The Role of Long Monitoring EEG in Idiopathic Epilepsy

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Abstract

Despite the advances in imaging, EEG remains a critical test for the diagnosis of epilepsy. Not only can it confirm the diagnosis, but it can also clarify the type of epilepsy. There are many different types of EEG recordings depending on duration, the presence of video, and inpatient or outpatient setting, each with its pros and cons. Interictalepileptiform abnormalities are very specific to epilepsy, but they can be over-interpreted by inexperienced readers. In addition to diagnosis of epilepsy, EEG also has a role in the decision to discontinue treatment in seizure-free patients, and in assessing critically ill patients for possible status epilepticus and encephalopathies. EEG reports should be relatively standardized and clear to the clinician who requested the EEG.

KeyWords:EEG, Epilepsy, IED.

DOINUMBER:10.48047/NQ.2022.20.19.NQ99430 **NEUROQUANTOLOGY2022;20(19):4671-4677**

Introduction.

In adult epilepsy centres, the sensitivity of an initial routine EEG to reveal IEDs ranges from 29 to 55%. IEDs should be distinct, standing out from the background, and usually appear as spikes, sharp waves and spike-wave complexes **(1).**

Depending on the type of epilepsy, the sensitivity of the routine EEG can vary. For example, patients with Lennox-Gastaut syndrome nearly always have abnormal routine EEG with IEDs, while a frontal lobe epilepsy patient may never have IEDs on interictal EEG. Other factors such as age, sleep deprivation, level of consciousness, focal vs generalized epilepsy, temporal versus extratemporal epilepsy, antiseizure medications (ASM), seizure frequency, activation procedures, proximity of the EEG to recent seizure activity, and additional electrodes can also affect the sensitivity of routine EEG. Generally, the sensitivity of routine EEG is around 50% for the initial EEG and increases to 82–92% with repeated studies **(2).**

Extended EEGs can also increase sensitivity. For example, the mean duration to an initial IED was 56 minutes in patients with temporal lobe epilepsy and 22 minutes in patients with a generalized epilepsy **(3).**

Others found that only 36% of patients had IEDs within the first 20 minutes of long-term monitoring, while 89% had IEDs in the first 24 hours. Other studies compared a repeat 30-minute EEG to extended two-hour EEG and found that the diagnostic yield is similar in patients with a previous normal routine EEG **(4).**

Table (1): The role of EEG in epilepsy **(5).**

Diagnosis of epilepsy
- Differential diagnosis of paroxysmal neurological events (spells)
- Distinction between a focal and generalized seizure disorder (classification)
- Diagnosis of epileptic syndromes and predicting the prognosis
- Recognition of photosensitivity (triggering factors)
- Identification of the refractory epilepsies

Management of epilepsy
- Assessing risk of recurrence after an unprovoked seizure
- Selection of antiepileptic treatment
- Likelihood of seizure relapse if medication is withdrawn
- Identifying the surgically remediable epilepsies
- Determining probability of seizure recurrence after medication withdrawal
- Predication of seizure recurrence after medication withdrawal following surgery
- Investigation of cognitive decline
- Detection of nonconvulsive status and seizure quantification

Uses Of EEG In Diagnosis Of Epilepsy:

EEG helps to determine seizure type and epilepsy syndrome in patients with epilepsy, and thereby choice of antiepileptic medication and prediction of prognosis. EEG findings contribute to the multi-axial diagnosis of epilepsy, in terms of whether the seizure disorder is focal or generalised, idiopathic or symptomatic, or part of a specific epilepsy syndrome (**6).**

In practice, the clinician will be reasonably certain about seizure type based on the account provided by the patient and witness. However, when history is unclear (unwitnessed ‘‘blackouts’’ or brief impairment of awareness), EEG can help distinguish between a complex partial seizure with focal IED, and an absence type seizure with generalised IED **(7).**

Interictal EEG:

While the background EEG is usually normal in patients with epilepsy, abnormal interictal EEG waveforms may include nonepileptiform abnormalities and interictalepileptiform discharges (IEDs). Patients with epilepsy may show generalized or focal slowing of the background, but the presence of focal or generalized IEDs is the most supportive of a diagnosis of epilepsy **(8).**

Epileptiform discharges:

IEDs are highly specific for epilepsy, yet approximately 1%–3% of the general population may have epileptiform discharges on EEG. Morphologies of IEDs may include focal and generalized spikes, sharp waves, spike-and-slow wave complexes, and polyspikes, occurring either as isolated discharges or in brief runs. In some cases, the neural network that generates spikes may not be the same as the network that generates seizures, and seizure may arise in a different location from where epileptiform discharges occur. Interictal spikes are so highly correlated with spontaneous seizures that their presence supports a diagnosis of epilepsy. At the cellular level, neurons undergo a paroxysmal depolarizing shift in membrane potential during detection of an interictal spike **(9, 10).**

Frequent IEDs may disrupt cognitive or neurologic function; however, the precise long-range impact of chronic repetitive IEDs on the normal neuronal circuitry and on cognitive outcome remains to be clarified. IEDs must be carefully distinguished from benign variants and normal brain waveforms to avoid over interpretation. The type, localization, and frequency of IEDs are of significant diagnostic and prognostic value in specific epilepsy syndromes. Table 2 summarizes features of abnormal epileptiform discharges **(11).**

Table (2): Criteria for interictal spikes and sharp waves **(11)**

Paroxysmal and clearly distinguished from background activity
An abrupt change in polarity occurring over several milliseconds
Duration less than 200 ms: 70–200 ms for a sharp wave and 20–70 ms for a spike
Asymmetric contour with steep upslope, with down stroke usually being less steep and deepening below the baseline
Has a physiologic field (seen in ≥2 nearby electrode sites) with voltage gradient
Are typically negative in polarity
Aftergoing slow wave
Appears in a location with an associated area of abnormality (e.g., focal slowing)
Persists during slow wave sleep (in contrast to benign variants)

Focal epileptiform discharges:

Focal IEDs may occur anywhere in the brain but are most often associated with temporal lobe epilepsy (TLE). Also patients with unilateral anterior or mid temporal IED were found to have better surgical outcome compared with those who have bilateral temporal or extratemporalepileptiform discharges) (Fig. 1). Frontal, parietal, and occipital epilepsies tend to have fewer identifiable epileptiform discharges with scalp EEG recording **(12).**

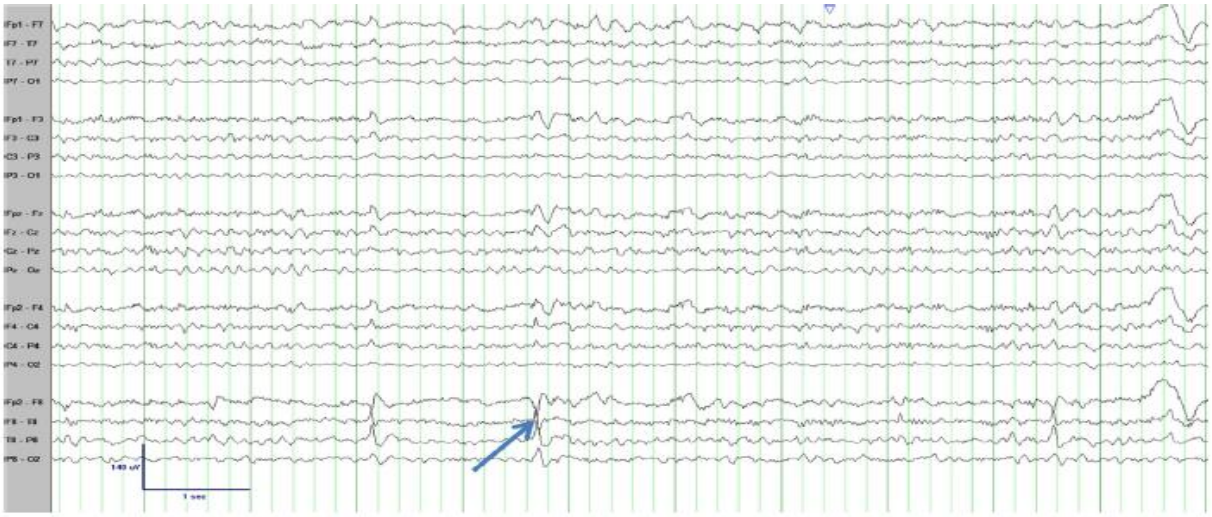

Figure (1): Temporal spikes with an electrographic maximum at the right anterior temporal electrode (T8) followed by low-voltage slow waves **(12).**

Table (3): Interictal and ictal EEG findings in focal epilepsy (**13**)

	Interictal EEG	Ictal EEG
Temporal lobe epilepsy		
Mesial TLE	Anterior or basal temporal EDs, bilateral EDs (30%)	Inferotemporal rhythmic regular 5–9 Hz discharge
Neocortical TLE	Broadly distributed EDs (often extending to the parasagittal area)	Temporal and/or frontocentral irregular rhythm 2–5 Hz discharge
Frontal lobe epilepsy		
Mesial FLE	Often negative	Often nonlateralizing with muscle or motion artifact or appear pseudo-generalized
Lateral FLE	Midline or bilateral EDs with asymmetric amplitude	Lateralized with/without false localization to ipsilateral temporal lobe
Parietal lobe epilepsy	Nonlateralizing/nonlocalizing (majority); centroparietal EDs (10%–15%)	<10% Localized to parietal lobe, 30% associated with bilateral synchrony; falsely localizing to temporal lobe
Occipital lobe epilepsy	EDs restricted in occipital lobe (<20%); falsely localizing to the ipsilateral temporal lobe or synchronous or bilateral EDs	Rhythmic occipital discharge (10%–20%); rapid speared or falsely localizing to ipsilateral temporal lobe

Generalized epileptiform discharges:

Generalized IEDs are usually typical or atypical generalized spike-and-wave (GSW) complexes, poly spike discharges, or poly spike-and-wave discharges. Other epileptiform discharges include "slow" GSW (2.5 Hz and less), "typical" GSW (3 Hz), and "fast" GSW (>3 Hz) occurring as generalized spikes or polyspikes with or without after-going slow waves, and at times as a single complex or burst of repetitive epileptiform discharges. Epileptiform discharges are seen in roughly 50% of patients with generalized tonic–clonic seizures (GTCs) but have been described in nonepileptic populations. An increased prevalence in nonepileptic patients having a first-degree relative with generalized epilepsy has a higher rate of GSW (**13).**

Several authors have reported atypical EEG abnormalities in generalized epilepsy. These include focal, unilateral, and asymmetric discharges, as well as generalized paroxysmal fast activity and distortion of spike–wave morphology during NREM sleep. Since focal spike–wave discharges may also occur in patients with genetic generalized epilepsy, they should not be mistaken as definitive evidence for underlying focal epilepsy unless consistent and persistent focal epileptiform discharges are identified. Secondary bilateral synchrony (SBS) is an EEG phenomenon in which focal spikes evolve to bisynchronous spike waves and could mimic a generalized epileptiform discharge **(14, 15).**

The identification of SBS, despite the appearance of generalized epileptiform discharges, is critical in the evaluation of drug-resistant epilepsy. In this group of patients, proper classification may allow for potentially curative treatment with focal resective surgery. Alternatively, the presence of focal epileptiform discharges and subtle asymmetries may lead the clinician to misdiagnose focal epilepsy and result in unnecessary evaluation, which can lead to treatment with inappropriate ASDs **(16).**

Table (4):EEG features in adult generalized epilepsy **(16).**

Common GGE	EEG features
Absence epilepsy	3-Hz generalized spike-and-slow wave; often repetitive trains of discharges; normal background; activation of IEDs and seizures with hyperventilation
Atypical absence	1.5- to 2.5-Hz generalized spike- and-slow wave discharges; IEDs may be asymmetric, with shifting focal features
Juvenile myoclonic	4- to 6-Hz generalized spike and multiple spike-and-slow wave; Normal background; activation of IEDs with photic stimulation is common; more typical 2.5- to 3-Hz spike-and-slow wave may be seen; IEDs may be asymmetric, with shifting focal features
Lennox–Gastaut syndrome	<2.5-Hz generalized sharp-and-slow wave discharges; Generalized background slowing and paroxysmal fast activity; often multifocal spikes and sharp waves
Progressive myoclonic epilepsy	Generalized and multifocal spikes, multiple spikes, and sharp waves; Progressive background slowing with disease progression; photic activation of IEDs in some cases; photoparoxysmal response in some

They are typically seen in the setting of an acute or subacute focal cerebral lesion such as abscess, infarction, and intracranial hemorrhage, and reflect an acute injury pattern. They can also be seen in patients with chronic conditions including brain tumor. Periodic discharges are a characteristic EEG feature of herpes encephalitis and may also be seen in other central nervous system infections including meningoencephalitis, Creutzfeldt-Jakob disease, and subacute sclerosingpanencephalitis**(17).** Lateralized periodic discharges when compared with other rhythmic discharges have the highest association with seizures, whereas monomorphic delta morphology carries the lowest risk **(16).**

Long term EEG monitoring:

Long term video or ambulatory EEG has an important role in the assessment of patients who present diagnostic or management difficulties following clinical evaluation and routine EEG. The clinical applications of EEG monitoring are:

- Diagnosis of paroxysmal neurological attacks.
- Differentiation between nocturnal epilepsy and parasomnias.
- Diagnosis of psychogenic non-epileptic seizures.
- Characterisation of seizure type.
- Quantification of IED or seizure frequency.
- Evaluation of candidates for epilepsy surgery(**18).**

Ambulatory EEG is most suitable when concurrent synchronised video to document clinical features is not essential, or for monitoring in an outpatient setting or specific environment. Inpatient video EEG telemetry is expensive and labour intensive, and a limited resource. Specialised telemetry units have the advantage of dedicated ward based staff, experienced in the identification of subtle clinical events, and close management of patients during seizures. Duration of study depends on frequency of attacks; in practice, long term EEG monitoring is unlikely to be productive if the patient's events occur less than once per week. Methods to increase likelihood of seizures include antiepileptic drug reduction (utilising specific protocols, and best reserved for pre-surgical evaluation) and provocation techniques (**19).** However, there is a risk that provocation by suggestion may lead to false positive results particularly in psychogenic non-epileptic seizures, and use of other techniques such as saline injection or alcohol swabs carries ethical difficulties. Long term monitoring generates very large amounts of data for analysis, which can be reduced by use of commercially available spike and seizure detection algorithms. There is current research interest in methods that anticipate or predict seizures, by detection of non-linear changes in EEG data at least several minutes before an epileptic seizure. Specificity and sensitivity of these methods has not been fully evaluated, and their clinical role is as yet uncertain (**19).**

In partial epilepsies, the most important ictal EEG changes for seizure localisation are those that occur within the first 30 seconds after the seizure onset. Broadly speaking, localised changes are more common in temporal lobe epilepsy than in extratemporal seizures, and epileptiform or high frequency discharge is more likely to occur in neocortical epilepsy, particularly if the focus is relatively superficial. In mesial temporal epilepsy, the typical ictal onset pattern is a rhythmic h (5–7 Hz) discharge localised to the anterior mid temporal lobe, with up to 80–90% of patients showing such change (**20).**

In lateral temporal seizures, ictal onset EEG changes are usually lateralised, and more likely to have a repetitive epileptiform appearance than mesial temporal seizures. Frontal lobe epilepsy ictal EEG onset patterns are most often generalised or widespread, comprising high frequency activity or slow rhythms or attenuation(**21).**

Localised changes in frontal lobe epilepsy are rare, for a number of reasons inaccessibility of much of the frontal lobes to scalp electrodes, widespread anatomical connections, likelihood of bifrontal damage in post-traumatic frontal epilepsy, and variability in size and distribution of epileptogenic regions. Interictal discharges in frontal lobe epilepsies are often generalised or non-localised for similar reasons (**22).**

Ictal EEG changes may also be obscured by the hypermotor clinical manifestations of FLE. Ictal onset EEG patterns in parietal and occipital seizures vary, in part dependent on pathways of seizure propagation. Localised and lateralised

ictal onset may occur, particularly in non-mesially sited epileptogenic foci. However, the rate of false localisation and lateralisation is highest in these two seizure types, thus limiting the role of ictal recording in parietal and occipital lobe epilepsy (**22).**

Scalp EEG commonly shows no change in simple partial seizures, because the focal ictal discharge is distant or deep, or involves too small a neuronal aggregate for synchronised activity to register on the scalp. This is unfortunate given how difficult diagnosis of simple partial seizures can be on clinical grounds (**23).**

References:

1. **Louis, E. K. S., &Cascino, G. D. (2016).** Diagnosis of epilepsy and related episodic disorders. *CONTINUUM: Lifelong Learning in Neurology*, *22*(1), 15-37.
2. **Salinsky, M., Kanter, R. &Dasheiff, R. M. (1987).** Effectiveness of multiple EEGs in supporting the diagnosis of epilepsy: an operational curve. Epilepsia, 28, 331-4.
3. **Losey, T. E. & Uber-Zak, L. (2008).** Time to first interictalepileptiform discharge in extended recording EEGs. J ClinNeurophysiol, 25, 357-60.
4. **Mahuwala, Z., Ahmadi, S., Bozoky, Z., et al. (2019).** Diagnostic yield of 2-hour EEG is similar with 30-minute EEG in patients with a Normal 30-minute EEG. *Journal of Clinical Neurophysiology*, *36*(3), 204-208.
5. **Fisher, R. S., Cross, J. H., D'Souza, C., et al. (2017).** Instruction manual for the ILAE 2017 operational classification of seizure types. Epilepsia 58: 531–542.
6. **Anwar, H., Khan, Q. U., Nadeem, N., et al. (2020).** Epileptic seizures. Discoveries, 8(2).
7. **Das, J. C. (2014).** Electroencephalogram (EEG) in the Management of Epilepsy in. Mymensingh Med J, 23(2), 406-411.
8. **Krumholz, A., Wiebe, S., Gronseth, G. S., et al. (2015).** Evidencebased guideline: management of an unprovoked first seizure in adults: report of the Guideline Development Subcommittee of the American Academy of Neurology and the American Epilepsy Society. Neurology 84: 1705–1713.
9. **Pillai, J., Sperling, M. R. (2006).** Interictal EEG and the diagnosis of epilepsy. Epilepsia 47 (Suppl. 1): 14–22.
10. **So, E. L. (2010).** Interictalepileptiform discharges in persons without a history of seizures: what do they mean? J ClinNeurophysiol 27: 229–238.
11. **Ung, H., Cazares, C., Nanivadekar, A., et al. (2017).** Interictalepileptiform activity outside the seizure onset zone impacts cognition. Brain 140: 2157–2168.
12. **Verma, A., Radtke, R. (2006).** EEG of partial seizures. J ClinNeurophysiol 23: 333–339.
13. **Devinsky, O., Gazzola, D., LaFrance, Jr., W. C. (2011).** Differentiating between nonepileptic and epileptic seizures. Nat Rev Neurol 7: 210–220.
14. **Williamson, R., Hanif, S., Mathews, G. C., et al. (2009).** Generalized-onset seizures with secondary focal evolution. Epilepsia 50: 1827–1832.
15. **Seneviratne, U., Hepworth, G., Cook, M., et al. (2016).** Atypical EEG abnormalities in genetic generalized epilepsies. ClinNeurophysiol 127: 214–220.
16. **Rodriguez Ruiz, A., Vlachy, J., Lee, J. W., et al. (2017).** Association of periodic and rhythmic electroencephalographic patterns with seizures in critically ill patients. JAMA Neurol 74: 181–188.
17. **Hirsch, L. J., LaRoche, S. M., Gaspard, N. et al. (2013).** American clinical neurophysiology society's standardized critical care EEG terminology: 2012 version. J ClinNeurophysiol 30: 1–27.
18. **Feyissa, A. M., Worrell, G. A., &Lagerlund, T. D. (2021).** EEG and Epilepsy. Epilepsy, 77-98.
19. **Smith, S. J. (2005).** EEG in the diagnosis, classification, and management of patients with epilepsy. Journal of Neurology, Neurosurgery & Psychiatry, 76(suppl 2), ii2-ii7.

*e*ISSN1303-5150

20. **Khalil, M., Shukralla, A. A., Kilbride, R., et al. (2021).** Ictal asystole during long-term video-EEG; semiology, localization, and intervention. Epilepsy & Behavior Reports, 15, 100416.
21. **Andrade-Machado, R., Cuartas, V. B., & Muhammad, I. K. (2021).** Recognition of interictal and ictal discharges on EEG. Focal vs generalized epilepsy. Epilepsy & Behavior, 117, 107830.
22. **Chen, H., &Koubeissi, M. Z. (2019).** Electroencephalography in epilepsy evaluation. CONTINUUM: Lifelong Learning in Neurology, 25(2), 431-453.
23. **Ramakrishnan, S., &Rayi, A. (2022).** EEG Localization Related Epilepsies. In StatPearls [Internet]. StatPearls Publishing.