The Management of Status Epilepticus
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Status epilepticus is a major medical emergency associated with significant morbidity and mortality. Status epilepticus is best defined as a continuous, generalized, convulsive seizure lasting > 5 min, or two or more seizures during which the patient does not return to baseline consciousness. Lorazepam in a dose of 0.1 mg/kg is the drug of first choice for terminating status epilepticus. Patients who continue to have clinical or EEG evidence of seizure activity after treatment with lorazepam should be considered to have refractory status epileptics and should be treated with a continuous infusion of propofol or midazolam. This article reviews current information regarding the management of status epilepticus in adults.

Key words: anticonvulsants; barbiturates; lorazepam; midazolam; phenytoin; propofol; refractory status epilepticus; status epilepticus

Abbreviations: CI = confidence interval; GABA = γ-aminobutyric acid; NMDA = N-methyl-D aspartate; VA = Veterans Affairs
Many types of epileptic seizures have been described, and, therefore, it follows that there are many types of status epilepticus. This has led to complex classifications of status epilepticus. However, using electroclinical features, status epilepticus may be classified simply by the presence of motor convulsions (convulsive status epilepticus) or their absence (nonconvulsive status epilepticus). They may be further divided into status epilepticus that affects the whole brain (generalized status epilepticus) or only part of the brain (partial status epilepticus). This review will focus predominantly on generalized convulsive status epilepticus, which is the form most commonly observed in clinical practice.

Classification

Many types of epileptic seizures have been described, and, therefore, it follows that there are many types of status epilepticus. This has led to complex classifications of status epilepticus. However, using electroclinical features, status epilepticus may be classified simply by the presence of motor convulsions (convulsive status epilepticus) or their absence (nonconvulsive status epilepticus). They may be further divided into status epilepticus that affects the whole brain (generalized status epilepticus) or only part of the brain (partial status epilepticus). This review will focus predominantly on generalized convulsive status epilepticus, which is the form most commonly observed in clinical practice.

Epidemiology

It has been estimated that up to 150,000 cases of status epilepticus and 55,000 deaths from it occur annually in the United States. Regardless of geographic influences, status epilepticus appears to be more frequent among men, blacks, and the aged. Geographic, sex, age, and race influence the epidemiology of status epilepticus. An incidence of between 6.2 and 18.3 per 100,000 population has been reported in the United States. Regardless of geographic influences, status epilepticus appears to be more frequent among men, blacks, and the aged.

The incidence of status epilepticus in the elderly population is at least twice that of the general population. Status epilepticus in the elderly is of great concern because of the existence of concurrent medical conditions that are more likely to complicate therapy and worsen the prognosis.

Etiology

In many patients with a preexistent seizure disorder, no obvious precipitating factor can be determined. A fall in serum levels of antiepileptic drugs due to poor compliance with medications or to due to increased clearance associated with concurrent illness has been implicated in some patients. Adult patients with a new diagnosis of epilepsy may first present while in status epilepticus. Genetic factors likely play a role as twin studies have demonstrated a greater concordance in monozygotic as apposed to dizygotic twins. Table 1 depicts the most common causes of status epilepticus seen in "first-world" populations.

Pathophysiology

It is likely that the ineffective recruitment of inhibitory neurons together with excessive neuronal excitation play a role in the initiation and propagation of the electrical disturbance occurring in status epilepticus. \( \gamma \)-Aminobutyric acid (GABA) is the major inhibitory neurotransmitter in the CNS. It is released from GABAergic neurons and binds to several types of GABA receptors (ie, GABA-A, GABA-B, and GABA-C receptors). GABA receptors are macromolecular proteins that form a chloride ion channel complex and contain specific binding sites for GABA and a number of allosteric regulators, including barbiturates, benzodiazepines, and a number of anesthetic agents. GABA receptor-mediated inhibition may be responsible for the normal termination of a seizure. In addition, the activation of the \( \mathrm{N} \)-methyl-D-aspartate (NMDA) receptor by the excitatory neurotransmitter glutamate may be required for the propagation of seizure activity. The activation of NMDA receptors results in increased levels of intracellular calcium, which may be responsible for the nerve cell injury seen in patients in status epilepticus. A growing body of basic science and clinical observation supports the concept that status epilepticus becomes more difficult to control as its duration increases. It is postulated that this may occur due to a mechanistic shift from inadequate GABAergic inhibitory receptor-mediated transmission to excessive NMDA excitatory receptor-mediated transmission.

Table 1—Common Causes of Status Epilepticus

<table>
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<tr>
<th>Cause</th>
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<tr>
<td>Antiepileptic drug noncompliance</td>
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<tr>
<td>Alcohol related</td>
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<td>Cerebrovascular accidents</td>
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<td>Drug toxicity (ie, cephalosporins, penicillins, ciprofloxacin, tacrolimus, cyclosporin, theophylline, and cocaine)</td>
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<td>CNS infections (eg, meningitis and encephalitis)</td>
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<td>CNS tumors (primary or secondary)</td>
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<td>Metabolic disturbances (eg, electrolyte abnormalities, sepsis, and uremia)</td>
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<tr>
<td>Head trauma</td>
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<tr>
<td>Cerebral anoxia/hypoxia</td>
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<td>Hypoglycemia or hyperglycemia</td>
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did not have preexisting seizures or systemic abnormalities. Neuron-specific enolase, a marker for acute neurologic injury, has been demonstrated to be increased in patients with nonconvulsive status epilepticus who did not have preceding or coexistent cerebral injury. Thom and coworkers demonstrated evidence of acute neuronal injury using heat shock protein-70 and c-Jun immunohistochemistry in patients who had sudden and unexpected death from epilepsy. Neuronal death is probably caused by the release of excitatory neurotransmitters. In an experimental model, Mikati and coauthors have demonstrated that increased NMDA activation results in increased ceramide levels followed by programmed cell death.

**Diagnosis**

Status epilepticus may be divided into two stages. The first stage is characterized by generalized convulsive tonic-clonic seizures that are associated with an increase in autonomic activity that results in hypertension, hyperglycemia, sweating, salivation, and hyperpyrexia. During this phase, cerebral blood flow is increased due to increased cerebral metabolic demands. After approximately 30 min of seizure activity, patients enter the second phase, which is characterized by the failure of cerebral autoregulation, decreased cerebral blood flow, an increase in intracranial pressure, and systemic hypotension. During this phase, electromechanical dissociation may occur in which, although electrical cerebral seizure activity continues, the clinical manifestations may be restricted to minor twitching.

The diagnosis of status epilepticus is straightforward in patients with witnessed generalized convulsive tonic-clonic seizures. However, status epilepticus may not be considered in patients who have progressed to the nonconvulsive phase of status epilepticus and present in coma. All comatose patients should therefore be carefully examined for evidence of minor twitching, which may involve the face, hands, or feet, or may present as nystagmoid jerking of the eyes. Towne and colleagues evaluated 236 patients with coma and no overt seizure activity. Eight percent of patients in this study were found to have nonconvulsive status epilepticus, as determined by EEG monitoring. Therefore, it is essential that an urgent EEG be performed in patients with unexplained coma.

**Treatment**

Status epilepticus is a medical emergency that requires rapid and aggressive treatment to prevent neurologic damage and systemic complications. The longer status epilepticus remains untreated, the greater the neurologic damage. In addition, the longer an episode of status continues, the more refractory to treatment it becomes and the greater is the likelihood of chronic epilepsy. The management of status epilepticus involves the rapid termination of seizure activity, airway protection, the taking of measures to prevent aspiration, the management of potential precipitating causes, the treatment of complications, the prevention of recurrent seizures, and the treatment of any underlying conditions.

**General Measures**

As with any critically ill patient, the first step in the management of a patient with status epilepticus should be to ensure an adequate airway and to provide respiratory support. The patient should be positioned so that they cannot harm themselves during the seizure activity. Two large-gauge IV catheters should be inserted to allow fluid resuscitation and pharmacotherapy. Should peripheral venous access be difficult, the placement of a central venous catheter is recommended. Despite the periods of apnea and cyanosis that occur during the tonic or clonic phases of their seizure, most patients in status epilepticus breathe sufficiently well as long as the airway remains clear. An oral airway may be required once the seizure has terminated to prevent airway obstruction. Once the seizures are controlled, and if the patient is oxygenating and ventilating adequately, endotracheal intubation may not be required for airway protection, even if the patient remains comatose. However, in this situation precautions should be taken to avoid aspiration, and a nasogastric tube should be placed to ensure that the stomach is empty. Endotracheal intubation will be required in patients who continue to experience seizures despite receiving first-line therapy. There are no available data as to the pharmacologic agents that are preferred for achieving endotracheal intubation. As these patients will be comatose and would already have received therapy with lorazepam, a hypnotic agent is usually not required. However, an anesthetic induction dose of propofol, midazolam, or etomidate may terminate the seizure activity and facilitate intubation.

Neuromuscular blockade will be required to facilitate intubation in patients who continue to have tonic-clonic seizure activity despite these pharmacologic interventions. Rocuronium (1 mg/kg), a short-acting, non-depolarizing muscle relaxant that is devoid of significant hemodynamic effects and does not increase intracranial pressure, is the preferred agent. Guercynylcholine should be avoided, if possible, as the patient may be
hyperkalemic as a consequence of experiencing rhabdomyolysis. Prolonged neuromuscular blockade should be avoided.

Hypoglycemia must be excluded rapidly, and corrective measures must be instituted if serum levels of glucose are low. If the prompt measurement of blood glucose levels is not possible, the patient should receive 100 mg IV thiamine followed by a 50-mL bolus of 50% dextrose. BP, ECG, and temperature should be monitored. If the patient develops significant hyperthermia (ie, temperature > 40°C), then passive cooling is required. Blood specimens should be obtained for the determination of serum chemistry levels. Continuous motor seizures may lead to muscle breakdown, with the release of myoglobin into the circulation. The maintenance of adequate hydration is necessary to prevent myoglobin-induced renal failure. Forced saline solution diuresis and urinary alkalinization should be considered in the presence of myoglobinuria or significantly elevated serum creatine kinase levels (ie, > 5,000 to 10,000 U/L). Brain imaging with a CT scan and/or MRI as well as a lumbar puncture will be required in patients presenting with a previously undiagnosed seizure disorder once the seizure activity has been controlled. It is important to emphasize that the first priority is to control the seizures. Imaging studies should be performed only once the seizure activity has been controlled. Endotracheal intubation and neuromuscular paralysis for the sole purpose of imaging the patient may increase morbidity and is strongly discouraged.

PHARMACOTHERAPY

Because only a small fraction of seizures go on to become status epilepticus, the probability that a given seizure will proceed to status is small at the start of the seizure and increases as the seizure duration increases. If a seizure lasts > 5 min, clinical experience suggests that the likelihood of spontaneous termination decreases. The goal of pharmacologic therapy is to achieve the rapid and safe termination of the seizure, and to prevent its recurrence without adverse effects on the cardiovascular and respiratory systems or altering the level of consciousness. Diazepam, lorazepam, midazolam, phenytoin, fosphenytoin, and phenobarbital have all been used as first-line therapy for the termination of status epilepticus. These drugs have different pharmacodynamic and pharmacokinetic properties, which determine their rapidity of clinical effect, their efficacy in terminating status epilepticus, and their duration of action. The benzodiazepines bind to the benzodiazepine binding site on the GABA receptor complex, increasing GABAergic transmission, while the barbiturates act directly on the GABA receptor. The antiseizure activity of phenytoin is complex, however, its major action appears to block the voltage-sensitive, use-dependent sodium channels.

The publication of the Veterans Affairs (VA) cooperative trial in 1998 and the San Francisco Emergency Medical Services study in 2001 allows for an evidence-based approach to the choice of the first-line agent to use in terminating status epilepticus. The VA cooperative study randomized 384 patients with overt generalized status epilepticus into four treatment arms, as follows: lorazepam, 0.1 mg/kg; diazepam, 0.15 mg/kg, followed by 18 mg/kg phenytoin; phenytoin, 18 mg/kg; and phenobarbital, 15 mg/kg. Successful treatment required both clinical and EEG termination of seizures within 20 min of the start of therapy, and no seizure recurrence within 60 min from the start of therapy. Patients who did not respond to the first treatment received a second choice of treatment drug and, if necessary, a third choice. The latter choices were not randomized, because this would have resulted in some patients receiving two loading doses of phenytoin, but the treating physician remained blinded to the treatments being given. Status epilepticus was terminated in 64.9% of patients randomized to lorazepam, 55.2% of those randomized to phenobarbital, 55.8% of those randomized to diazepam and phenytoin, and 43.6% of those randomized to phenytoin (p = 0.002 for lorazepam vs phenytoin). There was no difference between the arms in recurrence rates.

The San Francisco Emergency Medical Services study was a randomized, double-blind trial to evaluate IV benzodiazepine administration by paramedics for the treatment of out-of-hospital patients with status epilepticus. In this study, 205 patients were randomized to IV diazepam (5 mg), lorazepam (2 mg), or placebo. An identical second injection was administered if needed. Status epilepticus had terminated at arrival in the emergency department in 59.1% of the patients treated with lorazepam, in 42.6% of the patients treated with diazepam, and in 21.1% of patients treated with placebo (lorazepam vs diazepam: odds ratio, 1.9; 95% confidence interval [CI], 0.9 to 4.3). The duration of the status epilepticus was shorter in the lorazepam group compared to the diazepam group (adjusted relative hazard, 0.65; 95% CI, 0.36 to 1.17). These data are supported by a double-blind study reported by Leppik et al in 1983 in which 78 patients with status epilepticus were randomized to receive one or two doses of either lorazepam (4 mg) or diazepam (10 mg). Seizures were controlled in 89% of the episodes treated with lorazepam and in 76% of those treated with diazepam. Although the
dosages of lorazepam and diazepam differed in these three studies and phenytoin was added to diazepam in the VA study, the summed data indicate that lorazepam is significantly more effective in terminating seizures than is diazepam (odds ratio, 1.74; 95% CI, 1.14 to 2.64; p = 0.01). Furthermore, the pharmacokinetic properties of lorazepam favor its use over that of diazepam. The anticonvulsant effect of a single dose of diazepam is very brief (20 min), whereas that of lorazepam is much longer (> 6 h), and the risk of respiratory depression may be greater with diazepam. Although diazepam has a much longer elimination half-life, due to its high lipid solubility it is rapidly redistributed from the brain to the peripheral fat stores, accounting for its shorter antiseizure activity.

Based on these data, lorazepam in a dose of 0.1 mg/kg is recommended as first-line therapy for the control of status epilepticus. Although refrigeration is recommended for lorazepam, but not for diazepam, Gottwald and coworkers have demonstrated that lorazepam retains 90% of its original concentration when stored without refrigeration in ambulances (in San Francisco) for 5 months. Based on this information, lorazepam should replace diazepam in hospital code carts and “orange bags,” it should be stored in light-proof containers, and should be re-stocked every 4 to 6 months. Many authorities recommend phenytoin, 20 mg/kg (or fosphenytoin), following the administration of lorazepam. While there are no data that demonstrate that phenytoin increases the response rate following the use of lorazepam, this agent may prevent recurrent seizures and is recommended in patients without a rapidly reversible process (eg, the effect of subtherapeutic antiepileptic drug concentrations).

Continuous EEG monitoring is required in patients who do not recover consciousness once the convulsive seizure has aborted. In a study by DeLorenzo and colleagues, after the cessation of convulsions, 48% of patients continued to have seizure activity and 14% of patients had persistent nonconvulsive status epilepticus.

**Management of Refractory Status Epilepticus**

In the VA cooperative study, 55% of patients with generalized convulsive status epilepticus did not respond to first-line therapy. The aggregate response rate to a second first-line agent (eg, lorazepam, diazepam, phenytoin, or phenobarbital) was 7%, and to a third first-line agent it was 2.3%. Only 5% of patients with status epilepticus who did not respond to lorazepam and phenytoin therapy, responded to phenobarbital administration. These data suggest that refractory status epilepticus is much more common than is generally appreciated and that phenobarbital should not be used as a second (or third-line) agent in patients who have failed to respond to lorazepam. Furthermore, the limited data available suggest that the administration of further doses of lorazepam will not be useful.

A variety of agents has been recommended for the treatment of refractory status epilepticus, including midazolam, propofol, high-dose thiopentone or pentobarbital, IV valproate, topiramate, tiagabine, ketamine, isoflurane, and IV lidocaine. Treatment guidelines are difficult to formulate as refractory status epilepticus has not been studied in a prospective clinical trial. Currently, however, a continuous IV infusion of midazolam or propofol together with continuous EEG monitoring is the preferred mode of treatment. Both agents have been reported to be successful in the control of patients with refractory status epilepticus. It should, however, be pointed out that this recommendation is based on limited clinical data, with just > 100 cases of treatment with these agents having been reported.

Claassen and colleagues reported a “systematic review” that compared the outcome of patients with refractory status epilepticus who had been treated with pentobarbital, propofol, or midazolam. In this report, there were fewer treatment failures and breakthrough seizures with the use of pentobarbital than with the use of propofol or midazolam. As this study was a summation of 28 individual case series that did not control for the underlying medical condition, the cause of seizure, type of seizure, length of time prior to treatment, prior therapy, and end points of therapy, it is difficult to make any definitive conclusions regarding drug efficacy from this report.

The goal regarding the activity on the EEG remains a matter of debate. There is no prospectively collected evidence that a burst-suppression EEG pattern is required for, or is efficacious for, the termination of status epilepticus. Many patients can achieve complete seizure control with a background of continuous slow activity and do not incur the greater risks associated with higher doses of medication required to achieve a burst-suppression pattern.

Midazolam is a fast-acting, water-soluble benzodiazepine with a half-life of 4 to 6 h. The drug undergoes hepatic transformation into an active metabolite that is renally cleared. One of the major disadvantages of midazolam is tachyphylaxis. After 24 to 48 h, the dose of the drug often must be increased several fold to maintain seizure control. Furthermore, the drug accumulates with prolonged infusion, which may result in a prolonged time to
awakening. Midazolam is given as a loading dose of 0.2 mg/kg, followed by an infusion of 0.1 to 2.0 mg/kg/h titrated to produce seizure suppression by continuous EEG monitoring.

Propofol is an IV alkyphenol (2,6-diiisopropylphenol), which has been used extensively for the induction and maintenance of anesthesia and for sedation in the ICU. Propofol is a global CNS depressant. It directly activates the GABA receptor. In addition, propofol inhibits the NMDA receptor, modulates calcium influx through slow calcium ion channels, and has antioxidant activity. Experimental data have shown propofol to have strong anticonvulsant properties, which have proved to be very effective in controlling refractory status epilepticus. Propofol is highly lipophilic with a large volume of distribution. This property results in rapid uptake and elimination from the CNS, resulting in rapid onset of action and rapid recovery when discontinued. Recovery is rapid even with prolonged use. Propofol is metabolized by glucuronide and sulfate conjugation, and does not accumulate with long-term infusion. Dose reduction is not required in patients with hepatic or renal disease. Furthermore, the drug is easily titratable. A loading dose of 3 to 5 mg/kg is recommended followed by an infusion of 30 to 100 μg/kg/min titrated to EEG seizure suppression. After 12 h of seizure suppression, the dose is gradually titrated by 50% over the next 12 h and then titrated to 0% over the subsequent 12 h. If seizure activity should recur during the weaning period, a further loading dose of 1 to 3 mg/kg should be administered followed by infusion with the rate increased to obtain another 12-h seizure-free period.

Propofol has been administered to > 40 million patients with a remarkable safety record. The most severe complication associated with propofol is the propofol infusion syndrome, a very rare complication reported predominantly in pediatric patients and associated with high-dose propofol infusion. The propofol infusion syndrome is characterized by severe metabolic acidosis, rhabdomyolysis, and cardiovascular collapse frequently leading to death. Circumstantial data suggest that this disorder is due to interference with mitochondrial respiration. It is possible that the full-blown propofol infusion syndrome occurs only in those individuals with a genetic susceptibility. However, the risk appears to be higher in children, in whom the drug is contraindicated. It is currently recommended that the dosage not exceed 100 μg/kg/min in adults. Hyperlipidemia may result from the failure of free fatty acid metabolism and hence may be a useful early marker of the development of the syndrome. Consequently, triglyceride and creatine kinase levels (a marker of rhabdomyolysis) should be monitored in patients receiving prolonged high-dose infusions of propofol.

High-dose barbiturate therapy is associated with hemodynamic instability and immune paresis. Due to their side effects, therapy with barbiturates is reserved for those patients who do not respond to midazolam or propofol. Pentobarbital therapy, in a dose of 10 to 15 mg/kg/h followed by a dose of 0.5 to 1.0 mg/kg/h, is recommended. The pharmacologic approach to a patient in status epilepticus is outlined in Figure 1.

The Management of Nonconvulsive Status Epilepticus

Nonconvulsive status epilepticus constitutes approximately 20 to 25% of status epilepticus cases, occurring in about 8% of all comatose patients without clinical signs of seizure activity, and persisting in 14% of patients after generalized convulsive status epilepticus. Some have suggested that nonconvulsive status epilepticus is a benign condition that does not require aggressive therapy. However, the prognosis of nonconvulsive status epilepticus depends on the etiology and the level of consciousness. These are associated with significant morbidity in those patients with a depressed level of consciousness. Furthermore, experimental and clinical data suggest that nonconvulsive status epilepticus may cause ongoing neuronal injury. Shneker and Fountain reviewed their experience with 100 cases of nonconvulsive status epilepticus. In this report, nonconvulsive status epilepticus was associated with a high mortality rate (18%) and a significant morbidity rate (39%), with the mortality rate correlating with the underlying etiology of nonconvulsive status epilepticus, the degree of impairment in mental status, and the development of acute complications. The mortality rate was 18% in those patients with cryptogenic nonconvulsive status epilepticus, attesting to the serious sequelae of ongoing seizures. Based on this information, we suggest that comatose patients with nonconvulsive status epilepticus be treated aggressively as outlined above for refractory convulsive status epilepticus.

Prevention of Seizure Recurrence Once Status Epilepticus Is Terminated

Once status epilepticus is controlled, attention turns to preventing its recurrence. The best regimen
for an individual patient will depend on the cause of the seizure and any history of antiepileptic drug therapy. A patient who develops status epilepticus in the course of ethanol withdrawal may not need antiepileptic drug therapy once the withdrawal has run its course. In contrast, patients with new, ongoing epileptogenic stimuli (e.g., encephalitis) may require high dosages of antiepileptic drugs to control their seizures.

**Prognosis**

The prognosis of status epilepticus depends on several factors including the clinical presentation, the duration of seizures, the age of the patient, and, most importantly, the underlying disorder causing the seizures. Refractory status epilepticus has a mortality rate of up to 76% in elderly patients. In a population-based, long-term mortality study, the 10-year cumulative mortality rate among 30-day survivors was 43%, with a standardized mortality ratio of 2.8. However, the mortality rate of those patients with idiopathic status epilepticus was not increased (standardized mortality ratio, 1.1).

**Conclusions**

Patients who have generalized seizures that continue for more than 5 min should be considered to have status epilepticus and should be treated with a single IV dose of lorazepam (0.1 mg/kg). Patients who continue to have clinical or EEG evidence of seizure activity after receiving treatment with lorazepam should be considered to have refractory status epilepticus and should treated with a continuous infusion of propofol or midazolam.

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