**INTRODUCTION** — The diagnosis of epilepsy is often not straightforward, and misdiagnosis is not rare [1]. A detailed and reliable account of the event by an eyewitness is the most important part of the diagnostic evaluation, but may not be available [2].

Electroencephalography (EEG) is an important diagnostic test in evaluating a patient with possible epilepsy. It can provide support for the diagnosis of epilepsy and also assists in classifying the underlying epileptic syndrome.

However, there are several reasons why EEG alone cannot be used to make or refute a specific diagnosis of epilepsy:

- While many EEG patterns are recognizable, most patterns can be caused by a wide variety of different neurologic diseases.

- Many diseases can cause more than one type of EEG pattern.

- Intermittent EEG changes, including interictal epileptiform discharges, may be sufficiently infrequent so as to not appear during the relatively brief period of routine EEG recording.

- The EEG can be abnormal in some persons with no other evidence of disease.

- Not all cases of brain disease are associated with an EEG abnormality, particularly if the pathology is small, chronic, or located deep in the brain.

In order to make the best clinical use of EEG in the evaluation of patients with possible epilepsy, it is critical for the clinician to understand the strength and weakness of EEG, specifically as it relates to the diagnosis of seizures and epilepsy.

This topic discusses the use of EEG in the diagnosis of seizures and epilepsy. A more general discussion of EEG, and the use of other diagnostic tests in the evaluation of patients with seizures and epilepsy are presented separately. (See "Clinical neurophysiology", section on 'Electroencephalogram' and "Video and ambulatory EEG monitoring in the diagnosis of seizures and epilepsy" and "Evaluation of the first seizure in adults".)

**EEG FINDINGS IN PATIENTS WITH EPILEPSY** — Different EEG findings are variably associated with epilepsy. In the setting of potential epilepsy, it is useful to classify EEG abnormalities as epileptiform and nonepileptiform.

- Examples of epileptiform activity include interictal epileptiform discharges (IEDs), periodic lateralized epileptiform discharges (PLEDs) (graph 1), and generalized periodic epileptiform
discharges (GPEDs) (graph 2)

- Nonepileptiform abnormalities include slowing, which may be diffuse, regional, or localized; amplitude changes or asymmetries; and other deviations from normal patterns. (See 'Slowing' below.)

Only epileptiform discharges are associated with epilepsy at rates sufficiently high enough to be clinically useful, and only when benign variants have been excluded. Nonspecific (nonepileptiform) EEG abnormalities are relatively common, especially in elderly individuals, patients with migraine, and those on centrally-acting medications. These should not be interpreted as supporting a diagnosis of epilepsy.

**Interictal epileptiform discharges** — To qualify as an interictal epileptiform discharge (IED), discharges should meet the following criteria:

- They must be paroxysmal and distinct from the patient's normal background activity.
- They must include an abrupt change in polarity occurring over several milliseconds.
- The duration of each transient should be less than 200 milliseconds (ms). A spike has a duration of less than 70 ms; sharp waves have a duration between 70 and 200 ms.
- The discharge must have a physiological field.
- They must not be one of the known benign variants or normal discharges such as wicket spikes, small sharp spikes, or vertex waves (table 1 and graph 3A-G)

Most IEDs are of negative polarity at the scalp, and are followed by a slow wave (ie, a spike-wave complex). While these two features are not required criteria, they are helpful in distinguishing IEDs from other types of paroxysmal activity, including electrode or other artifacts. They also relate closely to the underlying physiological phenomena occurring at the cellular level [3]. (See "Pathophysiology of seizures and epilepsy", section on 'Paroxysmal depolarization shift'.)

**Sensitivity** — An IED is found in 20 to 55 percent of persons with epilepsy on a first "routine" EEG [4-7]. A number of factors can influence the sensitivity of a finding of IED for the diagnosis of epilepsy.

- The number of EEG studies — With repeated recordings, the sensitivity of IED increases from 20 to 50 percent to as high as 80 to 90 percent when four or more EEGs are obtained [5,6,8-10].
- EEG duration — A routine EEG is 30 to 45 minutes in length. EEG monitoring over several hours to days also increases the yield of the study. In one study of 100 adult patients with confirmed epileptic seizures, EEG monitoring for seven days revealed IEDs in 81 percent [11]. (See "Video and ambulatory EEG monitoring in the diagnosis of seizures and epilepsy".)
- Seizure frequency — More frequent seizures are associated with a higher frequency of IEDs on an EEG tracing [6,12]. In this regard, a finding of IEDs may also be predictive of seizure recurrence. In one study of 157 patients with a first idiopathic unprovoked seizure, two-year seizure recurrence rates were 83 percent in those with IEDs on EEG versus 41 percent in those with nonepileptiform abnormalities and 12 percent in those with normal EEGs [7].
- Timing in regard to recent seizure — Clinical seizures are temporally associated with more frequent IEDs [5,6,13]. Among 141 patients evaluated after a first seizure, IEDs were observed more often when the EEG was obtained within 24 hours of the seizure than when obtained later (51
versus 34 percent) [5]. One study suggested that this phenomenon is more often seen in seizures of medial temporal versus neocortical origin [14].

- Antiepileptic drug therapy — There is limited information regarding the suppressant effect of AEDs on IED detection [15-19]. Treatment with valproate, levetiracetam, and probably ethosuximide reduce the rate of generalized IEDs. Diazepam and phenobarbital can suppress IEDs acutely, but chronic therapy may have little impact. Another study found that AED withdrawal was associated with reduced spikes on EEG [14].

- Epilepsy syndrome — The EEG is more likely to be abnormal in certain epilepsy syndromes. As examples, IEDs are almost invariably present in children with untreated infantile spasms, Landau-Kleffner syndrome, and benign rolandic epilepsy. While medial temporal lobe epilepsy is usually associated with an abnormal interictal EEG, patients with frontal lobe epilepsy may have a normal interictal EEG [17,20]. However, the presence of IEDs often influences both the diagnosis of epilepsy itself, as well as the specific epilepsy syndrome; as a result, this may be a source of substantial bias.

- Specialized techniques — The use of activation procedures (hyperventilation, photic stimulation, sleep deprivation, induced sleep, and medication withdrawal) and special electrode placement can increase the yield of IEDs on an interictal EEG. Their use is recommended on most follow-up EEGs in order to improve the sensitivity of the test. (See 'Specialized techniques' below.)

- Factors that are variably reported to be associated with the prevalence of IEDs in persons with epilepsy include a younger age at the time of EEG, a longer duration of epilepsy, and an earlier age at epilepsy onset [6,12].

Specificity — IEDs are rare in patients without a history of seizures. Studies in healthy flight personnel reveal IEDs in 0.5 percent [21,22]. The prevalence of IEDs has been recorded to be somewhat higher in healthy children (3.5 percent) and in hospitalized adults with neurologic or psychiatric illness (2.0 to 2.6 percent) [23-25]. A finding of IEDs is most helpful if the clinical history strongly suggests epileptic seizure [2]. However, certain caveats apply:

- The pattern of IEDs, along with the patient’s age, impact the specificity of these findings. Spikes and sharp waves are common in normal neonates during quiet (nonREM) sleep, but disappear over the first six to eight weeks of life. In contrast, focal or multifocal IEDs in adults are almost always associated with epilepsy [6,26-29]. In children, IEDs are less specific for epilepsy. In particular, central-midtemporal discharges, generalized spike-wave discharges, and photoparoxysmal responses may be asymptomatic manifestations of genetic traits [21,22,24,30]. In one series of EEG studies, only 40 percent of children with central-midtemporal spikes had epileptic seizures [29,31].

The Table (table 2) lists IEDs that may be seen on EEG, their associated clinical significance, and their likelihood of association with epilepsy [17].

- Some conditions are associated with the presence of IEDs on EEG, but do not imply epilepsy. These include occipital spikes seen in blind people (especially those who are congenitally blind) [32].

- Withdrawal from short-acting barbiturates and benzodiazepines, certain metabolic derangements (eg, hypocalcemia, uremia, dialysis disequilibrium), as well as high drug levels of lithium, neuroleptics (especially clozapine), bupropion, and tricyclic antidepressants have been associated with IEDs even in the absence of accompanying seizures [18,33,34]. These conditions are also associated...
with a lower seizure threshold.

- Inexperienced interpreters of EEG, unaware of benign variants and normal fluctuations of EEG activity that are commonly seen on EEG, can misread EEGs and limit the specificity of the findings (see 'Pitfalls in interpretation' below).

**Periodic lateralized epileptiform discharges** — Periodic lateralized epileptiform discharges (PLEDs) are defined by lateralized, persistent spikes, sharp waves, or sharply contoured slow waves that occur with an almost regular repetition rate, typically 0.5 to 2 Hz (graph 1).

These are most often seen in the setting of acute, relatively large, destructive lesions, such as cerebral infarction or hemorrhage, encephalitis, abscess, or rapidly growing cerebral malignancy [35-40]. In children, PLEDs are also associated with chronic diffuse encephalopathies [41]. Acute symptomatic seizures are seen in about 70 to 85 percent of patients with PLEDs [37,42]. This EEG pattern usually resolves over several days to weeks with recovery from the acute illness. Some of these patients develop remote symptomatic epilepsy.

Bilateral independent PLEDs (BIPLEDs) have also been described, most often with acute central nervous system (CNS) infections (especially herpes simplex encephalitis), anoxic encephalopathy, and severe chronic epilepsy [37,43]. This pattern is also highly associated with seizures. Compared to PLEDs, BIPLEDs are associated with more severe cerebral injury, worse neurological status and a worse prognosis.

Generalized periodic epileptiform discharges (GPEDS) (graph 2) are less common than PLEDs, but are still often observed in critically ill patients [44]. This pattern can be seen after convulsive status epilepticus, when it is usually considered "postictal" (although this is controversial). GPEDs at a frequency of greater than 1 Hz sometimes signify nonconvulsive status epilepticus. A similar pattern can be seen in toxic-metabolic encephalopathy (eg, "triphasic waves"), Creutzfeldt-Jakob disease, Hashimoto's encephalopathy, and from medications such as lithium, baclofen and ifosfamide. Slow GPEDs (recurring every 4 to 12 seconds) accompanied by time-locked myoclonus in an awake person are highly characteristic of subacute sclerosing panencephalitis.

**Slowing** — Focal slow-wave activity and generalized slowing of background rhythms are common postictal and interictal findings in patients with partial seizures and symptomatic epilepsies. However, they are also frequently seen in other neurologic disorders, especially focal structural lesions, regardless of whether there are associated seizures [45-47]. As an example, two-thirds of patients with continuous, focal, polymorphic, delta activity have a structural lesion, but seizures occur in only about 20 percent [45]. In patients with no clinical history of neurologic injury and a normal neuroimaging study, focal slowing is more likely to suggest epilepsy.

Bilateral, synchronous, slow activity in the delta frequency range, or intermittent rhythmic delta activity, does not usually imply epilepsy. In adults, this pattern usually has an anterior predominance and is known as frontal intermittent rhythmic delta activity (FIRDA). Once thought to be associated primarily with deep midline cerebral lesions, FIRDA is now recognized to be a nonspecific finding that is associated with encephalopathy and neurodegenerative disease, and that can occasionally be seen in normal individuals as well [48].

Occipital intermittent rhythmic delta activity (OIRDA) is more common in young children and is rarely seen in patients older than 15 years [49]. It is a frequent interictal finding in generalized epilepsy syndromes, occurring in 15 to 38 percent of all patients with childhood absence epilepsy and implies a good prognosis [49-52].

Temporal intermittent rhythmic delta activity (TIRDA) is a particular form of focal slowing that is more specific for epilepsy. TIRDA is observed in as many as 25 to 40 percent of patients being
evaluated for temporal lobe resection [53,54]. TIRDA is often associated with temporal IEDs and has a high positive predictive value for temporal lobe epilepsy. (See "Localization-related epilepsy: Causes and clinical features", section on 'Mesial temporal lobe epilepsy'.)

**Epilepsy syndrome diagnosis** — The classification of seizures and epilepsy can be important for prognosis and treatment. In adult patients, the most important distinction is between primary-generalized and partial epilepsy.

The clinical history can be unhelpful or misleading in this regard. As examples, a patient with staring spells may have absence or complex partial seizures, and a patient with generalized convulsions may have primary or secondarily-generalized epilepsy. Although the presence of an aura strongly suggests partial-onset seizures, one study reported that symptoms interpreted by the patient as a seizure aura (often brief, nonspecific dizziness) occurred in up to 70 percent of those with primary generalized epilepsy [55].

Specific interictal EEG findings that are associated with specific epilepsy syndromes are listed in the Table (table 3). Clinical correlation between the clinical seizure type and EEG findings is also important. When these agree, this is generally sufficient to distinguish generalized from partial epilepsies [56]. In addition, when focal IEDs are strongly lateralized (more than 90 to 95 percent), they predict the side of seizure onset [27]. However, focal IEDs can manifest as secondary bilateral synchronous discharges, while generalized epilepsy can have fragmentary expression and appear more focal [57]. As a result it is important to consider the clinical as well as the EEG manifestations.

**SPECIALIZED TECHNIQUES** — Specific methods can be employed to improve the detection of IEDs and the sensitivity of the test.

**Routine activating techniques** — A standard routine EEG usually includes hyperventilation and photic stimulation.

- Hyperventilation increases the rate of generalized discharges in childhood absence epilepsy and other generalized epilepsies. It is less productive in partial epilepsies, increasing the yield of focal IEDs by less than 10 percent [17,58]. Two studies have suggested that the yield of hyperventilation in generalized epilepsy may also be low, approximately 12 percent [59,60]. However, this activation procedure is very effort-dependent, and yield may vary as a result.

- Photic stimulation induces IEDs in some individuals with idiopathic generalized epilepsy, and infrequently in patients with focal seizures arising from the occipital lobe [17]. A trait of photoparoxysmal response can also run in families, and in this setting in particular, is a less specific finding for epilepsy than spontaneous IEDs. A photoparoxysmal response that is both generalized and sustained (outlasting the period of photic stimulation) and occurs at a different frequency than the photic stimulation is more likely to be associated with epilepsy than when these features are absent [58].

**Sleep and sleep deprivation** — Sleep is a neurophysiologic activator of epilepsy; 20 to 40 percent of epilepsy patients with an initial normal recording will have IEDs on a subsequent recording that includes sleep [61-63]. Sleep is sometimes captured on a routine EEG, but sleep deprivation increases this likelihood. Sleep is also usually captured on prolonged EEG monitoring and alternatively, can be induced by administration of a sedative, usually chloral hydrate.

Sleep deprivation appears to increase IEDs to an extent not fully explained by the greater chance of recording sleep [4,28]. One study found that the additional yield of a sleep-deprived EEG was similar among EEGs performed after sleep deprivation whether or not sleep was recorded [62]. Another study found that a sleep-deprived EEG had significantly higher yield compared with drug-induced sleep [64]. In this study, the sleep-deprived patients were also more likely to have a
seizure during the EEG compared with the sedated patients. Sleep deprivation may not be as helpful in children, particularly in those less than three years old [65]. (See "Clinical and laboratory diagnosis of seizures in infants and children", section on 'Sleep EEG'.)

When sleep-deprived EEG was compared to 24-hour ambulatory EEG monitoring in 46 patients with "presumed epilepsy", IED detection was similar (24 versus 33 percent) [29]. However, clinical seizures were also captured in 15 percent of the ambulatory EEGs and in none of the sleep-deprived EEG. (See "Video and ambulatory EEG monitoring in the diagnosis of seizures and epilepsy".)

It is generally agreed that a follow-up EEG in a patient with possible epilepsy and a normal routine EEG should include sleep. Clinicians can order full or partial sleep disruption, but it is not clear how much this affects the yield [4]. The choice of test (sleep-deprived, sleep with oral sedation, or prolonged EEG monitoring) should be individualized to the patient's circumstances. Because sleep deprivation can be quite disruptive and carries some risk of seizure exacerbation, we generally prefer 24-hour-ambulatory EEG studies over sleep-deprived studies. (See "Video and ambulatory EEG monitoring in the diagnosis of seizures and epilepsy".)

**Special electrode placement** — Some highly epileptogenic areas, such as the mesial temporal lobes, are not well explored by the scalp electrodes placed on scalp. The classic temporal chain electrodes (T3/T5 and T4/T6) lie over the sylvian fissure and record from the infra- and suprasylvian regions.

Specialized electrode placement can improve detection of IEDs. However, these electrodes can be uncomfortable for patients and are associated with increased artifact, which increases the potential for misinterpretation. (See 'Pitfalls in interpretation' below.)

- Sphenoidal electrodes are wires inserted through a needle cannula inferior to the zygomatic arch, perpendicular to the sagittal plane, and parallel to the coronal plane, in an attempt to record activity from the anterior tip of the temporal lobe.
- Nasopharyngeal electrodes are inserted through the nostrils into the nasopharynx to record from the anterior mesial surface of the temporal lobe.
- Ear electrodes are inserted into the ear canal to lie next to the tympanic membrane.
- Superficial anterior temporal (T1) electrodes are placed 1 cm above and one-third the distance along the line from the external auditory meatus to the external canthus of the eye.
- Inferior temporal chain electrodes (F9/T9/P9 and F10/T10/P10) are an extra chain of three electrodes placed a standard electrode distance inferior to the standard temporal chain and record from the temporal lobes.

Of these, sphenoidal electrodes have the highest yield and are associated with considerably less artifact than nasopharyngeal electrodes [66]. In one study, sphenoidal electrodes detected 99 percent of discharges, compared with a 57 percent yield for nasopharyngeal electrodes and 54 percent yield for ear electrodes. However, these electrodes are invasive and uncomfortable for patients and are not used routinely for diagnostic purposes.

The superficial anterior temporal electrodes are somewhat less sensitive than sphenoidal electrodes, but are noninvasive and are equivalent or superior in sensitivity and patient comfort to the nasopharyngeal, minisphenoidal, and ear electrodes [67-70].

**PITFALLS IN INTERPRETATION** — Misinterpretation of EEG findings or over-reliance on the EEG frequently contributes to misdiagnosis [9,13,61,62,71].
It is important to remember that a normal EEG never rules out epilepsy [72]. Even with repeated EEGs, use of specialized techniques, or prolonged monitoring, a significant number of patients with epilepsy (10 to 20 percent) will not have IEDs [63,72]. Even ictal recordings may not have an identifiable scalp correlate in many frontal lobe seizures, as well as in simple partial seizures from any location [20].

It is also important to note that "abnormal" EEG does not define epilepsy; most abnormal findings are nonspecific. IEDs are the most specific finding for epilepsy, but these can occur in about 0.5 percent of healthy adults and in 1.9 to 3.5 percent of normal children [63].

There is also wide variation in how EEGs are interpreted. When EEGs are read by clinicians without special training, a number of benign or normal patterns are often misinterpreted as epileptiform [61,62,71,73]. These include (table 1) [62,74,75]:

- Benign epileptiform transients of sleep (BETS), also termed small sharp spikes (SSS) (graph 3A)
- Wickle spikes (graph 3B)
- Rhythmic midtemporal theta of drowsiness (psychomotor variant) (graph 3C)
- Six-Hz "phantom" spike and wave complex (graph 3D)
- Subclinical rhythmic EEG discharge in adults (SREDA) (graph 3E)
- Positive occipital sharp transients of sleep (POSTS) (graph 3F)
- Breach rhythm (graph 3G)
- Hyperventilation-induced high voltage paroxysmal slow waves
- Artifacts (such as over-filtered muscle potentials)
- Repetitive vertex waves, especially in children

Repeating the EEG or having it reinterpreted at a tertiary epilepsy center by a board-certified electroencephalographer can be helpful. One meta-analysis found that a more restrictive interpretation style that limits false positives improves diagnostic accuracy [76]. However, even among experienced, board-certified neurophysiologists, interobserver agreement is only moderate [2,7,77].

SUMMARY AND RECOMMENDATIONS — Electroencephalography (EEG) remains an important diagnostic test in evaluating a patient with possible epilepsy, providing evidence that helps confirm or refute the diagnosis. EEG also assists in classifying the underlying epileptic syndrome and thereby guides management.

- A single routine EEG has low sensitivity for detection of interictal epileptiform discharges (IED) (20 to 50 percent) for patients with epilepsy. The sensitivity can be increased by repeating the study, recording for a longer period of time such as overnight, including a recording of sleep (spontaneous, after sleep deprivation, or via administration of a sedative), performing the EEG within 24 hours of a seizure, and by using special electrodes for temporal lobe epilepsy. (See 'Sensitivity' above and 'Specialized techniques' above.)

A normal EEG, however, can never rule out epilepsy; 10 to 20 percent of patients with definite epilepsy never have IEDs.

- Overall, the specificity of IEDs for epilepsy is high, more than 90 percent in adults. However, inexperienced EEG interpreters can mistake artifact or benign EEG patterns for IEDs, lowering the specificity of the study. The specificity of this finding is also influenced by the pattern of IEDs, and by the patient's age, family history, and comorbid conditions. (See 'Specificity' above and 'Pitfalls in interpretation' above.)

- Periodic lateralized epileptiform discharges (PLEDs) are usually seen in the setting of acute,
relatively large cerebral injury, such as stroke, encephalitis, or rapidly growing cerebral malignancies. Acute symptomatic seizures are common in patients with PLEDs. (See 'Periodic lateralized epileptiform discharges' above.)

- Generalized or focal slowing on EEG is nonspecific and does not suggest epilepsy. One exception is the pattern of temporally-located intermittent rhythmic delta activity (TIRDA), which is highly associated with temporal lobe epilepsy. (See 'Slowing' above.)

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REFERENCES

17. Pillai, J, Sperling, MR. Interictal EEG and the diagnosis of epilepsy. Epilepsia 2006; 47 Suppl


47. Marshall, DW, Brey, RL, Morse, MW. Focal and/or lateralized polymorphic delta activity. Association with either 'normal' or 'nondelta' computed tomographic scans. Arch Neurol 1988; 45:33.


Right hemisphere PLEDs: periodic lateralized epileptiform discharges

<table>
<thead>
<tr>
<th>Patient: age / state</th>
<th>Waveform: morphology / frequency</th>
<th>Duration</th>
<th>Distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Benign spike-like patterns</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small sharp spikes</td>
<td>Adults / drowsiness and light sleep</td>
<td>Monophasic or diphasic spikes</td>
<td>Phase &lt;50 ms &lt; 11 Hz</td>
</tr>
<tr>
<td>Wicket spikes</td>
<td>Adults / drowsiness and light sleep</td>
<td>Sharply contoured arch shaped / 6 to 11 Hz</td>
<td>Few seconds</td>
</tr>
<tr>
<td>14- and 6-Hz positive bursts</td>
<td>Children and adolescents / drowsiness and light sleep</td>
<td>Arch shaped positive sharp component / 4-7 Hz and 13-17 Hz range</td>
<td>Less than 1 to 2 seconds</td>
</tr>
<tr>
<td>6 Hz spike and wave</td>
<td>Adolescents and young adults / relaxed wakefulness and drowsiness</td>
<td>Low voltage spike-high amplitude slow wave / 5 to 7 Hz</td>
<td>1 to 2 seconds</td>
</tr>
<tr>
<td><strong>Rhythmic patterns with an epileptiform morphology</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rhythmic temporal theta bursts of drowsiness</td>
<td>Adolescents and young adults / relaxed wakefulness and drowsiness</td>
<td>Notched flat topped / 5 to 7 Hz</td>
<td>Seconds</td>
</tr>
<tr>
<td>Subclinical rhythmic EEG discharge in adults</td>
<td>Adults &gt;50 / hyperventilation, drowsiness</td>
<td>Rhythmic sharply contoured / 5 to 6 Hz</td>
<td>40 to 80 seconds</td>
</tr>
<tr>
<td>Midline theta rhythm</td>
<td>Children and adults / awake, drowsy</td>
<td>Sinusoidal, arciform / 5 to 7 Hz</td>
<td>4 to 20 seconds</td>
</tr>
</tbody>
</table>

Small sharp spikes also known as benign epileptiform transients of sleep (BETS)

Small sharp spikes (also known as benign epileptiform transients of sleep, or BETS; see boxed area). This is a normal variant.

Courtesy of Drs. Lawrence Hirsch and Hiba Arif.
Wicket spikes

These may appear in clusters or singly in the temporal regions.

*Courtesy of Drs. Lawrence Hirsch and Hiba Arif.*
Rhythmical midtemporal theta of drowsiness (RMTD; see arrows); also known as psychomotor variant. This is a normal variant.

*Courtesy of Drs. Lawrence Hirsch and Hiba Arif.*
Six-Hz spike-and-wave bursts ("Phantom spike and wave"; see arrows): three 1 second bursts from one routine EEG recording. (A) This pattern is accentuated in an average referential montage, sensitivity 5 uV/mm. Note the low amplitude, occipitally predominant "phantom" spikes (arrows), and the frequency of approximately 6 per second (B) in a longitudinal bipolar montage (7 uv/mm), this pattern is not as well-appreciated. This is a normal variant.

*Courtesy of Drs. Lawrence Hirsch and Hiba Arif.*
Subclinical rhythmic EEG discharge of adults (SREDA) in a 52 year old woman with headaches, preceded by a widespread, moderate-to-high amplitude, biphasic waveform. This is a rare but normal variant.

A) POSTS (Positive occipital sharp transients of sleep), in runs, in a bipolar montage (red arrows). B) A referential recording confirms the positivity in the occipital regions (downgoing waves; green arrows). These are normal discharges.

*Courtesy of Drs. Lawrence Hirsch and Hiba Arif.*
Breach rhythm noted at the C3 electrode in a 61-year-old man with history of head trauma and a left frontoemporal craniotomy.

### Spike location and probability of epilepsy

<table>
<thead>
<tr>
<th>Probability:</th>
<th>Most often associated with:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High probability of epilepsy (&gt;85 percent):</strong></td>
<td></td>
</tr>
<tr>
<td>Anterior-mid temporal spikes*</td>
<td>Mesial temporal lobe epilepsy</td>
</tr>
<tr>
<td>Midline spikes•</td>
<td>Tonic-clonic seizures</td>
</tr>
<tr>
<td>Hypsarrhythmia</td>
<td>Infantile spasms (West syndrome)</td>
</tr>
<tr>
<td>Slow spike-wave</td>
<td>Lennox-Gastaut Syndrome</td>
</tr>
<tr>
<td>Generalized paroxysmal fast activity</td>
<td>Lennox-Gastaut Syndrome</td>
</tr>
<tr>
<td><strong>Moderate probability of epilepsy (&lt;75 percent):</strong></td>
<td></td>
</tr>
<tr>
<td>Frontal spikesΔ</td>
<td>Frontal lobe epilepsy</td>
</tr>
<tr>
<td>Generalized spike-wave (≥3 Hz)</td>
<td>Absence epilepsy (3 Hz; CAE, JAE), juvenile myoclonic epilepsy (&gt;3 Hz), and other primary generalized epilepsies</td>
</tr>
<tr>
<td>Centro-temporal spikes◊</td>
<td>Benign rolandic epilepsy of childhoodhood (BREC)</td>
</tr>
<tr>
<td>Occipital spikes§</td>
<td>Benign focal epilepsy of childhood (Gastaut and Panayiotopolous syndromes: the 2 variants of CEOP)</td>
</tr>
<tr>
<td>Photoparoxysmal response</td>
<td>Primary generalized epilepsy</td>
</tr>
</tbody>
</table>

CAE: childhood absence epilepsy; JAE: juvenile absence epilepsy; BECTS: benign focal epilepsy of childhood with centrotemporal spikes; BREC: benign rolandic epilepsy of childhood; CEOP: childhood epilepsy with occipital paroxysms.

* >90 percent probability of seizures/epilepsy (adults).¥

• 76-91 percent probability of seizures/epilepsy (children).†

Δ ~75 percent probability of seizures/epilepsy (children).†

◊ ~40 percent probability of seizures/epilepsy (children).†

§ 50 percent probability of seizures/epilepsy (children).†


Table modified and expanded from: Pillai, J, Sperling, MR. Interictal EEG and the diagnosis of epilepsy. Epilepsia 2006; 47 Suppl 1:14.
# Electroencephalographic features of some epilepsy syndromes

<table>
<thead>
<tr>
<th>Epilepsy syndromes</th>
<th>EEG findings</th>
<th>Additional EEG features</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Generalized</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absence epilepsy</td>
<td>3-Hz generalized spike-and-slow wave; often repetitive trains of discharges</td>
<td>Normal background; activation of IEDs and seizures with hyperventilation</td>
</tr>
<tr>
<td>Atypical absence epilepsy</td>
<td>1.5 to 2.5-Hz generalized spike-and-slow wave discharges</td>
<td>IEDs may be asymmetric, with shifting focal features</td>
</tr>
<tr>
<td>Juvenile myoclonic epilepsy</td>
<td>4 to 6-Hz generalized spike and multiple spike-and-slow wave</td>
<td>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</td>
</tr>
<tr>
<td>Infantile spasms</td>
<td>Hypsarrhythmia and multifocal spikes and sharp waves</td>
<td>No clearly normal background activity since the EEG is dominated by the hypsarrhythmia pattern</td>
</tr>
<tr>
<td>Lennox-Gastaut syndrome</td>
<td>&lt;2.5-Hz generalized sharp-and-slow wave discharges</td>
<td>Generalized background slowing and paroxysmal fast activity; often multifocal spikes and sharp waves</td>
</tr>
<tr>
<td>Progressive myoclonic epilepsy</td>
<td>Generalized and multifocal spikes, multiple spikes, and sharp waves</td>
<td>Progressive background slowing with disease progression; photic activation of IEDs in some cases</td>
</tr>
<tr>
<td><strong>Partial</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Benign rolandic epilepsy</td>
<td>Large-amplitude spikes or sharp waves, maximal over the centrotemporal region</td>
<td>Normal background; often prominent activation of IEDs with sleep; the discharge can be bilateral or unilateral and often has an anterior-posterior field of a tangential dipole</td>
</tr>
<tr>
<td>Benign occipital epilepsy</td>
<td>Bilateral or unilateral occipital spike-and-slow wave discharges</td>
<td>Occipital IEDs often attenuate with eye opening; photic stimulation may precipitate seizures</td>
</tr>
<tr>
<td>Temporal lobe epilepsy</td>
<td>Temporal lobe spikes, sharp waves, and temporal intermittent rhythmic delta activity; often activated with drowsiness and sleep</td>
<td>Often intermittent or persistent temporal slowing; may see independent IEDs from contralateral temporal lobe</td>
</tr>
<tr>
<td>Frontal lobe epilepsy</td>
<td>IEDs in the frontal region</td>
<td>Mesial frontal discharges often are not detected by scalp EEG; secondary bilateral synchrony can occur</td>
</tr>
</tbody>
</table>

EEG: electroencephalographic; IED: interictal epileptiform discharge.

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