Epilepsy & Behavior Reports 16 (2021) 100454

Contents lists available at ScienceDirect

Epilepsy & Behavior Reports

journal homepage: www.elsevier.com/locate/ebcr

When should we obtain a routine EEG while managing people with epilepsy?

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ARTICLE INFO

Article history:
Received 20 January 2021
Revised 24 March 2021
Accepted 22 April 2021
Available online 3 May 2021

Presented 12/2/2018 in part at the American Epilepsy Society Meeting in New Orleans, LA.

Keywords:
EEG
Routine
Stat
Video
Epilepsy
ASM
Taper
Electrodes
Montage
ILAE

ABSTRACT

More than eight decades after its discovery, routine electroencephalogram (EEG) remains a safe, noninvasive, inexpensive, bedside test of neurological function. Knowing when a routine EEG should be obtained while managing people with epilepsy is a critical aspect of optimal care. Despite advances in neuroimaging techniques that aid diagnosis of structural lesions in the central nervous system, EEG continues to provide critical diagnostic evidence with implications on treatment. A routine EEG performed after a first unprovoked seizure can support a clinical diagnosis of epilepsy and differentiate those without epilepsy, classify an epilepsy syndrome to impart prognosis, and characterize seizures for antiseizure management. Despite a current viral pandemic, EEG services continue, and the value of routine EEG is unchanged.

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Contents

Introduction . . . 2
Stat EEG . . . 2
When should a routine EEG be performed? . . . 2
What is the yield of a routine EEG? . . . 2
Optimal recording for routine EEG . . . 3
Source localization . . . 4
Activation techniques . . . 4
Routine EEG and epilepsy diagnosis . . . 4
When are EEG findings typical for generalized genetic epilepsy? . . . 5
When are EEG findings typical for focal epilepsy? . . . 5
When are EEG findings typical for epileptic encephalopathies and developmental disorders? . . . 6
When is routine EEG useful in people with psychogenic nonepileptic seizures? . . . 6
Routine EEG during epilepsy management . . . 6
When do pitfalls occur following routine EEG? . . . 7
Conclusion . . . 8
Ethical statement . . . 8

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https://doi.org/10.1016/j.ebr.2021.100454
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Declaration of Competing Interest 8
References 8

Introduction

Routine electroencephalogram (EEG) is a common test of neurophysiologic function that is performed to diagnose and monitor several conditions affecting the brain [1,2]. Routine EEG is foundational for the diagnostic process in people with epilepsy [3], extracting complex brain signals and applying them to clinically relevant features using visual analysis [4]. Normally, a comprehensive epilepsy center may perform > 4,000 EEG studies per year [5]. This was substantially reduced as COVID-19 influenced all aspects of hospital care. As regions rebound, the routine use of EEG in the interictal or asymptomatic period has remained a powerful test for diagnosis and management [4,6].

Today, routine EEGs are a sophisticated multi-channel microprocessor that is digitally based with sampling rates of > 200 Hz, 128 gigabytes of internal memory space, and resolution of at least 12-bits. Video-assisted EEG is now routine and is used to correlate behavioral activity with electrocerebral activity to provide even greater yield. The primary aim of routine EEG analysis is to support the clinician's evaluation of patients with objective data to support a clinical hypothesis for diagnoses.[7]

The benefits of long-term EEG are well established.[2] During the pandemic when "lock down" forced epilepsy monitoring units to close temporarily, the gap created by a lack of monitoring was augmented in our center (and probably many others) and met with a rise in routine and ambulatory EEGs to meet the needs of the patients. When used appropriately, ambulatory EEG is a valuable surrogate for inpatient video EEG monitoring without compromising the quality of the recording. The challenges to maintain a long-term EEG during COVID-19 have proven to facilitate usage of a routine EEG in cases where antiseizure medication (ASM) reduction and presurgical evaluation are not necessary.

In this review, we focus on when to perform and repeat a routine EEG during the evaluation of seizures and epilepsy and other neurological disorders by posing clinically relevant questions and answers.

Stat EEG

Stat EEGs are reserved for emergent situations, especially when non-convulsive status epilepticus (NCSE) is suspected. EEG is the only means of diagnosing NCSE and ongoing electrographic seizures that are potentially detrimental to the brain and may affect prognosis.[8] A stat EEG may also be justified when a patient fails to fully recover within an expected amount of time following treatment for acute seizures[9,10] or when there is no underlying process to explain the change in mental status with coexisting subtle motor activity (e.g., twitches or jerks) which may be suggestive of seizures.[11,12] A careful estimate is that a stat EEG will lead to medication change in at least 1 out of 8 intensive care unit (ICU) patients.[13]

While approximately 10% of EEGs are ordered stat, a vast majority do not result in emergent care.[14,15] The request for stat EEG may be more for convenience as opposed to a medical indication (e.g., pseudostat).[16] The most common reason cited for a stat EEG is often for the assessment of brain death.[9,10] Stat EEGs are not indicated when the patient has had a seizure and is now recovering, when an obvious clinical seizure is in progress, when a readily explained alteration of mental status is present, when hospitalized patients with seizures are awaiting discharge, or to confirm brain death. In these patients, as well as those in the emergency room or the observation unit, a routine EEG would be more suitable either during their hospitalization or as an outpatient procedure after discharge if the patient has returned to their baseline.

When a stat EEG is ordered, it should be interpreted in a timely fashion - usually less than 3 hours following completion.[16] However, earlier reading would be warranted if the technologists see any suspicious patterns of behavior during the recording. Other intermediary designations also exist at some institutions between stat and routine EEG.[16] A routine EEG refers to those recordings that are performed and interpreted within 1–2 days. Overuse of stat EEG orders results in poor use of time and human and material resources, may divert attention from others requiring emergent care, and is cost inefficient.

When should a routine EEG be performed?

A routine EEG is the cornerstone in providing support for a clinical diagnosis of neurological disorders but is especially suited for people with epilepsy (TABLE 1). In people with epilepsy, routine EEG has been applied to diagnosis, classification, quantification, and characterization of interictal epileptiform abnormalities. A routine EEG is generally performed for a seizure diagnosis and can aid in selecting treatment options with ASM **(**Fig. 1**).**

What is the yield of a routine EEG?

The American Academy of Neurology (AAN) and the American Epilepsy Society provide evidence-based guidelines regarding the management of an unprovoked first seizure in adults. There is level A evidence that adults presenting with an unprovoked first seizure are at a greater risk for recurrent seizures within the first two years after the first seizure (21–45%) and factors associated with an increased risk for seizure recurrence include prior brain insult such as stroke or trauma and an EEG with epileptiform abnormalities.[17] Similarly, there is level B evidence for immediate ASM therapy following a second seizure as it is likely to reduce the risk for seizure recurrence in the two years following a first seizure.[17] EEG demonstrates predictive value in determining the risk of seizure recurrence following a single unprovoked seizure.[18] Nonetheless, a normal interictal EEG is commonly seen after a first unprovoked seizure,[19] with a slightly higher yield of recording interictal epileptiform discharges (IED) when the routine EEG contains sleep, sleep-deprivation, or when the duration of the recording is increased to 60 minutes.[20,21] An abnormal EEG with generalized spike-and-wave (GSW) discharges is a consistent predictor for seizure recurrence.[22–26] IEDs present in the EEG have a high specificity for people with epilepsy **(**Fig. 2**)** and is associated with a 2–3 times higher risk for seizure recurrence than a normal EEG.[22–26] In 105 abnormal EEGs containing 6,923 IEDs in consecutive patients diagnosed with generalized genetic epilepsy (GGE), the density and duration of IEDs were used to subtype epilepsy syndromes.[27]

Pretest probability is an important consideration when selecting the appropriate EEG study for a particular patient. Low pretest probability of a diagnosis will have a low yield as a result following an EEG. High pretest probability of any epileptic or nonepileptic diagnosis can increase the diagnostic yield of a routine and ambulatory EEG. To that end, video EEG has demonstrated benefit by having a pretest question.[28] For example, sleep will activate

Table 1
When Should We Perform a Routine EEG?

Indications		Conclusions
Diagnosis and Management of Seizures	First unprovoked seizure	Presence of unequivocal IEDs equates with a new-onset epilepsy diagnosis
	Classification of focal and generalized epilepsy	IEDs help in the choice of ASM for the seizure type(s) and epilepsies
	Diagnosis of epilepsy syndromes	Focal IEDs in focal epilepsy, SSW in epileptic encephalopathies, "fast" GSW in GGE
	Selection of ASM and monitoring response to treatment	Reduction in the spike burden of GSW or seizure burden may be present as a response to therapy
	Head injury	IEDs may occur that suggest untreated seizures
	Brain tumor	Focal slowing loosely correlates with location of abnormality. IEDs suggest a greater potential for seizures
	Stroke	May identify IEDs (especially hemorrhagic strokes) to predict a higher incidence of post-infarction seizures
	Cognitive and memory problems (e.g., transient epileptic amnesia)	May help suggest seizures as a substrate for cognitive impairment when IEDs are present
	Episodic anxiety/mood disturbances	May occur in temporal lobe seizures suggested by anterior temporal IEDs
Diagnosis of Other Neurological Disorders	Paroxysmal neurological events	IEDs suggest seizures independent of bizarre paroxysmal behavior (especially when found in the frontal region)
	CNS infection	Supports a diagnosis of encephalitis
	Encephalopathy	Supported by diffusely slow background
	Sleep disorder	Presence of IEDs suggest nocturnal seizures versus parasomnia
	Behavioral conditions (e.g., PNES with provocation and normal EEG)	May provide a definitive diagnosis when suggestion during routine EEG provokes a habitual attack to differentiate them from epileptic seizures
	Brain death	May be an indirect confirmatory test

Abbreviations: ASM = antiseizure medication; CNS = central nervous system; EEG = electroencephalogram; GGE = generalized genetic epilepsy; GSW = generalized spike-and-wave; IED = interictal epileptiform discharges; PNES = psychogenic nonepileptic seizures; SSW = slow spike-and-wave.

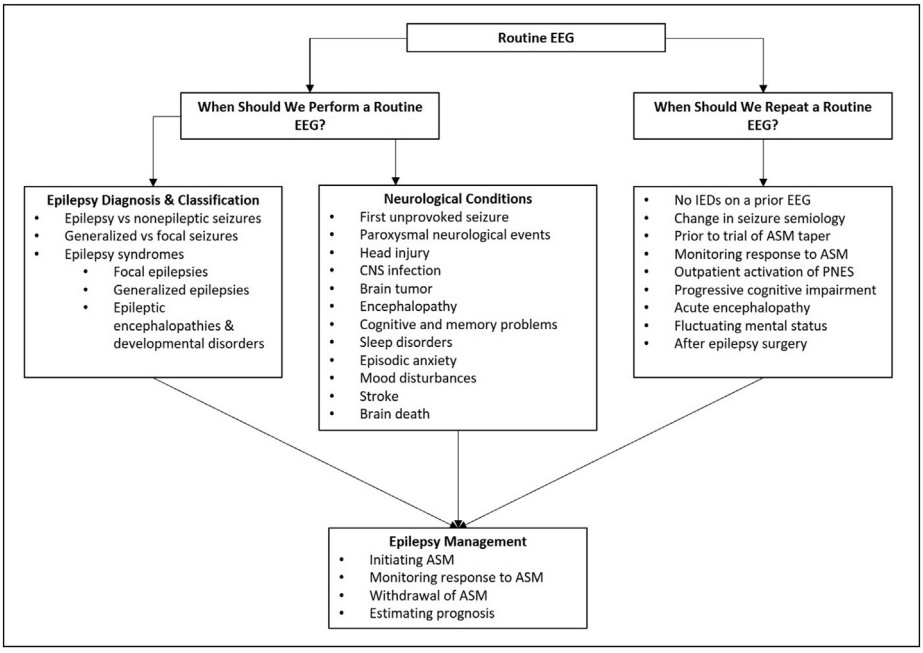

Fig. 1. When should we perform and repeat a routine electroencephalogram?. ASM = antiseizure medication; CNS = central nervous system; EEG = electroencephalogram; IED = interictal epileptiform discharge; PNES = psychogenic nonepileptic seizures. Copyright William O. Tatum.

epileptiform activity when it is obtained during a routine or sleep-deprived EEG. Pretest probability of establishing a diagnosis of sleep-related epilepsies is therefore higher when an EEG is obtained and includes N3 and stage R sleep.

Optimal recording for routine EEG

More than 21 electrodes and a single channel of electrocardiogram are typically used during a routine EEG depending upon the clinical context.[29,30] Silver-silver chloride electrodes are common reusable electrodes using the International 10–20 or 10–10 system of electrode placement. However, gold electrodes have the best properties for recording routine scalp EEG. *Dry electrodes* have become available for clinical EEG used in children $\geq$ 4 years and in adults for faster setup times, remote accessibility, and reduced overhead and time requirements.[31] Special electrodes and montages improve IED detection in focal epilepsies, concentrating on recording from the basal temporal lobe (A1 and A2), anterior temporal lobe (T1 and T2), and electrodes forming an inferior temporal chain (F9/T9/P9 and F10/T10/P10).[32,33] Midline electrodes (e.g., Cz, Fz, or Pz) are favorable for mesial foci deep to the surface of the scalp.

Guidelines recommend the use of at least one longitudinal bipolar, one transverse bipolar, and one referential montage for routine

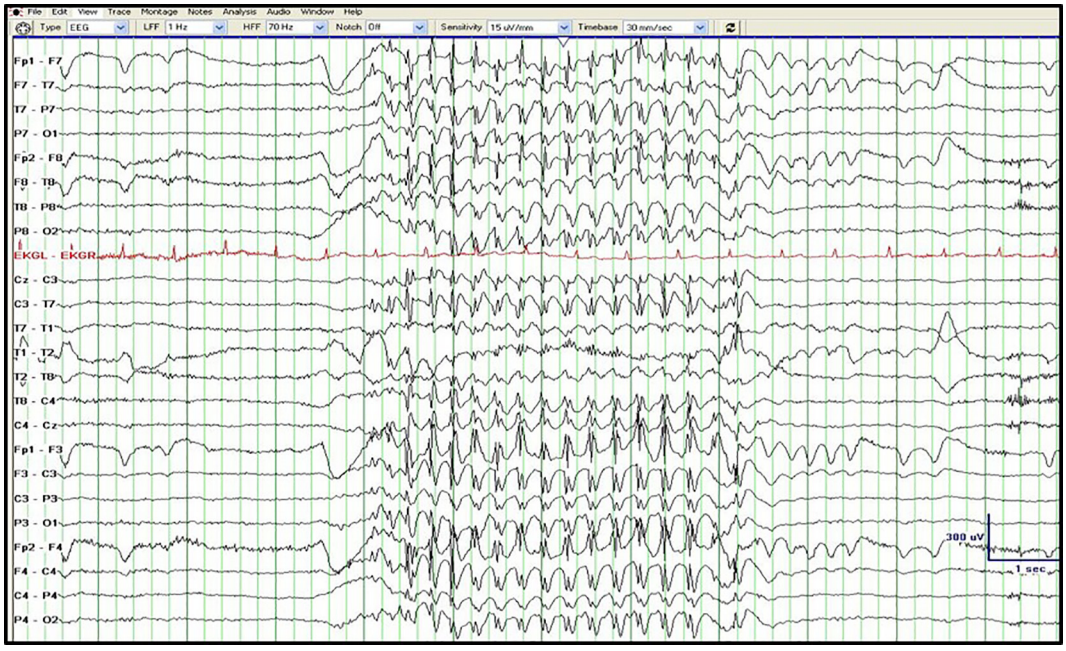

Fig. 2. Electroencephalogram was performed in a 12-year-old female for academic decline. Note the 4 second burst of 4-Hz generalized spike-and-waves. During this time, no clinical signs were observed, though treatment was initiated for juvenile absence epilepsy. Copyright William O. Tatum.

EEGs.[34] Referential montages are useful to identify absolute voltage and the field of distribution of the signal when determining a focal epileptogenic zone. Ear or mastoid references may provide optimal information when frontocentral abnormalities are suspected. However, avoiding their use during sleep will limit contamination from sleep elements. Referential montages unlike bipolar montages do not distort the shape or amplitude of the waveform, however, referential montages may be limited by widespread contamination from intrinsic and extrinsic artifact. Common average references minimize artifact at a single electrode but may reduce the amplitude in the region of interest when many electrodes are involved.[33]

Caps and forms of limited electrode "rapid EEG" are increasingly used during routine recording in the EEG laboratory and in the ICU, [35] with potential application in high-risk patients with COVID-19. Dense arrays up to 256 electrodes (high-density EEG) can aid with source localization.[35]

Source localization

Routine scalp EEG represents the combined electrical activity of billions of neurons, yet only records one-third of the cerebral cortex due to spatial limitations.[2] Certain highly epileptogenic regions may be inaccessible to standard scalp electrodes, including the mesial temporal lobe, and other buried brain regions, including the insular cortex, peri-rolandic areas, the interhemispheric and midline regions, and the basal and deep sulcal areas (e.g., orbitofrontal and opercular areas). The International 10–20 system only detects 65% of IEDs from the temporal lobes using standard temporal electrodes (F7/F8 and T7/T8) overlying the Sylvian fissure and recording from the infra- and supra-sylvian regions.[36] Summated dipoles from multiple neurons are required to become measurable at the scalp as a single dipole source and a combined synchronous cortical source composed of approximately 10^8 neurons is required to generate an IED on scalp EEG.[37] The magnitude reflects the summation of the pooled number and synchronicity of neuronal dipoles.[2] With a purely radial source, the EEG field maxima is placed at a position directly above it. With propagation of electrical activity into adjacent cortical regions, the geometry of the source changes. Scalp EEG requires at least 10 cm^2 or more of synchronized area of neuronal activity for spike detection for a spatial resolution of source localization of 7–10 mm. [38,39]

Activation techniques

Activation techniques during routine EEG include spontaneous sleep and sleep-deprivation in addition to hyperventilation, intermittent photic stimulation, and other personalized stimuli in reflex epilepsies. Many EEG recordings obtained during wakefulness are often contaminated by muscle and movement artifacts. An EEG recording during sleep will demonstrate IEDs in nearly 40% of epilepsy patients, where no IEDs were previously observed during wakefulness.[40] Additionally, nearly all patients with IEDs during daytime nap often have their first discharge within 15–30 minutes of sleep onset.[41] Furthermore, sleep-deprivation may also increase the detection rate of IEDs by 30–70%.[41–43]

Hyperventilation is especially useful to increase the rate of generalized epileptiform discharges in patients with generalized epilepsies, particularly, in absence seizures prior to treatment with ASM.[44,45] Hyperventilation appears to have limited use in focal epilepsies as it only increases the yield of focal IEDs by < 10%. [40,46] Intermittent photic stimulation may induce IEDs in patients with GGE and in those patients with occipital focal epilepsy syndrome.[40,44] Photoparoxysmal response occurs when intermittent photic stimulation generates bilateral synchronous epileptiform discharges. When the photoparoxysmal response outlasts the stimulus by several seconds, this is more often associated with epilepsy than when discharges are self-limited.[47]

Routine EEG and epilepsy diagnosis

The primary aim of a routine EEG is to support the clinician's evaluation with objective data for diagnosis and classification of patients suspected to have epilepsy. Among neurophysiologists, the inter-rater agreement for the visual analysis of an EEG is only

moderate,[4,48,49] with a high inter-rater variability for IEDs. [48,50,51] Specificity of an IED to reflect people with epilepsy is notably high, in contrast to low-moderate sensitivity. However, overinterpretation of benign variants and artifacts on EEG by neurophysiologists may in fact result in low specificity. In a longitudinal cohort study of 521 patients with no history of unprovoked seizures and a follow-up of 230 person-years, 12.3% of these patients demonstrated IEDs on their EEG.[52] Therefore, focal and generalized IEDs may increase the false-positive rate of diagnosing epilepsy.[40,53]

When the clinical history and seizure semiology is unclear, routine EEG may help to classify the seizure type or the epilepsy syndrome to guide appropriate selection of an ASM and predict prognosis. However, EEG has a better predictive value to determine the type of epilepsy in patients with a convincing history of a seizure but a low predictive value to determine if a spell is likely epileptic, at least when it comes to interictal findings. Routine EEG may facilitate a change in ASM treatment through reclassification of an epilepsy syndrome.[2] Further, EEG can define the distribution of epileptiform abnormalities and quantify the frequency of seizure occurrence.[2] Quantification of IED burden is critical for ASM management and can be challenging at times, particularly when patients are not self-aware of their seizures (Fig. 3). In one study, valproate therapy with plasma levels between 50–60 μg/ml was shown to reduce GSW discharges in 19 patients, of which, 11 patients had a reduction of > 75%, thus, reinforcing the impact of ASM treatment.[54] This is important in the context of driving and utilizing heavy machinery where frequent 3-Hz GSW discharges without reported clinical seizures may be associated with a greater likelihood in lapse of awareness and may warrant ASM therapy. [55] In another study, the effect of levetiracetam (LEV) on IED was quantified in 21 patients with generalized epilepsy, in terms of total number, total duration, maximal duration, and median duration of IEDs. Eleven patients received LEV as monotherapy and the reduction of IEDs was highly variable, while those who received LEV as an adjunct therapy demonstrated a significant reduction in IEDs.[56]

When are EEG findings typical for generalized genetic epilepsy?

The prototypic EEG feature of GGE is generalized IEDs.[40,57] However, they may also be seen in epileptic encephalopathies and developmental disorders with or without an underlying structural etiology. In GGE, the IEDs are symmetrical, synchronous, frontally predominant, GSW and generalized polyspike-and-wave (GPSW), recurring at $\geq$ 3 Hz with normal background activity. Discharges may be altered in frequency and morphology by age and stage of sleep.[58] Some features to suggest an epilepsy syndrome are noteworthy. Absence epilepsy is characterized by a typical 3-Hz GSW pattern,[59] while juvenile myoclonic epilepsy more often shows 3–5 Hz GPSW with a diagnostic sensitivity on routine EEG ranging from 54.0-73.3%.[60,61] GPSW may also be seen in patients with generalized tonic-clonic seizures alone, epilepsy with sporadic generalized tonic-clonic seizures, and in patients with absence seizures.[62] Nonetheless, activating procedures and performing the EEG early morning or during N2 sleep can lead to a significant activation of IEDs in GGEs.[44,63,64] The presence of IEDs upon awakening may also be an indirect biomarker for GGE[65] but lacks specificity for distinguishing between specific epilepsy syndromes.

When are EEG findings typical for focal epilepsy?

The most epileptogenic area of the brain is the temporal lobe. [66] The mesial aspect is often distant and inaccessible to routine scalp EEG electrodes. In a study of 300 consecutive patients, initial EEGs only revealed characteristic abnormalities in 44% of the 116 patients and 60% with sleep-deprivation in patients clinically diagnosed with focal epilepsy.[20]

Medial temporal lobe epilepsy is the most common focal epilepsy syndrome,[67] with focal slowing on EEG and temporal intermittent rhythmic delta activity (TIRDA) often ipsilateral to the side of seizure onset. More than 90% of patients have surface-negative anterior temporal spikes or sharp waves that typically predict the side of seizure onset.[68] Further, patients with neocortical

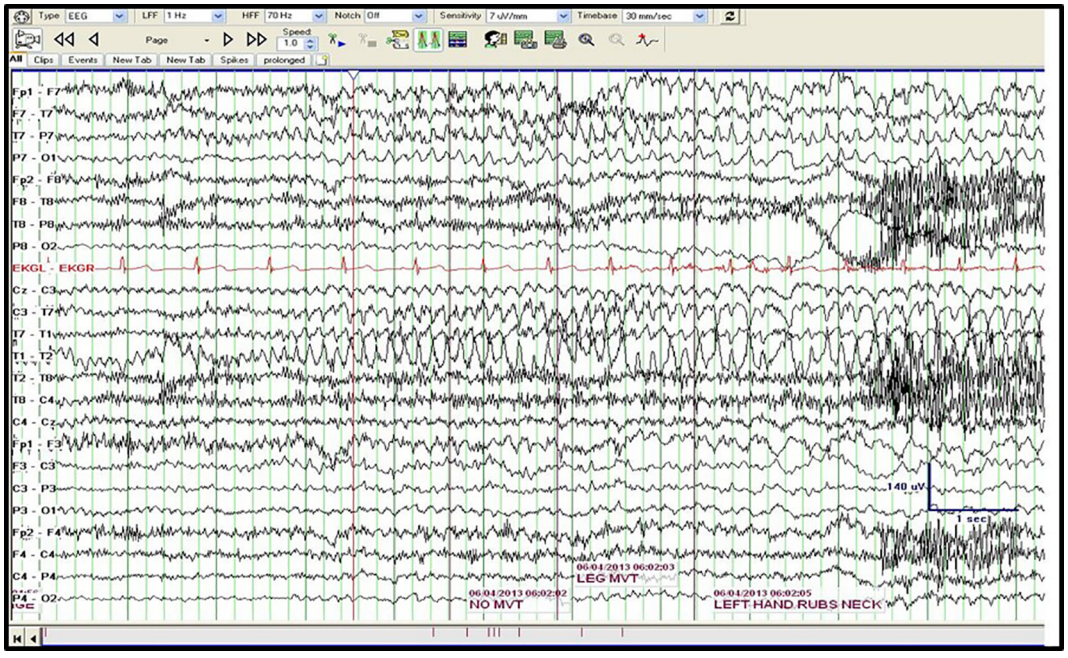

Fig. 3. Routine electroencephalogram was performed in a college student for episodes witnessed by others manifesting as behavior unbefitting her personality. During this time, she would spit but then deny that it occurred. Copyright William O. Tatum.

or lateral temporal lobe epilepsy demonstrate IEDs in the mid-temporal region with a broad spatial field of distribution over the ipsilateral hemisphere.

Frontal lobe epilepsy (FLE) is the second most common focal epilepsy syndrome, but often presents an electrographic challenge for diagnosis as 30% of patients with FLE do not demonstrate IEDs on routine EEG. However, the yield improves on prolonged EEG monitoring. False localization of an extratemporal source such as frontal lobe seizures to the temporal regions may also occur.[69] Generalized IEDs seen in FLE may be due to secondary bilateral synchrony with or without a structural lesion from a lateralized source.[2] Similarly, ictal scalp EEG recordings may be affected by the site of origin in the frontal lobes. For example, signals from distant, mesial, or basal gyri produce IEDs that may be attenuated by long distances and evade scalp detection or low voltage signals may be contaminated by significant muscle artifact, rendering the EEG useless.[70,71] When IEDs are detected, the spatial resolution or discharge localization is often a poor reflection of the source. In one study, localizing IEDs were found in only 12% of patients with FLE, of which the IEDs were lobar in 32%, multilobar in 25%, hemispheric in 9%, bifrontally independent in 9%, and bilaterally synchronous in 37% of patients.[72]

Parietal lobe epilepsy may demonstrate bilateral IEDs that may be falsely localized to the temporal region and even falsely lateralized.[2] Less than 10% of ictal EEGs are well-localized. Similarly, in patients with occipital lobe epilepsy, IEDs may be well-defined in $\leq$ 20% of patients over one occipital lobe, while most are seen bilaterally with a significant majority being falsely localized to the posterior temporal region.[73]

When are EEG findings typical for epileptic encephalopathies and developmental disorders?

Epileptic encephalopathies and developmental disorders often have extensive EEG abnormalities that contribute to cognitive and behavioral impairment beyond of what is expected from the underlying pathology.[74] Epileptic encephalopathies and developmental disorders include a broad range of epilepsies including some that are progressive.[75,76] An interictal EEG demonstrating multifocal independent spike-and-wave discharges, 1.5–2.5 Hz slow spike-and-wave (SSW), generalized paroxysmal fast activity (GPFA), and diffuse slowing of the background activity are defining features of the prototypic epileptic encephalopathy, Lennox-Gastaut Syndrome (LGS). In a study of 64 LGS patients conducted over 42 years, the mean duration of slow spike-waves was 8.2 years.[77] EEG features (and seizures) in LGS tend to evolve over time, especially when transitioning from childhood to adulthood.[78] Additionally, GPFA and generalized bursts of 15–25 Hz or prolonged polyspikes coupled with GSW may be present in some patients with drug resistant GGE.[79] GPFA is frequently present on EEG during non-rapid eye movement sleep, and may be associated with clinical and "subclinical" generalized tonic seizures.

When is routine EEG useful in people with psychogenic nonepileptic seizures?

Routine EEG may be helpful to facilitate a diagnosis of psychogenic nonepileptic seizures (PNES).[80] One study of 74 patients suspected of having PNES on clinical grounds analyzed the yield of short-term, outpatient EEG with video monitoring (OVEM). In 66% of patients, the suspected diagnosis of PNES was confirmed, thereby obviating the need for prolonged inpatient EEG monitoring.[81] Another study also investigated the diagnostic yield of OVEM and retrospectively analyzed 175 OVEM records of adults referred over a period of 5 years. The mean length of recording was 3.8 hours. The highest yield was found in PNES (37.1%), followed by IEDs (17.2%), and epileptic seizures (6.9%). Before OVEM, the provisional diagnosis was epilepsy in 77.7% and PNES in 22.3%. However, after OVEM, the pretest diagnosis was changed in 30.9% of patients. As a result, OVEM with activation can be considered a useful diagnostic test for PNES with a higher yield than in the routine use of EEG for people with epilepsy.[82]

Routine EEG during epilepsy management

I. When should we initiate ASMs after an EEG?

The definition of epilepsy was recently revised by the International League Against Epilepsy (ILAE).[83] Epilepsy may be defined now when a single unprovoked seizure has a $\geq$ 60% likelihood of experiencing recurrence.[83] The presence of any EEG abnormality including focal slowing of the background rhythm may carry a slightly increased risk of seizure recurrence, although the presence of epileptiform discharges is most predictive.[23,84] An EEG with epileptiform activity doubles the risk of seizure recurrence beyond a normal EEG increasing the risk for recurrent seizures to > 60%.[85] Therefore, a routine EEG with IEDs provides electrographic support following a single seizure for a working clinical diagnosis of epilepsy with level A evidence.[18,83] As such, in the appropriate clinical setting, the presence of IEDs on a routine EEG following a single unprovoked seizure has implications for treatment with ASM.

II. When should we repeat a routine EEG?

A repeat routine EEG may be helpful to classify or support a diagnosis of an epilepsy syndrome, especially when the clinical diagnosis is unclear or when the initial EEG has been non-diagnostic (TABLE 2).[18,86,87] However, when the diagnosis has been established, repeat EEGs are unnecessary unless a change in management is in question. Similarly, a routine EEG should not be used to exclude a diagnosis of epilepsy and should be avoided in patients with syncope because of the possibility of an over-interpretation.[88,89]

Repeat routine EEGs have shown to increase the yield of IEDs.[20,50,90] EEG has a relatively low sensitivity (32–59%),[19] though over time, serial routine EEGs increase the sensitivity to include > 90% of people with epilepsy.[91] In population-based studies, the yield for epileptiform abnormalities in people with epilepsy was 53% after the first routine EEG and 72% after the third. Patients with a single unprovoked seizure had a yield of 29% after the first routine EEG and 68% after the third.[19] As a result, the sensitivity in detecting IEDs increases when $\geq$ 3 serial EEGs are performed,[91] or when sleep or sleep-deprivation are utilized.[20,92] The specificity of EEG is superior to its sensitivity and ranges from 78-98%.[53] However, a routine EEG may unfortunately be persistently negative in roughly 10% of people with epilepsy.[93]

Moreover, an ambulatory EEG may be utilized in the outpatient setting instead of serial routine EEGs to enhance the yield of IEDs or when the initial routine EEG is nondiagnostic.[94] In a study of 180 patients undergoing ambulatory EEG, the median latency to the first IED was 316 minutes, where 44% were detected within 4 hours, 58% within 8 hours, 85% within 24 hours, and 95% within 48 hours.[95]

After the diagnosis of epilepsy and initiation of therapy, patients should be followed clinically to monitor the effectiveness of their ASM. A repeat routine EEG is not necessary after achieving seizure freedom unless considering a trial of ASM taper. Evaluating the seizure and spike burden on an EEG are effective longitudinal means to monitor ASM effectiveness.[96]

Table 2
When Should We Repeat a Routine EEG?

Indications		Conclusions
Diagnosis & Classification	Normal EEG	Serial EEGs increase the likelihood of recording an IED
	IEDs on prior EEG	May aid in the classification of IEDs versus seizures to be used for ASM management
	People with epilepsy syndrome (e.g., GGE, absence, or frequent seizures)	Clarification of the diagnosis and classification of epilepsy syndromes
	Behavioral disorders and those with impaired communication	May be used to differentiate seizures from cognitive impairment when IEDs are captured and/or psychogenic nonepileptic seizures from epileptic seizures if events are captured during provocation
Management	Considering a trial of ASM taper in patients who are prolonged seizure-free	IEDs suggests a greater risk for seizure recurrence
	Monitoring ASM response and effectiveness	The IED/seizure burden will suggest adjustment to the ASM regimen in effort to obtain better seizure control
	Hospitalized patients with change in mental status or spells	May suggest unrecognized seizures or subclinical seizures and warrant ASMs
	Change in seizure semiology	Facilitates identifying whether a change in prevalence, frequency, or duration of IEDs may influence ASM management
	After epilepsy surgery	IEDs suggest greater likelihood of seizure recurrence and further need for ASMs

Abbreviations: ASM = antiseizure medication; EEG = electroencephalogram; GGE = generalized genetic epilepsy; IED = interictal epileptiform discharges.

Repeat EEG in critically ill patients should be sought when there is high suspicion for NCSE. NCSE is a challenging condition, and the diagnosis is entirely supported by an EEG. NCSE may be seen in up to 20% of the critically ill patients.[2] In the medical ICU, sepsis is more frequently associated with seizures and periodic discharges than compared to other conditions.[97] In a prospective study, after controlling the initial convulsive status epilepticus, 48% of patients continued having electrographic seizures while 14% had NCSE.[98] In an investigation involving 570 critically ill adults, seizures were detected in 19% of patients, of which, 56% had their first event within one hour of initiating the continuous EEG, increasing to 82%, 88%, and 93% at 12, 24, and 48 hour, respectively.[99] A recent multicenter randomized clinical trial of critically ill adults with impaired consciousness but no recent seizures demonstrated that continuous EEG led to increased seizure detection and modification of ASM but was not related to improved outcome when compared to repeat routine EEG.[100] The use of repeat routine EEGs for the detection of NCSE may be relevant at hospitals with limited resources.

III. When does a routine EEG help judge ASM timing for withdrawal and estimate prognosis?

Patients who experience prolonged seizure freedom (1–2 years for children; 2–5 years for adults) are considered for ASM withdrawal.[101–104] Similarly, patients who have undergone epilepsy surgery may also be considered for ASM withdrawal after 2 years of seizure freedom.[105–108] The AAN identified several factors that suggested a greater chance of ASM withdrawal without risk of relapse: 1) seizure freedom of 2–5 years on ASMs; 2) single seizure type; 3) normal neurological examination and intelligence quotient; and 4) normal EEG with treatment.[103] Similarly, the ILAE has also provided evidence-based guidelines to help neurologists with their decision to withdraw ASMs in seizure-free patients.[109]

The likelihood of seizure recurrence increases when epileptiform abnormalities appear on EEG, when worsening EEG patterns after ASM withdrawal are present, and when specific EEG patterns such as GPSW are encountered.[104,110,111] Similarly, withdrawal of valproate was found to carry the worst prognosis for relapse.[112] When performing a routine EEG, especially after withdrawal of valproate which acts as a spike suppressant, the appearance of GSW dictates a high risk of seizure recurrence and should prompt re-initiation of ASMs. However, suppression of generalized epileptiform discharges may be apparent up to 3 months after discontinuation of valproate therapy.[113,114] Therefore, a repeat routine EEG 3 months after the valproate withdrawal period may be necessary to reevaluate the presence of GSW and influence decision-making for ASM discontinuation.[113,114]

Several studies have shown that epileptiform EEG abnormalities at the time of ASM withdrawal confer a higher risk for relapse, emphasizing the role of an EEG in predicting seizure recurrences.[112,115] In one study of young adults with focal epilepsy who were seizure-free for > 2 years and $\leq$ 6 years, EEG performed at 3, 12, 24, and 36 months predicted seizure recurrences during the ASM withdrawal but not when treatment was maintained.[116] As a result, 63% of patients relapsed within 3 years from complete ASM withdrawal, while 24% relapsed during the ASM withdrawal period.[116] One prospective study found that an abnormal EEG prior to ASM withdrawal in people with epilepsy carried a poorer prognosis for relapse shortly after 2 years of seizure freedom.[112]

While there is no consensus on when and how fast to taper ASMs in seizure-free patients, slowly tapering and avoiding abrupt discontinuation of ASMs is recommended during the withdrawal period.[110] Different rates of ASM withdrawal have been suggested with no difference in seizure recurrence including 4 weeks, 6 weeks, 3 months, 9 months, and 1 year.[117–119] Nonetheless, recurrence of seizures seem to occur relatively soon after ASM withdrawal, with nearly half of the recurrences occurring within < 6 months.[110]

When do pitfalls occur following routine EEG?

Some traps and pitfalls in obtaining a routine EEG include lost time, lack of a specific question or reason for the study, expense, and the potential for misinterpretation.[120–122] It is essential for interpreters to understand that a normal EEG does not rule out epilepsy. In addition, an abnormal EEG may not necessarily correlate with clinical seizures. The clinical correlation depends upon the context of the individual patient situation.[123]

Misinterpretation of benign variants and artifacts as abnormality can result in misdiagnosis of epilepsy.[122,124,125] Nearly 30% of patients presenting to an epilepsy center for drug-resistant seizures in fact do not have seizures and are misdiagnosed.[126] An "abnormal" EEG may serve as the rationale, even though their clinical history may suggest otherwise.[122,127–132] The underlying reason for overinterpretation of EEGs is a lack of training, inexpe-

rience, and not applying strict criteria when interpreting waveforms.[120] The less experience one has interpreting EEGs, the lower the threshold for over-interpreting an abnormality.[120] Of note, when a patient presents with nonspecific symptoms or has an "equivocal" EEG, the interpretation should be conservative to avoid an inappropriate diagnosis of epilepsy.[122,129–132] Interpreters may be unconsciously biased by the clinical history if the patient had a seizure, or because the technologist's impression suggested epileptiform activity.[120] A temporal location and "phase reversals" are common substrates for misinterpreted EEG mimicking abnormal epileptiform discharges.[122,131,132] Therefore, a more conservative approach to EEG interpretation is recommended.[133] Enhancing neurology residency training with mandatory rotations in clinical neurophysiology that encompasses EEG education should be a requirement.[120]

Conclusion

Routine EEG remains an essential bedside test for people with neurological disorders but is especially useful for those with seizures and epilepsy. It can define epilepsy after a first unprovoked seizure and aid in the initiation and withdrawal of ASMs. A repeat routine EEG has shown promise as an alternative to continuous EEG in critically ill adults at centers with limited resources. Despite technological advances in the field of clinical neurophysiology and the occurrence of a viral pandemic, temporarily reducing access, routine EEG will continue to occupy a central role in the assessment of neurological function.

Ethical statement

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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