

# EEG interpretation: common problems

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## Practice Points

- Certain waveforms in the EEG are 'epileptiform' in appearance and mimic pathological discharges, but are physiologic potentials.
- Pathological epileptiform discharges have a high specificity for seizures but may be mimicked by variations and variants of normal and by artifact.
- The presence of pathological epileptiform discharges in the EEG supports the clinical diagnosis of epilepsy while recording seizures is definitive.
- Misinterpretation of the routine scalp EEG is due to inherent subjectivity and is based upon limited training and experience.
- A normal routine EEG does not exclude a diagnosis of epilepsy because of the low-to-moderate sensitivity in demonstrating epileptiform discharges.
- Patients with an 'abnormal' EEG and a clinical diagnosis that is atypical for epilepsy should be evaluated by a seizure specialist for treatment.
- The proper treatment of patients with epilepsy is based on a correct clinical diagnosis of epileptic seizures and not based on interictal EEG.
- Video-EEG monitoring is the gold standard in defining the clinical diagnosis when it is necessary to distinguish epilepsy from nonepilepsy, classify seizure types or when selecting patients for neurosurgical treatment.

**SUMMARY** An EEG that contains epileptiform discharges has a high specificity in supporting the clinical diagnosis of epilepsy. The ability to correctly identify and distinguish a pathological epileptiform discharge is a skill based upon the subjective and qualitative nature of EEG. There are common pitfalls that exist when interpreting the EEG. Physiological aspects noted during drowsiness, normal variants and artifact can mimic interictal epileptiform activity and challenge the EEG interpreter. A clinician that lacks exposure to EEG during residency training or interprets with a known clinical bias is at risk for overinterpreting the EEG. The rami-

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fications of this may be enormous. The incorrect identification of interictal epileptiform activity may lead to a misdiagnosis of epilepsy and subsequently to the risks of antiepileptic drug treatment. Recognizing and understanding the common traps and pitfalls associated with misinterpretation will underscore the importance of a conservative approach to reading EEG and serve as a means of reducing error that may impair correct interpretation.

By conservative estimates, 2.3 million Americans have epilepsy [1]. The EEG has revolutionized the study and treatment of epileptic seizures and syndromes [2]. The objective support it provides remains key in the diagnosis of epilepsy. It assists antiepileptic drug (AED) selection by classifying seizures and syndromes [3]. It may help predict recurrence after a first seizure [4], and relapse after prolonged seizure freedom [5]. EEG is a quality indicator for physicians who treat patients with epilepsy [6].

The EEG is misused when obtained to prove or rule out the diagnosis of epilepsy [7]. The foundation for the diagnosis of epilepsy and epilepsy syndromes remains a clinical one. Overreliance on EEG is perhaps the greatest pitfall leading to improper diagnosis and treatment, especially when the EEG is interpreted out of the clinical context [8].

It is incumbent upon the interpreter to distinguish features in the EEG that are pathological from those due to physiological causes, artifact or normal variants.

#### Pathological epileptiform discharges

Epileptiform discharges (EDs) rarely occur in patients who do not experience epileptic seizures [9,10]. In a study of 3726 children without epilepsy,

3.5% had focal or generalized interictal EDs that disappeared by early adolescence [11]. Most EDs in the absence of clinical epilepsy consists of centrottemporal spikes, generalized spike-and-wave patterns, and a photoparoxysmal response with higher yields noted depending upon certain features (Box 1) [9]. The prevalence in adults is lower than children although it may be seen in people without a history of seizures (Box 2) [12].

The definition of an ED and the means of identifying them are not homogeneous nor are they standardized (Figure 1). Identifying ED is based upon subjective interpretation. Therefore, a quantitative and objective means to reliably define apart from nonepileptiform waveforms with comparable changes in waveform location, amplitude, frequency, duration and morphology is lacking. Hence, the definitions to validate an abnormal finding and distinguish it from normal are limited. Criteria have been previously suggested to identify an ED [13]. Guidelines exist on how an EEG is to be performed [101], although no guidelines exist on interpretation of an EEG. Currently, studies on inter-rater reliability of how to interpret the EEG are scarce, or focus on aspects other than EDs. Considerable inter-reader variability exists even among experts [14]. The principles of recording EEG have been greatly advanced with the evolution to digital recording. Newer EEG systems are able to make modifications electronically with changes in montage, filter settings and sensitivity that help interpreters avoid common problems. Computer-based spike-detectors aid in recognizing 'suspicious' transients, but suffer from over-identification compared with human interpreters [15]. Neural networks may become useful to judge candidate waveforms as EDs but still suffer from initial expert opinion that identifies the patterning of the computer [16].

#### Physiological activity mimicking pathological EDs

Many normal physiological frequencies in the EEG possess features that mimic EDs and may prompt confusion [17]. The limited representation

#### Box 1. Features producing a higher yield of interictal epileptiform activity detection on scalp EEG.

- Age (younger)
- Location (temporal)
- Sleep
- Special electrodes and greater number of electrodes
- Performed within 24 h after a seizure
- Syndromes (i.e., Lennox–Gastaut syndrome and TLE)
- Antiepileptic drugs (i.e., VPA with lower recovery)
- Greater number of recordings
- Longer duration of recordings

TLE: Temporal lobe epilepsy; VPA: Sodium valproate. Adapted from [9].

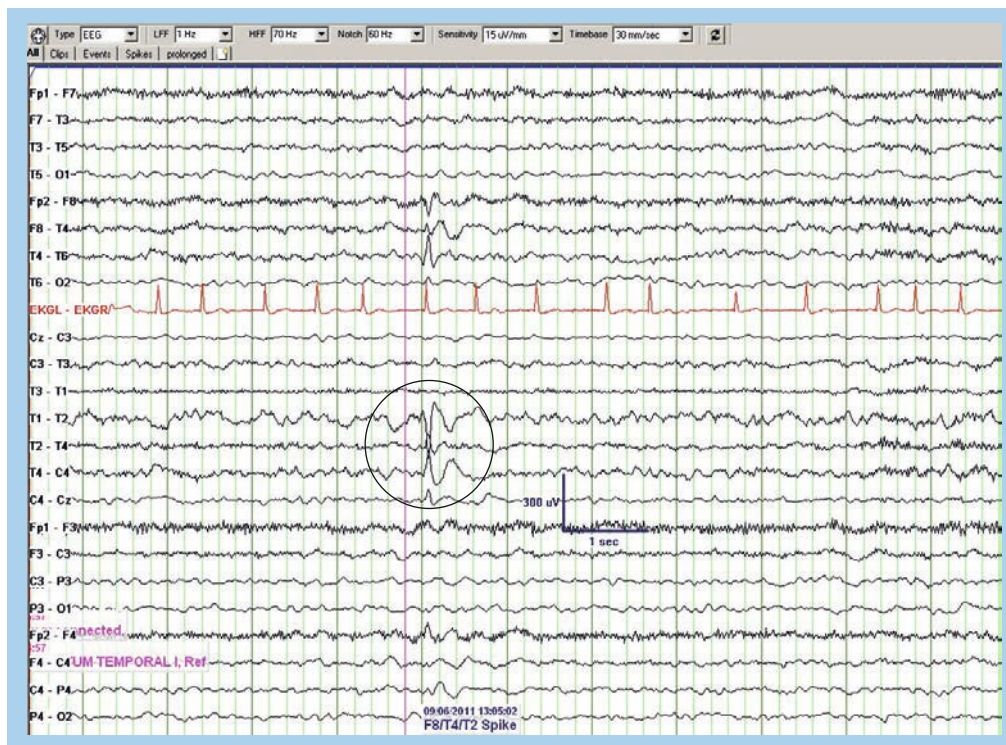
of waveforms by frequency, duration and amplitude gives rise to the same waveform or bandwidth being normal in one setting and abnormal in another (i.e., alpha coma). Some normal waveforms in the EEG are 'epileptiform' by morphological appearance but do not reflect a pathological ED. One example is vertex 'sharp' transients, which are 'epileptiform' by appearance (some even appear 'spiky') although they reflect normal physiological features of sleep (and not epilepsy). Spikes and sharp waves may be normal if morphology is the only criterion used to determine them. Spiky vertex is normal in children (Figure 2) and 'spike-driving' is a normal response to intermittent photic stimulation at low flash frequencies (Figure 3). Vertex sharp transients are epileptiform yet are a normal physiologic aspect of stage 1 sleep. The appearance of v-waves in the EEG is usually not difficult when other components of sleep are present. However, a spiky morphology or use of an unfamiliar montage may produce a contrast and highlight the waveform to allow it to stand out from the background and lead to overidentification. The rationale for attributing abnormal characteristics to the EEG because 'it just looks that way' is a trap associated

**Box 2. Conditions where interictal epileptiform activity is reported to have been increased without epileptic seizures.**

- Autistic spectrum disorder
- Attention deficit disorder
- Cerebral palsy
- Neurobehavioral disorders
- Blindness
- Following head trauma
- Preschool/employment screen
- Dementia
- Associated with electroconvulsive therapy/psychiatry
- Drugs (i.e., clozapine, lithium, tricyclics and alcohol)
- Syncope
- Psychogenic nonepileptic attacks

Adapted from [12].

with pattern recognition [18]. The caveat is that when pattern recognition alone is the foundation for abnormality it serves as a potential pitfall for correct differentiation of waveforms as EDs. This concept applies to electrical fields in the EEG and underlies the foundation for correct interpretation [19,20].



**Figure 1. Right temporal spike-and-wave (circle) in a patient with focal seizures with dyscognitive features. Note the concomitant focal slowing that occurs in the right temporal region.**





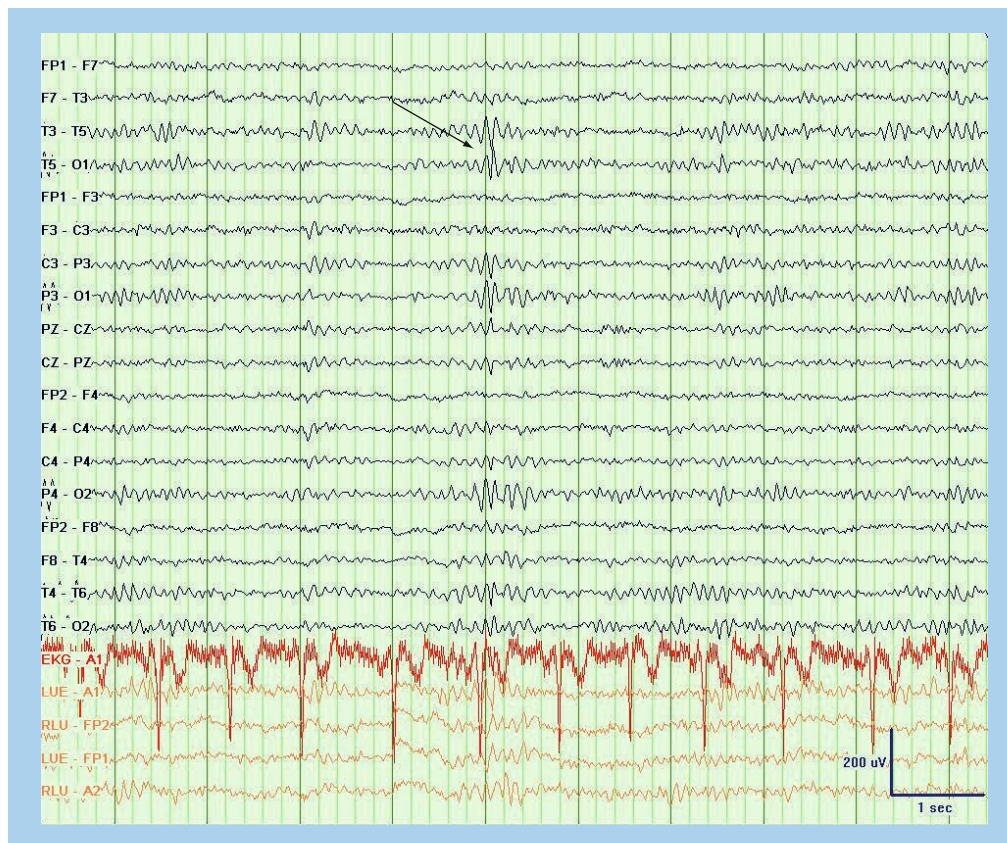
**Figure 2. Spike-driving during low-frequency photic stimulation in a normal individual referred for episodes of altered awareness (arrows).**

Another pitfall of interpretation is an over-emphasis on phase reversals [21]. The site of a phase reversal in a bipolar montage signifies only maximal electronegativity (or positivity) independent of whether it is a normal or abnormal feature of the EEG. Drowsiness tends to produce normal paroxysmal features [17,22]. Sharp

transients may be identified in more than 90% of healthy adults when they are drowsy, with 'sharp' waves often appearing in the temporal and frontal regions [23]. One study found normal fluctuations of the alpha rhythm (Figure 4) to be the most common reason for misidentifying EDs on the EEG, most of which were identified



**Figure 3. 'Spiky' vertex in a 15-year-old patient with seizures. Note the phase-reversal and 'spike' identified by the computer software (oval). Reproduced with permission from [25].**



**Figure 4. Normal alpha rhythm that has phase reversals, which appear more prominent at T5 (arrow).**

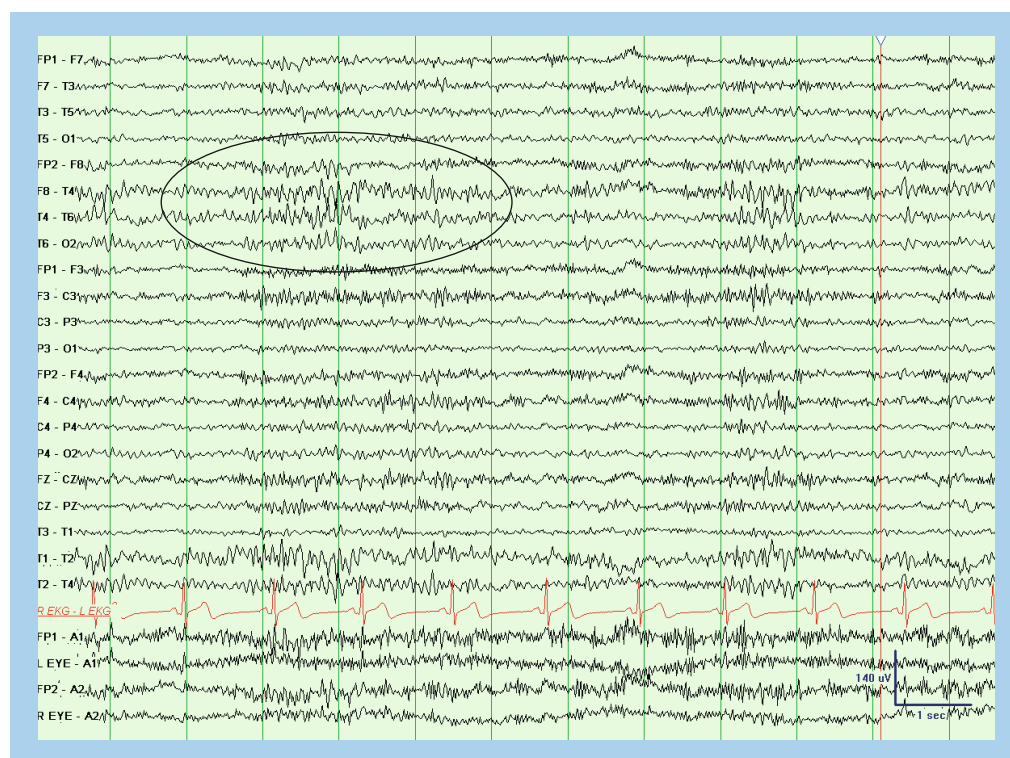
during drowsiness. In this series of patients with video-EEG-documented psychogenic nonepileptic seizures, 41 out of 127 (32%) patients had a history of an ‘abnormal’ EEG erroneously described as containing EDs [8]. When a misinterpreted EEG results in a misdiagnosis of epilepsy, the risks from AED treatment as well as driving and other social restrictions will be instituted. Variations in the condition of recording the EEG may also create confusion. Skull defects that occur often following craniotomy enhance beta activity under the site of ‘breach’ (Figure 5). These waveforms may contain prominent phase-reversals of all underlying activity that can attract the reader’s eye. Due to the higher amplitude rhythm with over-riding beta activity, the appearance may resemble an ED and lead to misinterpretation. Still more worrisome is the potential for abnormal physiological waveforms to be misinterpreted as an ictal manifestation. When clinical symptoms are present that suggest a behavioral seizure, the EEG interpretation should be supplemented with an electroclinical correlation, but if nonconvulsive seizures are

encountered then treatment including iatrogenic coma relies solely on the interpretation of the EEG [24].

#### Normal variant EDs

Patterns that are epileptiform or rhythmic are often features associated with an abnormal EEG in patients with epilepsy. The patterns of uncertain significance (or normal variants) may possess these same characteristics and constitute a potential pitfall for those interpreting the EEG [25]. Beyond finite waveforms that reflect the normal variants, there are normal variations and fluctuations in the routine scalp EEG over time [26]. In addition, many normal variants and variations have a predilection for the temporal lobe and appear to be most prevalent in drowsiness [17]. When they are a normal variant, they will disappear during slow-wave sleep. This can be a helpful method in distinguishing pathological ED (that persists), from normal variants that disappear during deeper stages of sleep that may be obtained with prolonged EEG recording. Most physicians that interpret EEG have a clinical





**Figure 5. Right midtemporal breach rhythm during drowsiness with phase reversals at T4 and superimposed beta activity that gives the false appearance of repetitive epileptiform discharges. Reproduced with permission from [25].**

background. Most patients (~60%) have localization-related epilepsy and focal seizures [27]. Of those with focal seizures, temporal lobe epilepsy comprises the bulk of patients and this region in the EEG is also the location that is most subject to misinterpretation [28,29]. Most of the normal variants have the temporal lobe as the site of maximal expression and less often involve the frontal or occipital location. Benign epileptiform transients of sleep (Figure 6) and sharply contoured rhythmic midtemporal theta bursts of drowsiness appear maximal in the temporal derivations and mimic EDs [30]. If bursts of rhythmic midtemporal theta occur in drowsiness, and repeat with a frequency that is less than 5 Hz, the interpreter should be suspicious that these do not reflect a normal variant. 14- and 6-Hz positive bursts appear as polyspike discharges in the posterior temporal derivations, but have a positive polarity. The 6-Hz spike-and-waves are epileptiform and reflect a generalized pattern that is thought to be a benign variant of uncertain significance in most cases [17,30]. Wicket waves are temporal waveforms that mimic EDs and the most commonly

misread normal variant (Figure 7). In one study 25 out of 46 (54%) of patients referred for epilepsy had wicket waves interpreted as EDs often appearing as fragmented discharges in the temporal regions [29]. Wicket waves usually appear in brief trains but may be isolated and give the appearance of an ED. The difference is that these sharply contoured waveforms, at times appearing as isolated wicket spikes, are recorded over the temporal regions during drowsiness and are usually symmetrical in their rate of rise and fall. Additionally, they appear identical in frequency to similar waveforms in the bursts, do not have an aftergoing slow wave and are not associated with other abnormal focal features. This normal variant closely mimics an ED and further underscores the need for a conservative approach to interpretation of the EEG. It is rare to record a seizure during a routine EEG; however, sometimes physiological features may be misinterpreted as seizures [31]. Also rare is a unique normal variant known as subclinical rhythmical EEG discharges of adults that takes on the appearance of an electrographic seizure [17].

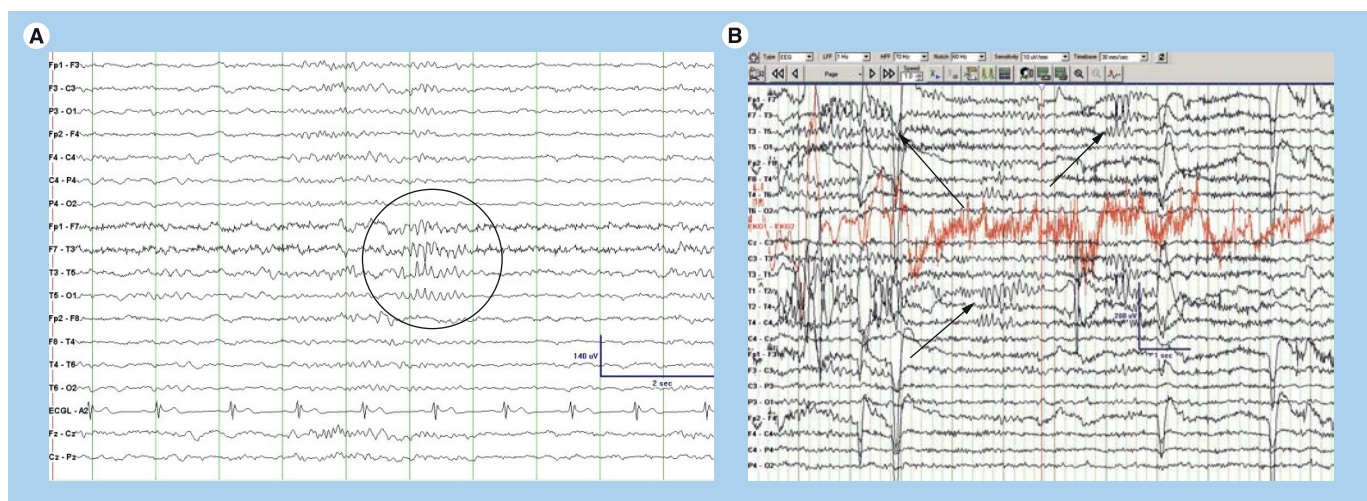


**Figure 6.** Left temporal benign epileptiform transient (arrow) during drowsiness in a patient with headache and episodes of tingling.

### Artifact

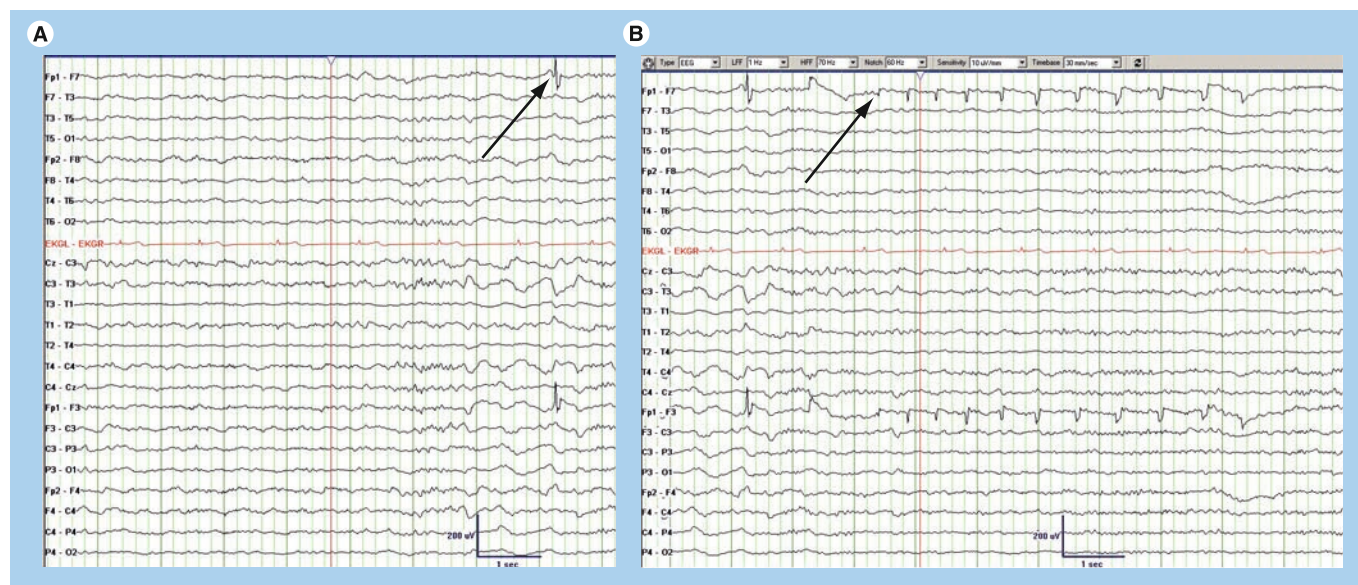
Artifact can mimic almost any type of electrocerebral activity on the EEG, including focal and generalized EDs, leading to confusion [18]. The most common artifacts are due to an insecure connection between the electrodes and the

machine (Figure 8). Biological artifacts arising from the patient are contained in essentially every routine study and are a necessary part of the recording (e.g., eye movement artifact and sleep staging); however, some artifacts serve as ‘contaminants’[32]. Many artifacts exist that challenge the interpreter, including interictal and



**Figure 7.** Interictal EEG demonstrating midtemporal wicket waves and the third rhythm in an epileptic patient. (A) A burst of left midtemporal wicket waves (circle) that simulates polyspikes. (B) The third rhythm (arrows) in a patient treated for epilepsy based upon this EEG. Neither of these patients had epilepsy.

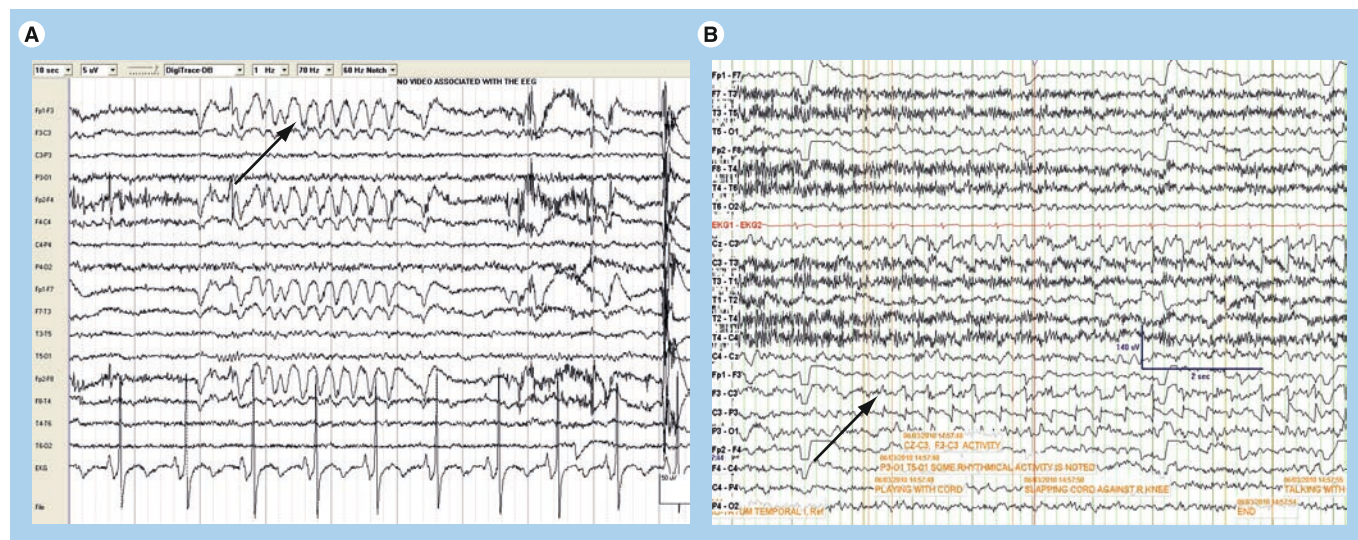




**Figure 8.** Interictal EEG showing a focal FP1 spike and the isolation in a single electrode. Focal FP1 spike in (A) that was clarified later in the recording as a single electrode artifact (arrow). (B) Note the isolation in a single electrode by the lack of a discernible field and the change in polarity in (arrow).

ictal mimics [33]. As medical care becomes more complex and technology advances, newer types of artifacts become apparent [34]. Nevertheless, common artifacts such as myogenic artifact generated by the frontalis (Figure 9A) or temporalis muscles may produce focal or generalized muscle spikes or even polyspikes, serving as a source for misinterpretation of the EEG [102].

Artifacts in the EEG can be particularly challenging when no annotation by the technologist or ability for video review is available to support an artifact. With artifact, intermittency is the rule, and regularity the clue, regardless of the morphology [18]. There are many unique and complex artifacts (Figure 9B) that may jeopardize correct interpretation of the EEG, especially with



**Figure 9.** Misinterpreted EEG with pseudo-generalized spike-and-waves and EEG 'pseudoseizure'. (A) Misinterpreted EEG with pseudo-generalized spike-and-waves due to superimposition of vertical eye blink artifact and myogenic 'spikes' (arrow). (B) Note the EEG 'pseudoseizure' due to repetitive electrode artifact at C3 (arrow) and the adjacent double and triple phase reversals indicative of artifact.



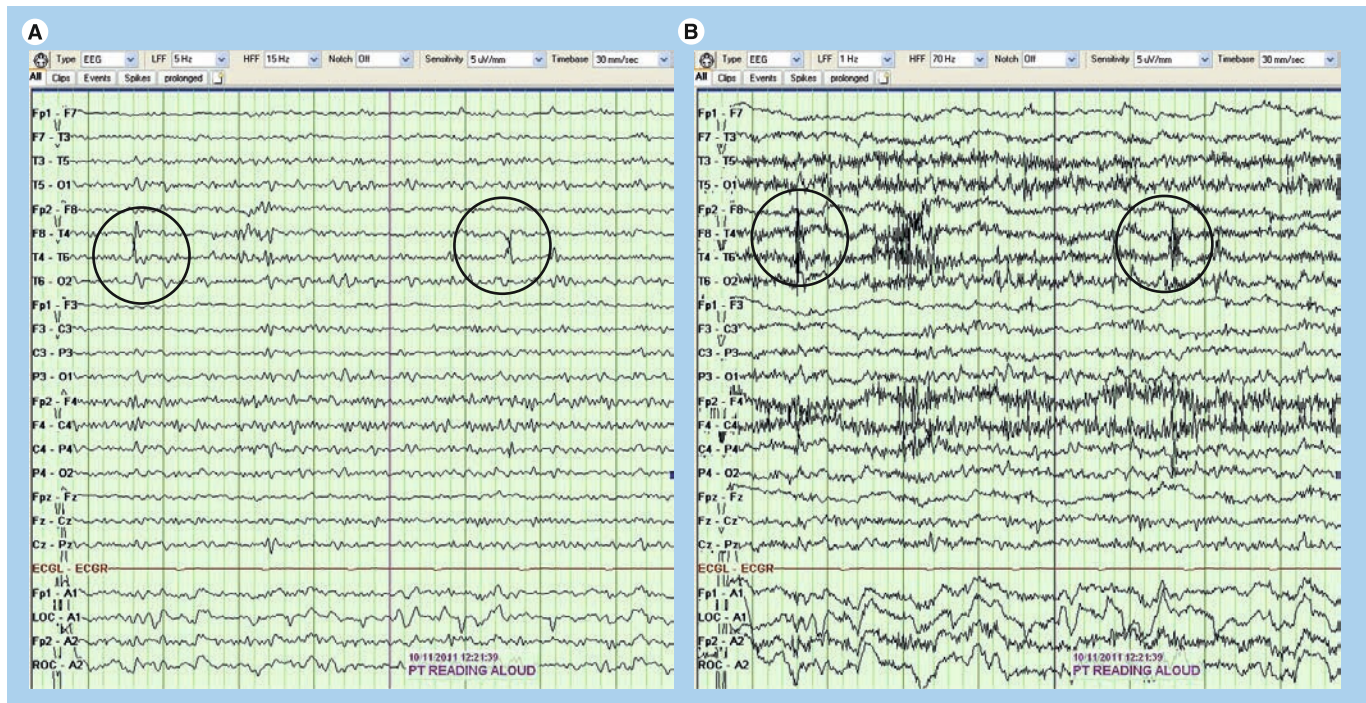
prolonged recordings or in an electrically 'hostile' environment such as the intensive care unit. The means to judge the cerebral–extracerebral mismatch is based upon a believable localization, polarity and field. Even in the course of routine EEG recording, the use of *post-hoc* analyses that were impossible with prior analogue recordings have led to improvement in recognition of cerebral activity. When appropriately used, *post-hoc* filtering can help eliminate the source of electrical 'interference' and clarify the EEG for correct interpretation. Similarly, modifying the sensitivity will optimize the amplitude to facilitate correct interpretation of the EEG. When inappropriately utilized however, these *post-hoc* methods may also act as a pitfall (Figure 10). When in doubt, it is incumbent upon the EEG interpreter to assume that the source is artifact until proven otherwise.

### Case report

A 27-year-old female had a single febrile seizure as a child. Besides depression, she had no significant past medical history. At age 17 she began to develop staring spells. Her husband noted that she would sometimes stare out into space for

up to 45 min. During this time she would not interact with him and though she was able to talk, the speech was curt and did not maintain active conversation. Her primary care physician thought she was depressed and prescribed sertraline. The episodes continued and she was referred to neurology. A brain MRI was normal, but an EEG recorded "clear left temporal sharp waves" (Figure 11). She was prescribed carbamazepine, but the episodes continued on a weekly basis without response. Topiramate was added to her treatment and the episodes worsened. She was implanted with the vagus nerve stimulator. She improved by 25% and further drug manipulation resulted in incomplete improvement. The patient complained of difficulty tolerating her medication and she was referred by her primary care physician for another opinion.

On evaluation, a single uncomplicated febrile seizure occurred at 2 years of age without additional risk factors for epilepsy. The semiology did not suggest focal seizures due to an atypical clinical course and prolonged episodes. During in-patient video-EEG monitoring, multiple typical events were captured after AEDs were discontinued.



**Figure 10. A misinterpreted EEG interpreted as an 'abnormal' recording. (A)** Demonstrates right temporal spikes in a patient treated for drug-resistant epilepsy. The 'right temporal spikes' (circles) were due to inappropriate filter settings of 5–15 Hz. Psychogenic nonepileptic seizures were later diagnosed by video EEG. **(B)** The use of appropriate filter settings demonstrated similar waveforms due to myogenic artifact.



**Figure 11. EEG demonstrating left temporal ‘spikes’ (circles) in a patient refractory to two antiepileptic drugs and the vagus nerve stimulator. She was subsequently diagnosed with nonepileptic seizures and is now off all antiepileptic drugs, had the vagus nerve stimulator explanted and remains event-free and is driving. Her ‘abnormal’ EEG was unable to be recovered.**

No scalp ictal EEG changes were evident and the semiology suggested nonepileptic staring. She was released with a tapering schedule for her AEDs and later had the vagus nerve stimulator explanted. She has remained event-free for 2 years off AEDs.

The EEG during video-EEG demonstrated left temporal wicket waves. After obtaining the outpatient EEG the morphologies and findings were the same. While the EEG was incorrectly interpreted as abnormal due to ED when it was normal with a normal variant, the profound effect that the diagnosis of epilepsy had upon the patient’s life with loss of driving privileges, compromised employment, side effects from treatment and overall impairment in the quality of life were so significant that she was thrilled with the news. However, a delay in the true diagnosis may be costly and prolonged. Similar to our patient, the delays may extend up to 7–10 years before a definitive diagnosis is finally made [35].

#### Future perspective

Currently, many perils and pitfalls exist during visual interpretation of the EEG in clinical

practice [28]. The method for most interpretation is based upon subjective visual analysis that relies upon qualitative waveform pattern recognition alone. The problem has been clarified by video-EEG monitoring that has revealed an epilepsy misdiagnosis rate of approximately 25–30% of hospitalized patients that may be predicated upon a misread EEG [8,29,31,33,36,37].

The potential problem of the EEG being interpreted out of context will remain despite advances in education and technology. In one study of outpatient EEG interpretation, multiple unexplained symptoms were the most common reason identified in one study that led to the misinterpretation of an EEG with EDs [36]. In the EEGs that were recovered, 30 out of 37 had temporal waveforms that were misinterpreted as EDs. It is, therefore, essential that the interpretation of the EEG is conservative given that there is more harm done by an EEG misinterpreted as abnormal, than by one misinterpreted as normal [8,17,25,36].

In the future, identifying common errors of EEG interpretation and reinforcing the electrical concepts of localization and fields in formal



neurology education will ensure greater skill. The accuracy of a qualitative assessment of EEG fluctuates from person to person, but also in the same person over time [37]. The digital age of EEG technology now allows for 'cut-and-paste' examples of 'borderline' EEG features to be included in reporting for the clinician to judge interpretation reliability. We may see this with greater frequency in the future. Transmission of EEG for second opinions may grow in popularity for a 'low-tech' validation of an EEG interpretation. This is similar to cardiologists overreading ECGs, which is routinely performed and has been suggested by others [36]. In a survey of attendees at a clinical neurophysiology meeting, 96% of respondents felt overinterpretation was the reason for misread EEGs [38]. A second opinion with a board-certified clinical neurophysiologist would provide support for an ancillary study that holds such strong implications for treatment in patients with recurrent spells. When controversial epileptiform waveforms are encountered, a conservative approach is warranted. For diagnostic purposes, we use the 2 min rule: if after 2 min following review of the EEG a 'discharge' is unable to be clearly categorized as an ED, then a conservative interpretation should apply and the waveform interpreted as nonepileptiform. Apparent and true ED may be encountered during EEG interpretation, although this should not be equated with an automatic clinical association

with epilepsy [39]. Reporting reliability is suggested when a confident description of ED distribution, morphology, frequency, duration and field is rendered. If the report appears confusing to the clinician, then experience and reliability of the one interpreting the EEG may be questioned. This is especially true when the report is at odds with the clinical state of the patient. Future perspectives will include improved development of 'high-tech' methods to identify waveforms more appropriately for the electroencephalographer. However, this requires more work. Greater definition of what constitutes a clear ED with high reliability are needed. Development of software with sensitivity and specificity for the varieties of waveforms that are epileptiform will need to be enhanced. The area of computer identification of EDs is evolving [14] and we are closing the gaps that limit our abilities to identify EDs beyond duration alone [17].

#### Financial & competing interests disclosure

*The author has no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.*

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