Seizures are a common emergency presentation, accounting for approximately 1% of all ED visits. Presentations include patients with epilepsy, new-onset or first-time seizure (whether provoked or unprovoked), and other diagnostic entities that can mimic seizure but are not a true epileptic seizure. Even after a detailed and comprehensive evaluation, correctly determining the diagnosis can still be a challenge.

Seizure Phases

The International League Against Epilepsy (ILAE) defines epileptic seizures as “a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain.” There are typically three phases of a seizure—the aural, ictal, and postictal states.

Ictal Phase. The second stage of seizure, the ictal phase, is the typical cognitive or motor manifestations of seizure activity. Seizures can last several seconds to minutes, but the majority has a duration of less than 1 minute.

Postictal Phase. The postictal period occurs after the active phase of seizure and is characterized by confusion, altered mental status, and somnolence. The postictal period can last from several minutes to hours and can result in suppression of function; including cognitive or motor deficits such as Todd’s paralysis wherein a patient experiences transient paralysis confined to one hemisphere.

Etiology and Classification

Seizures can be subdivided based on two different categories: etiology or origin of abnormal electrical impulses within the brain. To categorize seizures based on etiology, the clinician must determine whether the seizure was brought on by an identifiable cause.

Provoked Seizure

Provoked seizures are also referred to as acute symptomatic seizures, because they...
present within 7 days of a systemic insult, whether it be secondary to an electrolyte abnormality (e.g., hyponatremia, hypoglycemia, hypercalcemia), substance withdrawal (e.g., alcohol, benzodiazepines), toxic ingestion, infection, central nervous system lesions, or head injury. The aforementioned does not represent a comprehensive list, but rather some of the more common etiologies of seizures.2,5

Unprovoked Seizure
An unprovoked seizure occurs without an identifiable acute precipitating insult. These types of seizures are generally more consistent with epilepsy or are due to a remote systemic insult greater than 7 days prior. Examples include patients who have a history of stroke, traumatic brain injury, or congenital brain malformation.2,5

Epilepsy is described as a seizure disorder where recurrent, usually unprovoked seizures occur. Determining the probable etiology of a seizure can be important when pursuing proper objective evaluation and work up, as we will discuss in this article.

Seizure Type
Seizures can also be classified as being generalized or focal, depending on the probable origin of the abnormal electrical discharges within the brain. This classification system is widely used and was developed by the ILAE.6

Generalized Seizures
Generalized seizures have bilateral cortical involvement at the onset of presentation and are associated with loss of consciousness. This is determined through electroencephalogram (EEG) monitoring because focal seizures, where the initiation of abnormal electrical discharges are located in one cortical hemisphere or localized area of the brain, may rapidly spread to both hemispheres and appear very similar to a primary generalized seizure.

Tonic-Clonic Seizures. The most colloquial type of generalized seizure is a tonic-clonic seizure. “Tonic” refers to the muscle stiffness or rigidity that occurs during this type of seizure, and “clonic” describes the rhythmic jerking of these muscles.

Nontonic-Clonic Seizures. Other types of generalized seizures include absence seizures (brief staring episodes or an arrest in behavior), atonic seizures (loss of muscle tone), and myoclonic seizures (brief, sudden muscular contractions).5

Focal Seizures
Focal seizures are diagnosed when the history, clinical presentation, and EEG findings support the localization of abnormal electrical neuronal discharges to one hemisphere of the brain. Loss of consciousness does not always occur during a focal seizure, and the ILAE recently updated the terminology in this regard to this distinction in 2017. Instead of classifying focal seizures as simple partial or complex partial in relation to the preservation of consciousness, the terminology has now changed to focal aware (no loss of consciousness) and focal impaired awareness (affected consciousness). Focal seizures can have not only motor manifestations, but may also present with sensory, autonomic, or psychic symptoms, depending on the anatomic location of the abnormal neuronal activity.5-6

Evaluation in the ED Setting
The classification of a seizure does not often change the ED management of seizures, but it is important to be able to recognize that seizures may present with different clinical appearances. It is also important to remember that not all seizure-like activity is due to epilepsy or abnormal neuronal discharges. There are several other conditions that can present with physical symptoms and characteristics similar to seizure, and are often misdiagnosed as seizures. The next section describes several of these seizure mimics and how to recognize or differentiate them from seizures through a careful history, physical examination, and
laboratory evaluation; as one diagnostic tool, the EEG, is not routinely available to the emergency physician (EP).

**Seizure Mimics**

**Syncope**

Syncope is secondary to decreased cerebral perfusion, which results in brief loss of consciousness and postural tone, and often with brief convulsions. Myoclonic jerking lasting a few seconds can be seen in many syncopal episodes, and if present is termed convulsive syncope. Following any syncopal episode, patients generally return to their baseline mental status without a postictal period. A prodrome of pallor and sweating can be helpful clues to identify a syncopal episode. In addition, a patient’s eyes may remain open during the event.

There are several types of syncope: cardiac, orthostatic, or neurocardiogenic (vasovagal). History and physical examination can help distinguish syncope from seizure.

**Cardiac Syncope.** Cardiogenic causes of syncope may be seen in elderly patients who lack a prodrome prior to the event, chest pain may have been present, the event may occur with exercise, or there is evidence of underlying heart disease. An electrocardiogram (ECG) should be done to detect cardiac dysrhythmias.

**Orthostatic Syncope.** Vital signs may be useful in assessing for an orthostatic cause of syncope (drop in systolic blood pressure [BP] by 20 mm Hg or more and drop in diastolic BP by 10 mm Hg or more within 3 minutes of standing), though orthostatic hypotension is common in the elderly. Dysautonomia as a cause of orthostatic hypotension may show a delayed drop in BP after standing 5 to 10 minutes, in contrast to hypovolemia which tends to be present with immediate standing.

**Neurocardiogenic Syncope.** Neurocardiogenic syncope, a somewhat confusing term, is perhaps better described as a reflex syncope, or simple faint. Often this is referred to as “vasovagal” syncope. Typically, there are physical or psychological noxious stimuli prior to the brief loss of consciousness and postural tone. Pain or strong emotions are common triggers.

**Convulsive Concussion**

Another seizure mimic is convulsive concussion in which the patient exhibits non-epileptic movement following a closed head injury. It is hypothesized that these post-traumatic convulsions are due to transient functional abnormalities, rather than structural brain injury. In one study, 22 cases of concussive convulsions were identified in which tonic-clonic convulsions began within 2 seconds of impact, and lasted for up to 150 seconds. These patients generally have good outcomes and do not require antiepileptic treatment; they also do not need to abstain from sports or other physical activities.

**Movement Disorders**

Certain movement disorders can appear similar to seizures with sustained muscle contractions, repetitive movements, dystonias, or even abnormal posturing. However, these abnormal movements are generally painful and there is often impairment of consciousness. They may be genetic in nature or secondary to a neurologic disease or medications such as neuroleptics or antipsychotics.

**Psychogenic Nonepileptic Seizures**

Psychogenic nonepileptic seizures (PNES) are defined as episodes of altered movements or sensations that appear similar to epileptic seizures, but have an underlying psychological etiology rather than abnormal neuronal discharges. Seventy percent of these patients have a psychiatric illness, such as depression, post-traumatic stress disorder, or personality disorders. Features that can help distinguish PNES from epileptic seizures include long duration, fluctuating symptoms, asynchronous or non-rhythmic movements, pelvic thrust-
ing, side-to-side head or body movements, closed eyes, lack of tongue biting, memory recall, crying, or suppression by distraction. Laboratory testing provides little benefit, aside from a lactate level, which if elevated can suggest a possible epileptic etiology. These cases may require consultation with neurology and psychiatry or video-EEG monitoring to correctly diagnose.

Other non-epileptic and possible seizure mimic diagnoses to be considered include stroke, transient ischemic attack, migraine headache, and sleep disorders.

**Evaluation**

When assessing a patient presenting with seizure-like activity or altered mental status, the clinician must keep a broad differential diagnosis. The first step is to evaluate the ABCs. Once that is completed, a blood glucose should be obtained, as it is a quick test and can determine whether hypoglycemia is the likely cause. Intravenous (IV) access should be obtained and routine labs ordered, including a complete blood count (CBC), a comprehensive metabolic profile (CMP), magnesium, urinalysis, ECG, and lactate. Other labs that may be of clinical utility in certain cases include anticonvulsant levels (in patients that are on these medications), toxicology screens, and cerebrospinal fluid studies, if indicated. It is important to note that anticonvulsant reference ranges are trough values, so levels that are drawn within a few hours of the last dose taken reflect a peak and falsely elevated level. Useful imaging may include computed tomography (CT) scan of the head and magnetic resonance imaging (MRI) of the brain. The American Academy of Neurology recommends cross-sectional imaging via CT of the head if there is a focal seizure onset, persistent neurologic deficit, if the patient is immunocompromised, or if the patient does not return to their baseline mental status. If a patient does not show progressive signs of increasing arousal or awareness within 30 minutes, an immediate EEG is indicated to assess for non-convulsive status epilepticus.

**Patient Disposition**

The management and ultimate disposition of a patient with a seizure depends on the underlying cause, and whether the patient has neurologic deficits and/or is back to their baseline mental status. For patients presenting with a first-time seizure and have returned to baseline, the American College of Emergency Physicians’ (ACEP) clinical policy states that precipitating medical conditions should be identified and addressed accordingly if it is a provoked seizure.

If patients present with a first-time unprovoked seizure and do not have evidence of brain injury or neurologic disease (ie, persistent altered mental status or abnormal neurologic examination), then the EP does not need to initiate antiepileptic medications. If there is evidence of neurologic disease or brain injury in an unprovoked seizure, then the EP may either choose to initiate antiepileptic medications or choose to defer, pending consultation with neurology. This group of patients (first-time unprovoked seizure back to baseline) do not need to be admitted to the hospital under the premise that they have a negative workup, to include glucose, CT scan (if indicated), ECG, CBC, and CMP. They must also have normal vital signs, be advised regarding seizure precautions such as not to drive until further medical evaluation (with duration perhaps being set by state law), and have good social support. They will require close follow up for further evaluation and definitive diagnosis, which may include head imaging, if not already performed in the ED, and EEG.

Patients with a history of epilepsy can have recurrent seizures that are either provoked or unprovoked. If the seizure appears clinically similar to their previous seizures, then causes that could lower the seizure threshold should be investigated; including compliance with antiepilep-
tic medications (obtain serum levels), infection (urinalysis and/or chest X-ray), sleep deprivation, electrolyte imbalances, or medications known to lower seizure threshold (eg, certain antibiotics such as fluoroquinolones, antidepressants such as bupropion and venlafaxine, and antipsychotics such as clozapine). These underlying causes should be treated accordingly. If a patient has been noncompliant with their medications, a loading dose can be given in the ED, although there are no definitive studies that either support or debunk this practice. If it is a true, unprovoked seizure and the patient is compliant with their antiepileptic medication, it is reasonable to discuss medication regimen changes with the patient’s neurologist.

A patient that presents with seizure-like activity and does not return back to their baseline mental status requires a more immediate and comprehensive evaluation. Persistent altered mental status has a vast differential diagnosis, and is outside the scope of this article, but if seizures were part of the clinical presentation, the possibility of non-convulsive epileptics should be considered. These patients may require treatment with medications (usually IV benzodiazepines), admission to the hospital, neurology consultation, EEG, imaging (CT vs MRI), and +/- lumbar puncture depending on the clinical scenario.

Status epilepticus will be further discussed in part 2 of this review.

References