EEG Essentials

By William O. Tatum IV, DO, FAAN, FACNS, FAES

REVIEW ARTICLE

CONTINUUM AUDIO INTERVIEW AVAILABLE ONLINE

VIDEO CONTENT AVAILABLE ONLINE

ABSTRACT

PURPOSE OF REVIEW: EEG is the best study for evaluating the electrophysiologic function of the brain. The relevance of EEG is based on an accurate interpretation of the recording. Understanding the neuroscientific basis for EEG is essential. The basis for recording and interpreting EEG is both brain site–specific and technique-dependent to detect and represent a complex series of waveforms. Separating normal from abnormal EEG lies at the foundation of essential interpretative skills.

RECENT FINDINGS: Seizures and epilepsy are the primary targets for clinical use of EEG in diagnosis, seizure classification, and management. Interictal epileptiform discharges on EEG support a clinical diagnosis of seizures, but only when an electrographic seizure is recorded is the diagnosis confirmed. New variations of normal waveforms, benign variants, and artifacts can mimic epileptiform patterns and are potential pitfalls for misinterpretation for inexperienced interpreters. A plethora of medical conditions involve nonepileptiform and epileptiform abnormalities on EEG along the continuum of people who appear healthy to those who are critically ill. Emerging trends in long-term EEG monitoring to diagnose, classify, quantify, and characterize patients with seizures have unveiled epilepsy syndromes in patients and expanded medical and surgical options for treatment. Advances in terminology and application of continuous EEG help unify neurologists in the diagnosis of nonconvulsive seizures and status epilepticus in patients with encephalopathy and prognosticate recovery from serious neurologic injury involving the brain.

SUMMARY: After 100 years, EEG has retained a key role in the neurologist's toolkit as a safe, widely available, versatile, portable test of neurophysiology, and it is likely to remain at the forefront for patients with neurologic diseases. Interpreting EEG is based on qualitative review, and therefore, the accuracy of reporting is based on the interpreter's training, experience, and exposure to many new and older waveforms.

INTRODUCTION

Initial concepts and clinical observations were described by Hans Berger in his work on the "Elektroenkephalogram des Menschen" (the EEG of man) that depicted electrical currents produced by the brain represented in graphic form.[1] EEG can be recorded in the hospital, intensive care unit (ICU), operating room (OR), and ambulatory setting in patients of all ages. Since the first description in the 1920s, EEG has remained the most relevant testing modality to evaluate people with seizures. Approximately 1 in 10 people will have at least one seizure at some point in their lives, and 1 in 26 Americans will be

CITE AS:
CONTINUUM (MINNEAP MINN) 2022;28(2, EPILEPSY):261-305.

Address correspondence to Dr William O. Tatum IV, Mayo Clinic College of Medicine & Science, Mangurian Building, Fourth Floor, 4500 San Pablo Rd, Jacksonville, FL 32224, tatum.william@mayo.edu.

RELATIONSHIP DISCLOSURE:
Dr Tatum has received personal compensation in the range of $500 to $4999 for serving as an Editor-in-Chief for *Epilepsy & Behavior Reports* and as an expert witness for a defense law firm on behalf of a patient with epilepsy with funds donated to the Epilepsy Foundation of America; has received personal compensation in the range of $10,000 to $49,999 for serving as a consultant for BioSerenity, Holberg EEG AS, Neurelis, Inc, Zimmer Biomet; and has received publishing royalties from Demos and Springer Publishers. The institution of Dr Tatum has received research support from Cerevel Therapeutics, Engage Pharma, Esai Inc, LivaNova PLC, the Mayo Clinic, Medtronic, and Xenon.

UNLABELED USE OF PRODUCTS/INVESTIGATIONAL USE DISCLOSURE:
Dr Tatum reports no disclosure.

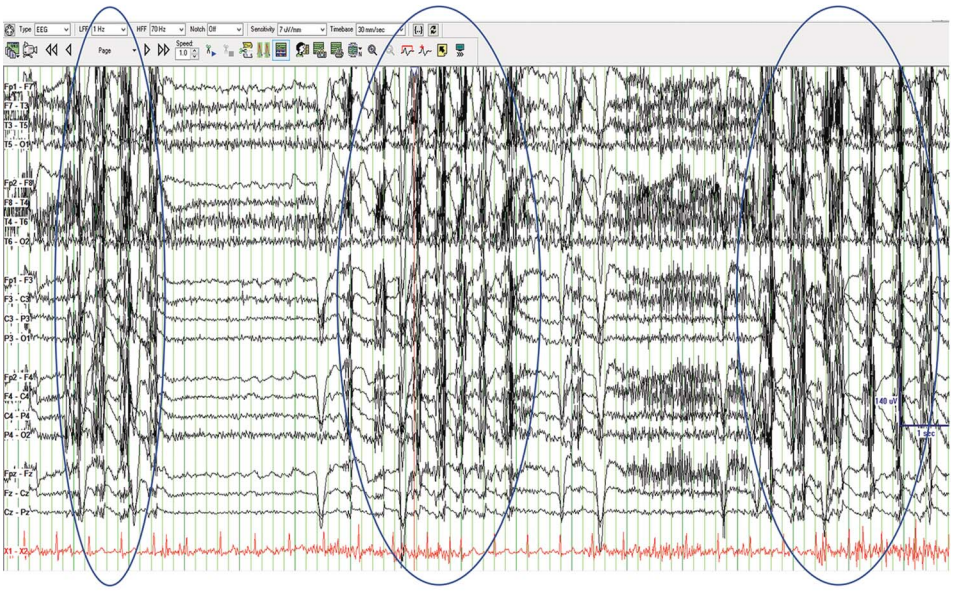


FIGURE 2-1
Chewing artifact (*ovals*) mistaken for frequent and prolonged bursts of polyspike and waves in a 24-year-old driver involved in a car accident. He did not recall the impact when he was jettisoned from the vehicle. Although he never experienced a seizure, he was unnecessarily placed on antiseizure medication "just in case."

TABLE 2-1 **Selected Diagnostic Indications for EEG**

High-yield EEG

- Seizures and epilepsy
- Possible seizures/"spells"
- Head injury, stroke, central nervous system infection, and brain tumor
- Parasomnias and sleep disorders
- Movement disorders
- Progressive dementias and encephalopathy
- Acute altered mental status and coma

Low-yield EEG

- Headache
- Pain
- Chronic behavioral disorders
- Isolated dizziness/vertigo
- Regular EEGs in patients who are seizure free

EEG = electroencephalography.

diagnosed with epilepsy,[2] imparting a significant burden of disease. EEG is foundational for people with epilepsy, and the clinical utility is well established.[3] EEG abnormalities form the physiologic basis when evaluating patients with paroxysmal neurologic events. Qualitative assessment is the standard method of interpreting standard EEG for routine clinical use. However, interpretation of EEG recordings has only moderate interrater reliability using visual analysis and is therefore subject to misinterpretation (FIGURE 2-1). When epileptiform features are present on the EEG, the frequency, discharge duration, and spatial distribution are defining characteristics of interictal epileptiform discharges and clinical impact. EEG abnormalities in patients with epilepsies aid diagnosis but also impact management. EEG in the ICU can identify nonconvulsive seizures in patients with altered mental status. Long-term EEG monitoring can clarify the interictal–ictal continuum and provide prognostic information.

With its versatility and adaptability, EEG is useful in many conditions (TABLE 2-1). It is also important for interpreters to understand the limits of EEG, including the lack of specificity for disease states, insensitivity in some patients to detect abnormalities, and "interference" by artifacts and the physiologic effects incurred from medication or prior surgery.[4] This article on EEG functions as a primer and highlights common examples across a broad range of neurologic disease states focused on the epilepsies as a roadmap to aid competency and as an introduction to formal curricula dedicated to EEG education.[5]

KEY POINTS

- Since the first description in the 1920s, EEG has remained the most relevant testing modality to evaluate people with seizures.
- Signals detected and ultimately recorded by EEG are generated by dynamic extracellular currents produced by transmembrane ion flow.

NEUROSCIENCE CONCEPTS UNDERLYING EEG

Physicians who treat patients with epilepsy need to understand biophysical aspects of signal generation and recording to accurately interpret the results of the clinical EEG.[4] Signals detected and ultimately recorded by EEG are generated by dynamic extracellular currents produced by transmembrane ion flow. These currents are initiated in the apical dendrites of the pyramidal neurons located in layers IV and V of the cerebral cortex. Excitatory and inhibitory postsynaptic potentials are arranged in palisades and summate through passive current flow. The local field potentials generated are volume conducted through extracerebral tissues to the recording sensor. Excitatory postsynaptic potentials have an actively maintained separation of electrical charge and extracellular negativity at the superficial synapse. This serves as a "sink" to actively attract sodium ions. Compensatory current flow from the extracellular space at the opposite end of the neuron becomes relatively positive as a passive "source." In contrast, inhibitory postsynaptic potentials have extracellular positivity due to the active influx of chloride ions or efflux of potassium ions at the source and extracellular negativity at a passive sink resulting from current flow. Dendrites in the superficial cortical layers have an anatomical separation with excitatory inputs at the distal dendritic arborization whereas inhibitory inputs are closer to the soma, resulting in an electrophysiologic dipole. When a large pool of neurons is synchronized, excitatory postsynaptic potentials or inhibitory postsynaptic potentials are summated and amplified and can be recorded by the EEG electrodes. When numerous dipoles are summated, currents are generated that are large enough to conduct across the brain tissue and connect an electrophysiologic generator with a recording electrode.

To record EEG signals (ie, interictal epileptiform discharges), at least 10 cm^2 of cortex needs to be activated. Dipoles generating abnormal electrophysiologic potentials have a specific orientation, with the negative

end lying in the superficial cortical layers and the positive end closer to the cell body in deeper layers of the cortex. Scalp topographic representation by EEG is determined by the area of the cortex generating the signal and the orientation of the negative and positive ends of the dipole that determine the spatial distribution of the recorded signal.[4]

RECORDING EEG

The American Clinical Neurophysiology Society has published guidelines regarding EEG monitoring in addition to minimal standards for recording.[6,7] Most standard EEGs are obtained using standard scalp electrodes and acquired in the interictal period when patients are asymptomatic. Standard EEG is a safe, noninvasive, routine study that is the most common type of EEG recording obtained. Ambulatory EEG, video-EEG monitoring, critical care continuous EEG, quantitative EEG, intracranial EEG, and electrocorticography all record the same brain signals, differing in technique and site of recording. Standard EEG recording involves a multichannel microprocessor, high sampling rates of 512 Hz or higher, 128 gigabytes of internal memory or more, and resolution of at least 16 bits.[3] Clinical EEG is recorded by using the international 10-20 system of electrode placement as a universal standard (FIGURE 2-2). Twenty-five channels of EEG are recommended for clinical use by the International Federation of Clinical Neurophysiology, incorporating a single channel of ECG.[8] Bipolar montages (two active electrode sites) use phase reversals, and referential montages (one active electrode site) use absolute voltage to measure electrical field maxima. Anterior-posterior longitudinal bipolar montage (also known as the *double banana*) is commonly used as a screening montage. Standard parameters for recording EEG include sensitivity (adult, 7 μV/mm), filter settings of 1 to 70 Hz (notched filter with 60-Hz artifact), and a time base of 30 mm/s.

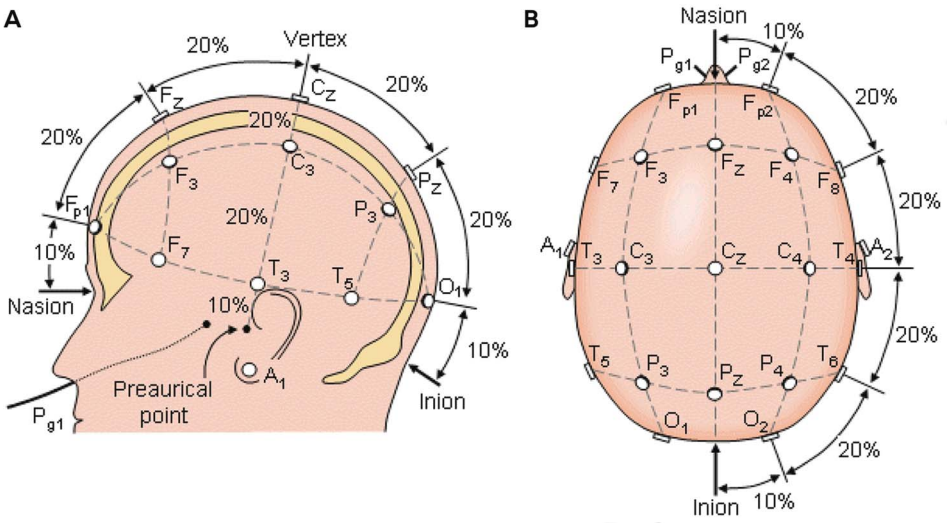


FIGURE 2-2

Electrode placement for scalp-recorded EEG. The international 10-20 system of electrode placement (*A*) uses anatomic landmarks on the skull with sites subdivided by intervals of 10% and 20% to designate the site where an electrode will be placed. The modified combinatorial system (*B*) uses more closely spaced electrodes in a 10-10 system.

Advantages of altering recording parameters include changing the display speed (FIGURE 2-3), montage (FIGURE 2-4), sensitivity (FIGURE 2-5), and filter settings to enhance the EEG recording.

Sleep, sleep deprivation, and sedated sleep may be useful to activate interictal epileptiform discharges during standard EEG recording. Therefore, obtaining sleep in every routine EEG recording is desirable. Hyperventilation and intermittent photic stimulation are standard activation procedures to enhance detection of interictal epileptiform discharges. This is especially applicable in patients with generalized genetic epilepsy, where activation is most likely to occur. Hyperventilation performed for 3 minutes at a rate of about 20 breaths/min normally produces a "buildup" (FIGURE 2-6). Nearly every person with untreated absence seizures has hyperventilation-provoked 3-Hz generalized spike-and-wave discharges. Intermittent photic stimulation, using a series of stroboscopic photic flashes 20 to 30 cm from the patient's eyes, can identify abnormal photosensitivity if a photoparoxysmal response (FIGURE 2-7) is recorded.[9] However, an isolated occurrence may also occur in people without clinical seizures. If a photoparoxysmal response is seen during EEG recording, intermittent photic stimulation should be discontinued to prevent a generalized tonic-clonic seizure. Use of video cameras may reveal subtle symptomatology (or artifact) during review that is not reported by patients and increases the interpretative potential over EEG alone. Real-time remote EEG and wireless network connections with high-speed data transfer are possible with most modern EEG systems (FIGURE 2-8).

KEY POINT

- **Most standard EEGs are obtained using standard scalp electrodes and acquired in the interictal period when patients are asymptomatic.**

NORMAL EEG

A wide range of background frequencies can be seen in the EEG. Interpreting abnormal EEG requires understanding when and what studies are normal.[10,11]

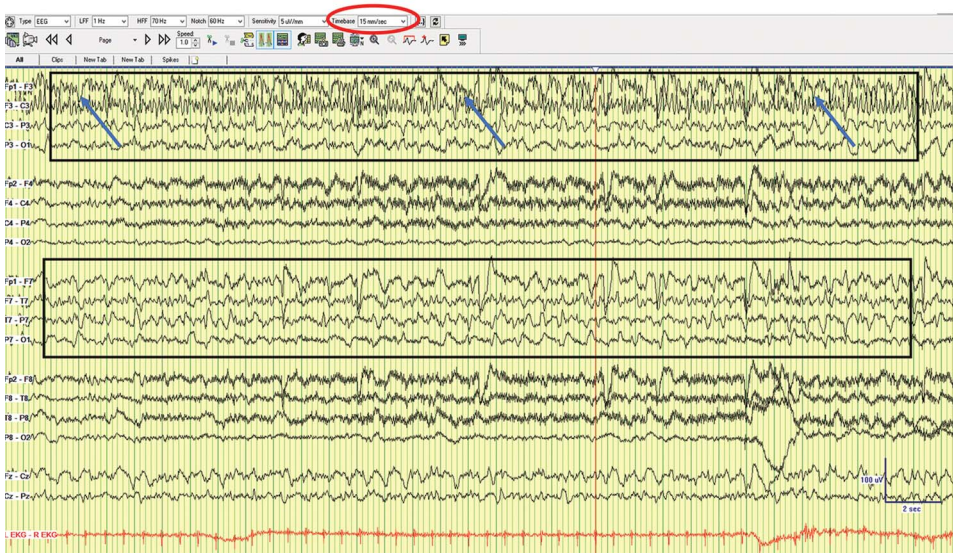


FIGURE 2-3

Continuous EEG demonstrating nonconvulsive status epilepticus in a 45-year-old woman after left hemicraniectomy for left frontocentral intraparenchymal hemorrhage during pregnancy. Note the slow display speed of 15 mm/s (*oval*) that enhances continuous left hemispheric slowing (*rectangles*) and F3 ictal fast activity under a breach rhythm (*arrows*).

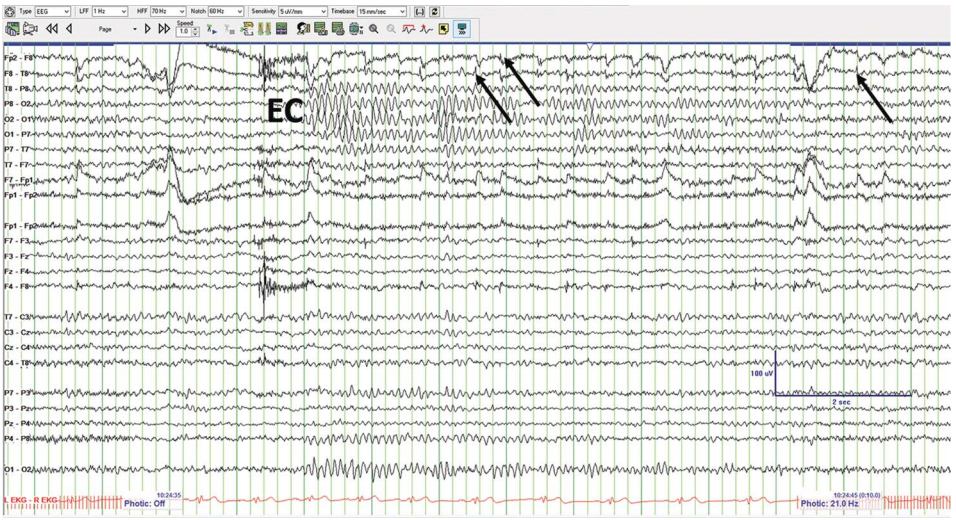


FIGURE 2-4

Combined circle and transverse bipolar montage accentuating the alpha rhythm in the occipital region. Note the lateral rectus spikes (myogenic artifact, *arrows*) and reactivity with return of the alpha when the eyes are closed (*EC*).

A normal EEG is a common result when patients obtain a standard study. However, a normal result does not exclude the possibility a patient has epilepsy, and EEG should not be used to make an epilepsy diagnosis independent of the clinical context of recording.

Interpretation involves the qualitative process of visually extracting a complex series of brain signals.[4] Most clinical EEG interpretations identify frequencies

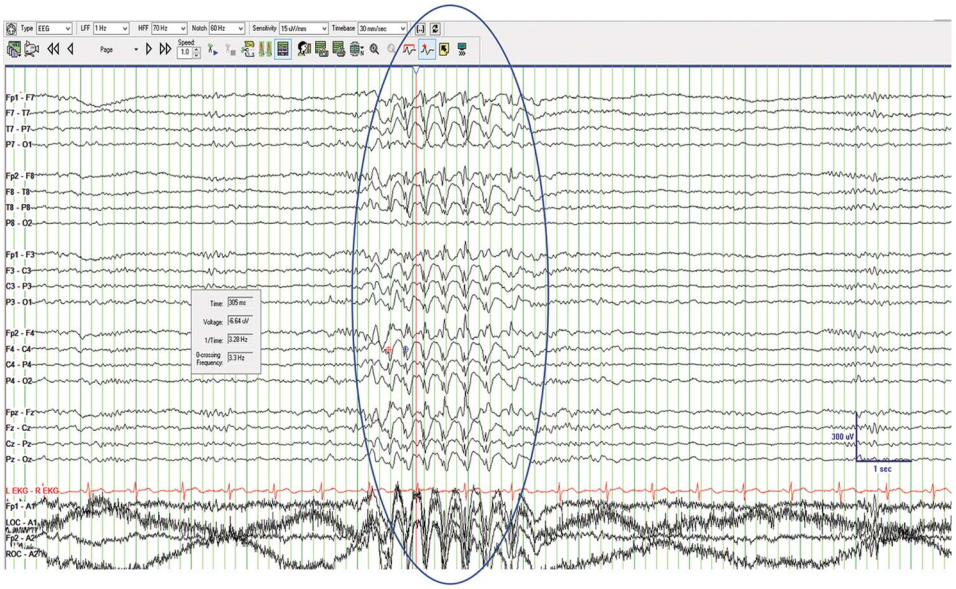


FIGURE 2-5

EEG with a 2.5-second burst of 3.3-Hz generalized frontally predominant spike and waves (*oval*) without clinical signs in N2 sleep. Note the sensitivity of 15 μV/mm, which makes the background look suppressed, but which allows visualization of the spike phase reversals in the frontal region.

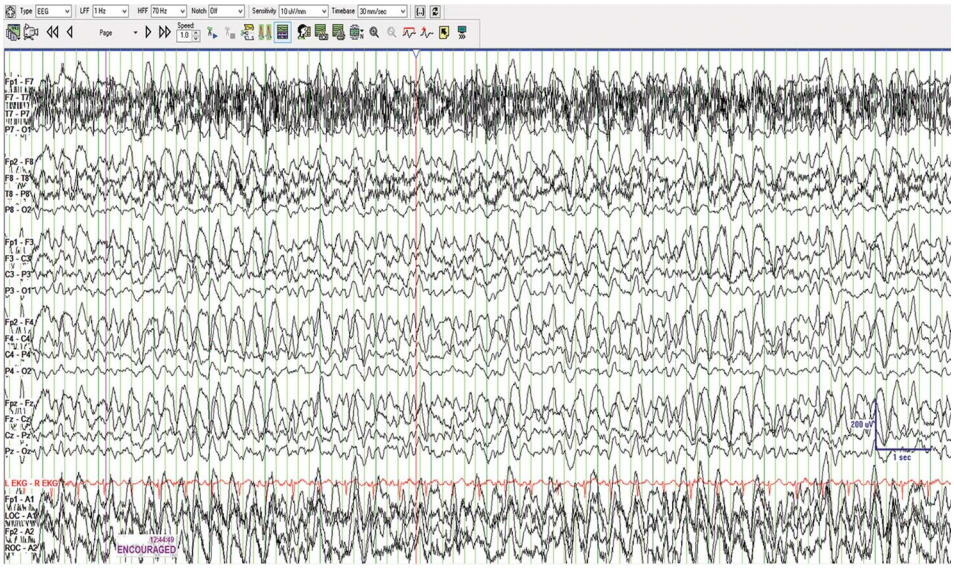


FIGURE 2-6

A robust but normal buildup with bursts of frontal intermittent rhythmic delta activity (FIRDA) intermixed with theta and delta during hyperventilation. This 17-year-old girl had migraines and had last eaten the day before the EEG recording.

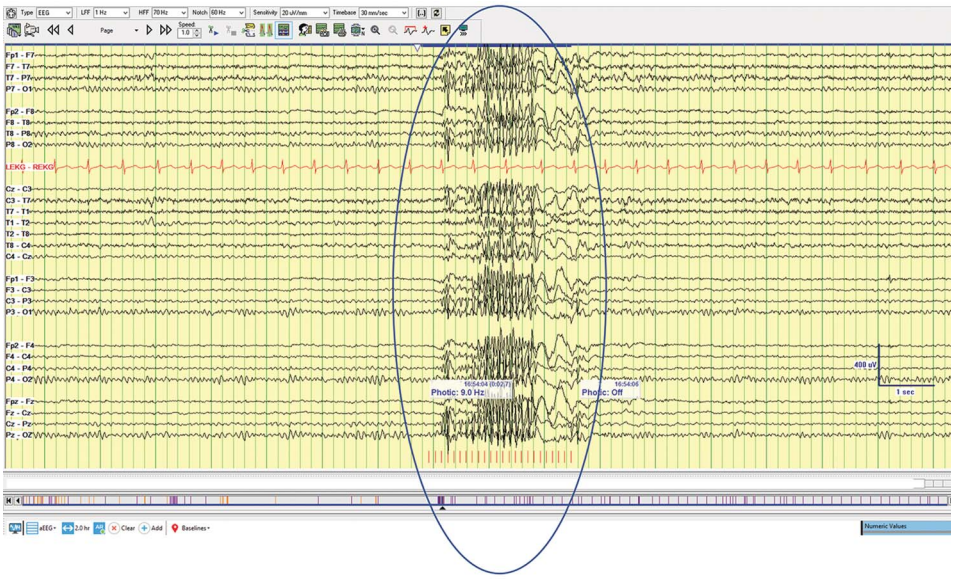


FIGURE 2-7

Self-limited photoparoxysmal response (*oval*) in a patient with idiopathic (presumed genetic) photosensitive occipital lobe epilepsy. During intermittent photic stimulation, an incremental series of flashes is delivered for about 4 seconds, with at least 4 seconds in between each train increasing to 30 Hz.

KEY POINT

- **A normal EEG is a common result when patients obtain a standard study. However, a normal result does not exclude the possibility that a patient has epilepsy, and EEG should not be used to make an epilepsy diagnosis independent of the clinical context of recording.**

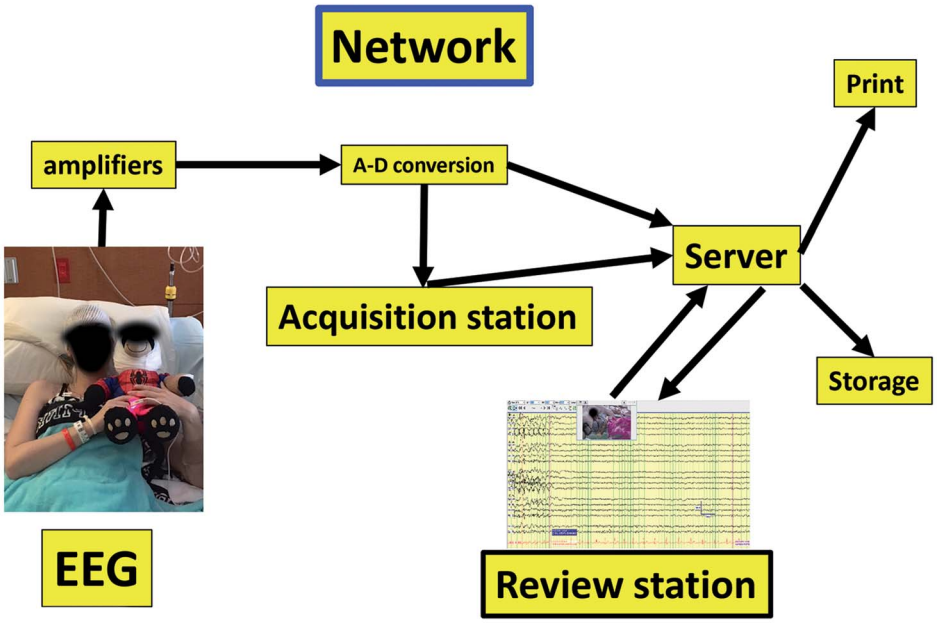


FIGURE 2-8
Modern EEG systems.
A-D = analog to digital.

between 1 and 35 Hz (also known as the *Berger band*), including a mixture of frequencies involving alpha (8 to 13 Hz), beta (13 to 30 Hz), theta (4 to 8 Hz), and delta (less than 4 Hz). The alpha rhythm (posterior-dominant rhythm) is the starting point for interpretation (FIGURE 2-9). Each frequency bandwidth can have normal and abnormal implications depending on the clinical context of recording (TABLE 2-2).[12]

A crucial aspect of interpreting EEG lies in differentiating pathologic from physiologic waveforms (TABLE 2-3). Normal aspects of the EEG (FIGURE 2-9 and FIGURE 2-10), variations of normal (FIGURE 2-11), and benign variants in the EEG may be epileptiform (FIGURE 2-12) or rhythmic patterns (FIGURE 2-13). These may look abnormal but are not despite appearing "suspicious" to novice interpreters.[11] Overinterpretation of normal features of the EEG is a common reason for misinterpretation.[11,13] Some normal variations in frequency (eg, left temporal delta during wakefulness in older adult patients) or morphology (eg, posterior temporal "apiculate" alpha; FIGURE 2-11) may lead the interpreter to misinterpret a normal EEG as abnormal and result in incorrect treatment (CASE 2-1).

Artifact occurs from a wide range of sources and is present in essentially every EEG recording. Artifact is an essential means of identifying normal levels of wakefulness and sleep, although in electrically hostile environments, artifacts can completely obscure a tracing (FIGURE 2-15) and interfere with the ability to interpret underlying electrocerebral activity. Both physiologic and nonphysiologic sources of artifact may mimic interictal and ictal abnormalities (FIGURE 2-16). Experienced technologists are invaluable in ensuring optimal EEG recording and annotation of events.[14] In expert hands, interpreting a quality home video recording has the potential to separate epileptic and nonepileptic events although it remains only an indirect supplement to seizure diagnosis without the

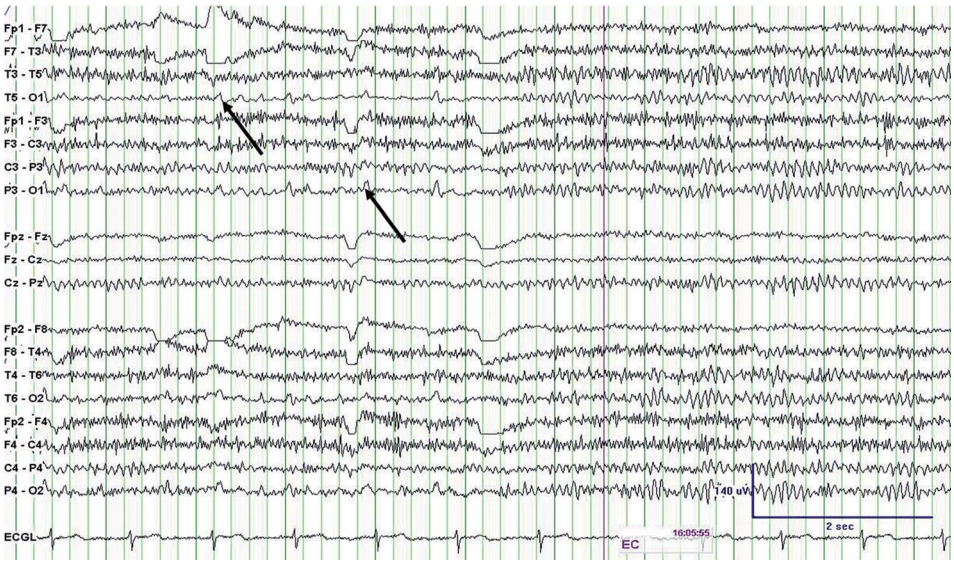


FIGURE 2-9

Awake EEG. In the first half of the tracing, horizontal and vertical eye blink artifact is present generating lambda waves (*arrows*). Note the reactivity and return of alpha after eye closure (*EC*). Lambda waves have a "sharp" appearance, are located bilaterally over the occipital region, and are evoked by scanning eye movements.

KEY POINTS

- **Benign variants of uncertain significance, normal waveform variations, and artifacts may be pitfalls to overinterpreting a normal record as abnormal leading to inappropriate treatment with antiseizure medication.**
- **Standard EEG is the diagnostic test of choice to provide electrophysiologic information about the presence of neurophysiologic dysfunction.**

definitive means provided by EEG.[15] During video-EEG monitoring, reviewing the video can clarify waveforms when EEG is challenging. For example, movements identified on video may correlate with a sustained rhythmic discharge signifying an artifact as opposed to a seizure (VIDEO 2-1).

Wicket spikes are unilateral or bilateral independent benign variants composed of 7- to 11-Hz bursts of anterior-mid temporal, medium- to high-voltage, monophasic waveforms (FIGURE 2-12). Bursts of wicket spikes have a negative polarity, repetitive rhythmic spikey morphology, and duration of 0.5 to 1 second. They are common in older adults, accentuated by light sleep, and appear with left temporal predominance. Isolated, sharply contoured single waveforms may occur over any head region as spikey "fragments" of a burst. The pattern may be deceiving when it is unilateral and has a temporal location and can be mistaken for a pathologic interictal epileptiform discharge.

NONEPILEPTIFORM ABNORMALITIES

Abnormal EEGs include both nonepileptiform and epileptiform activity. Nonepileptiform abnormalities include diffuse slowing in encephalopathy and focal slowing with a structural brain lesion involving the white matter tracts.[16] Neither of these abnormalities connotes epilepsy.[17] Nonepileptiform abnormalities are common in hospital and ICU EEGs. Prognosis is relative to the underlying cause of the slowing. Standard EEG is the diagnostic test of choice to provide electrophysiologic information about the presence of neurophysiologic dysfunction.

Diffuse slowing of the background activity is nonspecific and may be intermittent (FIGURE 2-17) or continuous (FIGURE 2-18). Diffuse slowing is associated with a toxic-metabolic-systemic etiology but also may be due to a

diffuse or multifocal structural cause, neurodegenerative and posttraumatic injury, or postictal state. The degree of background slowing reflects the severity of encephalopathy. A mild to moderate encephalopathy is present when the normal posterior-dominant rhythm is intermixed with theta and/or delta activity. When the posterior-dominant rhythm is in the delta range, a moderate to severe encephalopathy is present. Other features include loss of an anterior-posterior gradient, cyclical alternating patterns, discontinuous patterns with background attenuations, and asymmetries in patients with moderate to severe encephalopathy. The severity of encephalopathy is proportional to the degree of slowing of the posterior-dominant rhythm, with/without periods of voltage attenuation (FIGURE 2-19), periods of suppression, and reactivity of the electrocerebral activity to external stimulation. Burst attenuation, electrocerebral inactivity, diffuse low-voltage (less than 20 μV) or suppressed (less than 10 μV) recording (FIGURE 2-20), and coma with a monomorphic

TABLE 2-2 **Frequency Analysis and Clinical Correlates**[a]

Bandwidth	Frequency (Hz)	Selected conditions
Alpha[b]	8-13	Normal: posterior-dominant rhythm, mu rhythm, wicket waves Abnormal: alpha coma, focal (temporal) seizures
Beta[b]	13-30	Normal: medication (benzodiazepines/barbiturates), drowsiness Abnormal: drug use and abuse, breach rhythm, focal seizures (extratemporal)
Theta[b]	>3.5 to < 8	Normal: drowsiness, children, older adults, concentration Abnormal: nonspecific (mild) encephalopathy, theta coma
Delta[b]	0.5-3.5	Normal: N3 sleep, hyperventilation, posterior slow waves of youth, focal delta complexes in older adults Abnormal: encephalopathy (diffuse), white matter dysfunction (focal)
Infra-slow	<0.5	Normal: artifact (eg, sweat, movement, electrode) Abnormal: focal seizures (invasive EEG)
Gamma	30-80	Normal: volitional movement, learning, memory Abnormal: focal seizures onset (invasive EEG)
Ripples (high gamma)	80-250	Normal: cognitive processing and memory consolidation; identified in patients with chronic pain Abnormal: high-frequency oscillations, focal seizures (invasive EEG)
Fast ripples	250-500	Normal: undetermined if fast ripples are associated with normal function Abnormal: focal seizures (invasive EEG)
Very fast ripples	500-1000	Normal: acquisition of sensory information Abnormal: focal seizures (invasive EEG)

EEG = electroencephalography.
[a] Modified with permission from Tatum WO, et al, MedLink Neurology. © 2021 MedLink, LLC.[12]
[b] Bandwidths included in the "Berger band."

unreactive frequency (eg, alpha, theta coma) usually portend a poor prognosis when they occur in the appropriate clinical context (eg, hypoxic injury, subarachnoid hemorrhage) and persist off IV sedation. Loss of reactivity (FIGURE 2-3) is an unfavorable sign when present.[18] Slowing of the background activity and the absence of reactivity and periodic discharges (FIGURE 2-21) have prognostic implications.[19]

Morphologic differences of waveforms occur between recordings and within the same EEG recording of a single patient. Focal slowing that is irregular or polymorphic (FIGURE 2-22A) may suggest an underlying structural lesion involving white matter, although morphology is neither sensitive nor specific for an underlying etiology and does not suggest the potential to generate seizures

Waveforms That May Be Confused With Interictal Epileptiform Discharges

TABLE 2-3

Normal variations in EEG

- Normal frequencies (predominantly involving alpha and beta morphology and frequency)
- Vertex sharp waves (eg, spiky vertex)
- Lambda waves
- Mu rhythm
- Breach rhythm[a]
- Positive occipital sharp transients of sleep
- Frontal intermittent rhythmic delta activity (FIRDA) during hyperventilation[b]

Benign EEG variants

- Wicket waves (wicket spikes)
- 6-Hz spike-and-wave discharges
- 14- and 16-Hz positive bursts
- Benign epileptiform transients of sleep
- Rhythmic midtemporal theta of drowsiness (sharp form)
- Subclinical rhythmic EEG discharges of adults
- Frontal midline theta

Artifacts

- Over-filtering (eg, fast activity and myogenic artifact)
- Single electrode artifact
- Myogenic "spikes"
- Chewing
- Rhythmic patient-generated movements
- ECG (eg, confusion with periodic epileptiform discharges)
- Ocular flutter/rapid eye blinking

ECG = electrocardiogram; EEG = electroencephalography.

[a] Normal for the physiologic conditions of recording in the context of a skull disruption (eg, craniotomy and skull fracture).

[b] Increased intracranial pressure, recent cerebrovascular disease, severe cardiopulmonary disease, recent surgery, hyperviscosity syndromes, and pregnancy are relative contraindications to hyperventilation.

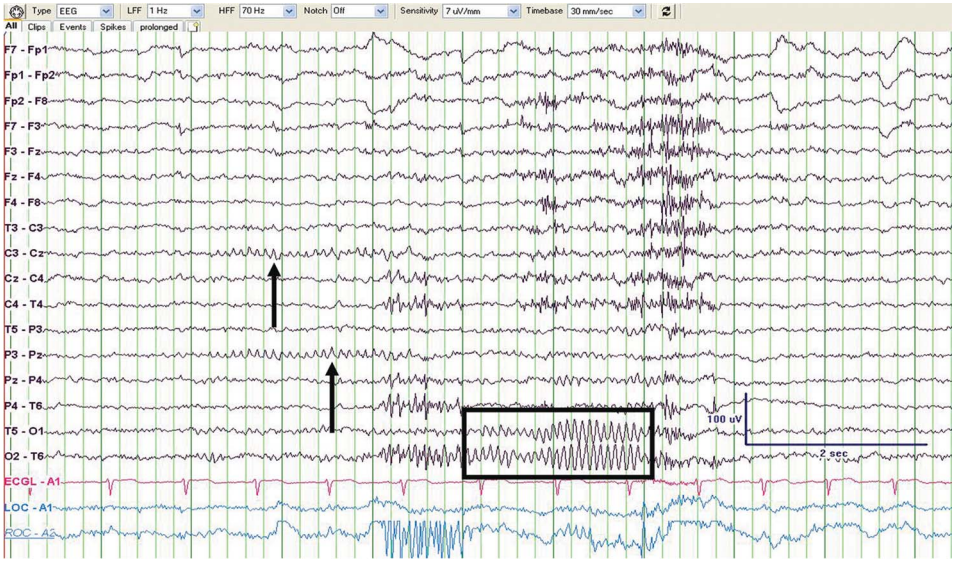


FIGURE 2-10

EEG demonstrating the contrast between mu (*arrows*) and alpha (*rectangle*). The frequency is the same, but the location (central mu versus occipital alpha) and stimulus for reactivity (eye-opening with alpha versus contralateral limb movement with mu) differ. In people with a mu rhythm, asymmetry may appear abnormal. However, both may appear "spiky."

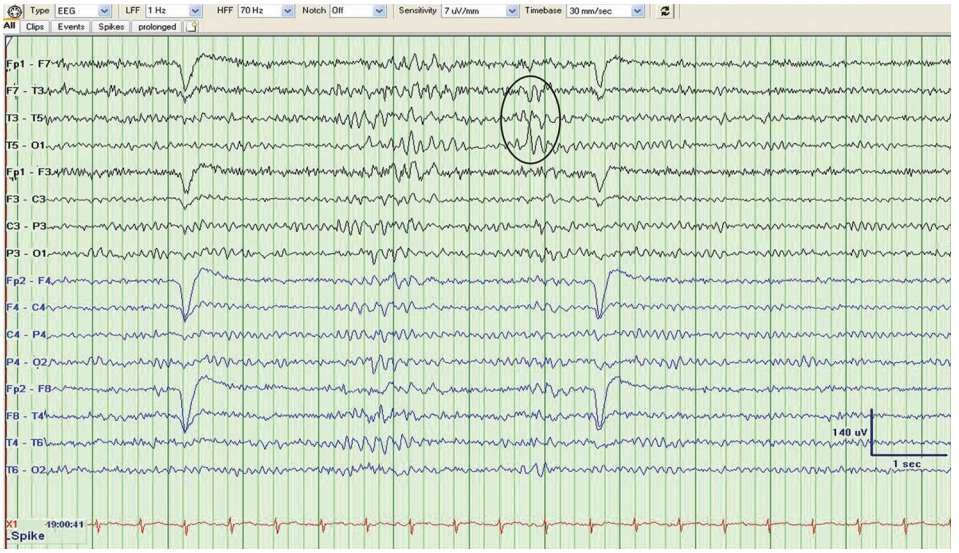


FIGURE 2-11

EEG with a normal variation of "sharp" alpha (T5) mimicking an abnormal sharp wave (*oval*) as the patient becomes drowsy. This 19-year-old woman was referred for migraine and did not have a history of seizures. EEG is not indicated for headache diagnosis; if EEG is obtained, it may serve as a pitfall that leads to overdiagnosis and mismanagement.

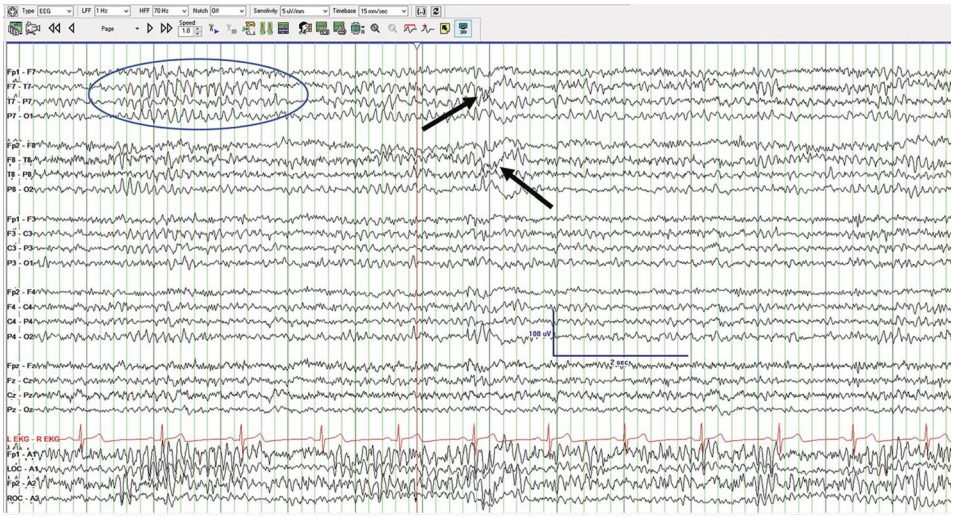


FIGURE 2-12

Normal EEG with a left temporal burst of wicket waves (*oval*). Note the single complexes of bitemporal delta (*arrows*) that are normal during drowsiness in this 68-year-old woman evaluated for suspected convulsive syncope.

(FIGURE 2-22B). However, the greater the persistence of polymorphic delta, the greater the likelihood of being associated with a destructive lesion. Intermittent generalized slowing may also appear rhythmic and may be a normal feature of the EEG during hyperventilation (FIGURE 2-6) and drowsiness (FIGURE 2-17). Temporal intermittent rhythmic delta activity (TIRDA) is unlike other forms of slowing (FIGURE 2-23), including occipital and frontal intermittent rhythmic delta activity (FIRDA). TIRDA is a unilateral or bilaterally asynchronous EEG pattern that predicts focal seizures with high likelihood in patients with temporal

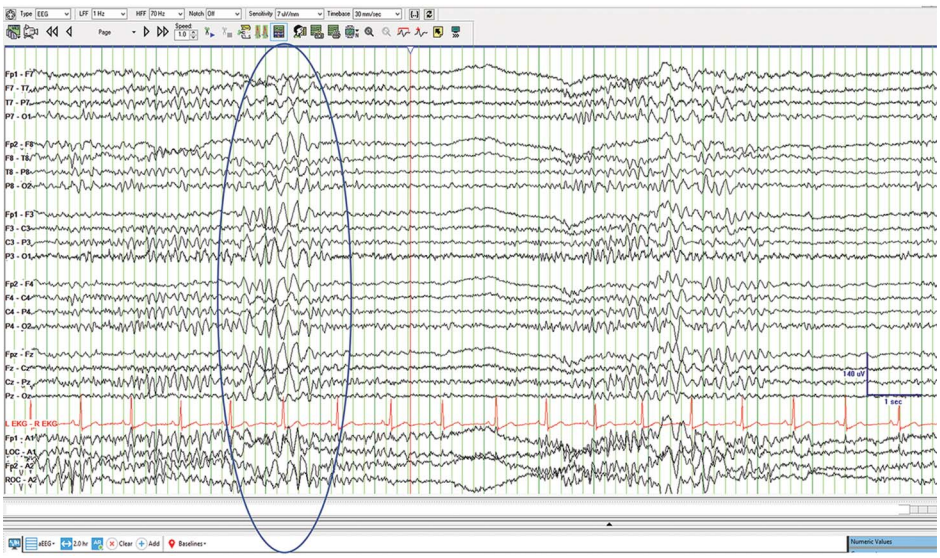


FIGURE 2-13

EEG with normal bursts of sharply contoured theta (*oval*) during transitioning between the awake and drowsy state. Some bursts may appear "sharp" but are normal. These may be pronounced and prominent in younger people.

CASE 2-1

A 65-year-old right-handed man with a past history of hypertension, transient ischemic attack, chronic dizziness, and major depression was evaluated for recurrent "blackouts." The patient described a warning of light-headedness, and coworkers described him as having sudden loss of consciousness and "shaking all over." Over the past few months, he developed an asymmetric tremor, bradykinesia, and falls. He was diagnosed with parkinsonism due to chronic neuroleptic use, and a head-up tilt table test was reported as "positive." Brain MRI had subcortical microvascular ischemic changes.

An EEG obtained in the office was interpreted as showing "seizure activity" over the left temporal region (FIGURE 2-12). Levetiracetam was begun and increased, but the patient became violent. Lamotrigine was then added, but episodes continued.

The patient then presented for a second neurologic opinion. Video-EEG monitoring was performed. Interictal EEG was normal, and abundant wicket waves were present during light sleep (FIGURE 2-14). A spell was captured during orthostatic blood pressure assessment that was typical of the patient's previous events. As a result, he was diagnosed with syncope/convulsive syncope, and rereview of the outside EEG demonstrated wicket waves. Antiseizure medication was withdrawn, and midodrine was begun without recurrence of events.

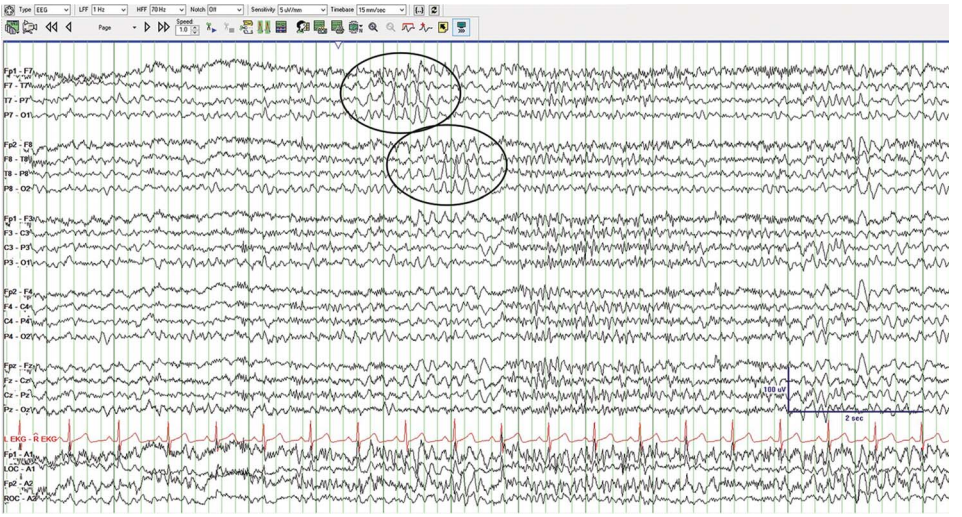


FIGURE 2-14
Independent left and right rhythmic (5 Hz) temporal theta discharges of drowsiness (*ovals*) in a patient referred for evaluation of syncope. Note the sharply contoured morphology in this patient with a benign EEG variant.

COMMENT

Benign variants of uncertain significance as in this case, normal waveform variations, and artifacts may be pitfalls to overinterpreting a normal record as abnormal, leading to inappropriate treatment with antiseizure medication.

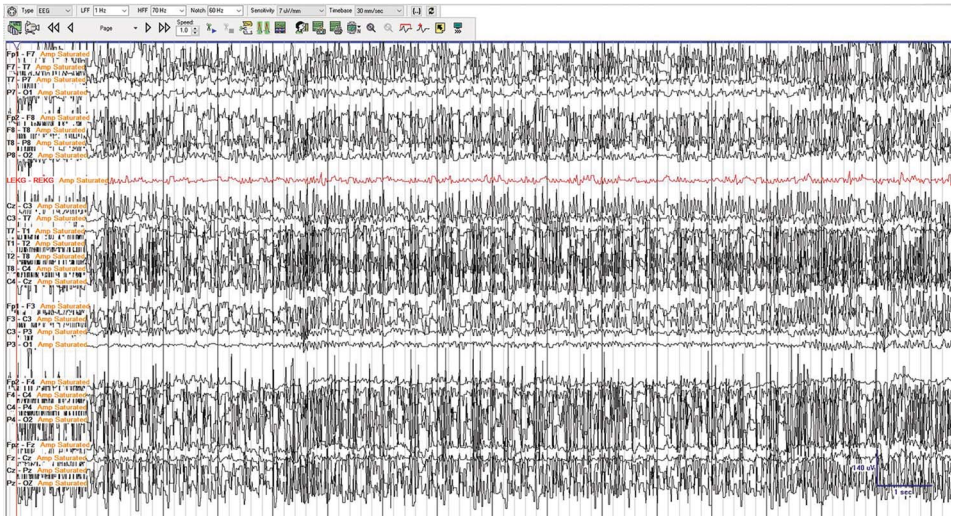


FIGURE 2-15

Diffuse myogenic and instrumental artifact due to loss of the reference electrode. Note the montage was reformatted into an anterior-posterior longitudinal bipolar array.

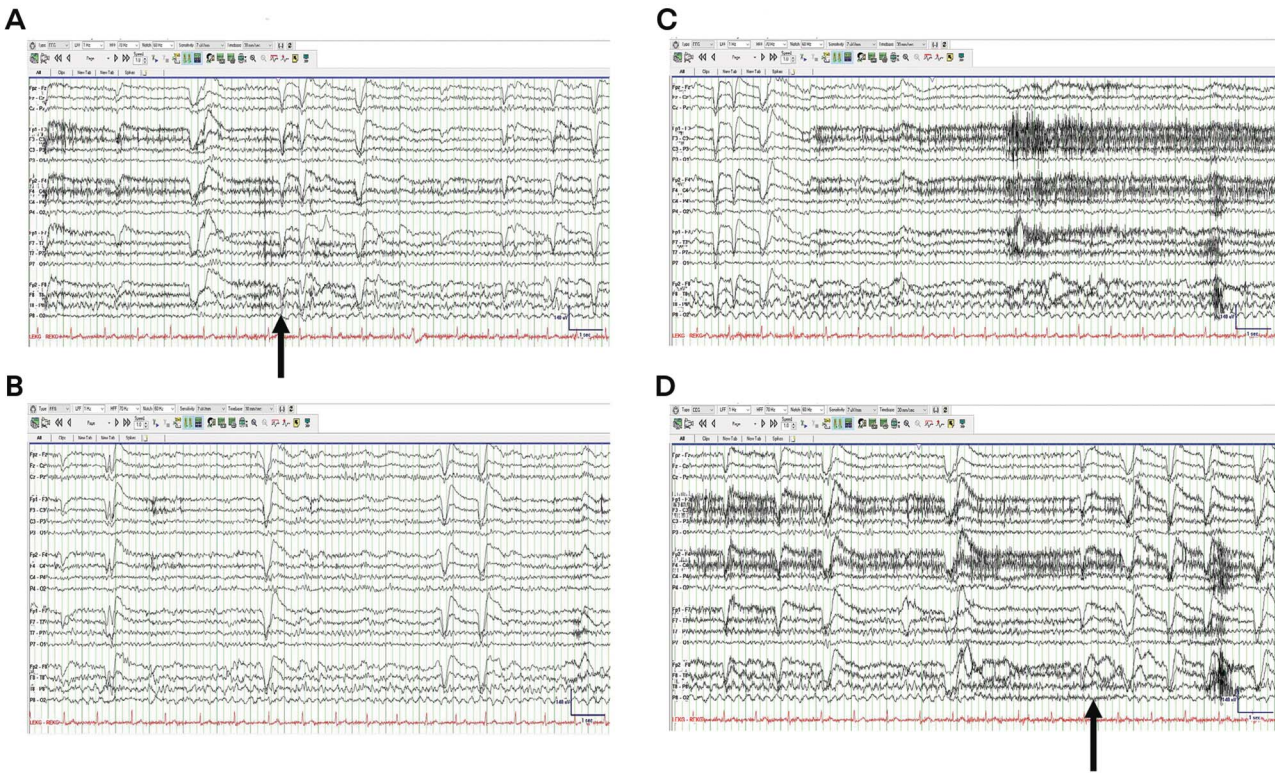


FIGURE 2-16

Epochs of EEG during a "seizure" that was suspected during initial interpretation of the EEG. Note the onset (*A, arrow*), rhythmic pattern (*B*), pseudo-evolution (*C*), and termination (*D, arrow*) in the images. The persistent focal distribution without propagation to adjacent regions, pseudo-evolution in amplitude without change in frequency, and lack of postictal slowing reflecting a hand tremor during a telephone call that produced artifact on the EEG.

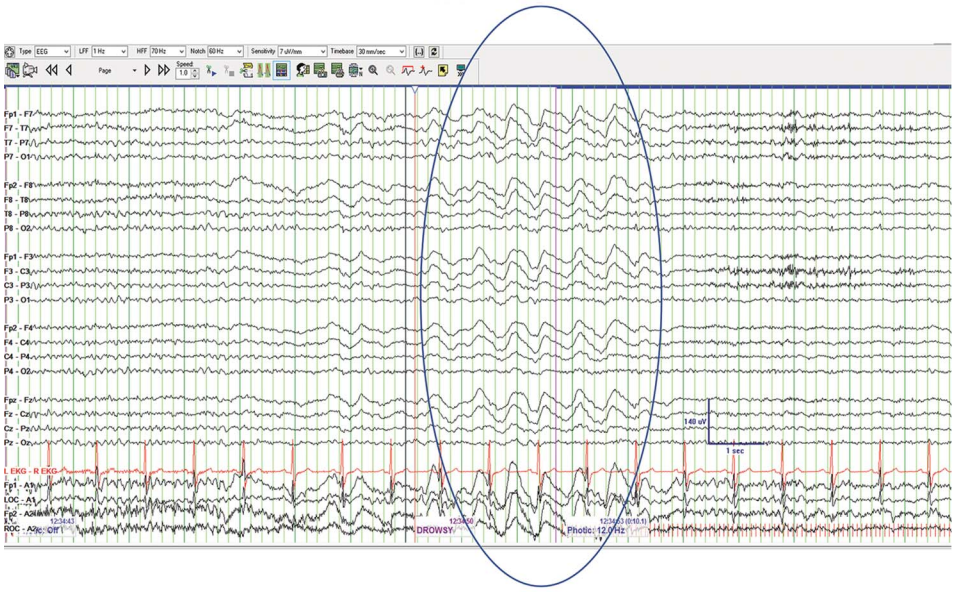


FIGURE 2-17

Brief 4-second burst of frontal intermittent rhythmic delta activity (FIRDA) during drowsiness (*oval*). This EEG was obtained in an 81-year-old man after an acute confusional state. FIRDA during the awake state is abnormal, yet it may be normal during drowsiness and hyperventilation.

lobe epilepsy.[20] Normal EEG waveforms are not perfectly symmetric. In some cases, attenuation of the waveform amplitude suggests localized gray matter dysfunction within the ipsilateral hemisphere. Amplitude differences are considered abnormal asymmetries with greater than 50% side-to-side difference (FIGURE 2-24). Voltage attenuation of brain signals normally occurs because of the effect of overriding skull and pericranial tissues limiting propagation of

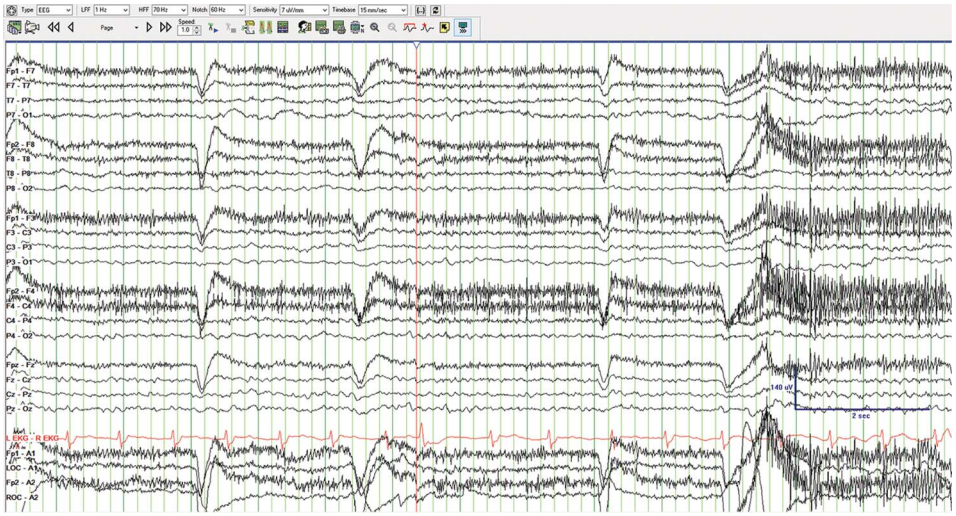
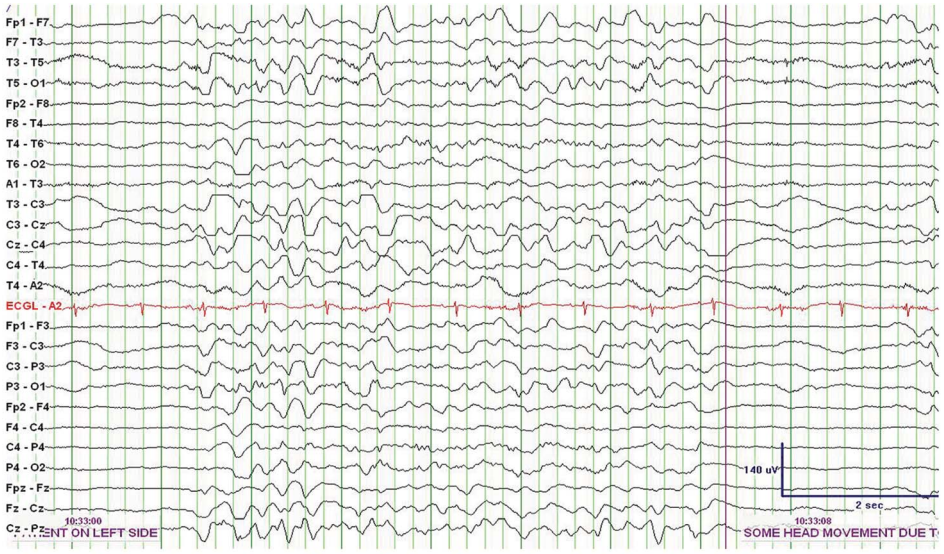


FIGURE 2-18

EEG with diffuse slowing of the posterior-dominant rhythm to 6 to 7 Hz in a 56-year-old man evaluated for mild cognitive impairment.



Sensitivity 7 μV/mm, Filters 1-70 Hz, Display speed 30 mm/second

FIGURE 2-19

Encephalopathy during rewarming from iatrogenic hypothermia with EEG showing diffuse mixed-frequency irregular background slowing coupled with attenuations (first and last 1 to 2 seconds) in a patient with hypoxic-ischemic encephalopathy and left hemispheric ischemic infarction after cardiac arrest.

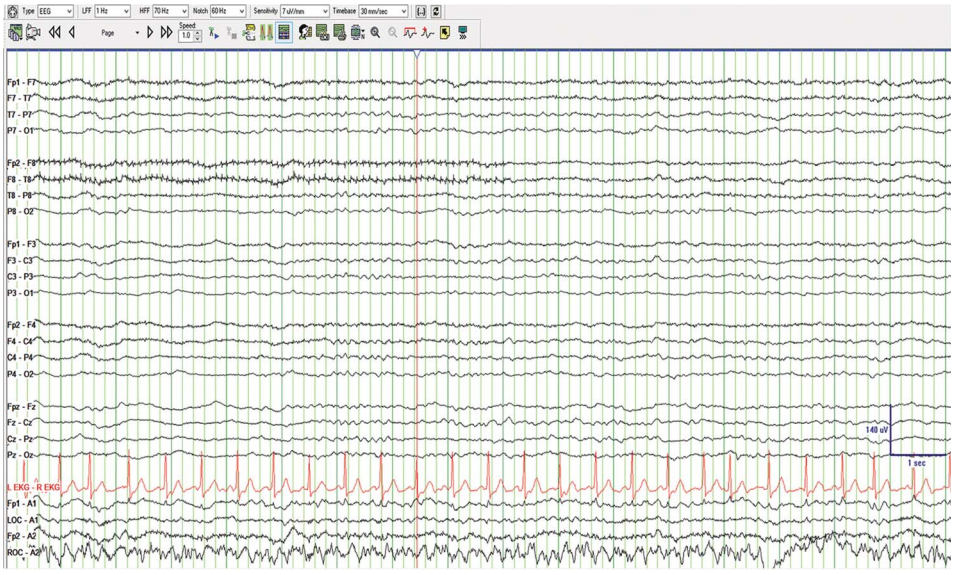


FIGURE 2-20

Low-voltage recording (10 to 15 μV) associated with a subarachnoid hemorrhage in a 56-year-old man with uncontrolled hypertension.

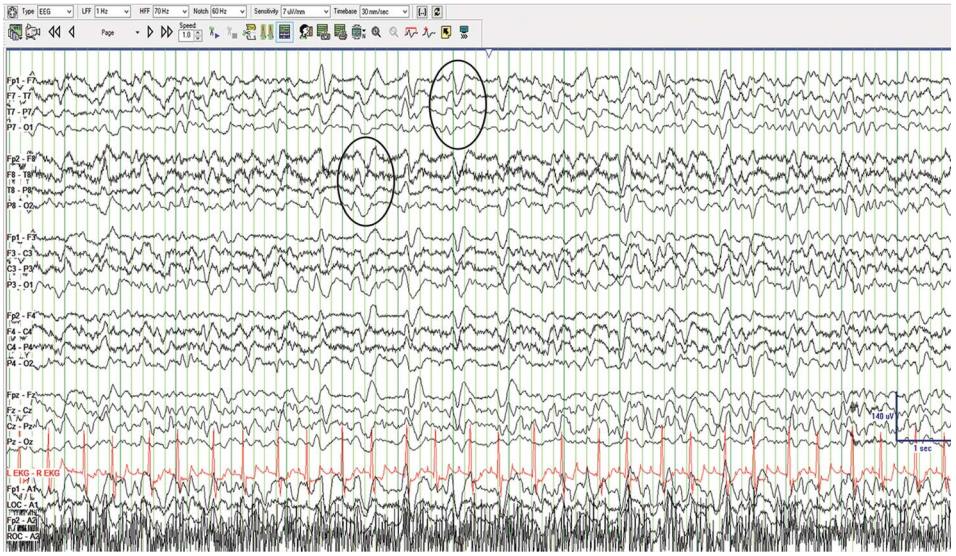


FIGURE 2-21

Generalized periodic discharges with triphasic morphology. Note the diffusely slow background activity in addition to the triphasic morphology and anterior-posterior lag (*ovals*) that supports a toxic-metabolic-systemic etiology. This was a 46-year-old woman with an orthotopic liver transplant evaluated for acute mental status changes due to sepsis.

signals to the scalp. A focal area of high-voltage electrocerebral activity (often beta) is present where the skull is breached (FIGURE 2-25).

INTERICTAL EPILEPTIFORM ACTIVITY

The interictal EEG is useful to differentiate focal from generalized seizures and epilepsies. Diagnostic criteria (TABLE 2-3) differentiate epileptiform activity from nonepileptiform transients.[21,22] Spikes and sharp waves are interictal epileptiform discharges and are surrogate biomarkers of epilepsy. Spikes have a duration of 20 to 70 milliseconds, and sharp waves have a duration of 70 to 200 milliseconds. When EEG is viewed using a display speed of less than 30 mm/s or lowering the high-frequency filter below 70 Hz, a false "spiky" appearance may be produced. Therefore, an understanding of the parameters of recording is essential for accurate interpretation (FIGURE 2-26). An EEG that contains spikes and sharp waves supports a clinical diagnosis of epilepsy. When EEG records an electrographic seizure (FIGURE 2-27) in patients with a history of recurrent unprovoked seizures, this is diagnostic of epilepsy.

The likelihood of an EEG recording interictal epileptiform discharges depends on the presence of several factors: a lesion, the location, state of arousal, spatial distribution of interictal epileptiform discharges, and the patient's clinical course. Factors that influence the ability to record interictal epileptiform discharges in the routine EEG include age, location, sleep, use of special electrodes, recording within 24 hours after a seizure, specific syndromes, antiseizure medication, and the number and length of EEG recordings.[17] Temporal interictal epileptiform discharges have a strong association with epilepsy. In contrast, central interictal epileptiform discharges are associated with clinical seizures less than 50% of the time. An underlying structural basis (eg, cerebral palsy with central spikes) and an inherited trait independent of seizures (eg, a photoparoxysmal response) are possible reasons for the difference.

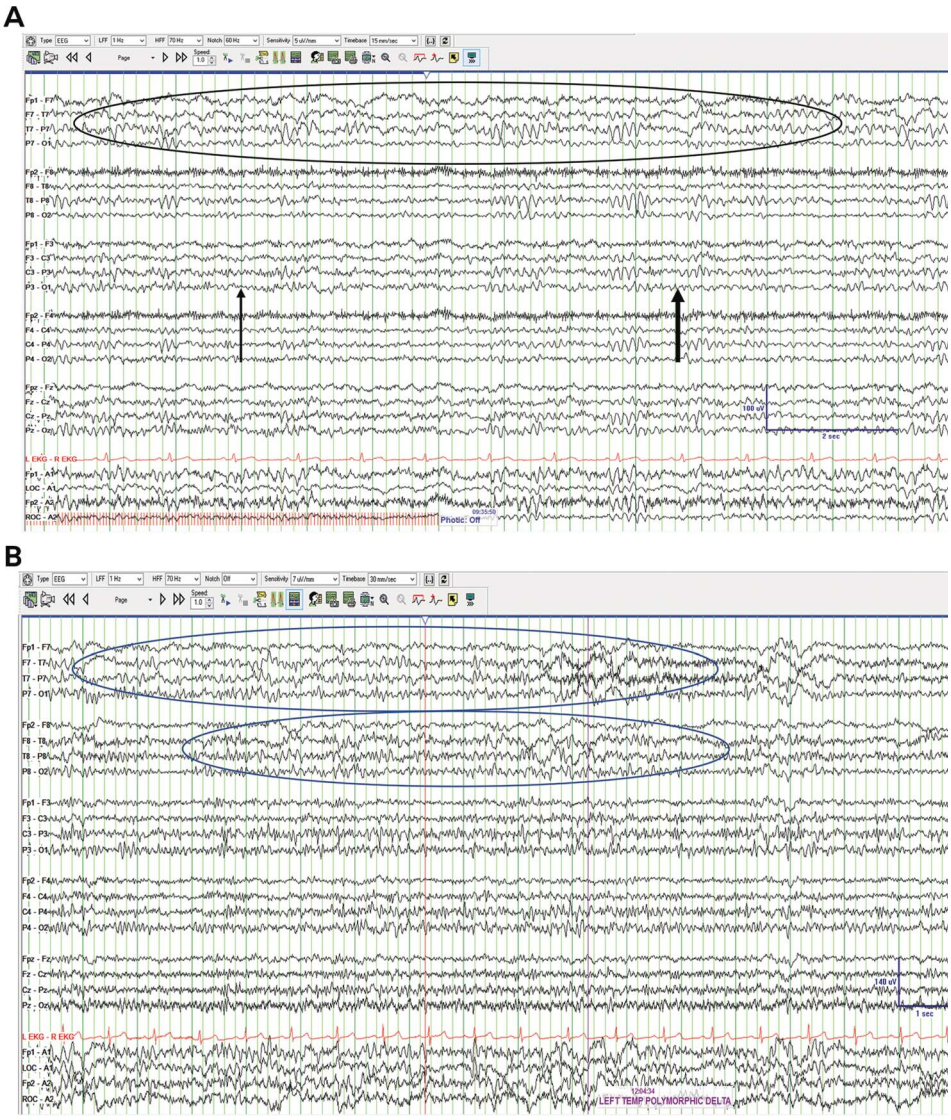


FIGURE 2-22

EEG with focal slowing. *A*, Abnormal EEG with continuous left anterior temporal slowing (*oval*) in a 60-year-old man with a normal MRI referred for memory loss and possible seizures. Note the variable hemispheric spatial field of distribution (with hemispheric involvement [*thin arrow*]; time of less involvement [*thick arrow*]). *B*, Irregular bitemporal delta (*ovals*) present in the same patient. Note the midtemporal localization and appearance of right midtemporal delta slowing at different times during video-EEG monitoring performed for quantification of subclinical seizures and subtle seizures without awareness.

KEY POINTS

- **An EEG that contains spikes and sharp waves supports a clinical diagnosis of epilepsy.**
- **When EEG records an electrographic seizure in patients with a history of recurrent unprovoked seizures, this is diagnostic of epilepsy.**

The prevalence of interictal epileptiform discharges on EEG is related to multiple factors. Nevertheless, EEG is persistently "negative" in about 10% to 15% of people with epilepsy.[3] Sleep is the best modulator of interictal epileptiform discharges and seizures and is activating in about one-third of patients with epilepsy and preferentially affecting those with genetic generalized epilepsies.[3] However, sleep may alter the frequency, morphology, and location of interictal epileptiform discharges. In patients with focal epilepsies, bilateral

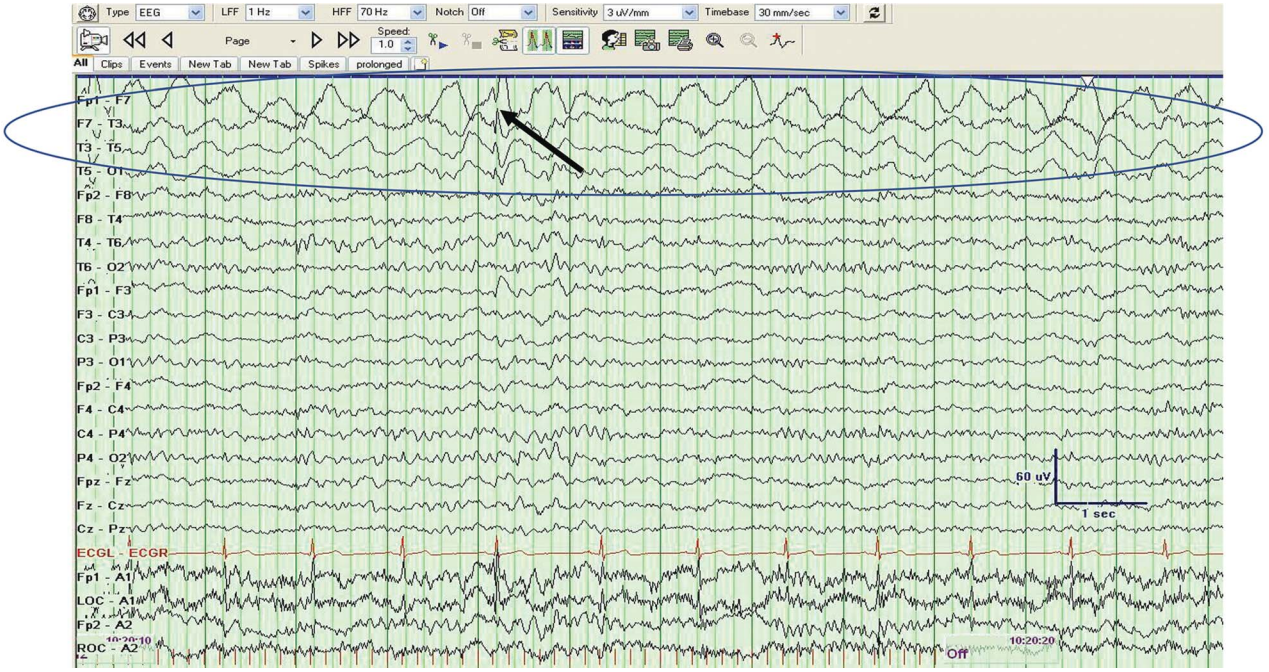


FIGURE 2-23

Left anterior temporal intermittent rhythmic delta activity (TIRDA) (*oval*) in the EEG with a left regional temporal spike (*arrow*) in second 5. TIRDA is strongly associated with temporal lobe epilepsy, unlike other forms of intermittent rhythmic delta activity. In this case, "intermittent" appears continuous during an epoch of a prolonged burst.

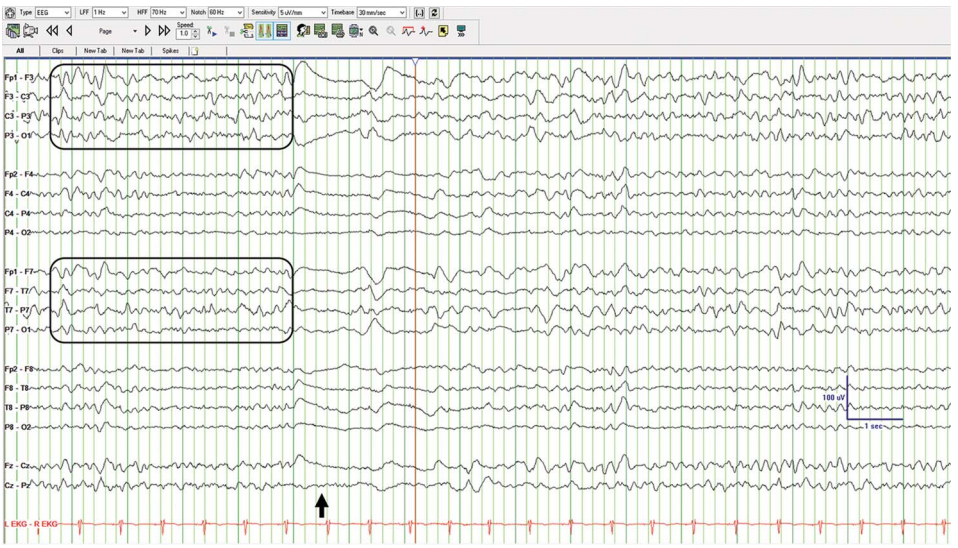


FIGURE 2-24

Amplitude asymmetry (*rectangles* over the left hemisphere) with greater than 50% difference in the left-right voltage. Diffuse attenuation (*arrow* in second 6) and background slowing is also present in a 46-year-old woman with an intraparenchymal hemorrhage after rupture of a berry aneurysm with a subsequent left hemicraniectomy. Focal asymmetries associated with focal slowing are abnormal.

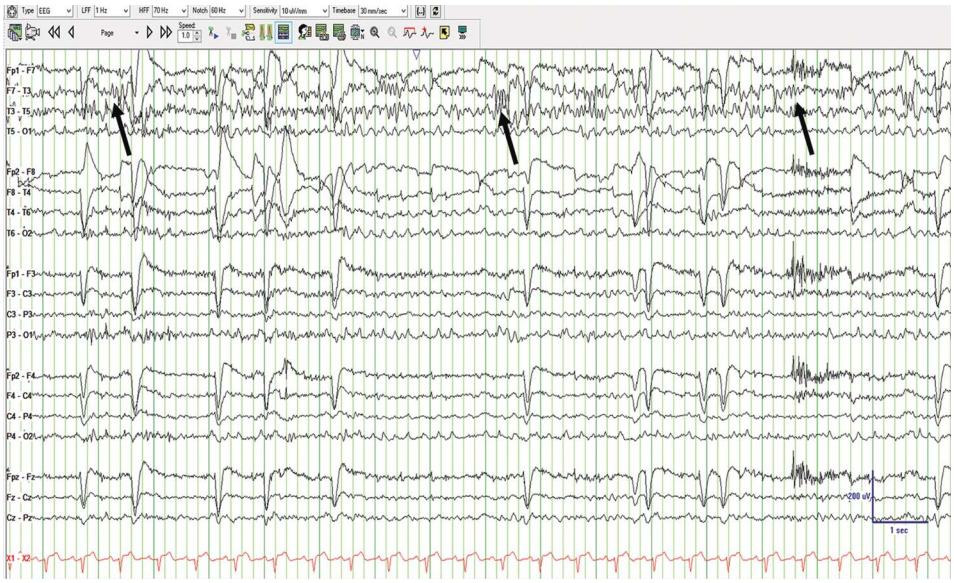


FIGURE 2-25

Left midtemporal breach rhythm in a 26-year-old woman with prior left temporal lobectomy for drug-resistant temporal lobe epilepsy. She was seizure free for 2 years, and EEG was obtained to evaluate the risk of antiseizure medication taper. No interictal epileptiform discharges were present. Note the breach rhythm (*arrows*) were associated with left temporal theta and delta slowing.

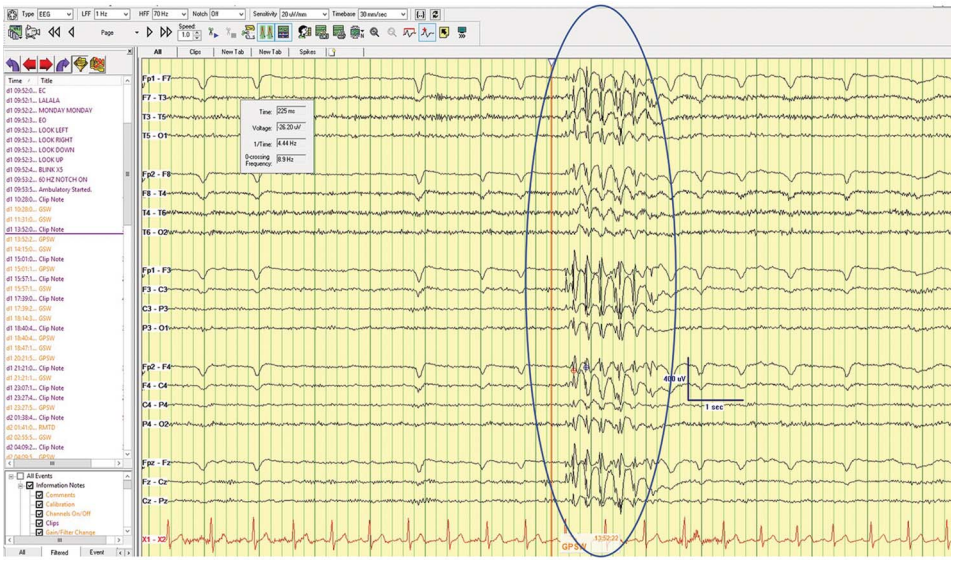


FIGURE 2-26

EEG in a 21-year-old with juvenile myoclonic epilepsy with 4.5-Hz left greater than right generalized spike-and-wave (*oval*) 1-second burst. A "fast" frequency is present at 4.44 Hz (see inset). Note the written entries made by the technologist on the left-hand side of the EEG (the annotation viewer) indicating frequent generalized spike and waves. Left hemispheric predominance can be seen in this discharge but no focal evidence to support secondary bilateral synchrony.

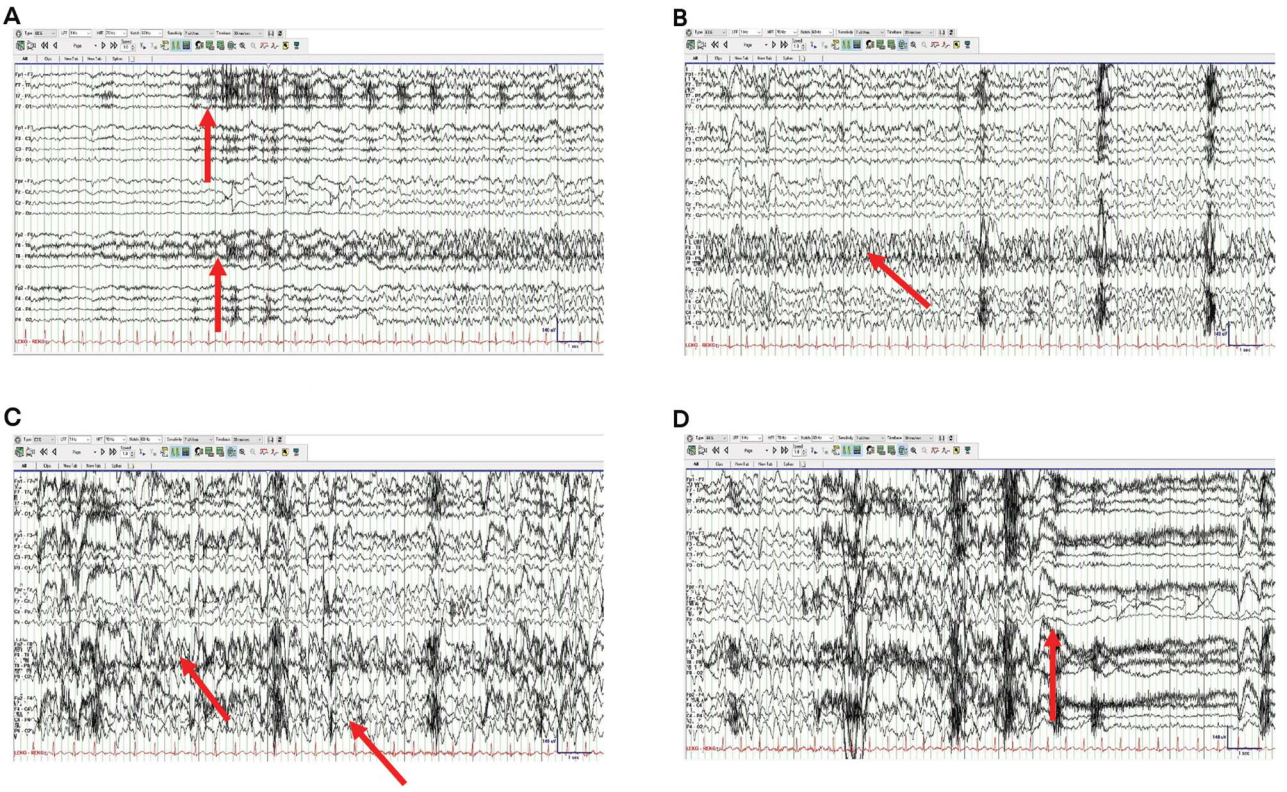


FIGURE 2-27

Right temporal seizure evolving. Note the obscuration by bitemporal artifact at onset (*A*, *arrows*), right temporal maximum with T8 phase reversal (*B*, *arrow*), right hemispheric predominance (*C*, *arrows*) before bilateral rhythmic ictal theta, and abrupt termination and postictal background attenuation (*D*, *arrow*).

discharges may appear, and polyspike formation and slower interspike intervals may emerge in deeper stages of sleep as interictal epileptiform discharges may become irregular. Overnight recording with ambulatory EEG may reveal interictal epileptiform discharges on awakening in patients with genetic generalized epilepsy (FIGURE 2-28). Patients with genetic generalized epilepsy treated with valproate may suppress generalized spike and wave on EEG. Lamotrigine may reduce photosensitivity, and benzodiazepines may reduce interictal epileptiform discharges acutely. However, in patients with focal epilepsies, antiseizure medication does not significantly alter the interictal epileptiform discharge frequency. Electrographic or electroclinical focal seizures have a characteristic EEG pattern beginning abruptly at onset, evolving in frequency and spatial distribution, followed by an abrupt offset with postictal slowing (FIGURE 2-27). With scalp EEG, the location of interictal epileptiform discharges and focal seizures does not always indicate the same location for the source of abnormality or epileptogenicity.

Asymptomatic individuals may rarely demonstrate interictal epileptiform discharges. EEGs with centrotemporal spikes, occipital spikes, and a self-limited photoparoxysmal response may manifest in people as an inherited trait, independent of clinical seizures. Congenital blindness ("needle spikes"), cerebral palsy (central spikes), autism spectrum disorder (temporal spikes), a sibling/

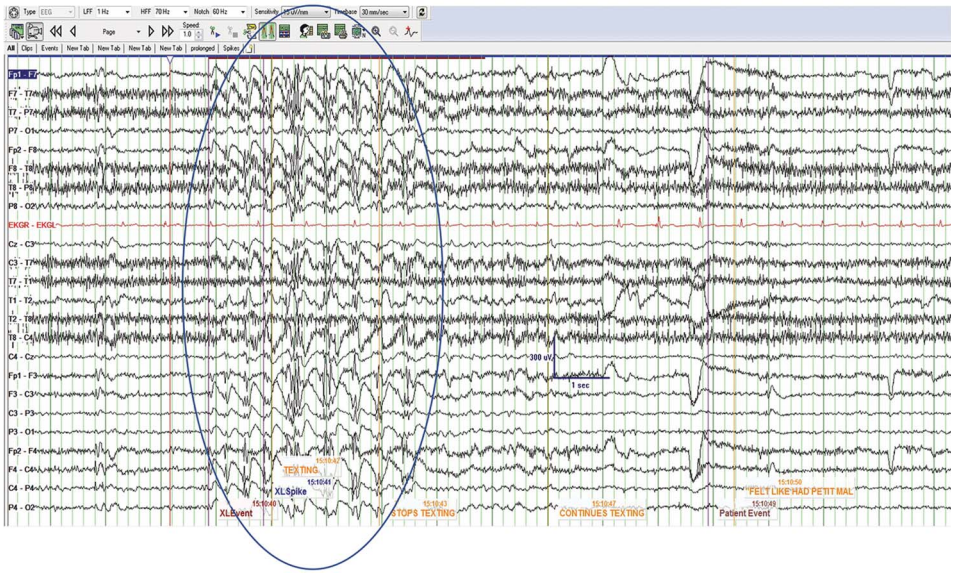


FIGURE 2-28
Ambulatory EEG without video demonstrating a 5-second burst of generalized spike-and-wave discharges (*oval*) detected in a patient with sleep-related epilepsy shortly after awakening with episodes of morning confusion.

KEY POINTS

- **People who experience a first seizure are at risk for recurrence when EEG demonstrates abnormal interictal epileptiform discharges.**
- **When history and other imaging modalities are considered in addition to an ictal EEG, epilepsy syndromes can usually be defined for the purpose of providing optimal treatment.**

family member with epilepsy (generalized spike and wave), and medications (eg, antipsychotics, lithium, baclofen) (atypical generalized spike and wave) may demonstrate interictal epileptiform discharges as an epiphenomenon of a nonepileptic condition.

People who experience a first seizure are at risk for recurrence when EEG demonstrates abnormal interictal epileptiform discharges (CASE 2-2). Overall, routine EEG yields interictal epileptiform discharges in approximately one-third of initial recordings in patients who are referred for evaluation of seizures. For some patients, the yield increases when performed within 24 hours of a seizure, after sleep deprivation, or when the recording duration is prolonged.[24] Repeating several EEGs increases the yield even further. When interictal epileptiform discharges are present on EEG, level A evidence supports the likelihood of seizure recurrence within the first 2 years.[25] An abnormal nonepileptiform EEG increased the risk of recurrence to 40%, but interictal epileptiform discharges increased the risk to more than 60% to establish a working diagnosis of epilepsy,[23] prompting discussion with patients regarding antiseizure medication.[25] Reevaluating patients who are seizure free for 2 to 5 years with EEG is less predictive when antiseizure medication taper is considered.[3,25,26] The risk is favorable when interictal epileptiform discharges remit and greater risk is present when interictal epileptiform discharges persist, especially when they include generalized polyspike and wave and generalized spike and wave (FIGURE 2-31).[27]

ICTAL EEG CLASSIFIES EPILEPSIES

Seizure type(s) and epilepsy syndromes are classified based on event-related signs and symptoms of an event, supplemented with EEG, MRI/CT, and laboratory findings.[27] When history and other imaging modalities are

considered in addition to an ictal EEG, epilepsy syndromes can usually be defined for the purpose of providing optimal treatment.[28] Focal seizures are the most common form of human adult epilepsies, and temporal lobe epilepsy is the most common syndrome.[29] In temporal lobe epilepsy, spikes are located over the anterior temporal regions in more than 90% of patients. Bitemporal independent interictal epileptiform discharges are present in more than one-third of cases of temporal lobe epilepsy when recording is prolonged.

A focal aware seizure (also known as "aura") will be visible in a standard EEG in only 30% of cases. Focal impaired awareness seizures associated with temporal

CASE 2-2

A 45-year-old man with a history of hypertension and bipolar disorder had no risk factors for epilepsy. He attended a social event for his job where he reportedly drank several alcoholic beverages. He returned home inebriated and later experienced a spell during sleep. At 2:00 AM, his wife awoke to a guttural noise. When she turned on the light, she saw her husband "flailing around in bed." He was initially unarousable, but after a minute, she noted blood trickling down the right side of his mouth, which was caused by a tongue laceration (FIGURE 2-29). She called emergency medical services, and he was taken by ambulance to the closest emergency department.

He had partial recall of the transport but denied loss of awareness, noting "I just had a bad dream." Brain CT was normal. The patient was sleepy, but an EEG was normal. Overnight, bedside EEG demonstrated bitemporal spike and waves (FIGURE 2-30). He was placed on antiseizure medication with seizure precautions and remained seizure free.

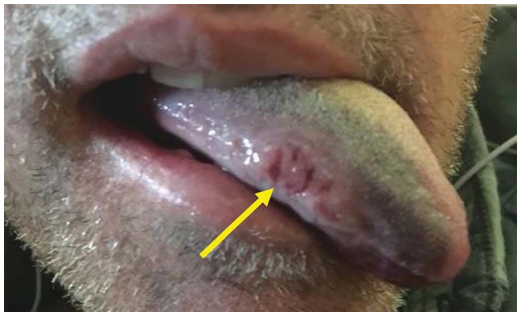


FIGURE 2-29
Right lateral tongue laceration (*arrow*) in the patient in CASE 2-2 after a first nocturnal "spell" suspected to be caused by a generalized tonic-clonic seizure.

COMMENT

When encountering a limited historical description for spells, a witnessed paroxysmal event, congruent symptomatology, occurrence out of sleep, lateral tongue laceration, partial amnesia for the event, and potential triggers portend a high risk for seizure recurrence. This establishes a working diagnosis of epilepsy despite the occurrence of a single seizure and should prompt discussion with the patient regarding implementation of antiseizure medication.[23] When EEG is repeated or prolonged, the yield of identifying interictal epileptiform discharges is enhanced (TABLE 2-4). In this case, the patient was appropriately diagnosed and successfully treated for epilepsy.

lobe epilepsy manifest a unilateral temporal rhythmic 5- to 9-Hz ictal theta discharge (VIDEO 2-2). Lateral (neocortical) temporal lobe epilepsy is more likely to demonstrate evolving rhythmic ictal delta at seizure onset with a broad hemispheric field of spatial distribution present. Focal or lateralized postictal slowing is often present but is less localizing than EEG at seizure onset. Localizing ictal EEG in patients with extratemporal epilepsies seldom occurs. Diffuse, nonlocalized seizures are more likely to be seen with various morphologies present. In frontal lobe epilepsies, interictal EEG is often normal and may appear hemispheric or bifrontal, diffuse, or as midline interictal epileptiform discharges. Secondary bilateral synchronous interictal epileptiform discharges appear

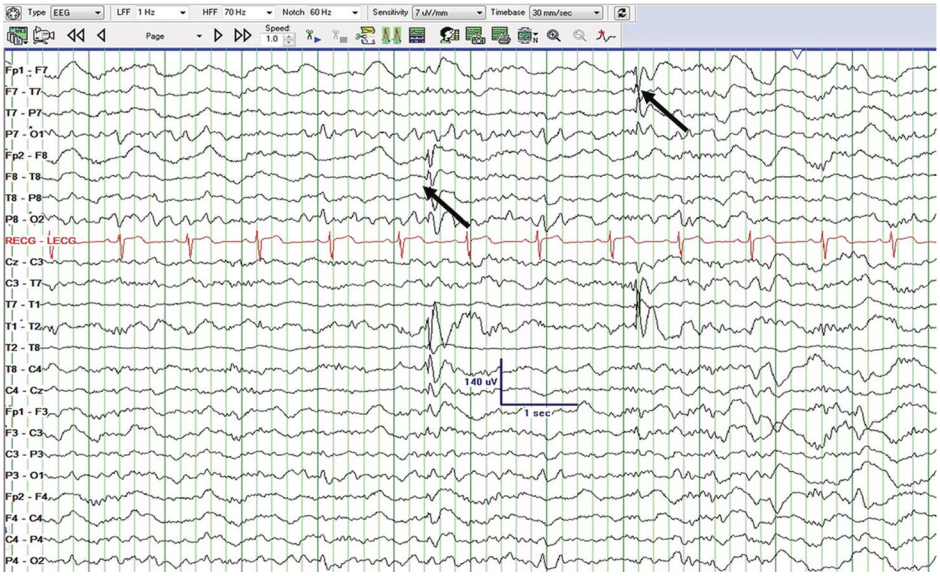


FIGURE 2-30

EEG from the patient in CASE 2-2. Bilateral independent anterior temporal spike-and-wave complexes (*arrows*) with a regional temporal field of spatial distribution activated by N2 and N3 sleep are seen on overnight video-EEG monitoring. The cross-chains of the anterior-posterior and transverse bipolar montages intersect, localizing interictal epileptiform discharges to the temporal region.

"generalized" but reflect focal onset. Focal epilepsies with interictal epileptiform discharges and secondary bilateral synchrony can be differentiated from generalized spike and wave in genetic generalized epilepsy[30]; a lead-in time of the initial discharge should occur for 2 seconds or more, and the morphology of the triggering spikes should resemble other focal spikes from the same region. Also, the morphology of focal interictal epileptiform discharges should differ from the bisynchronous interictal epileptiform discharges. When not obscured by artifact, the ictal EEG in frontal lobe epilepsy may demonstrate bilateral nonlateralized voltage attenuation, slowing, or epileptiform features. Low-voltage fast activity at seizure onset (eg, gamma activity and high-frequency oscillations) favorably suggest a dorsolateral frontal localization. Normally it is attenuated by the skull (or obscured by artifact) until propagated to higher-amplitude slower frequencies detectable by scalp EEG. Interictal epileptiform discharges in parietal and occipital lobe epilepsies are infrequent. Like the ictal EEG of frontal lobe epilepsy, the EEG is poorly localized, bilateral, or even falsely lateralized to the ipsilateral temporal region and rarely has well-localized seizures (FIGURE 2-32) and interictal epileptiform discharges.[31]

In generalized epilepsies, the discharges present in EEG are symmetrical, synchronous, frontally dominant, generalized spike and wave, and generalized polyspike and wave recurring at 3 Hz or higher. Interictal epileptiform discharges on EEG in conjunction with a normal background typically are present in genetic generalized epilepsy syndromes, yet no generalized interictal epileptiform discharges are specific for a seizure type or epilepsy syndrome.[32] Most seizures provoked by activation are absences or myoclonic seizures and rarely a generalized tonic-clonic seizure.[33] Interictal features of genetic generalized epilepsy are nonspecific and may demonstrate generalized spike and wave, generalized polyspike and waves, or a combination that is associated with one or a combination of generalized seizures including myoclonus, absence, and generalized tonic-clonic seizures. Generalized interictal epileptiform discharges appear bilateral with maximal voltage present in the anterior head regions of the EEG. Lateralized (FIGURE 2-26) or "fragmented" generalized interictal epileptiform discharges, like the appearance of lateralized seizure behavior, may

TABLE 2-4

Recommendations to Identify Interictal Epileptiform Discharges

- Waveforms differ in duration (frequency) from the surrounding background activity
- The amplitude of the waveform stands out from the background, and the waveform disrupts the ongoing background activity
- The waveform has an asymmetric contour with a steep upslope relative to the downstroke
- Waveforms may be either diphasic or triphasic with a pointed peak
- The waveform is often followed by an aftergoing slow wave
- The waveform coexists in the area of abnormality (eg, a sharp wave occurs in the same region as focal slowing), and the orientation of the dipole corresponds with a source in the brain

Independently, none of the criteria qualify individually to distinguish an interictal epileptiform discharge or not.[22]

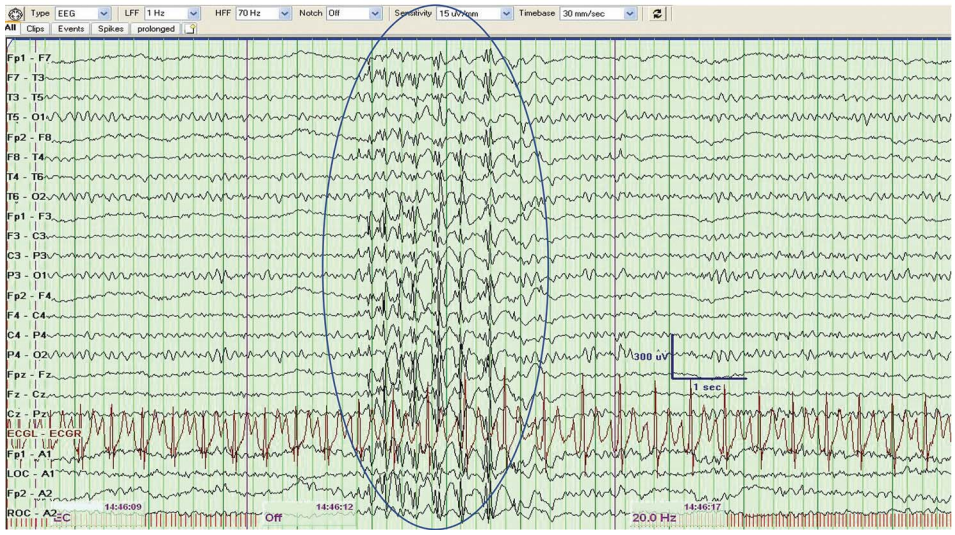


FIGURE 2-31

Burst of generalized bifrontally dominant 5- to 5.5-Hz polyspike and spike-and-slow-wave discharges (*oval*) asymptomatic during drowsiness. This 22-year-old woman reported generalized tonic-clonic seizures at night beginning at age 12. Video-EEG monitoring demonstrated absence seizures on awakening and a syndromic diagnosis of juvenile absence epilepsy.

confuse accurate classification of generalized seizures and incorrectly lead to the false diagnosis of focal epilepsy. Selection of antiseizure medication may be guided by EEG when the historical recount for an observed symptomatology is unable to classify the seizures or an epilepsy syndrome.

Diffuse or multifocal structural brain pathology is present in patients with epileptic encephalopathies and developmental disorders. Cognitive comorbidity

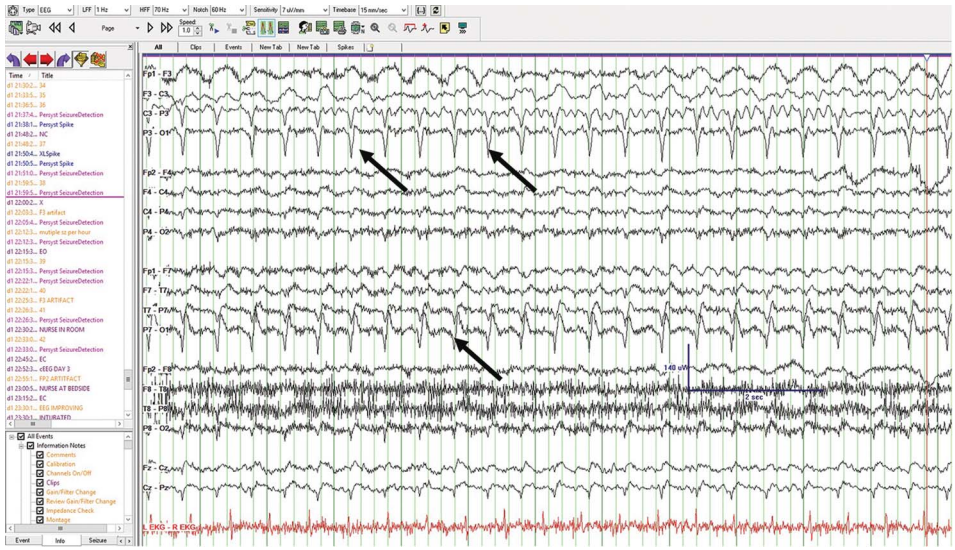


FIGURE 2-32

Continuous EEG in the intensive care unit with left occipital focal subclinical seizure (*arrows*). Occipital seizures are rarely well localized to one hemisphere as in this case. Note the evolving field and the annotation viewer with more than 40 nonclinical seizures.

KEY POINT

- **Selection of antiseizure medication may be guided by EEG when the historical recount for an observed manifestation is unable to classify the seizures or an epilepsy syndrome.**

is felt to be exacerbated by frequent interictal epileptiform discharges on EEG.[34] A symptomatic etiology is represented by diffusely slow background activity and focal or multifocal nonepileptiform and epileptiform features. An interictal EEG demonstrating slow spike and waves (FIGURE 2-33) is the hallmark of Lennox-Gastaut syndrome, the prototypic epileptic encephalopathy. A diffusely slow background, multifocal independent spike discharges, and bursts of generalized paroxysmal fast activity (FIGURE 2-34) are characteristic features. EEG with generalized paroxysmal fast activity manifest as 10- to 25-Hz frontally dominant spikes during non–rapid eye movement (REM) sleep correlates with tonic seizures.

LONG-TERM EEG MONITORING

Standard EEG has utility for diagnosis and treatment in patients with epilepsy (TABLE 2-5). Long-term EEG monitoring encompasses several forms of video-EEG monitoring including short-term scalp video-EEG, ambulatory EEG with video, and inpatient continuous EEG with video in the epilepsy monitoring unit or ICU.[35] Ambulatory EEG with video recorded over 24 to 72 hours is a portable, outpatient strategic alternative to inpatient video-EEG monitoring when a high diagnostic pretest probability is present (FIGURE 2-28), but inpatient video-EEG monitoring is required for safe withdrawal of antiseizure medication to characterize the electroclinical seizure onset zone for epilepsy surgery.
The yield of video-EEG monitoring for nonspecific "spells" is high. A definitive diagnosis of epilepsy is established when epileptiform activity is present during a seizure, and a nonepileptic event is supported by the absence of physiologic changes in the EEG background activity during a paroxysmal neurologic event where awareness is lost (FIGURE 2-35). When a clinical seizure is accompanied by an ictal discharge on EEG, an epilepsy diagnosis is confirmed, and seizure classification (FIGURE 2-36) may be deduced for management.

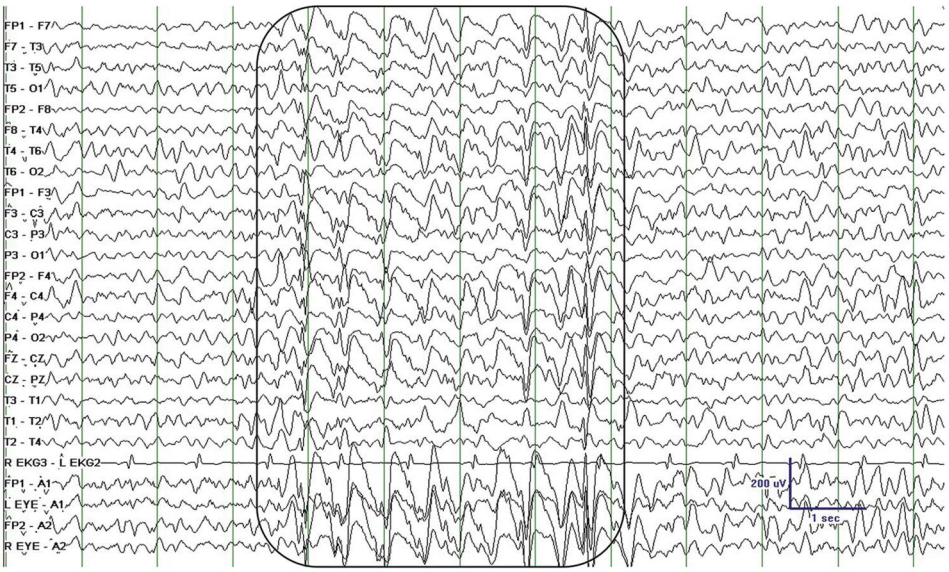


FIGURE 2-33
Slow spike and waves (*rectangle*) in a patient with Lennox-Gastaut syndrome. Note the 2-Hz interspike frequency and diffusely slow background of 5 Hz.

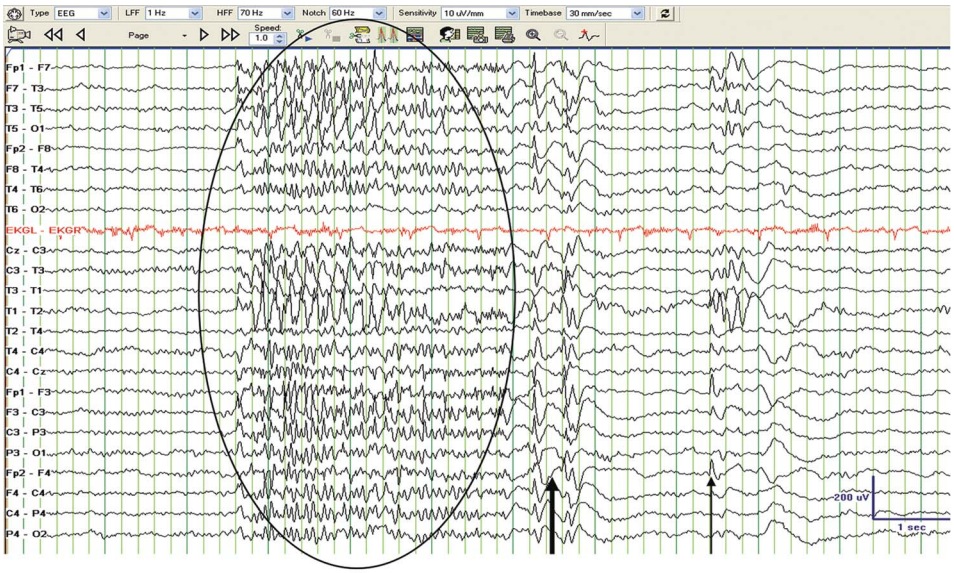


FIGURE 2-34

Generalized paroxysmal fast activity (*oval*) with mixed left temporal polysharp and slow waves followed by a burst of slow spike and waves (*thick arrow*) and bifrontal sharp waves (*thin arrow*).

Utility of EEG in People With Epilepsy

TABLE 2-5

Diagnosis

- Provide objective support for a clinical diagnosis of epilepsy
- Evaluate patients with paroxysmal neurologic events for the presence or absence of epileptiform activity
- Classify focal versus generalized seizure type(s) and epilepsy syndrome(s)
- Quantify seizure frequency and burden of epileptiform activity

Treatment

- Assess recurrence risk after a first seizure to determine treatment need
- Select antiseizure medication based on seizure/epilepsy classification
- Estimate risk of seizure relapse when planning antiseizure medication withdrawal
- Characterize the electroclinical features of seizures for surgical therapy[a]
- Monitor the course of antiseizure medication treatment (eg, during critical care)[a]
- Aid neurosurgeons during brain surgery to identify regions of importance[a]

[a] Continuous EEG is needed.

The cumulative yield of EEG to record interictal epileptiform discharges during long-term EEG monitoring increases relative to the duration of recording.

A significant minority of people are self-unaware of experiencing seizures despite impaired consciousness and overt signs that are visible to other individuals. Approximately 20% to 30% of patients are never aware of their seizures.[36] Nocturnal seizures (FIGURE 2-28), subclinical (electrographic) seizures (FIGURE 2-37), and focal seizures without self-awareness may be objectively quantified with long-term EEG monitoring. In patients experiencing frequent seizures or events, such as those with Lennox-Gastaut syndrome (FIGURE 2-33 and FIGURE 2-34), short-term outpatient EEG may provide information on event frequency and duration.[37] EEG may be modified by antiseizure medication, which can reduce interictal epileptiform discharges.[38] Selected patients with drug-resistant focal epilepsy who have their source localized on scalp ictal EEG patterns in noneloquent regions confirmed by long-term EEG monitoring are favorable candidates for epilepsy surgery, whereas those with a multifocal or poorly localized seizure onset have a less successful outcome (CASE 2-3).[39]

Classification of focal versus generalized seizures and epilepsies may at times be challenging. Juvenile myoclonic epilepsy (JME) is a "great mimicker" and the most common genetic generalized epilepsy, representing about 10% of all epilepsies. Fast spike-and-wave runs and prolonged epileptiform bursts of 3 seconds or longer, including photoparoxysmal and hyperventilation-induced runs, were associated with persistent seizures in JME.[40] Seizure onset in adolescence and myoclonus are the cornerstone of JME, but generalized tonic-clonic seizures occur in more than 90% of patients and usually prompt evaluation and management. Standard EEG is often normal, but interictal epileptiform discharges composed of "fast" (3 to 6 Hz) generalized spike-and-wave and generalized polyspike-and-wave discharges (FIGURE 2-28) arise mostly

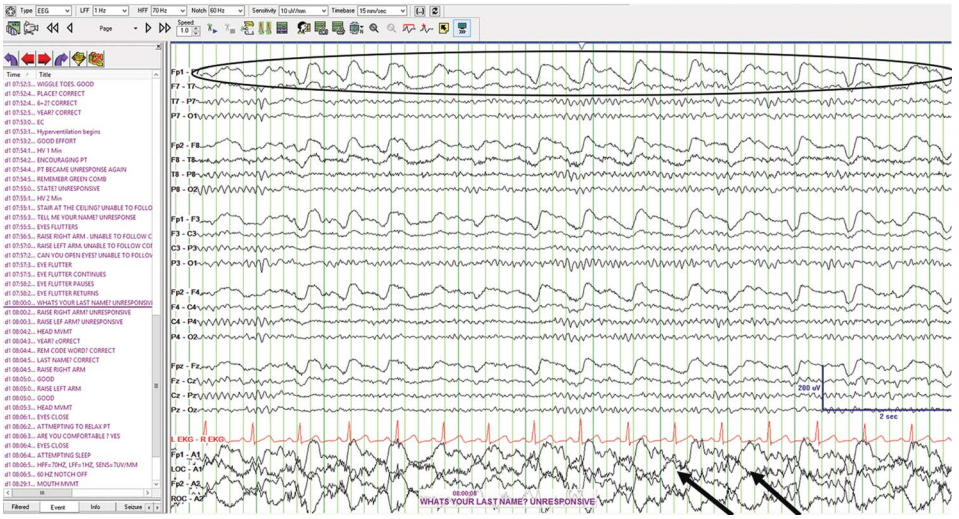


FIGURE 2-35

EEG during a psychogenic nonepileptic attack with unresponsiveness with normal EEG. During nonepileptic movements, artifact may impair the ability to observe the underlying cerebral activity. Anterior "slowing" (*oval*) is caused by eye flutter artifact validated by eye movement monitors that show "out-of-phase" deflection (*arrows*).

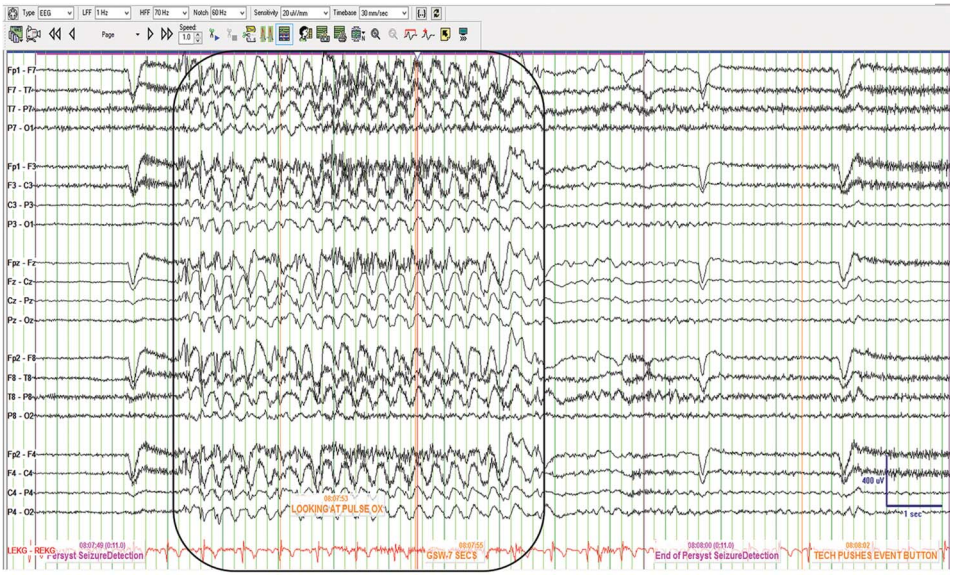


FIGURE 2-36

Prolonged burst of generalized 3- to 4-Hz generalized spike and waves (*rectangle*) without response testing. The duration of 7 seconds carries a concern for brief alteration of awareness. Spike-and-seizure burden can be quantified during long-term EEG monitoring.

from sleep. Ictal EEG in JME demonstrates symmetrical generalized frontally dominant 10- to 16-Hz polyspike discharges coalescing into continuous myogenic artifact with tonic stiffening; these are followed by phasic myogenic artifact during clonic jerking (FIGURE 2-38), terminating with postictal generalized EEG suppression. Photosensitivity is observed in 30% of patients. Although focal and generalized epilepsies can coexist, it is quite rare.[30]

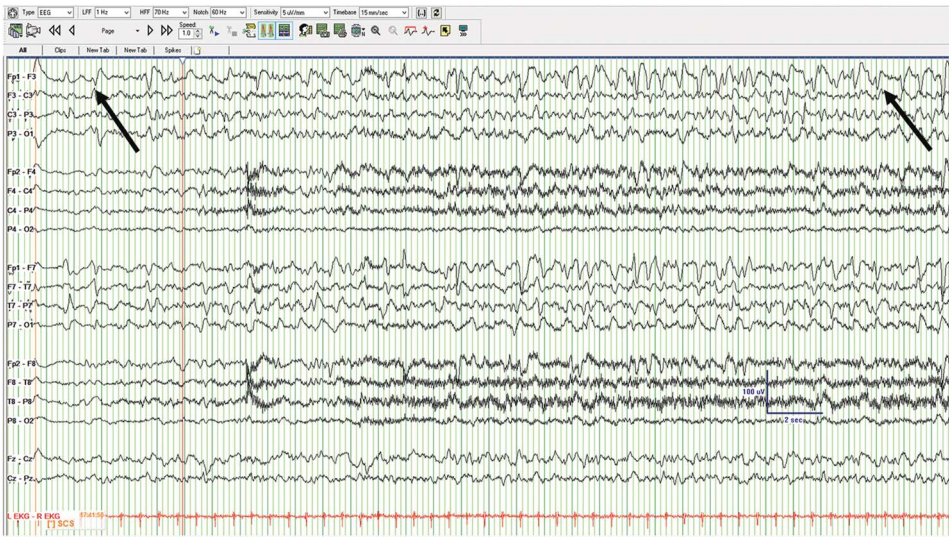


FIGURE 2-37

Subclinical seizure (*arrows*) due to a left hemispheric intraparenchymal hemorrhage in a 59-year-old comatose man. Evolution was subtle and enhanced by using a 15-mm/s (slow) display speed.

KEY POINTS

- **A significant minority of people are self-unaware of experiencing seizures despite impaired consciousness and overt signs that are visible to other individuals.**
- **Video-EEG classifies focal and diffuse cerebral dysfunction and can support an epilepsy syndromic diagnosis that aids in medical and surgical management.**

CRITICAL CARE EEG

Standard EEG is helpful when evaluating hospital and ICU patients with encephalopathy, changes in cognition and behavior, disorders involving ischemic brain disease, intermittent and episodic movements, and coma and has been used as an adjunct in the clinical determination of brain death.[41] Continuous EEG monitoring in critically ill patients in the ICU has been increasingly used to identify and quantify nonconvulsive seizures and status epilepticus and assist with management decision-making in refractory cases. Standardized critical care terminology has been validated for clinical use.[15] Background abnormalities are common in hospital-based EEG and continuous EEG recordings. Background slowing of the EEG ranges from mild to severe and

CASE 2-3

A 32-year-old man had seizure onset at 12 years of age, which became uncontrolled by antiseizure medication resulting in his referral for presurgical evaluation. When he presented for evaluation after 20 years of recurrent seizures, they were manifested by a "warning" right before he would experience a convulsion with a frequency that occurred every other month. Brain MRI revealed a right temporal lobe lesion consistent with the radiographic appearance of a meningioma. Phenytoin, oxcarbazepine, levetiracetam (brief trial limited by side-effects), and lacosamide in low doses had been ineffective. Standard EEGs were normal.

During video-EEG monitoring, oxcarbazepine 600 mg orally 2 times a day and lacosamide 100 mg orally 2 times a day were tapered, and overnight long-term video-EEG recorded frequent (hourly) bursts of 4- to 4.5-Hz generalized spike-and-wave and generalized polyspike-and-wave discharges lasting up to 12 seconds. Intermittent photic stimulation produced a self-limited photoparoxysmal response with subtle upper body myoclonus reported by the patient to represent his "warning." On the morning of day 3 of video-EEG monitoring, a generalized tonic-clonic seizure of nonfocal origin was captured and preceded by myoclonus.

Drug-resistant juvenile myoclonic epilepsy (JME) was successfully treated with valproate monotherapy, and surgery was deferred to neurosurgical follow-up for ongoing surveillance by annual brain MRI with intervention if growth occurred or the lesion became symptomatic.

COMMENT

Warnings are usually associated with focal seizures. In this case, focal epilepsy was initially suggested in association with a focal structural lesion on brain MRI. Furthermore, the presence of resistance to more than two antiseizure medications was redefined as "pseudo-drug resistance" following confirmation of a genetic generalized epilepsy syndrome by video-EEG monitoring. By accurately identifying the epilepsy syndrome as JME, the appropriate antiseizure medication could be instituted. By defining and classifying a genetic generalized epilepsy, the meningioma was determined to be asymptomatic, and unnecessary surgery was therefore avoided.

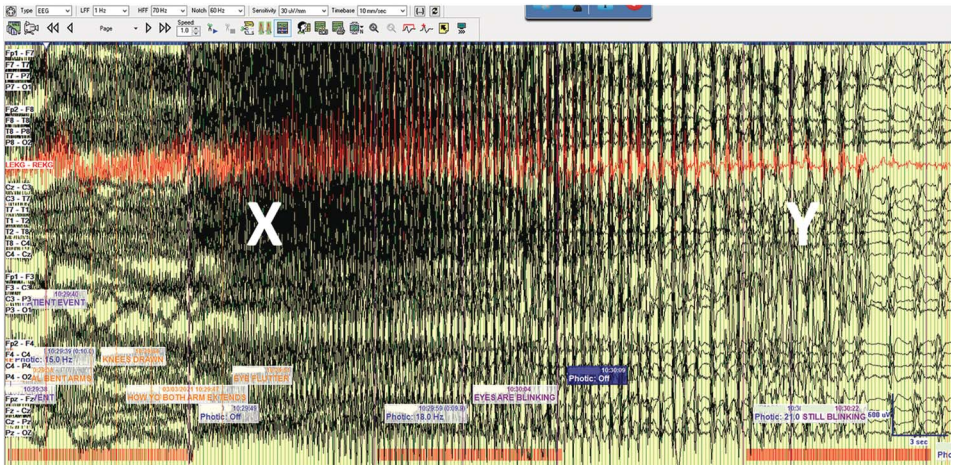


FIGURE 2-38

Generalized tonic-clonic seizure in a 19-year-old man with reflex photosensitive epilepsy triggered during photic stimulation. The *red bars* at the bottom reflect stimulations. The stimulator was moved away from the patient to allow the technologist to attend to the patient. Display speed was 10 mm/s. Note the tonic phase (*X*) and clonic phase (*Y*).

includes focal, hemispheric, diffuse, and multifocal slowing; low-voltage EEGs; burst suppression; and electrocerebral inactivity.[42] Postsurgical EEGs may demonstrate a breach rhythm (FIGURE 2-25) where higher amplitudes and faster frequencies make normal rhythms appear "spikier," mimicking a pathologic interictal epileptiform discharge.

Periodic patterns (a discharge repeating itself for at least six cycles) occurring in critically ill patients are well established.[15] Morphology of the periodic patterns varies. They may or may not be associated with fast activity (FIGURE 2-39).

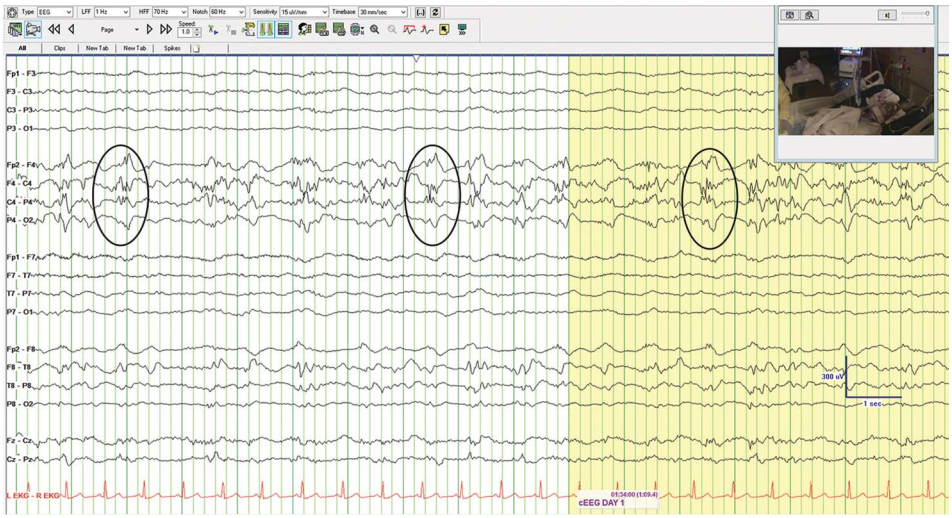


FIGURE 2-39

Right central lateralized periodic discharges (LPDs) plus fast activity at 1 Hz (*ovals*) during standard EEG. Later, continuous EEG performed in this 55-year-old man with persistent unexplained altered mental status after operation for a right perirolandic glioblastoma recorded serial nonconvulsive seizures.

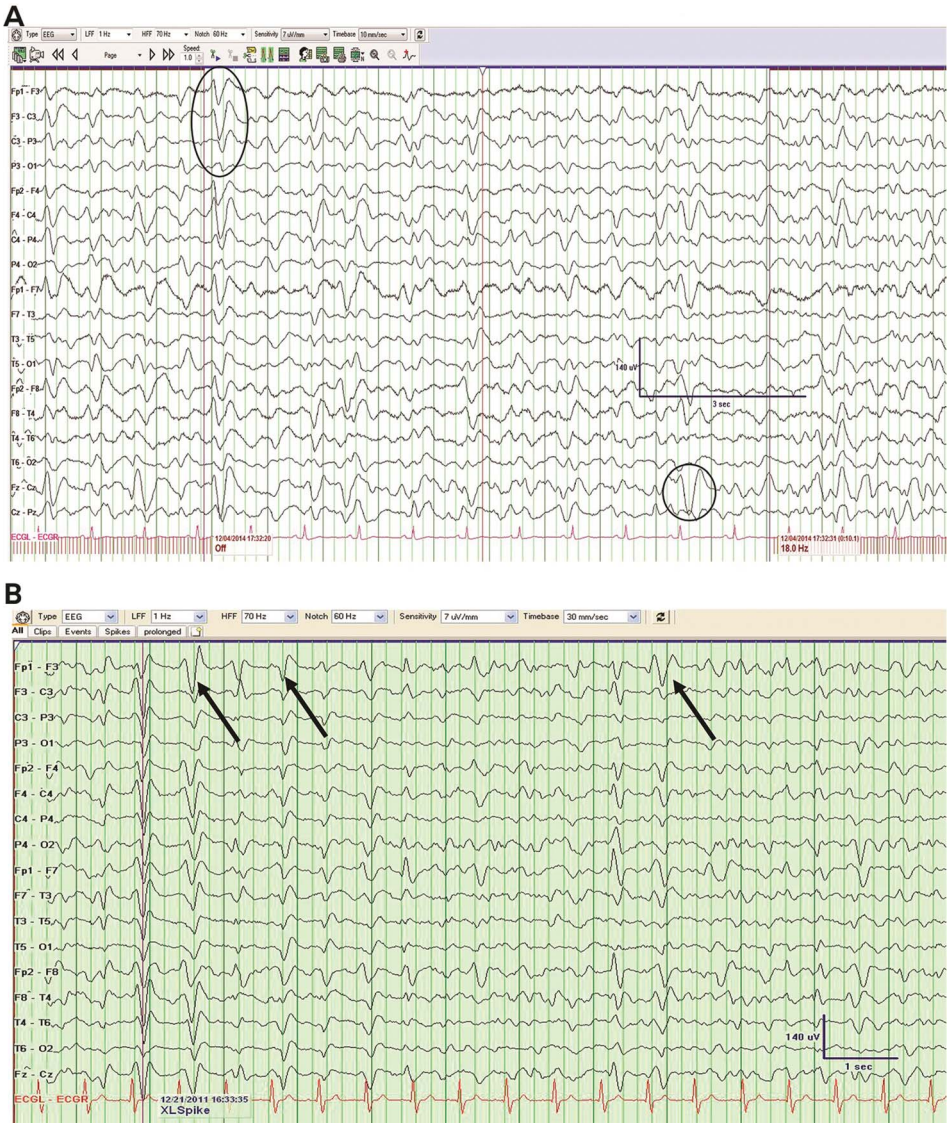


FIGURE 2-40

EEG depicting encephalopathy. *A*, Diffuse slowing of the posterior-dominant rhythm to 5 Hz with continuously intermixed delta and triphasic waves in a 78-year-old woman with end-stage renal disease and a syncopal episode during hemodialysis. Note the triphasic morphology (*circle*) and "anterior-posterior lag" of the waveform from the frontal to the occipital derivations (*oval*). *B*, EEG in a 91-year-old man with generalized periodic discharges with triphasic morphology (*arrows*) associated with postinfarction focal epilepsy. He was admitted for acute mental status changes and urosepsis after he was found on the floor at home by his daughter. Despite a treatment challenge with benzodiazepine administration, the patient did not improve. The clinical course ultimately resulted in a fatal outcome.

Lateralized periodic discharges (LPDs) and LPDs plus fast activity (LPD+F) are strongly associated with seizures and status epilepticus.[43] Independent bilateral LPDs have a periodic pattern; the periods and morphologies arising from each hemisphere differ and reflect diffuse cortical injury (ie, hypoxia). Lateralized rhythmic delta activity, like LPDs, is an EEG pattern strongly associated with seizures[43,44] Generalized periodic discharges (GPDs) are bilateral synchronous symmetrical discharges. GPDs include many morphologies, including GPDs with

a triphasic morphology (FIGURE 2-40A), that reflect diffuse cerebral gray matter dysfunction.

When nonconvulsive seizures and nonconvulsive status epilepticus occur, EEG is the only means of arriving at the diagnosis when no clinical signs are present. Therefore, continuous EEG is useful in demonstrating ongoing electrographic seizures and nonconvulsive status epilepticus to implement or modify treatment.[45] Criteria for diagnosing nonconvulsive status epilepticus include the presence of at least 25 epileptiform discharges/10-second epoch or 2.5 discharges/s. When discharges are 2.5/s or less or rhythmic delta/theta exceeds 0.5/s, an additional criterion must be present. This includes clinical and EEG improvement from antiseizure medication administration (FIGURE 2-40B), the presence of subtle ictal clinical features, or spatiotemporal evolution of epileptiform activity.[46] Quantitative EEG and trend analysis of the ICU EEG (FIGURE 2-41) is a useful adjunct to raw continuous EEG when utilized over long periods of time (CASE 2-4).[19]

INTRACRANIAL EEG

During noninvasive presurgical evaluations with scalp EEG (FIGURE 2-45), attempts to localize the source may fail. Deep-seated and interhemispheric foci, discordant noninvasive evaluations, and cases of false localization merit intracranial EEG to lateralize and localize one or more seizure onset zones.[49] Intracranial EEG recording methods vary by institution, although depth electrodes and stereo-EEG, subdural grids and strips, foramen ovale electrodes, and epidural pegs may be used alone or in combination. Intracranial EEG has a high signal-to-noise ratio when recording brain signals, and therefore, it is less subject to artifact but prone to sampling biases. Waveform frequencies are the same as those on scalp EEG. Interictal epileptiform discharges on intracranial EEG are more likely to accurately reflect the seizure onset zone when they are

KEY POINTS

- EEG is the only test in critically ill patients with altered mental status that can result in the diagnosis of nonconvulsive seizures and nonconvulsive status epilepticus.
- The goals of therapy for ongoing nonconvulsive seizures and nonconvulsive status epilepticus aim at achieving seizure suppression on continuous EEG and reducing cerebral metabolic rates by achieving a burst-suppression pattern.

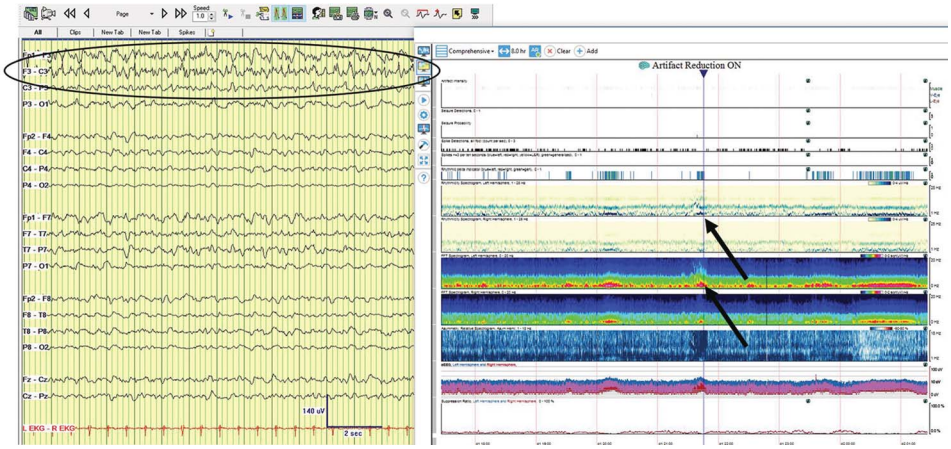


FIGURE 2-41

Intensive care unit EEG and trending. A 45-year-old woman with aphasia and right hemiparesis underwent a left craniotomy for resection of a high-grade glioma. Standard EEG showed left frontal lateralized periodic discharges (LPDs). Continuous EEG in the intensive care unit (raw EEG on the left, trends on the right) detected serial subclinical seizures with waxing and waning ictal EEG (*oval*) that persisted after treatment. Here, the fast Fourier transform and rhythmicity spectrogram (not the seizure probability algorithm) best reflected ongoing seizures (*arrows*).

CASE 2-4

A 67-year-old man presented as a stroke alert after awakening with "garbled speech" and left-sided weakness (last well time 12 hours prior). He had a history of hypertension, coronary artery disease, and hyperlipidemia. A prior transient ischemic attack (transient aphasia) occurred 2 months before admission.

In the emergency department, he was aphasic with right hemiplegia. Diffusion-perfusion brain MRI showed early signs of a left middle cerebral artery ischemic infarction. He underwent emergency angiography with thrombectomy and was transferred to the intensive care unit (ICU) on thrombolytics. He began to be unresponsive with continuous right face and head twitching. A stat EEG demonstrated left frontotemporal lateralized periodic discharges (LPDs) and LPDs plus fast activity (LPD+F) at 1 Hz. Subsequently, he was administered levetiracetam, and continuous EEG demonstrated serial nonconvulsive seizures despite treatment with lorazepam, levetiracetam, and valproate. Ketamine was then begun and titrated to burst suppression on continuous EEG. The goals of therapy for ongoing nonconvulsive seizures and nonconvulsive status epilepticus aim at achieving seizure suppression on continuous EEG and reducing cerebral metabolic rates by achieving a burst-suppression pattern. However, despite maintaining burst suppression with administration of anesthetics and antiseizure medication, seizures were intermittently present (FIGURE 2-42) as anesthetics were withdrawn. With continued treatment, the seizures became infrequent, and the patient's mental status improved with reduction of anesthetics. He reached the point where he was able to be stabilized and discharged from the hospital to a rehabilitation facility for ongoing therapy.

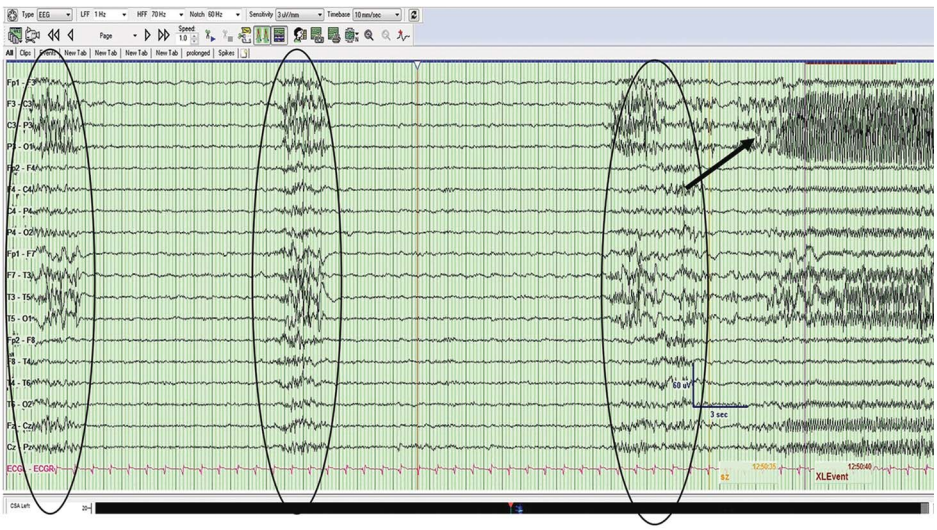


FIGURE 2-42

EEG of the patient in CASE 2-4 who had a left hemispheric focal seizure arising from iatrogenic burst suppression pattern. This 67-year-old man in a coma had super-refractory status epilepticus with ongoing electrographic seizures arising from the left parietal region (*arrow*). Compressed display speed (10 mm/s) highlights EEG bursts (*ovals*) in seconds 2 to 3, 15 to 16, and 32 to 33 before the seizure.

An EEG demonstrating LPDs usually reflects an acute or subacute injury of the cortex. Infrequently, LPDs reflect a postictal feature or acute reactivation of an underlying chronic structural lesion. Stroke is the most commonly encountered structural etiology generating LPDs but probably a reflection of the prevalence as opposed to specificity. LPDs on EEG are high-voltage periodic or semiperiodic discharges, often every 1 to 2 seconds, correlated with focal seizures.[44] LPD+F on EEG is a high risk for status epilepticus; their finding on standard EEG should prompt further investigation with continuous EEG. Continuous EEG monitoring in the ICU and special care units provides dynamic information about seizures and brain function.[47] Electrographic status epilepticus must be 10 minutes or longer or a total duration of 20% or more of any 60-minute recording period.[48] Seizures on EEG are 10 seconds or longer but may be briefer when accompanied by clinical signs. Brief potentially ictal rhythmic discharges (BIRDs) can be focal or generalized rhythmic activity greater than 4 Hz and between 0.5 seconds and less than 10 seconds (**FIGURE 2-43**). Like stimulus-induced rhythmic periodic ictal discharges (**FIGURE 2-44**), BIRDs lie on the ictal-interictal continuum.

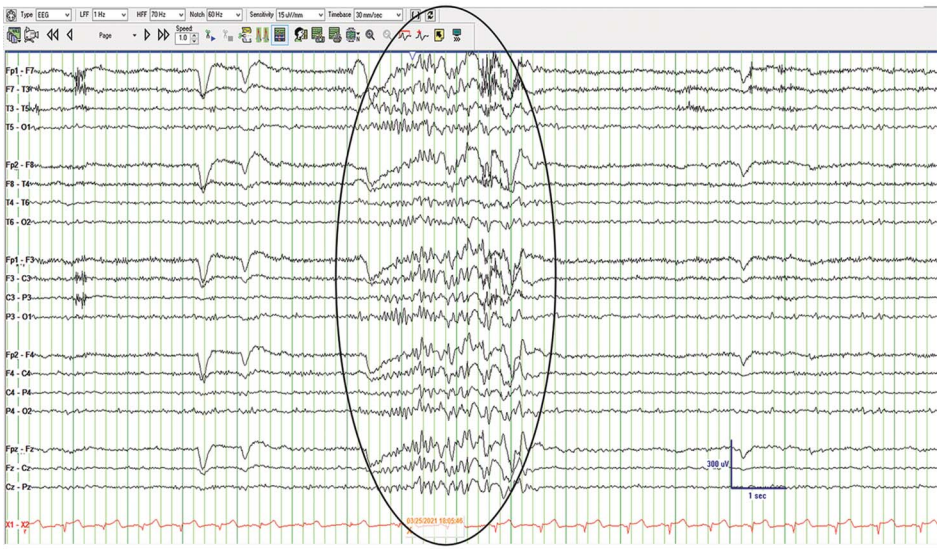


FIGURE 2-43

Brief potentially ictal rhythmic discharge (BIRD) (*oval*). This BIRD was seen multiple times in a patient with subclinical seizures and symptomatic occipital lobe epilepsy.

CONTINUED ON PAGE 298

CONTINUED FROM PAGE 297

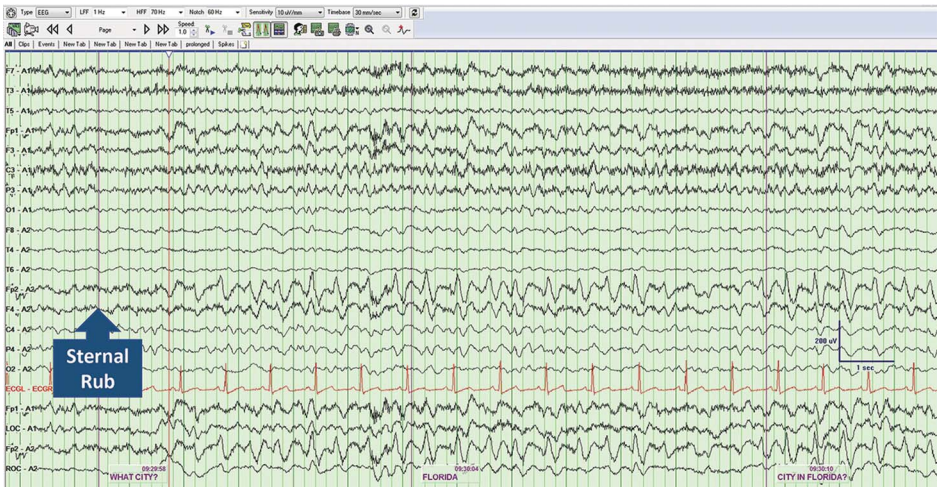


FIGURE 2-44

Stimulus-induced rhythmic, periodic, or ictal discharges (SIRPIDs) usually accompany other EEG abnormalities such as lateralized periodic discharges (LPDs) or lateralized rhythmic delta activity found in patients in the intensive care unit, including subclinical seizures. Notice the increase in right frontal spiking that occurs after applying a sternal rub.

high amplitude and frequent with a short interspike interval (FIGURE 2-46). Interictal epileptiform discharges are more frequent on intracranial EEG than scalp EEG and often extend outside the region of seizure onset. Because intracranial EEG has greater spatial-temporal resolution than scalp EEG, it is more likely to detect seizures early. Diffuse attenuation and multiple-electrode involvement of intracranial EEG at seizure onset suggest a propagated pattern.

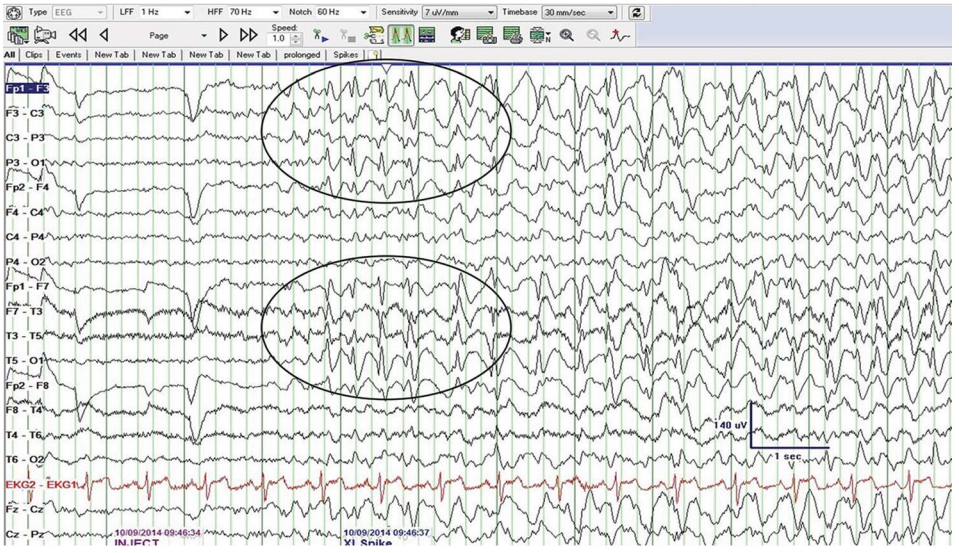


FIGURE 2-45

EEG taken from a Wada test by using methohexital where left hemispheric spike and waves (*ovals*) were precipitated in a prolonged run after injection in the left internal carotid artery.

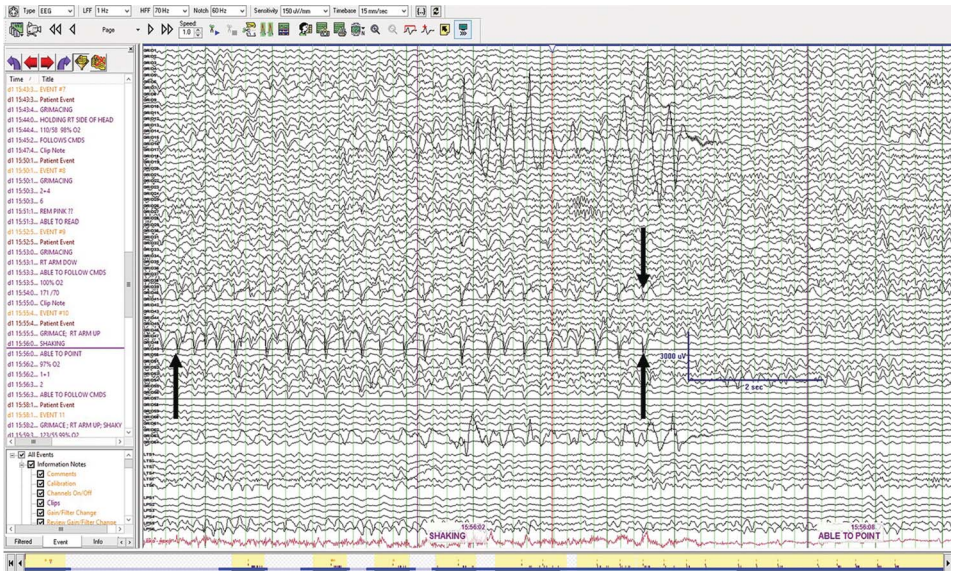


FIGURE 2-46

Subdural intracranial EEG with repetitive sharp waves (*single arrow*) and seizure termination (*double arrow*) during a phase 2 evaluation. A 64-channel grid over the frontoparietal region and two 1 x 6 strips in the left temporal and inferior parietal region approximated a region of possible focal cortical dysplasia. This patient had drug-resistant extratemporal focal epilepsy and recurrent right focal motor seizures.

Intracranial EEG is also used for functional brain mapping by using direct electrocortical stimulation (FIGURE 2-47) for prognostication before epilepsy surgery, and via neuromodulation and after localization using thermocoagulation to provide treatment.[3,49] Because of greater sensitivity, intracranial EEG can identify high-frequency oscillations (brief high-frequency bursts in bandwidths subserved by gamma, ripples, and fast-ripples bandwidths) that are felt to represent areas of epileptogenesis. (TABLE 2-2).

ELECTROCORTICOGRAPHY

Electrocorticography may be performed extraoperatively, although it usually refers to intraoperative EEG associated with direct brain recording. One use of electrocorticography in epilepsy (or lesional) surgery is mapping areas of the cortex associated with interictal epileptiform discharges before tailoring surgical resection or suggesting postresection prognosis.[50] Electrocorticography is also used to monitor for afterdischarges (FIGURE 2-47) during direct electrocortical stimulation performed for functional brain mapping.[51] Language, motor, and sensory cortices are defined by using direct superficial or deep electrical stimulation directed by a wand handheld by the neurosurgeon. Compared with intracranial EEG, intraoperative electrocorticography has brief recording periods, less sustained discomfort, and lower risk from electrode placement. However, commercially available electrocorticography has limited sampling from electrodes (FIGURE 2-48) as well as electrode placement. Resection of high-frequency oscillations present on electrocorticography (and intracranial EEG) has shown predictive value in localizing the seizure onset zone in neocortical epilepsies to forecast a favorable surgical outcome.[52]

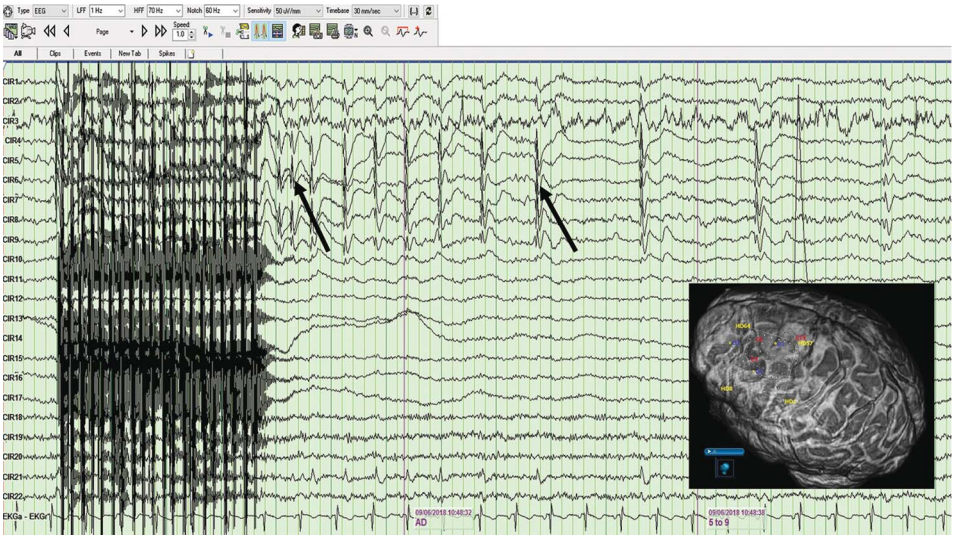


FIGURE 2-47

Direct electrocortical stimulation for 4 seconds at 2 mA during intraoperative electrocorticography that resulted in a regional afterdischarge (*arrows*). The insert displays the location of a 64-channel high-density grid, depth electrode placement that was evaluated before the use of a customized circular electrode for real-time electrocorticography during functional brain mapping and surgery.

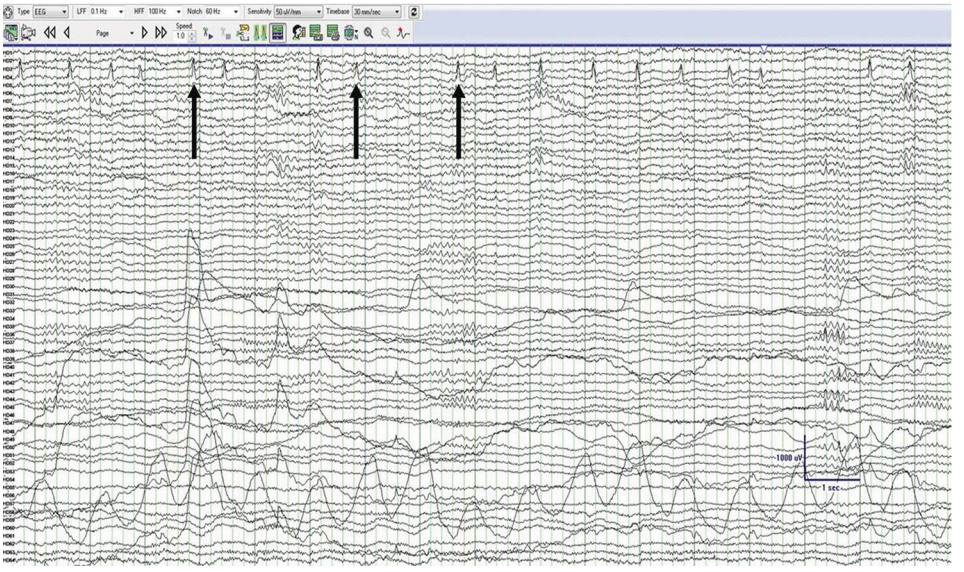


FIGURE 2-48

High-density 64-channel electrocorticography with 2-mm sensors, spaced 5 mm apart, recorded focal periodic epileptiform discharges (*arrows*) in a patient undergoing awake craniotomy for resection of a high-grade glioma. Note the restricted field of spatial distribution that is confined to 1 cm.

OTHER EEG RECORDING TECHNIQUES

EEG, among other neurophysiologic techniques, helps determine brain function.[19] The evidence is good for successful temporal and extratemporal source localization using EEG source localization/imaging to identify the "inverse solution" from a scalp EEG–generated location.[53,54]

Long-term intracranial electrocorticography is now possible on an outpatient basis with neuromodulation devices (FIGURE 2-49). High-density EEG augments the standard 10-20 international system of electrode placement and typically utilizes 64 channels or more to record EEG. High-density EEG can demonstrate highly focal abnormalities beyond the resolution of standard EEG.[55] Computerization enables high-density EEG recording with high temporal resolution and good spatial resolution of subcortical regions in the study of scalp-based brain dynamics.[56] Computer-assisted EEG source localization (FIGURE 2-50) of dipolar and distributed EEG sources and signal analysis localizes the source through enhanced computational capability seen with digital technology. Research use of microwires recording from single columns of neuronal activity has revealed microseizures at the neuronal level.[57]

CONCLUSION

To interpret abnormal interictal EEG, knowledge of the "gray areas" and boundaries of normal is essential. Patients with seizures and epilepsy are best suited for evaluation with EEG with practical implications for diagnosis, management, and prognosis. Long-term EEG with video provides dynamic information in patients who need a definitive diagnosis or those with drug-resistant epilepsy evaluated for surgery. Effective use of continuous EEG has identified nonepileptiform and epileptiform features in patients with acute and chronic brain disorders and can quantify nonconvulsive seizures to guide patient management. In the operating room, like in the epilepsy monitoring unit, electrocorticography may be used for localizing areas of functional brain via direct electrocortical mapping. Intracranial EEG has been associated with presurgical evaluation of patients with drug-resistant epilepsies but is increasingly used outside hospital-based epilepsy monitoring units with advances in neuromodulation. Whether EEG is performed in the clinical neurophysiology laboratory, special care units in the hospital, or the home setting, it provides unique information unparalleled by other testing modalities. The limits of

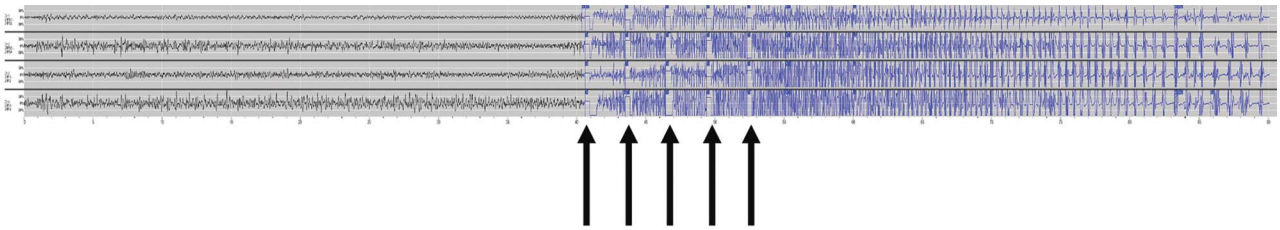


FIGURE 2-49

Electrocorticogram in a patient with a responsive neurostimulator implanted with recording and treating bitemporal depth electrodes. Note the initial detection and repetitive electrical stimulations (*arrows*) that did not abort this focal impaired awareness seizure.

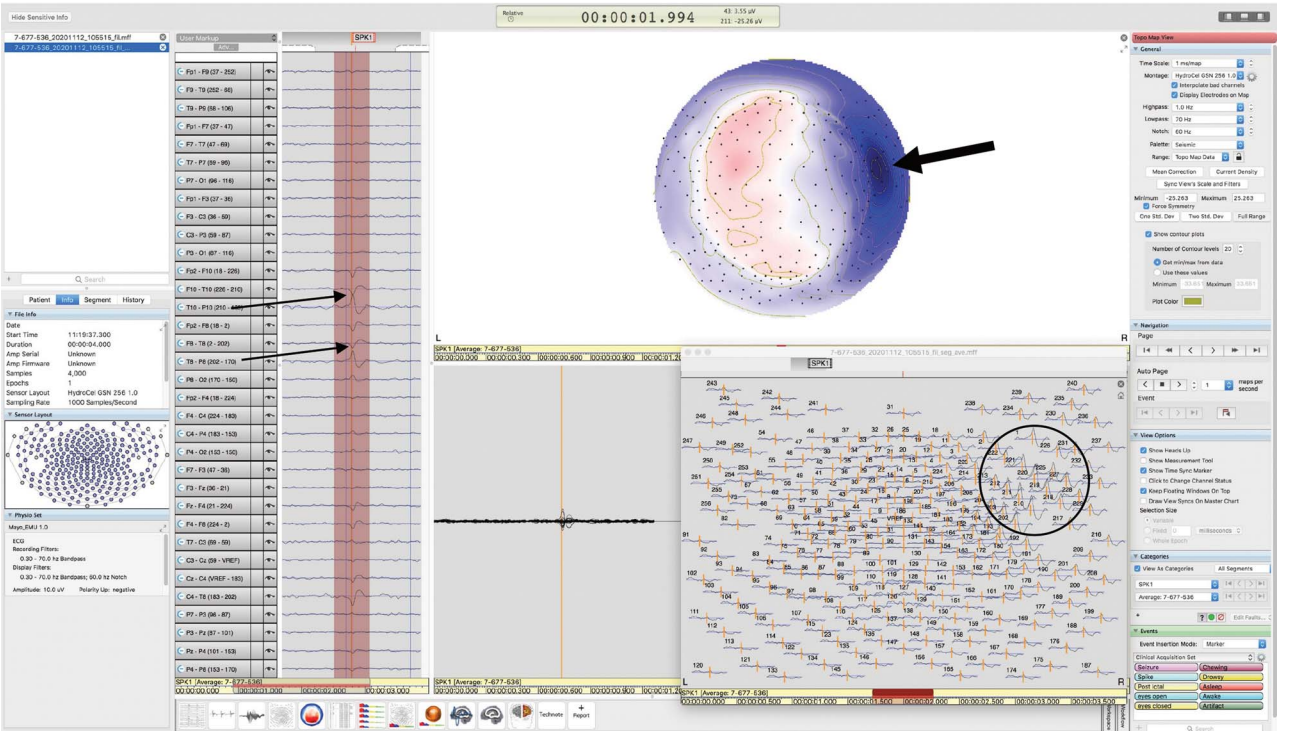


FIGURE 2-50

EEG source localization. Waveforms presented as raw EEG and modeled as a spherical source, topographic map, and butterfly chart. The 256-channel high-density EEG obtained in a 59-year-old woman after right temporal resection revealed source localization in the residual right lateral temporal neocortex. Note the topographic map with interictal epileptiform discharges (*circle*) reflects averaged interictal epileptiform discharges (*thin arrows*) on the spherical head model (*thick arrow*). Blue is the negative end of the dipolar source measured.

learning EEG from primers lies in visualizing a single "classic" example. With the myriad of waveform variations involving one example, the permutations of each waveform are infinite.

VIDEO LEGENDS

VIDEO 2-1
Right temporal artifact on EEG due to tremor. Video clarifying right temporal artifact on EEG. Note that the tremor while the patient is holding the telephone over his right ear produces a local multiple-electrode artifact simulating a right temporal electrographic seizure.

VIDEO 2-2
Focal impaired awareness seizure of right temporal lobe origin. Focal impaired awareness seizure of right temporal lobe origin during video-EEG monitoring for presurgical evaluation. Note the initial stare, vocalization, and oroalimentary and bimanual automatisms with the ability to retain "ictal speech." This symptomatology and ictal EEG support a temporal and nondominant hemispheric origin.

USEFUL WEBSITE

AMERICAN CLINICAL NEUROPHYSIOLOGY SOCIETY
This website provides links to guidelines pertinent to understanding EEG performance.

acns.org/practice/guidelines

REFERENCES

1 Berger HLFH. Hans Berger (1873–1941), Richard Caton (1842–1926), and electroencephalography. J Neurol Neurosurg Psychiatry 2003;74(9). doi:10.1136/jnnp.74.1.9

2 Bergey G. Management of a first seizure. Continuum (Minneap Minn) 2016;22(1, Epilepsy): 38-50. doi:10.1212/CON.0000000000000271

3 Tatum WO, Rubboli G, Kaplan PW. Clinical utility of EEG in diagnosing and monitoring epilepsy in adults. Clin Neurophysiol 2018;129:1056-1082. doi:10.1016/j.clinph.2018.01.019

4 Beniczky S, Schomer DL. Electroencephalography: basic biophysical and technological aspects important for clinical applications. Epileptic Disord 2020;22(6):697-715. doi:10.1684/epd.2020.1217

5 Blumcke I, Arzimanoglou A, Beniczky S, Wiebe S. Roadmap for a competency based educational curriculum in epileptology: report of the epilepsy education task force of the International League Against Epilepsy. Epileptic Disord 2019;21:129-140. doi:10.1684/epd.2019.1039

6 American Clinical Neurophysiology Society. Guidelines and consensus statements. Accessed December 17. acns.org/practice/guidelines

7 Sinha SR, Sullivan LR, Sabau D, et al. American Clinical Neurophysiology Society Guideline 1: Minimum Technical Requirements for Performing Clinical Electroencephalography. Neurodiagn J 2016;56(4):235-244. doi: 10.1080/21646821.2016.1245527

8 Seeck M, Koessler L, Bast L, et al. The standardized EEG electrode array of the IFCN. Clin Neurophysiol 2017;128(10):2770-2077. doi:10.1016/j.clinph.2017.06.254

9 Martins da Silva A, Leal B. Photosensitivity and epilepsy: current concepts and perspectives–a narrative review. Seizure. 2017;50:209-218. doi:10.1016/j.seizure.2017.04.001

10 Tatum WO, Husain AM, Benbadis SR, Kaplan PW. Normal adult EEG and patterns of uncertain significance. J Clin Neurophysiol. 2006;23: 194-207. doi:10.1097/01.wnp.0000220110.92126.a6

11 Tatum WO. Normal "suspicious" EEG. Neurology 2013;80(1):S4-S11. doi:10.1212/ WNL.0b013e31827974df

12 Tatum WO, Desai NN. EEG in epilepsy. In: Roos RP, editor. MedLink neurology. MedLink, LLC; 2021.

13 Kang JY, Krauss GL. Normal variants are commonly overread as interictal epileptiform abnormalities. J Clin Neurophysiol 2019;36(4): 257-263. doi:10.1097/WNP.0000000000000613

14 Tatum WO, Reinsberger C, Dworetzky B. Artifacts of recording. In: Schomer DL, Silva FH, eds. Niedermeyer's electroencephalography: basic principles, clinical applications, and related fields. Vol 2017. 7th ed. Wolters Kluwer Health/ Lippincott Williams & Wilkins: 266-316.

15 Tatum WO, Hirsch LJ, Gelfand MA, et al. Video quality using outpatient smartphone videos in epilepsy: results from the OSmartViE study. Eur J Neurol 2021;28(5):1453-1462. doi:10.1111/ene.14744

16 Kane N, Acharya J, Beniczky S. A revised glossary of terms commonly used by clinical electroencephalographers and updated proposal for the report format of the EEG findings: revision 2017. Clin Neurophysiol Pract 2017;2:170-185. doi:10.1016/j.cnp.2017.07.002

17 Pillai J, Sperling MR. Interictal EEG and the diagnosis of epilepsy. Epilepsia 2006;47(suppl 1): 14-22. doi:10.1111/j.1528-1167.2006.00654.x

18 Admiraal MM, Rootselaar AF, Horn J. Electroencephalographic reactivity testing in unconscious patients: a systematic review of methods and definitions. Eur J Neurol 2017;24: 245-254. doi:10.1111/ene.13219

19 Johnson EL, Kaplan PK. Clinical neurophysiology of altered states of consciousness: encephalopathy and coma. Chapter 4. Handb Clin Neurol 2019;161:73-88. doi:10.1016/B978-0-444-64142-7.00041-2

20 Tatum WO, Thottempudi N, Gupta V, et al. De novo temporal intermittent rhythmic delta activity after laser interstitial thermal therapy for mesial temporal lobe epilepsy predicts poor seizure outcome. Clin Neurophysiol 2019;130(1): 122-127. doi:10.1016/j.clinph.2018.11.012

21 Maulsby RL. Some guidelines for assessment of spikes and sharp waves in EEG tracings. Am J EEG Technol 1971;11:3-16. doi:10.1080/00029238.1971.11080808

22 Kural MA, Duez L, Hansen VS. Criteria for defining interictal epileptiform discharges in EEG: a clinical validation study. Neurology 2020;94: e2139-e2147. doi:10.1212/ WNL.0000000000009439

23 Fisher RS, Acevedo C, Arzimanoglou A, Bogacz A, Cross JH, Elger CE. ILAE official report: a practical clinical definition of epilepsy. Epilepsia 2014;55: 475-482. doi:10.1111/epi.12550

24 Burkholder DB, Britton JW, Rajasekaran V. Routine vs extended outpatient EEG for the detection of interictal epileptiform discharges. Neurology 2016;86(16):1524-1530. doi:10.1212/ WNL.0000000000002592

25 Gavvala JR, Schuele SU. New-onset seizure in adults and adolescents: a review. JAMA 2016;316: 2657-2668. doi:10.1001/jama.2016.18625

26 He RQ, Zeng QY, Zhu P, Bao YX, Zheng RY, Xu HQ. Risk of seizure relapse after antiepileptic drug withdrawal in adult patients with focal epilepsy. Epilepsy Behav 2016;64:233-238. doi:10.1016/j.yebeh.2016.08.006

27 Koutroumanidis M, Arzimanoglou A, Caraballo R. The role of EEG in the diagnosis and classification of the epilepsy syndromes: a tool for clinical practice by the ILAE Neurophysiology Task Force (Part 1). Epileptic Disord 2017;19(3):233-298. doi:10.1684/epd.2017.0935

28 Maganti RK, Rutecki P. EEG and epilepsy monitoring. Continuum (Minneap Minn) 2013;19(3, Epilepsy):598-622. doi:10.1212/01.CON.0000431378.51935.d8

29 Tatum WO. Mesial temporal lobe epilepsy. J Clin Neurophysiol 2012;29(5):356-365. doi:10.1097/WNP.0b013e31826b3ab7

30 Andrade-Machado R, Benjume Cuartas V, Muhammad IK. Recognition of interictal and ictal discharges on EEG. Focal vs generalized epilepsy. Epilepsy Behav 2021;117(107830). doi:10.1016/j.yebeh.2021.107830

31 Adcock JE, Panayiotopoulos CP. Occipital lobe seizures and epilepsies. J Clin Neurophysiol 2012;29(5):397-407. doi:10.1097/WNP.0b013e31826c98fe

32 Seneviratne U, Cook M, D'Souza W. The electroencephalogram of idiopathic generalized epilepsy. Epilepsia 2012;53(234). doi:10.1111/j.1528-1167.2011.03344.x

33 Craciun L, Varga ET, Mindruta I, Meritam P, Horvath Z, Terney D. Diagnostic yield of five minutes compared to three minutes hyperventilation during electroencephalography. Seizure 2015;30:90-92. doi:10.1016/j.seizure.2015.06.003

34 Ung H, Cazares C, Nanivadekar A, et al. Interictal epileptiform activity outside the seizure onset zone impacts cognition. Brain 2017;140:2157-2168. doi:10.1093/brain/awx143

35 Benbadis S. What type of EEG (or EEG-video) does your patient need? Expert Rev Neurother 2015;15(5):461-464. doi:10.1586/14737175.2015.1029918

36 Langston ME, Tatum WO. Seizures without awareness: a pure cohort. Epilepsy Res 2015;109:163-168.

37 Seneviratne U, Rahman Z, Diamond A, Brusco M. The yield and clinical utility of outpatient short-term video-electroencephalographic monitoring: a five-year retrospective study. Epilepsy Behav 2012;25(3):303-306. doi:10.1016/j.yebeh.2012.08.018

38 Yao J, Wang H, Xiao Z. Correlation between EEG during AED withdrawal and epilepsy recurrence: a meta-analysis. Neurol Sci 2019;40(8):1637-1644. doi:10.1007/s10072-019-03855-x

39 Tanaka H, Gotman J, Khoo HM, Olivier A, Hall J, Dubeau F. Neurophysiological seizure-onset predictors of epilepsy surgery outcome: a multivariable analysis. J Neurosurg 2019;1-10. doi:10.3171/2019.9.JNS19527

40 Arntsen V, Sand T, Syvertsen MR, Brodtkorb E. Prolonged epileptiform EEG runs are associated with persistent seizures in juvenile myoclonic epilepsy. Epilepsy Res 2017;134:26-32. doi:10.1016/j.eplepsyres.2017.05.003

41 Lewis A, Kirschen MP. Brain death/death by neurologic criteria determination. Continuum (Minneap Minn) 2021;27(5, Neurocritical Care):1444-1464. doi:10.1212/CON.0000000000000987

42 Stecker MM, Sabau D, Sullivan L. American Clinical Neurophysiology Society Guideline 6: Minimum Technical Standards for EEG Recording in Suspected Cerebral Death. J Clin Neurophysiol 2016;33(4):324-327. doi:10.1080/21646821.2016.1245575

43 Gaspard J, Manganas L, Rampal N, Petroff OA, Hirsch LJ. Similarity of lateralized rhythmic delta activity to periodic lateralized epileptiform discharge in critically ill patients. JAMA Neurol 2013;70:1288-1295. doi:10.1001/jamaneurol.2013.3475

44 Rodriguez Ruiz A, Vlachy J, Lee J. Association of periodic and rhythmic electroencephalographic patterns with seizures in critically ill patients. JAMA Neurol 2017;74:181. doi:10.1001/jamaneurol.2016.4990

45 Herman ST, Abend NS, Bleck TP. Consensus statement on continuous EEG in critically ill adults and children, part I: indications. J Clin Neurophysiol 2015;32(2):87-95. doi:10.1097/WNP.0000000000000166

46 Leitenger M, Trinka E, Gardella E. Diagnostic accuracy of the Salzburg EEG criteria for non-convulsive status epilepticus: a retrospective study. Lancet Neurol 2016;15:1054-1062. doi:10.1016/S1474-4422(16)30137-5

47 Trinka E, Cock H, Hesdorfer D. A definition and classification of status epilepticus-report of the ILAE Task Force on classification of status epilepticus. Epilepsia 2015;56:1515-1523. doi:10.1111/epi.13121

48 Hirsch LJ, Fong MWK, Leitinger M, et al. American Clinical Neurophysiology Society's standardized critical care EEG terminology: 2021 version. J Clin Neurophysiol 2021;38:1-29. doi:10.1097/WNP.0000000000000806

49 Jayakar P, Gotman J, Harvey AS, Palmini A, Tassi L, Schomer D. Diagnostic utility of invasive EEG for epilepsy surgery: indications, modalities, and techniques. Epilepsia 2016;57:1735-1747. doi:10.1111/epi.13515

50 Ritaccio A, Brunner P, Schalk G. Electrical stimulation mapping of the brain: basic principles and emerging alternatives. J Clin Neurophysiol 2018;35:86-97. doi:10.1097/WNP.0000000000000440

51 Tatum WO, McKay JH, ReFaey K, et al. Detection of after-discharges during intraoperative functional brain mapping in awake brain tumor surgery using a novel high-density circular grid. Clin Neurophysiol 2020;131(4):828-835. doi:10.1016/j.clinph.2019.12.416

52 van't Klooster MA, van Klink NEC, Leijten FSS, et al. Residual fast ripples in the intraoperative corticogram predict epilepsy surgery outcome. Neurology 2015;85(2):120-128. doi:10.1212/WNL.0000000000001727

53 Scherg M, Berg P, Nakasato N, Beniczky S. Taking the EEG back into the brain: the power of multiple discrete sources. Front Neurol 2019; 10(855). doi:10.3389/fneur.2019.00855

54 Lascano AM, Perneger T, Vulliemoz S, Spinelli L, Garibotto V, Korff CM. Yield of MRI, high-density electric source imaging (HD-ESI), SPECT and PET in epilepsy surgery candidates. Clin Neurophysiol 2016;127:150-155. doi:10.1016/j.clinph.2015.03.025

55 Tatum WO, Feyissa AM, ReFaey K, Grewal SS, Alvi MA, Castro-Apolo R. Periodic focal epileptiform discharges. Clin Neurophysiol 2019;130(8): 1320-1328. doi:10.1016/j.clinph.2019.04.718

56 Seeber M, Cantonas LM, Hoevels M, Sesia T, Visser-Vandewalle V, Michel CM. Subcortical electrophysiological activity is detectable with high-density EEG source imaging. Nat Commun 2019;10(1):1-7.

57 Worrell GA, Gardner AB, Stead SM. High-frequency oscillations in human temporal lobe: simultaneous microwire and clinical macroelectrode recordings. Brain 2008;131: 928-937. doi:10.1093/brain/awn006