptiform Discharges). MRI brain showed hyperintense signal in the left medial temporal region. CSF HSV PCR was positive.
Figure 16: Interictal

8-month-old baby with infantile spasm. EEG shows chaotic background activity with multifocal epileptiform discharges consistent with modified hypsarrythmia pattern.
Figure 17: interictal

Photoparoxysmal response in a girl with Juvenile myoclonic Epilepsy
Figure 18: Interictal

Burst suppression pattern in a child on thiopentone coma for refractory status epilepticus.
Reference:
18. Overweg J. Withdrawal of antiepileptic drugs (AEDs) in seizure-free patients, risk factors for relapse with special attention for the EEG. Seizure. 1995;4:19-36

Appendix-1: Interictal EEG patterns in some pediatric epilepsy syndromes

1. Benign Epilepsy of childhood with occipital paroxysms: Diphasic sharp waves from either occipital region without slowing of background activity. Activation of sharp waves during sleep.
2. Benign Epilepsy of Childhood with centrotemporal spikes (Benign Rolandic Epilepsy): Diphasic sharp waves from either centrotemporal regions without background slowing. Activation of sharp waves during sleep.
3. Childhood absence epilepsy: Generalized 3 per second spike and wave discharges with normal background activity.
4. Juvenile myoclonic epilepsy: Generalized spike, polyspike and wave discharges with normal background activity. Photoparoxysmal response may be seen.
5. Infantile spasm: Characteristic EEG pattern is hypsarrythmia (chaotic high amplitude background activity with multifocal epileptiform discharges). The typical may not be present in every case. Hypsarrythmia pattern may be seen only during sleep.
6. Lennox-Gestaut syndrome: Slow background activity with bifrontal or generalized slow (1-2.5 Hz) spike and wave discharges; also multifocal epileptiform discharges.