nonconvulsive status epilepticus (NCSE) is defined as prolonged or recurrent electrographic seizure activity lasting more than 30 minutes without prominent motor (i.e. convulsive) clinical symptoms. There are as many types of NCSE as there are nonconvulsive epileptic seizures (Table 1). The hallmark of NCSE is an altered level of consciousness, which may range from mild confusion, inattention, or bizarre behavior to stupor or coma. Motor movements such as automatisms, eye deviation or nystagmoid eye jerks, or limb or facial twitching may be seen. Because NCSE cannot be clinically distinguished from other conditions causing stupor or coma, the electroencephalogram (EEG) is an essential tool for the diagnosis and classification of NCSE. The EEG criteria for NCSE are subjective and somewhat arbitrary, however, and even experienced electroencephalographers often disagree on whether a particular EEG pattern represents NCSE. This article will review the EEG diagnostic criteria for NCSE and common EEG patterns that can be confused with NCSE.

Which patients with altered mental status should get an EEG?

Because NCSE has pleomorphic clinical manifestations, its diagnosis is commonly delayed for hours or days or even missed entirely. The problem of delayed diagnosis is compounded by lack of availability of emergency 24-hour EEG in many hospitals. Even when off-hours EEGs are available, there is often a prolonged interval (three-24 hours) between the initial EEG request and interpretation.

Clinical scenarios in which an EEG should be considered include: 1.) Patients with unexplained acute change in mental status or personality, especially if there is a history of epilepsy or remote neurological injury.
2.) Patients with an acute neurologic injury with alteration in consciousness out of proportion to injury severity. NCSE is particularly common in critically ill neurology patients, including those with intracerebral hemorrhage (18-28 percent), severe traumatic brain injury (28 percent), CNS infection (26 percent), brain tumor (23 percent), and subarachnoid hemorrhage (eight percent). In the intensive care unit, concurrent neurological deficits, sedation, or neuromuscular blocking agents may further confound the diagnosis of NCSE.

3.) Patients with persistent unresponsiveness after cessation of convulsive status epilepticus or other clinically-evident seizures. In convulsive status epilepticus, EEGs show persistent NCSE in 14-20 percent of patients after treatment and cessation of convulsive motor activity. Patients with persistent NCSE in 14-20 percent of patients after treatment and cessation of convulsive motor activity may further confound the diagnosis of NCSE.

4.) Comatose or stuporous patients with subtle limb twitching or nystagmoid eye movements. NCSE may be present in eight percent of comatose or obtunded patients with no or subtle clinical motor signs.

Which EEG patterns are diagnostic of NCSE?

NCSE can be classified into three groups based on the predominant EEG pattern:

1.) NCSE which is generalized at onset,
2.) NCSE which begins focally, with or without secondary generalization, and
3.) NCSE which has both focal
and generalized features or is otherwise poorly classified.\textsuperscript{17}

In any of these groups, NCSE may consist of continuous ictal activity for more than 30 minutes or of recurrent discrete electrographic seizures, each lasting more than 10 seconds, for more than 30 minutes. If NCSE is discontinuous, background activity between seizures is usually slow or attenuated and may show interictal epileptiform discharges. In generalized NCSE, the EEG pattern at onset shows continuous or recurrent generalized repetitive rhythmic spikes, sharp waves, spike-wave, or polyspike-wave complexes that wax and wane in amplitude and frequency, usually faster than 3Hz. Partial or focal NCSE begins in a single brain region and then evolves in field, morphology, amplitude, or frequency. Some cases of NCSE show bihemispheric ictal patterns, with either a widespread field with focal predominance over one hemisphere or with shifting predominance between hemispheres.\textsuperscript{18} Focal NCSE may evolve into secondarily generalized NCSE. Because the EEG is usually started in the middle of NCSE, it is often impossible to distinguish whether the seizures were focal or generalized at onset.\textsuperscript{17} Some apparently generalized patterns become focal once treatment is started.

Unfortunately, there is significant controversy regarding which EEG patterns are consistent with a diagnosis of NCSE. The medical history, clinical state of the patient, and response to antiepileptic drugs (AEDs) may be required to make an accurate diagnosis. Several groups have proposed EEG criteria,\textsuperscript{1,5,19,20} but there remains a great deal of overlap between “ictal” and “non-ictal” patterns. A recent Epilepsy Research Foundation workshop report proposed several “clear-cut patterns” of NCSE.\textsuperscript{1} Most electroencephalographers would agree that frequent or continuous focal electrographic seizures with ictal patterns that evolve in amplitude, frequency, and/or spatial distribution represent focal NCSE. Patterns in generalized NCSE are more controversial. Rhythmic or repetitive generalized spikes, polyspikes, or spike-wave complexes are usually considered to be ictal if the repetition rate is faster than 2.5-3Hz. Spikes at slower than 1.5Hz are usually interictal, but patterns between 1.5 and 2.5Hz remain indeterminate. Some interpreters would call these intermediate frequencies NCSE if there is significant variability in the frequency of the discharges, evolution in the morphology of the waveforms, or a clear-cut clinical and electroencephalographic response to intravenous AEDs. Patterns which are generally agreed not to represent NCSE include periodic discharges such as periodic lateralized epileptiform discharges (PLEDs), bilateral independent periodic lateralized epileptiform discharges (BiPLEDs), periodic epileptiform discharges (PEDs), and triphasic waves. Fig. 1 provides an algorithm for the EEG diagnosis of NCSE, focusing on several main features adapted from prior published criteria.\textsuperscript{5,10}

The EEG criteria for NCSE become even more difficult as the EEG patterns evolve with time or as treatment is initiated. Treiman\textsuperscript{21} proposed a stereotyped sequence of EEG changes in generalized convulsive SE, from 1.) discrete seizures, 2.) merging seizures, 3.) continuous ictal activity, 4.) ictal activity with “flat”
periods of background discontinuity, and finally to 5.) periodic epileptiform discharges (PEDs) on a flat background. Stage 4 has subtle or no motor manifestations and is usually considered to represent generalized NCSE. Stage 5, PEDs, is not accepted as ictal by most electroencephalographers. Others have not found an orderly temporal progression of EEG patterns in SE.\textsuperscript{22,23} Evolution of the EEG depends on the baseline condition of the patient, time from NCSE onset, etiology, and treatment. Such changes make classification of SE based purely on EEG criteria more problematic.

A clear clinical and electrographic improvement after administration of intravenous AEDs, usually benzodiazepines, is often helpful when the EEG pattern is indeterminate. An EEG response alone is not conclusive, as benzodiazepines may suppress triphasic waves.\textsuperscript{24} Many comatose or obtunded patients may not show much clinical improvement immediately after IV AEDs, even if the EEGs improve, or the benzodiazepines may blunt the clinical response. Therefore, many controversial EEG patterns remain ambiguous.

**Generalized NCSE**

**Typical absence status epilepticus (Typical ASE).** Clinically, typical ASE is characterized by impairment of consciousness sometimes described as a “twilight state.” Patients are confused, with slowed thinking, staring, repetitive blinking or myoclonus.\textsuperscript{25} Typical ASE occurs in individuals with a history of primary generalized epilepsies (childhood absence epilepsy, juvenile absence epilepsy, juvenile myoclonic epilepsy), and less often de novo. The EEG shows generalized high voltage 3Hz spike-and-wave discharges or polyspike-and-wave discharges, usually maximal over frontal or central head regions (Fig. 2). Ictal activity can be either continuous seizures or recurrent brief electrographic seizures.\textsuperscript{26,27} Typical ASE usually shows a prompt clinical and EEG response to IV AEDs.

**Atypical absence status epilepticus (Atypical ASE).** Atypical ASE is seen in patients with epileptic encephalopathies such as the Lennox–Gastaut syndrome.\textsuperscript{28} Because such patients often have mental retardation and severely abnormal EEGs at baseline, diagnosis of NCSE can be difficult. There must be a clear change from baseline in both clinical status and EEG features.\textsuperscript{1} The EEG of atypical ASE shows generalized spike-wave activity at frequencies of 1 to 2.5Hz (faster than baseline EEG), superimposed on a diffusely slow background (Fig. 3).\textsuperscript{2,29}

**Subtle or electrographic generalized convulsive status epilepticus (SGCSE).** SGCSE describes the late stages of GCSE (i.e. stages 4 and 5 as described by Treiman)\textsuperscript{21} in comatose patients with minimal or no motor manifestations. The EEG shows repetitive generalized periodic epileptiform discharges or...
bursts of polyspikes, with frequencies from 0.5 to 4Hz.\textsuperscript{21,30} An identical EEG pattern can be seen in patients with severe encephalopathy (e.g. anoxic or uremic) without a preceding history of seizures.\textsuperscript{31}

**Focal NCSE**

Complex partial status epilepticus (CPSE). CPSE can show a variety of clinical manifestations, including confusion, staring, unresponsiveness, and oral and limb automatisms. CPSE may arise from any brain region, but is most commonly frontal or temporal.\textsuperscript{32} CPSE nearly always shows discernable ictal patterns on scalp EEG, but EEG during frontal or parietal CPSE may be normal or obscured by artifact.\textsuperscript{33,34} The EEG patterns of CPSE are highly variable and similar to those seen in brief complex partial seizures, reflecting differences in ictal onset zones and propagation pathways.\textsuperscript{35} Common morphologies include repetitive spikes, spike-and-slow wave discharges, and rhythmic waveforms in the theta, delta, or alpha frequency range (Fig. 4). Early EEG manifestations are usually focal or lateralized, but ictal activity can spread to produce diffuse or generalized patterns difficult to distinguish from ASE.\textsuperscript{17,36,37} CPSE can either be continuous or recurrent.\textsuperscript{2,38,39}

**Focal NCSE in critically ill obtunded or comatose patients.** Electrographic partial SE occurs in stuporous or comatose patients with no clear clinical signs of seizure activity.\textsuperscript{20} The EEG patterns are similar to those in CPSE, often superimposed on a slow or attenuated background or alternating with periodic epileptiform discharges. Electrographic partial SE is commonly seen after strokes or other acute brain injuries and should be suspected when patients do not stabilize or improve as expected.\textsuperscript{35}

**Nonepileptic NCSE** *(Psychogenic or pseudo-SE)*

In some cases, patients with nonepileptic psychogenic seizures can present with altered mental status mimicking NCSE. The EEG shows normal background activity or artifact from muscle or eye movements,\textsuperscript{40} and an alpha rhythm can be elicited by passive eye opening and closure. Because missing this diagnosis can result in overly aggressive treatment with AEDs, EEG should be performed for any patient with suspected NCSE who does not respond to initial treatment.\textsuperscript{31,42}
Controversial Patterns

A number of EEG patterns exist at the borderland of NCSE. These are typically periodic focal or generalized discharges at repetition rates slower than 2.5 Hz in patients with altered mental status in the setting of acute brain injuries. Distinguishing these patterns from NCSE may be difficult or impossible. Because these patterns are associated with a high likelihood of electrographic seizures, continuous EEG monitoring for 24 hours should be considered when they are seen.

The following section provides examples of the most common periodic patterns and their relationship to NCSE.

Periodic lateralized epileptiform discharges (PLEDs and PLEDs plus). PLEDs are repetitive lateralized polyphasic sharp-and-slow wave or spike-and-wave complexes with repetition rates of 0.5-2 Hz. The discharges are typically broadly distributed over most of one hemisphere and background activity between the discharges is severely attenuated and slow (Fig. 5). PLEDs occur most commonly after acute large destructive lesions such as stroke and infection.43 PLEDs are usually not considered to be an ictal pattern, but are highly associated with seizures. Clinical seizures occur in 75-84 percent of patients with PLEDs43,44 and electrographic seizures in even higher proportions (Fig. 5). “PLEDs plus” is a term used to described PLEDs with complex morphology, prolonged afterdischarges, intervening fast frequencies, and rapid (>2 Hz) repetition rates.44,45 Occasionally, PLEDs themselves may be ictal, usually with a discharge frequency greater than 1.5-2 Hz and with clinical manifestations of focal motor status epilepticus.46 Determining whether PLEDs are ictal or not may require correlation with functional neuroimaging studies or response to AEDs.

Bilateral independent periodic epileptiform discharges (BiPLEDs). BiPLEDs are morphologically similar to PLEDs, but occur asynchronously and independently over the two hemispheres.47 Background activity is usually severely attenuated diffusely. BiPLEDs are seen in association with acute bilateral destructive lesions such as anoxic encephalopathy and CNS infection. BiPLEDs are associated with poorer mental status (coma in 72 percent vs. 24 percent) and higher mortality rates (61 percent vs. 29 percent) than PLEDs, but focal seizures are less common (55 percent vs. 80 percent).47

Generalized periodic epileptiform discharges (GPEDs). GPEDs are continuous generalized sharp-and-slow waves, spikes, polyspikes, or triphasic waves with a repetition rate of approximately 1 Hz, arising from a diffusely attenuated background (Fig. 6). GPEDs are most commonly seen after severe anoxia or other metabolic insults, or in the late stages of generalized convulsive status epilepticus. Treiman et al. consider GPEDs to be ictal and recommend aggressive AED therapy,21 while others believe that GPEDs are symptoms of severe acute neuronal injury rather than seizures, and do not require aggressive treatment.48 No features definitively distinguish GPEDs after status epilepticus from those after anoxia.31 Seizures are common in patients with GPEDs (Husain et al. 1999; Yemisci et al. 2003). A longer EEG recording can help to exclude electrographic seizures in patients in whom the initial pattern is equivocal.

Triphasic waves. Triphasic waves are named for their multiphasic morphology, with an initial low-amplitude, negative sharp wave followed by a high-amplitude positive sharp wave and a lower amplitude, negative broad slow wave (Fig. 7). They are broadly distributed, usually with maximal amplitude in frontal regions, but may also be maximal posteriorly. Repetition rate is usually 1-2 Hz, but may occasionally be faster. Triphasic waves occur in metabolic encephalopathies, most commonly hepatic encephalopathy, as well as uremia, hypothyroidism, toxic ingestions, and diffuse structural lesions.

Triphasic waves may be particularly difficult to distinguish from NCSE.49 Although there are no absolute EEG criteria for diagnosis, the epileptiform discharges in generalized NCSE typically show shorter wave 1 duration, higher frequency (mean 2.4 vs. 1.8 Hz), polyspikes, and sharper morphology than triphasic waves.50 A phase lag from anterior to posterior
channels is often seen with triphasic waves, but not NCSE. Triphasic waves are commonly state responsive, and may be abolished by administration of IV AEDs.24

**Stimulus-Induced Rhythmic, Periodic or Ictal Discharges (SIRPIDs).** Some periodic and quasi-periodic discharges can be elicited in stuporous or comatose patients by stimulation or occur with spontaneous arousal. State-related discharges can be focal or generalized. Hirsch et al.1,2,9,102 termed these “stimulus-induced rhythmic, periodic, or ictal discharges” or SIRPIDs. The discharges abate when the patient lapses back into unresponsiveness. Whether these discharges represent seizures or an abnormal arousal process is debated; in general, discharges with repetition rates greater than 3Hz with clear evolution may be thought to be ictal, while invariant patterns slower than 3Hz are likely not ictal. SIRPIDs are seen frequently during prolonged EEG monitoring in critically ill patients.

**Conclusion**

The diagnosis of NCSE by EEG can be difficult, as there are no universally accepted criteria for ictal EEG patterns in NCSE. Patterns with repetition rates faster than 2.5-3Hz are usually ictal, and those slower than 1Hz usually interictal. For the patterns in the border zone of 1.5-2.5Hz, the clinical and EEG response to intravenous AEDs may be helpful, but many patterns remain ambiguous.

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