

# Psychiatric Manifestations of Nonconvulsive Status Epilepticus

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## Abstract

Nonconvulsive status epilepticus (NCSE) is clinically characterized by altered mental status and the diagnosis is confirmed by electroencephalography. Absence status (AS) and complex partial status (CPS) are the two primary types of NCSE. Patients in NCSE may exhibit a wide range of clinical presentations including subtle memory deficits, bizarre behavior, psychosis, or coma. While prognosis is dependent on the underlying etiology and possibly related to duration of the event, there is limited research in this area. Treatment focuses on correcting underlying pathologic abnormalities such as hyponatremia or drug toxicity, and initiating pharmacologic therapy. The benzodiazepines are considered the first line treatment for both AS and CPS.

**Key Words:** Nonconvulsive status epilepticus, absence status, complex partial status.

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## Introduction

NONCONVULSIVE STATUS EPILEPTICUS is a term that incorporates a number of disparate conditions that have the common denominator of prolonged altered mental status due to ongoing seizure activity. Prognosis varies depending on the underlying etiology. The clinical presentation can vary from minimal confusion to bizarre behavioral manifestations to psychosis to coma. Consequently, the diagnosis can be missed and, if the entity is not considered, can at times misinterpreted to be psychiatric in origin. The recognition of this entity was made possible by the introduction of the electroencephalogram (EEG) in the 1930s, which allowed for clinical and EEG correlation. Nonetheless, to this day, it can still represent a diagnostic challenge for both the neurologist and the psychiatrist. Indeed, because its primary manifestation is an alteration in behavior, NCSE serves as a model condition defining the field of neuropsychiatry.

In 2004, a prestigious group of neuroscientists met to discuss the definition, diagnosis, and management of NCSE (1). That goal was to arrive at a consensus that would facilitate communication and research in this area. Interestingly, the group did not include a psychiatrist despite the fact that a significant number of patients with NCSE are misdiagnosed as having a psychiatric condition which at times leads to a delay in receiving the appropriate management (2). The objective of this article is to review the current understanding of NCSE, heighten awareness of the condition, and ultimately improve the care these patients receive.

## Definitions

The Epilepsy Research Foundation's proposed definition for nonconvulsive status epilepticus is: "A range of conditions in which electrographic seizure activity is prolonged and results in nonconvulsive clinical symptoms" (1). At first glance, this does not seem a useful definition, and yet it reflects the difficulty faced by the workgroup in finding commonalities between the wide range of disorders that result in NCSE. The definition builds off of past definitions that characterized NCSE as a state of continuous or intermittent seizure activity without a return to baseline, lasting more than thirty minutes. Historically, thirty min-

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utes has been used as a defining feature for status epilepticus, yet the time is arbitrary and recent definitions of status epilepticus have used time frames as short as five minutes (3). In general, NCSE differs from convulsive status by the lack of a predominant motor component (4). The hallmark of NCSE is a change in behavior or mental status that is associated with diagnostic EEG changes.

There are two main categories of NCSE: absence status, which is a primary generalized process, and complex partial status, which is focal in origin. A third category is subtle generalized convulsive status epilepticus, which describes a subset of patients who have sustained significant cerebral insult, often after an episode of convulsive status epilepticus, and are in a comatose state that is at least partially due to ongoing seizure activity (5). Since the etiology and prognosis of subtle convulsive status is distinctly different from that of AS and CPS, it will not be further discussed in this article.

Both absence and complex partial status are characterized by a change in the level of consciousness and behavior, with either no or minimal motor activity. Onset can be sudden or gradual, and the duration can vary from minutes to days or months (6). Unresponsiveness, once thought to be a distinctive feature of CPS (7), has also been described in association with absence status (8, 9); the same is true for cyclic patterns (4). Absence status is usually characterized by abrupt clearing of consciousness, though gradual postictal normalization has been reported (6). CPS is usually followed by a prolonged postictal state with depression.

Historically, absence status was reported to be more common than complex partial status but improved electroencephalographic techniques have brought this into question (4, 10). Rapid generalization of CPS on EEG is the most likely explanation for misdiagnosing cases of CPS as absence status, possibly compounded by unawareness of this issue (4, 11). Supporting this concept are reports of AS responding to pharmacologic interventions that are usually not indicated in AS (e.g., phenytoin and phenobarbital); it is possible that in these cases the actual diagnosis was CPS with rapid generalization (12).

In summary, clinical manifestations of AS and CPS overlap significantly and there are no clear clinical criteria that accurately differentiate the two. The overlap in clinical presentations and the potential for rapid generalization on EEG poses a challenge in clinical decision making.

### Epidemiology

Establishing the incidence of NCSE is challenging since the diagnosis requires availability of

an EEG. Most of the studies in the literature include only hospitalized patients, and even in those studies there is a selection bias, i.e., if the diagnosis had not considered, the EEG was not obtained. It is easier to establish the incidence of generalized convulsive status epilepticus (GCSE) since its clinical manifestations are recognizable: In a population based study, DeLorenzo et al. report an incidence of 41 per 100,000. Population-based epidemiological studies have indicated mortality from GCSE to be almost 22%, and that figure can increase to 30% in the elderly (13).

Regarding NCSE the Epilepsy Foundation Research workgroup amalgamated the data from the literature and reported an estimated incidence of 6–18 cases / 100,000 / year (1). Towne et al., in a prospective study of 236 patients with coma who had no clinical evidence of seizures, reported that 8% of the patients met criteria for NCSE on EEG (11); DeLorenzo et al. found that NCSE was present in 14% of patients after control of GCSE (13). In a prospective population-based study of status epilepticus, NCSE represented approximately 5% of status epilepticus cases presenting in Richmond, Virginia (14). Privitera and Strawsburg reported that in a prospective study of 198 patients with altered consciousness, 37% had EEG findings suggestive of NCSE (15). These studies emphasize the need to keep NCSE in the differential diagnosis of any patient with altered mental status when the etiology is unknown: These studies also bring to light the question of whether electrophysiology should be available 24/7.

NCSE has been reported in all age groups from the very young to the very old and in both sexes without a clear predominance in either sex (11, 16, 17). NCSE does not require a prior history of seizures, with the literature reporting that 10–100% of patients who present with NCSE having no history of a seizure disorder (4, 6, 7, 9, 17–19). It is estimated that 10% of adults with absence seizures will have at least one episode of absence status (6, 20). Though absence status has mostly been reported in children it can present *de novo* in later life (18, 21).

A variety of precipitating factors have been implicated in NCSE including metabolic abnormalities, infection, drug toxicity, alcohol intoxication / withdrawal, pregnancy, central nervous system (CNS) lesions, and electroconvulsive therapy treatment (8, 21, 22). Different series have identified a precipitating factor in 15–70% of cases, emphasizing the importance of a comprehensive assessment in the evaluation of these patients (7, 10).

### Clinical Characteristics

Clinical manifestations of NCSE include the full spectrum of mental status changes. Findings

can be so subtle that they are recognized only by family or by friends, while at the other end of the spectrum these patients can appear delirious or in coma; see Table 1 (6, 2, 7, 11, 23–29). Fluctuations of symptoms can occur with varying degrees of impairment, which at times contributes to obscuring the diagnosis.

Motor activity is normal in most cases; however, decreased response time, clumsiness, apraxia, focal jerks, twitching of facial muscles (in particular of the eyelids and automatisms such as licking, chewing or picking) have been reported (4, 30). When present, automatisms have been thought to be longer in duration and more complex in nature in CPS than in AS (31, 32). Automatisms in the form of gross movements such as positioning, raising, flexion or extension of the extremities, head deviation, though not common, have also been noted (4).

In view of the panoply of clinical presentations, it becomes clear how challenging this diagnosis can be in any circumstance, but especially with a patient who presents with cognitive impairment at baseline (e.g., mental retardation or dementia) or who carries a prior history of a psychiatric disorder.

### Diagnostic Approach

The differential diagnosis of altered mental status is extensive, and being able to determine the underlying cause on clinical grounds alone is at times almost impossible. Table 2 lists some situations in which a diagnosis of NCSE should be considered. After reviewing the wide range of presen-

**TABLE 1**  
*Clinical Presentations of Patients in Nonconvulsive Status Epilepticus*

- 
- Mild cognitive disturbances (e.g., impaired attention, difficulty in sequential planning of complex motor tasks)
  - Mild disorientation or confusion
  - Prolonged confusional states
  - Mood disturbance
  - Cortical blindness
  - Speech disturbance (e.g., verbal preservation, reduced verbal fluency, muteness, speech arrest or aphasia)
  - Echolalia
  - Confabulation
  - Bizarre behavior that is uncharacteristic or deviates from baseline (e.g., laughing, dancing and singing inappropriately)
  - Psychotic states
  - Autonomic disturbances (e.g., belching, borborygmi, flatulence)
  - Sensory and psychic phenomena
  - Coma
- 

**TABLE 2**

*Situations Where Nonconvulsive Status Should Be Considered*

- 
- Prolonged postictal period (more than two hours) after a generalized tonic-clonic seizure
  - Altered mental status associated with twitching or blinking and/or fluctuating mental status
  - Altered mental status of unexplained etiology, especially in patients with a history seizures
  - Unexplained altered mental status in the elderly
  - Stroke patients who look clinically worse than expected
- 

tations of NCSE, it is easy to understand how the diagnosis is often missed and confused with a psychiatric disorder or a neurologic insult. In order to put the patient's presentation into the proper context, a detailed history needs to be obtained, including change from baseline status, onset and duration of the events, presence or absence of lucid interval, timing in relation to the sleep/wake cycle, and presence or absence of motor activity or automatisms. Medical, neurologic and psychiatric history, family history, social history, and medication history are other important components of the diagnostic evaluation.

Currently, there are no clear criteria for deciding when an EEG should be requested. That said, when NCSE is suspected on clinical grounds, an EEG is indicated to confirm the diagnosis and direct management. Table 3 lists the types of EEG patterns identified by the Epilepsy Research Foundation's workshop (1).

In absence status, EEG is characterized by continuous or nearly continuous generalized, rhythmic, bilaterally synchronous, spike-and-wave discharges at intervals of 3/second with a maximum over the bifrontal region; however, variations in the EEG pattern can occur, including 2–3 per

**TABLE 3**

*Electrographic Criteria of Nonconvulsive Status Epilepticus*

- 
- Frequent or continuous focal electroencephalographic seizures
  - Frequent or continuous generalized spike wave discharges in a patient without a prior history of epilepsy
  - Frequent or continuous generalized spike discharges which show significant changes in intensity or frequency compared to baseline EEG
  - Periodic lateralized, or periodic bilateral, epileptiform discharges occurring in patient in coma after a generalized tonic clonic seizure
  - Frequent or continuous EEG abnormalities in patient with no previous similar abnormalities
  - Frequent or continuous generalized EEG abnormalities in patients with epileptic encephalopathies in whom similar interictal EEG patterns are seen but in whom clinical symptoms are suggestive of nonconvulsive status
-

second spike-waves complexes as well as bursts of rhythmic slow, rhythmic or arrhythmic spike waves or polyspike activity (4, 17, 33, 34). In CPS, various forms of less synchronous seizure activity, rhythmical slowing, rhythmic spikes, and rhythmic sharp and slow waves have been described (4, 30, 26). One should be aware that while several EEG patterns have been described to be suggestive of both types of NCSE, a clear pathognomonic pattern has not been defined. When (and if) an EEG is performed at the onset of the seizure, a clear focus may be identified that may be critical to differentiating CPS from AS. If secondary generalization is present, either because of rapid spread or because the EEG is performed when a patient has been in CPS for a long period of time, then there is the risk of misclassification (4, 35). This has significant clinical implications since long-term pharmacologic management is different for primary and secondary generalized seizure disorders.

### Case Presentations and EEG

#### Case #1

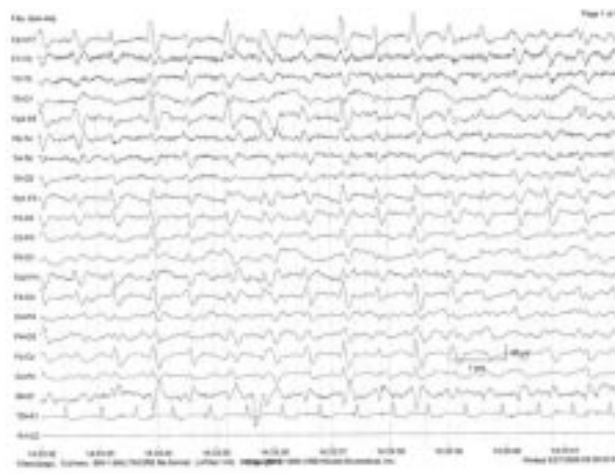
On the second hospital day, a 68-year-old man with a small bowel obstruction was found unresponsive to both verbal and painful stimuli. He had no other medical problems. The obstruction was secondary to adhesions from past surgery. He was taking no medications. His physical and neurologic examinations were non-focal. An EEG was obtained (Fig. 1). The EEG shows bilateral, asynchronous, almost continuous sharp wave discharges, which improved with 2 mg of lorazepam.

#### Case #2

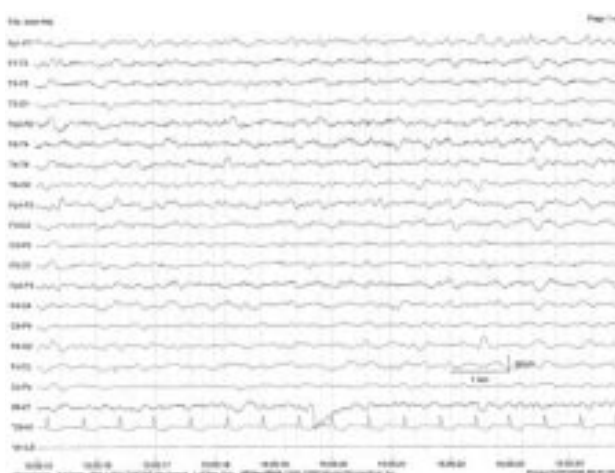
An 83-year-old woman with no history of seizure disorder developed a fever of 102°F. She developed a seizure characterized by focal left arm twitching followed by secondary generalization. The seizure lasted several minutes but the patient remained “post-ictal” for hours after the event leading to obtaining an EEG (Fig. 2). The EEG shows generalized bilaterally synchronous sharp waves at 2/second, which resolved after a loading dose of phenytoin was given.

### Prognosis

Prognosis of NCSE is dependent on the underlying etiology; however, good research in this area is limited. Without characterizing the type and etiology of NCSE, it is not possible to assign a predictable outcome. Outcomes do not appear to be as



A

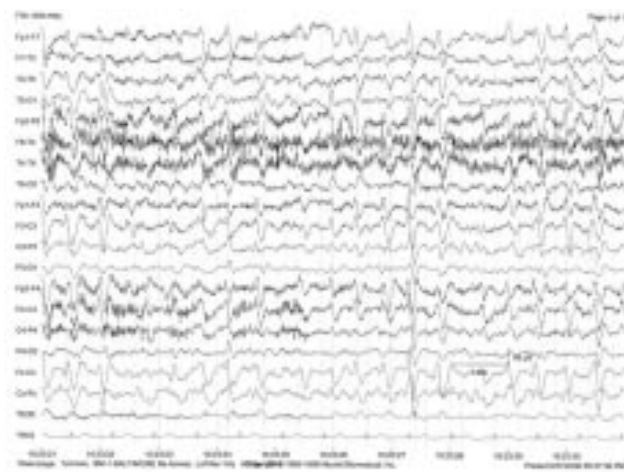


B

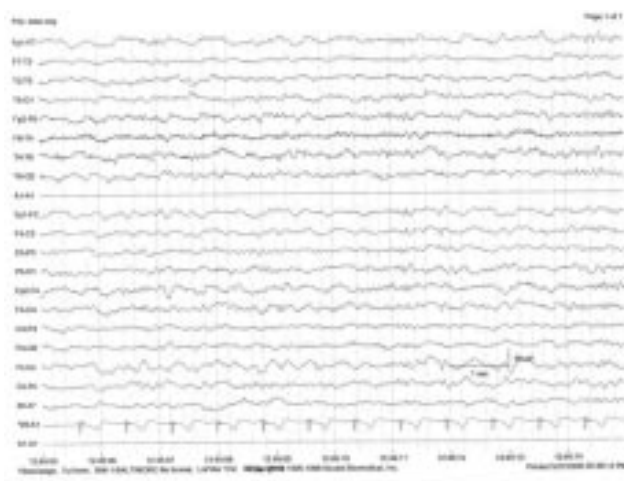
**Fig. 1.** EEG of a 68-year-old man with a small bowel obstruction: (A) before medication and (B) after 2 mg of lorazepam. EEG and case courtesy of Dr. James Rowan.

closely linked to duration of the event as in generalized convulsive status.

Prognosis is dependent on the population and type of NCSE; for example, elderly patients in subtle generalized status have an extremely poor prognosis and studies on this group recommend aggressive therapy (36). Shneker and Fountain found that in patients who had an acute medical problem as the underlying cause of NCSE, overall mortality was as high as 27% (37). A study of elderly patients with NCSE concluded that a prognosis NCSE for an elderly patient was worse because of the severity of the underlying etiology and because of hospital-acquired infection (38). On the other hand, other series on patients with CPS and AS suggest that NCSE is a relatively benign condition (37). Indeed, there is a report in the litera-



A



B

**Fig. 2.** EEG of an 83-year-old woman who developed a seizure: (A) before medication and (B) after loading dose of phenytoin. EEG and case courtesy of Dr. James Rowan.

ture of a case where NCSE lasted nine years, in which the patient did not exhibit any definite cognitive changes (39).

The question regarding prognosis relates to distinguishing between the reversible cognitive impairment that resolves when the NCSE is treated and permanent damage that results from the ongoing and persistent neuronal discharges. Since most patients do not have both pre- and post-seizure neurocognitive testing, it is difficult to ascertain cause and effect. However, studies looking of markers of brain injury suggest that NCSE does predispose to permanent injury at some level (1). In convulsive status, even when systemic effects are controlled with paralysis and ventilation, CNS damage in the hippocampus (40, 41) occurs relatively quickly. The mortality rate ranges from 5–50% (42); in this case

aggressive treatment is advocated since it is thought that the outcome is related not only to the underlying etiology of the episode but to its duration (32). In NCSE, it has been suggested that neuronal damage may occur (42), but controversy still exists regarding the need for aggressive therapy since there are reports of both complete recovery and of associated transient or permanent cognitive or neurologic deficit (28, 43).

The limiting factor in better assessing the true morbidity and mortality of NCSE is due to the variety of clinical symptoms, potential misinterpretation of EEG patterns, grouping of patients with different comorbidities, and lack of prior cognitive function or neuropsychological assessment for comparison. Due to the lack of well-designed studies there are still no clear guidelines for the treatment of NCSE and there is still significant controversy regarding the need for aggressive therapy.

In summary, the data suggests that prognostically NCSE generally does not result in permanent cognitive or neurologic deficit unless it occurs in the setting of an underlying medical problem. Further controlled prospective studies and clear guidelines are needed for the management of these difficult patients.

### Treatment

Despite several controversies regarding the importance of the timing in initiating treatment in NCSE, general principles in the management of NCSE include early identification of etiological and precipitating factors so that they can be corrected as soon as possible. Physiological stressors, including infections, toxins, metabolic abnormalities, structural lesions, drug interactions or withdrawal and pregnancy, should be sought in all patients presenting with either a new seizure or exacerbation of a known disorder.

NCSE had been identified for more than 50 years, though to this day it is still unknown if the morbidity that can occur in NCSE is secondary to NCSE *per se* or if it is secondary to underlying conditions. Clear guidelines have not been established on the importance of the timing in initiating treatment, choice of anticonvulsants, or how aggressively we should institute treatment, especially in case of treatment resistance. Ultimately, after a careful risk/benefit assessment, treatment is tailored to the individual situation. In order to assess any correlation between the EEG pattern and clinical symptoms, intravenous benzodiazepine should be administered during EEG monitoring. Confirmation of NCSE by EEG is recommended before instituting pharmacological treatment, whenever possible. Some clinicians have opted to give benzodiazepine without EEG guidance when NCSE is clinically

suspected, however, the pros and cons involved in this practice must be carefully weighed.

There have been cases described in the literature were spontaneous recovery or immediate response to i.v. benzodiazepines has been observed (13), although episodes of NCSE often tend to reoccur and require an additional anticonvulsant. The questions that are still unanswered are which anticonvulsants should be used as a second or third line treatment, how aggressive the treatment should be, and how to balance the risks and benefits of treatment.

Benzodiazepines, e.g., diazepam (4, 7), lorazepam (44), clonazepam (4), and midazolam (3), have all been used as monotherapy or in combination. It has been shown that NCSE responds to benzodiazepine, even if at times the response is delayed, or followed by a recurrence of symptoms hours or days later; therefore, the use of long-acting antiepileptic drugs (AED)s may be required in order to achieve lasting effects (4, 28).

Benzodiazepines are particularly indicated in case of *de-novo* absence when the precipitating factor is secondary to benzodiazepine withdrawal (21), or in cases of mitochondrial encephalomyopathy with lactic acidosis and stroke-like episodes (Melas syndrome), in which case valproate and phenytoin are not viable choices (45). The use of benzodiazepines should be carefully assessed in patients who are medically compromised and in whom possible hypotension or respiratory depression may occur. Of note, it has been reported that the response to i.v. benzodiazepines can be different in AS vs. CPS, in that in the former the response has been reported to be more abrupt, whereas in the latter it is more variable (44).

Once the diagnosis of NCSE is determined and seizures controlled, long-term treatment should be considered. Several antiepileptic drug options are available. Phenytoin, valproic acid, and phenobarbital have all been suggested. They can be administered intravenously in the attempt to control an acute event or orally when long-term treatment is required. Carbamazepine, primidone, and the new generation AEDs (e.g., lamotrigine, topiramate, levetiracetam) are also considerations for long-term treatment. Valproic acid is the drug of choice for managing patients with absence seizures (46). Ethosuximide and clonazepam have also been used (47). Vigabatrin and tiagabine have not been recommended due to concerns that they might play a role in worsening or precipitating NCSE (48).

### Conclusion

Nonconvulsive status epilepticus is more common than once appreciated and should be consid-

**TABLE 4**

*Key Concepts in Nonconvulsive Status Epilepticus (NCSE)*

- 
- Nonconvulsive status epilepticus is characterized by altered mental status ranging from subtle memory deficits to coma
  - Prognosis in NCSE is related to etiology
  - Altered mental status or coma of undetermined etiology and prolonged “postictal” periods should prompt an investigation of NCSE
  - Treatment of absence status and complex partial status is less emergent and less aggressive than for convulsive status epilepticus
  - NCSE persisting after the treatment of convulsive status epilepticus portends a poor prognosis
  - Benzodiazepines are the first-line treatment for NCSE
- 

ered in any patient presenting with an alteration in mental status of indeterminate etiology. Table 4 lists the key points that summarize concepts presented in this article. The clinician must remain aware of the different clinical characteristics of this disorder and remember that a seizure history is not necessary for the diagnosis, nor is there necessarily associated motor activity. The diagnosis is confirmed with an EEG, which is required in order to distinguish absence status from complex partial status. Treatment begins with correcting underlying etiologies and using a benzodiazepine. Response can be delayed, in which case other anticonvulsants are indicated. The overall prognosis is generally good for both AS and NCSE, although the literature is unclear if neurocognitive outcomes are related to duration of the status. There is still a great deal that is unknown about NCSE, and a need for well-designed studies in both diagnosis and therapeutics.

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