Using EEG in a Consultative Role

Joseph Drazkowski, M.D.¹

ABSTRACT

The electroencephalogram (EEG) is a long-used tool assisting health care providers with the diagnosis, management, and treatment of various neurological disorders. This article highlights several scenarios in which a consultant may utilize the routine EEG in managing specific neurological cases. Eight case scenarios from a tertiary referral hospital are presented for the reader’s consideration. Scenarios selected are new-onset seizures, encephalopathy, syncope, dementia, brain death, hypoxic-ischemic encephalopathy, status epilepticus, and migraine. A history in each condition is presented and is followed by a discussion of how useful an EEG may be in these specific situations. These eight cases highlight specific learning points where the EEG may be useful and how it can be practically incorporated into care of patients. Understanding how the EEG may be useful in the presented cases will allow the efficient and effective use of the EEG in similar clinical scenarios.

KEYWORDS: Electroencephalogram (EEG), seizures/status epilepticus, dementia, syncope, brain death, encephalopathy

Objectives: Upon completion of this article, the reader will have a renewed appreciation for the clinical usefulness of EEG.

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For more than six decades the electroencephalogram (EEG) has been a major tool for evaluating, diagnosing, and guiding therapy for neurological disorders.¹ The advent of modern imaging techniques has narrowed the usefulness of the routine EEG. Once used routinely, EEG remains a powerful investigative technique in selected clinical situations. This article describes the more common situations in which the EEG may provide useful diagnostic or prognostic information. It is important to remember that EEG interpretation is subjective and must be correlated with the clinical picture.

EEG IN SEIZURE DISORDERS

The most common contemporary use of EEG is in evaluation of patients with spells of uncertain etiology and new or ongoing seizures. The routine EEG may be particularly informative when epileptiform activity is identified in the context of episodic neurologic events.

Figure 1  Focal slowing; right temporal spike wave in a patient with partial seizures.

Table 1  Waveforms Associated with a Tendency for Seizures

<table>
<thead>
<tr>
<th>Waveform</th>
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<tr>
<td>Focal sharp wave</td>
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<td>Focal spike after-coming slow wave</td>
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<td>Generalized spike and polyspike with slow wave</td>
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<td>Rolandic (central)/occipital spike (children)</td>
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<td>Hypsarrhythmia (children)</td>
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<td>Atypical spike and wave</td>
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<td>Photosensitive response</td>
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Case 1

A 39-year-old traveling salesman is referred by a local physician after having a first-ever unprovoked seizure. Brain computed tomography (CT) and magnetic resonance imaging (MRI) scan were normal. After advising him on the recurrence rate of new-onset seizures and driving restriction, you consider whether an EEG will be helpful in the management of his case.

Diagnosis  New-onset unprovoked seizure.

DISCUSSION

An EEG may reveal epileptiform abnormalities associated with seizure disorders. EEG abnormalities associated with a tendency to seizures are listed in Table 1. The prototypical epileptiform discharge is a spike–polyspike with an aftercoming slow wave. Spikes are defined as sharply contoured waveforms, each of 20 to 70 msec duration, that stand out from the background; most have a surface negative polarity. Spike and wave can be focal, multifocal, generalized, or a combination of these patterns. Figure 1 is an example of focal spike and wave in the midtemporal region. The EEG in Figure 2 demonstrates a generalized spike and wave pattern consistent with a generalized seizure disorder. Generalized spike and polyspike and wave at about 4 to 5 Hz is associated with juvenile myoclonic epilepsy (JME) and implies a lifelong tendency for seizures. Temporal spikes and sharp waves are strongly associated with the presence of partial seizures. Some estimates place the correlation between partial seizures and temporal spike and sharp waves as high as 90 to 95%. Such findings may assist the practitioner with regard to the patient’s prognosis and likelihood of seizure recurrence and thus guide specific therapy.

A normal interictal EEG may be helpful in reassuring the clinician in cases in which the clinical suspicion of a seizure is low. However, even a series of normal EEGs does not completely rule out the presence of a clinical seizure disorder. The utility of routine EEG becomes significantly lower after a patient has four normal routine EEGs. Further EEGs generally provide little or no useful information, and the practitioner must therefore place even greater emphasis on the clinical history and other diagnostic tests to help guide therapy. Continuous monitoring through the use of ambulatory
EEG or admission to an epilepsy monitoring unit (EMU) to record an actual event may yield useful diagnostic information, but the events in question need to be frequent enough to capture during the average 3- to 5-day hospital admission. The obvious downside to long-term monitoring in an EMU is expense, but if the patient has sufficiently frequent events it is reasonable to consider such studies when the diagnosis is in question.7

The presence of an abnormal EEG pattern may provide helpful additional diagnostic and prognostic information about focality and recurrence in the patient in this case.

**EEG IN ALTERED MENTAL STATUS**

For patients with dementia, delirium, lethargy, or confusion, the EEG provides useful information in several ways: (1) determining a focal versus generalized dysfunction, (2) recognition of specific patterns associated with specific conditions, and (3) providing evidence of physiologic dysfunction of the nervous system. The following cases emphasize clinical situations in which a routine EEG may contribute to patient care.

**Case 2**

*A 78-year-old male nursing home resident is admitted to the hospital after he was found early one morning to be less responsive than usual. He is mildly febrile and has no focal neurological abnormalities. The CT scan of the head is normal, and routine laboratory tests including complete blood count, electrolytes, hepatic and renal function, and urinalysis are also normal. It is discovered that the patient has taken most of a large bottle of acetaminophen.*

**DISCUSSION**

In this situation, the EEG often reveals nonspecific diffuse slowing replacing normal background activity. Common causes of such nonspecific slowing are listed in Table 2. Diffuse slowing indicates that there is a generalized organic process contributing to the patient’s symptoms (Fig. 3). Figure 4 is an EEG tracing demonstrating triphasic waves, a recognizable diffuse pattern of abnormality often caused by hepatic encephalopathy. Other conditions associated with triphasic waveforms include toxic states, hypoxic injury, metabolic disturbances, and degenerative processes.2 Triphasic waveforms include:

<table>
<thead>
<tr>
<th>Table 2 Common Causes of Nonspecific Slowing on Routine EEG</th>
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<tr>
<td>Hypoxic-anoxic insult</td>
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<tr>
<td>Medication effect</td>
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<td>Metabolic disturbances</td>
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<tr>
<td>Postictal state</td>
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<td>Degenerative diseases of the brain</td>
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<td>Meningoencephalitis</td>
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forms may show a characteristic temporal “lag” from the anterior to posterior head regions (or posterior-to-anterior delay). They are usually 1- to 2-Hz complexes consisting of a short-duration initial low-amplitude negative wave, a positive sharp contour wave, and another longer duration negative wave in succession. On occasion, nonconvulsive status epilepticus may be characterized by a generalized pattern. Triphasic waves must be differentiated from epileptiform activity.

An EEG showing focal slowing is suggestive of a localized process (Fig. 5). The specific focal pattern known as periodic lateralized epileptiform discharges
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Figure 5 Focal right hemisphere slowing in a patient with a prior stroke.

(PLEDs) has been associated with several acute pathological processes including acute stroke, ongoing focal seizure, and herpes encephalitis. The EEG is very sensitive to acute changes in cerebral blood flow after a certain threshold is reached. PLEDs may be detected by EEG before structural changes are evident on neuroimaging. Figure 6 is a pattern from a patient with herpes simplex virus encephalitis. Focal slowing may also be seen in the postictal period following a partial seizure. Ongoing partial status epilepticus may manifest as recurrent or persistent focal ictal discharges. Examples of typical ictal discharges include rhythmic theta, repetitive sharp waves, spike and wave, or other evolving rhythmic activity.

Case 3
A 75-year-old man is brought in by his wife for evaluation of memory loss. Except for moderate global cognitive impairment, his physical and neurological examinations and routine laboratory studies are normal. Brain MRI shows mild atrophy and nonspecific white matter changes.

Diagnosis Dementia.

Discussion
The EEG has some utility in the evaluation of the patient with dementia. Although common, the differentiation of Alzheimer's disease (or other similar degenerative disorders) or depression with cognitive symptoms (pseudodementia) from other causes of dementia may be difficult. Significantly abnormal EEGs early in the course of the illness suggest the possibility of the presence of a treatable illness such as a chronic infection or toxic or metabolic disorder. The majority of patients with depression or depressive pseudodementia have normal or minimally abnormal EEGs during wakefulness. In the later clinical stages of generalized dementia, a normal or only slightly abnormal EEG suggests a predominantly subcortical process or pseudodementia, although exceptions do exist. In one series, 93% of patients with subcortical dementia had a relatively normal EEG versus only 29% with cortical dementias.

An EEG showing a generalized, mostly symmetrical periodic pattern of approximately 1 Hz triphasic or biphasic sharp waveforms strongly suggests, but is not specific for, Creutzfeldt-Jakob disease (CJD). There is a tendency for the amplitude to decline and the interval between discharges to lengthen as the disease progresses. This periodic pattern of sharp wave complexes is typically seen during the first 12 weeks of the illness.

EEG and Imitators of Epilepsy

Case 4
HH is a 62-year-old woman with diabetes and heart disease who "passed out" in a local department store after complaining of diaphoresis and nausea. She had no convulsive
movements and quickly recovered consciousness shortly after hitting the floor. No focal abnormalities were found on examination, and a CT scan in the emergency room was normal. Electrocardiography and screening laboratory tests were normal. A diagnosis of syncope was made and she was told to follow up with her primary physician. Would an EEG be helpful?

Diagnosis Syncope.

DISCUSSION
In this case of syncope, it is unlikely that a patient will suffer a repeated attack during a routine EEG; between attacks, the EEG is usually normal. In the unlikely event that syncope is captured during an EEG, one of two patterns may occur. The first pattern is initial suppression of alpha background activity followed by low-amplitude fast activity. The fast activity is replaced by increasing amplitude theta frequencies evolving into high-amplitude delta. The second pattern is diffuse delta, decreasing in amplitude evolving to electrical silence and eventually gradual EEG recovery. Figure 7 demonstrates the EEG in tussive syncope, in which delta waves emerge several seconds after coughing triggered by hyperventilation. Many patients with syncope also have coexisting cerebrovascular or cardiovascular disease with EEG abnormalities secondary to effects of these chronic conditions. Such nonspecific abnormalities should not be confused as implying a tendency toward seizures.

Case 5
JT is a 25-year-old woman with a 10-year history of migraine headaches. There is a family history of migraine headache. Her MRI and general medical and neurological evaluations are normal. Would an EEG provide clinically useful information?

Diagnosis Migraine

DISCUSSION
The utility of EEG in headache patients is controversial. Reports of abnormal EEG in migraine patients have been criticized for various methodological difficulties. The EEG is generally normal between attacks but may show minor nonspecific abnormalities of questionable diagnostic value. During an attack the EEG usually remains normal, but it can be severely abnormal in some cases such as hemiplegic migraine. The EEG is useful only when confounding features of the history raise the possibility of a seizure disorder, and its use is not recommended in uncomplicated cases of migraine or headache.

EEG IN THE INTENSIVE CARE UNIT AND OPERATING ROOM
The EEG is valuable in acute situations commonly encountered in the intensive care unit (ICU) or operating room (OR). The EEG is generally sensitive to acute changes in nervous system function due to blood flow,
toxic or metabolic abnormalities, and other physiologic derangement.

Case 6
GA is 32-year-old woman admitted to the hospital with what was described as the worst headache of her life. Brain CT and angiography confirmed aneurysmal subarachnoid hemorrhage. The aneurysm rebled, causing a massive intracerebral hemorrhage and coma. Examination shows apnea, absence of brain stem reflexes, and no motor responses. A clinical diagnosis of brain death was made, but one consultant inquires about using an EEG to confirm “no brain activity.” Is this necessary or useful?

Diagnosis  Brain death.

DISCUSSION
The EEG has long been used to determine brain death. The so-called Harvard criteria of 1968 included a “flat EEG” a part of the criteria to determine brain death. Current guidelines established by the American Academy of Neurology (AAN) do not require an EEG to determine brain death.24 When specific elements of clinical testing are not conclusive, the EEG can be used in a confirmatory role. The EEG has 90% sensitivity and specificity in brain death determination.25 Specific requirements, listed in Table 3, must be met when using the EEG in brain death determination.

An EEG for brain death determination is invariably performed in the ICU—a hostile electrical environment in light of the high gain and other technical requirements needed to meet brain death requirements. In this setting, EEGs run to determine brain death have residual activity in about 20% of cases that otherwise fulfill the criteria for clinical brain death.25 The presence of this residual EEG activity may persist from hours to days and might confuse health care providers or family members. Because of this possibility of confusion, an EEG should be performed only when necessary. The AAN guidelines for clinical brain death determination are listed in Table 4.

Table 3  EEG Requirements for Brain Death Determination

<table>
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<td>Recording time of 30 minutes or more</td>
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<td>Long interelectrode distances (&gt;10 cm)</td>
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<tr>
<td>No electrical activity of greater than 2 µv (2 µv/mm for &gt; 30 minutes)</td>
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<tr>
<td>Requires use of long time constants (high-pass filter)</td>
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<tr>
<td>Impedances of &lt; 10 kohms and &gt; 100 ohms</td>
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<td>Qualified technologist performing the test</td>
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<td>No reactivity to afferent stimulation</td>
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<td>Minimum of eight scalp electrodes</td>
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<td>Verification of the integrity of the entire recording system</td>
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<td>Utilization of additional monitors</td>
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American Academy of Neurology Guidelines for Brain Death Determination

- Demonstration of coma
- Known cause of coma
- No confounding factors such as drugs, hypothermia, electrolyte or endocrine abnormality
- Apnea
- No motor response to noxious stimulation (absent motor response)
- Absent brain stem reflexes
- Repeat evaluation in 6 hours (arbitrary)
- Use of confirmatory tests as needed (EEG, angiogram, single photon emission computed tomography)

Case 7
DF is a 69-year-old man who required emergency coronary angioplasty. He suffered cardiac arrest during the procedure and required 60 minutes of cardiopulmonary resuscitation before a stable cardiac rhythm was achieved. He was transferred to the ICU, placed on a ventilator, and remained unresponsive. Neurological consultation and an EEG were ordered.

Diagnosis Hypoxic-ischemic encephalopathy.

DISCUSSION
The EEG in this situation may be helpful in the evaluation and in arriving at a prognosis for this patient. The EEG is sensitive to alterations in cerebral blood flow. When cerebral blood flow falls below 15 mL/min/100 g of brain tissue, there is usually a loss of electrical activity. Some areas of the brain have a selective vulnerability to hypoxia-anoxia injury, including the hippocampus, cerebellum, and neocortical layers 3, 5, and 6. The long-term effects of this selective vulnerability may be reflected by the EEG, which may show persistent background slowing or development of temporal epileptiform activity.

In acute anoxic injury, the EEG usually shows diffuse changes with one or more of several distinct patterns. The EEG usually remains normal for the first 5 to 6 seconds of anoxia. At 7 to 13 seconds, the record shows increasing amplitude and progressive slowing of the background. If circulatory arrest is further prolonged, the EEG becomes flat and featureless. After restoration of blood flow, the EEG may show several specific patterns. Reactive or nonreactive diffuse slowing of the EEG has been described to be associated with anoxic-hypoxic injury with about 64% showing activity reminiscent of sleep spindles. When spindle activity is present, the background activity tends to be more reactive to afferent stimuli. Occasionally, frontal intermittent rhythmic delta activity (FIRDA) (Fig. 8) is seen in acute conditions, but it is more often seen in chronic ischemia.

Continuous spikes or periodic burst suppression activity is often seen in severe postanoxic cases (Fig. 9). Abnormal motor activity such as myoclonic jerks or major motor seizures can be associated with these pat-
Diagnosis  Status epilepticus.

DISCUSSION
In this situation, an EEG is required because the muscle paralysis may mask ongoing seizures. Convulsive seizures might persist despite therapy or may change to nonconvulsive status epilepticus. An EEG confirms that electrographic seizure activity has stopped. Even without muscular paralysis, the EEG should be completed to ensure complete resolution of status epilepticus, especially if the clinical recovery is delayed.

Continuous EEG monitoring at the bedside may be employed to provide feedback regarding persistent seizures and the effects of therapy. It is also necessary for monitoring medically induced coma utilizing barbiturates, benzodiazepines, propofol, or other agents as a part of therapy for status epilepticus. Effectiveness of this treatment can be determined if the EEG reflects a burst suppression pattern or flat tracing. The EEG helps assure that status has not recurred when the depth of the medically induced coma has been reduced.

SUMMARY
The EEG remains a useful tool to assist in the diagnosis and prognosis of specific neurologic conditions. Understanding the specific situations in which the EEG is
helpful will allow efficient and effective use of this relatively safe and low-cost tool.

REFERENCES