# Chapter 31
# Status Epilepticus


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## 31.1 Introduction

An epileptic seizure represents an isolated and self-limited episode, usually of short duration, characterized by synchronous and abnormal depolarization of a given group of cortical neurons. Occasionally, central inhibitory mechanisms fail to abort this phenomenon, or excitatory mechanisms are triggered, leading to abnormal prolonged seizures. This condition can cause neuronal injury, neuronal death, and long-term consequences. In the early sixties, status epilepticus (SE) was defined as an epileptic seizure which, due to its prolonged duration or frequent recurrence, generates a neurological damage [1]. Later, the International League Against Epilepsy (ILAE) Task Force for the classification of SE developed a revised definition of SE, but did not establish a seizure length for a definitive diagnosis of SE [2, 3]. Thus, several definitions can be found in the literature and an operational definition was adopted. For most investigators, SE is a single epileptic seizure or several recurrent seizures without recovery of consciousness, lasting at least 30 minutes [4, 5].

The length of an epileptic seizure considered SE was a topic of discussion in the literature [4]. Epileptic seizures with a minimum duration of 30 minutes were compared with seizures lasting between 10 and 29 minutes [5]. Although both groups of patients demonstrated similar epidemiological characteristics, 93% of seizures lasting 30 minutes or more required administration of antiepileptic drugs to cease them. In the group of patients with seizures lasting 10 to 29 minutes, the seizures ceased spontaneously in 43% of the patients. Mortality was also significantly different

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between the two groups, being 19% for the group with crises during at least 30 minutes, and 2.6% for the group with crises during 10 to 29 minutes. These data demonstrated that the 30-minute length seems to be ideal to define SE, although there is no rationale for waiting 30 minutes to start a specific therapy in the clinical practice. Treatment should start as soon as possible in the pre-SE phase, in order to avoid the evolution to established and refractory SE. However, from the point of view of classification, prognosis, and evolution, it should be considered that seizures lasting up to 29 minutes differ from those with minimal duration of 30 minutes [5].

In 2015, the task force came out with the following definition [6]: "Status epilepticus is a condition resulting either from the failure of the mechanisms responsible for seizure termination or from the initiation of mechanisms, which lead to abnormally prolonged seizures (after time point t1). It is a condition, which can have long-term consequences (after time point t2), including neuronal death, neuronal injury, and alteration of neuronal networks, depending on the type and duration of seizures" [6] This new definition of SE gives a good guidance on when to consider an emergency treatment. In general, t1 is the time point when treatment should be started, which is at 5 minutes for generalized tonic–clonic seizures, and at 10 minutes for focal seizures with or without impairment of consciousness. On the other hand, t2 marks the time point at which neuronal damage or self-perpetuating alteration of neuronal networks may begin, indicating that SE should be later controlled by that time (30 minutes in case of generalized tonic clonic seizures).

Operational definitions have been established to conduct the treatment appropriately, in each phase.

## 31.2 SE Classification

In 1962, during the "X European Conference of Epileptology and Clinical Neurophysiology", SE was first subdivided into subtypes convulsive and nonconvulsive [1].

More recently, there have been several proposals in the literature for new classifications encompassing all types of SE and, at the same time, incorporating information on semiology, anatomy, and etiology [7, 8], or even more specific classifications, as SE clinical presentation (focal or generalized onset, and either convulsive or nonconvulsive presentation) [9]. Although interesting, these classifications are complex and often ineffective for clinical purpose. Treiman et al. [10] introduced the term "subtle generalized convulsive" SE to identify patients who remain unresponsive after the apparent interruption of generalized epileptic crises. These patients are usually stuporous or comatose, showing subtle clinical manifestations such as minimal eyelid, facial or mouth movements, nystagmus, tremor or focal clonic movements of the trunk or limbs, or even total absence of movement.

In 2015, ILAE proposed a new classification considering semiology, etiology, electroencephalography (EEG) correlates, and age [6]. From the clinical perspective, the presence or absence of prominent motor symptoms and the degree

(qualitative or quantitative) of impairment of consciousness are used, which may be summarized as the initial classifications in convulsive SE (with motor signs) and nonconvulsive SE (NCSE) (without evident motor signs). Table 31.1 shows in detail the semiology-based classification.

Based on etiology (underlying cause), SE can be classified into SE with *known* or *symptomatic causes* (structural, metabolic, inflammatory, infectious, toxic, or genetic); *SE in defined electroclinical syndromes;* and *unknown SE.* The *known* group can be further subdivided according to its temporal relationship, into *acute* (e.g., stroke, intoxication, and encephalitis), *remote* (e.g., posttraumatic, postencephalitic, and poststroke), and *progressive* (e.g., brain tumor, Lafora's disease and other progressive myoclonic epilepsies, dementias) [6].

The EEG is very useful in SE and, although there is no specific pattern, its findings are crucial in the diagnosis of NCSE. It is recommended to describe the EEG

**Table 31.1** Classification of status epilepticus (SE) based on semiology

| |
|---|
| *With prominent motor symptoms* |
| Convulsive SE (CSE or tonic–clonic SE) |
| Generalized convulsive |
| Focal onset evolving into bilateral convulsive SE |
| Unknown whether focal or generalized |
| Myoclonic SE (prominent epileptic myoclonic jerks) |
| With coma |
| Without coma |
| Focal motor |
| Repeated focal motor seizures (Jacksonian) |
| Epilepsia partialis continua (EPC) |
| Adversive status |
| Oculoclonic status |
| Ictal paresis (i.e., focal inhibitory SE) |
| Tonic status |
| Hyperkinetic SE |
| *Without prominent motor symptoms (*i.e.*, nonconvulsive SE, NCSE)* |
| NCSE with coma (including so-called "subtle" SE) |
| NCSE without coma |
| *Generalized* |
| Typical absence status |
| Atypical absence status |
| Myoclonic absence status |
| Focal |
| Without impairment of consciousness (aura continua, with autonomic, sensory, visual, olfactory, gustatory, emotional/ psychic/experiential, or auditory symptoms) |
| Aphasic status |
| With impaired consciousness |
| Unknown whether focal or generalized |
| Autonomic SE |

findings according to the *location* of critical activity (generalized, lateralized, bilateral independent, multifocal), *pattern* (periodic discharges, rhythmic delta activity, or spike-and-wave/sharp-and-wave plus subtypes), *morphology* (sharpness, number of phases [e.g., triphasic morphology], absolute and relative amplitude, polarity), *time-related features* (prevalence, frequency, duration, daily pattern duration and index, onset, and dynamics) *modulation* (stimulus-induced or spontaneous), and *effect of the intervention* on EEG (e.g., medication) [6].

It is also recommended to classify SE according the age groups, in *neonatal SE* (0 to 30 days), SE of *infancy* (1 month to 2 years), SE of *childhood* (>2 to 12 years), SE of *adolescence* and *adulthood* (>12 to 59 years), and SE of *elderly* (≥60 years) [6].

The development of more detailed and specific systems to classify SE has been encouraged, since there is no classification system encompassing clinical and research needs so far.

## 31.3 Incidence and Mortality

It is very difficult to obtain accurate data on SE incidence grounded in community-based studies. The first population study on SE conducted in the USA [11] estimated an incidence of about 50 episodes of SE per 100,000 individuals per year. The cases of SE showed a bimodal distribution, with one peak occurring in the first year of life and another after 60 years of age [11]. This finding is in contrast to previous studies that identified only one peak in childhood [12].

The projection of the incidence of SE reported by DeLorenzo and collaborators in Richmond, Virginia [11], for the Brazilian population suggests the occurrence of approximately 100,000 cases of SE per year, which is much more frequent than usually assumed.

The highly variable mortality rate associated with SE can reach up to 58%, depending on the etiology, mostly in SE secondary to acute factors such as stroke, CNS infection, and metabolic disorders [13]. It is very dependent on the etiology and the age group of the patients [13]. The mortality exclusively related to prolonged epileptic seizure is fortunately much lower (between 1 and 2%) [11].

## 31.4 Pathophysiology

The systemic effects of convulsive SE can be divided into two stages: Stage I or Compensated Phase (0–30 minutes) and Stage II or Decompensated Phase (30–90 minutes).

In Stage I, the brain autoregulation and homeostasis are still preserved. The brain area in which the crisis originates requires a greater supply of glucose and oxygen, in addition to an adequate blood flow, to remove water and carbon dioxide. The

prolonged epileptic seizure causes a massive release of catecholamines and increases blood glucose, heart rate, and blood pressure, initially keeping cerebral perfusion at adequate levels and providing the muscles with the necessary substrates for exhaustive contraction. The increase in muscle activity produces large amounts of heat, as well as hyperthermia above 40 °C. The presence of hyperthermia may cause brain damage and worsen the prognosis [14, 15].

In Stage II, the mechanism of autoregulation of the cerebral blood flow is compromised, becoming dependent on blood pressure. The lactic acidosis leads to lack of responsiveness of peripheral vessels to circulating catecholamines and this effect, added to the drop in the levels of catecholamines, causes progressive hypotension, compromising the cerebral blood flow and further reducing the supply of glucose and oxygen. Hypoglycemia also occurs due to the exhaustion of glycogen stocks and the increased secretion of neurogenic insulin.

Experimental studies on pathophysiology also show that: (1) hippocampal activity is activated during SE; (2) loss of GABA-mediated inhibitory synaptic in the hippocampus is fundamental to establish the SE, and finally, (3) glutamatergic synaptic transmission maintains SE and causes cell death [15, 16].

The hippocampal CA1 and CA3 pyramidal neurons and the dentate hilus are highly and selectively vulnerable to SE neuronal injury, while other regions, such as neurons in the CA2 region and the granular cells of the dentate gyrus, are more resistant. Other cortical regions are affected in varying degrees, and the neuronal injury is similar to that observed in severe hypoxia and ischemia.

Epileptogenesis alone leads to neuronal hyperexcitability, and the excitotoxicity is mediated by glutamate and aspartate. The epileptic activity produces abnormalities in the neuronal membrane and failure of the calcium pump, leading to calcium influx into the cell; this, in addition to acidosis and the action of excitatory amino acids, can lead to cell death. In summary, diverse mechanism of action of several factors would lead to the common final pathway of cell death and brain inflamation[14,15.16].

## 31.5 Clinical Manifestations and Physical Examination

The clinical diagnosis of convulsive SE is straightforward. Patients present “episodes of excessive abnormal muscle contractions, usually bilateral, which may be sustained or uninterrupted” and last 5 minutes or more; alternatively, they can have recurrent seizures without complete recovery of consciousness between the events, lasting at least 30 minutes [17]. On the other hand, the diagnosis of both focal and generalized nonconvulsive status epilepticus requires identification of the impairment of consciousness associated with the finding of an ictal electrographic pattern.

Operational definition is important for classifying SE in stages based on seizure duration and to guide treatment.

Considering seizure duration, SE can be divided into four stages:

- *Stage I*—Pre-SE or imminent SE—epileptic seizures lasting more than 10 minutes and less than 30 minutes
- *Stage II*—Established SE—epileptic seizures lasting 30 minutes or more and less than 60 minutes
- *Stage III*—Refractory SE—epileptic seizures lasting longer than 60 minutes
- *Stage IV*—Super-refractory SE—epileptic seizures lasting more than 24 hours, despite the use of anesthetic drugs

Clinical signs depend on the semiology of the crisis, as well as of the SE stage. In convulsive SE, the tonic phase is usually followed by clonic movements, with massive sympathetic outpouring such as pupillary dilatation, tachycardia, hypertension, and hyperglycemia. Consciousness is impaired at the beginning and should be recovered as soon as the SE ends. Sometimes, convulsive SE evolves to an ongoing nonconvulsive seizure activity, that is referred to as subtle status epilepticus. Common manifestations include nystagmus, blinking, eye deviation, speech arrest, and stereotyped automatisms.

In nonconvulsive SE, the clinical manifestations are variable. In stage I, if the seizure does not compromise the consciousness, patients can be completely oriented and report their medical history, while patients in stage IV are in drug-induced coma.

Other clinical and neurological signs will depend on the etiology of SE. The neurological examination of a patient with a focal lesion may reveal focal signs, depending on the location of the lesion. Nuchal stiffness, fever, and mental confusion are data that can be found in patients with infection of the central nervous system (CNS).

## 31.6 Neuroimaging/ Electroencephalogram

Imaging exams such as noncontrast computer tomography (CT) scans of the head are useful to identify structural lesions in patients suspected of having hemorrhage or ischemic stroke, malformation, calcified lesions, tumor, or traumatic brain injury. The magnetic resonance imaging (MRI) of the brain has a higher sensitivity for identification of structural abnormalities such as cortical development malformation, heterotopia, small lesions not identified by CT scan, or even hippocampal sclerosis.

EEG is essential for the differential diagnosis and the clinical follow-up, especially in refractory SE, when its findings influence the choice and the aggressiveness of the treatment, as well as dictate the prognosis. In patients under treatment with benzodiazepines, barbiturates, general anesthetics, or similar drugs, it is practically impossible to determine, based only on clinical data, the persistence, or resolution of SE [18, 19]. Therefore, in this very critical condition, it is recommended to

monitor patients with EEG recordings as early as possible. This very easy and simple technology will help to properly handle these patients.

EEG can also be useful in differentiating focal SE with disperceptive seizures and generalized nonconvulsive SE, as well as to help diagnose "subtle" tonic seizures with axial movements only (ocular deviation or mild cervical tonic contraction) [6]. We also recommend that all patients suspected of having refractory SE should be submitted at least to a single EEG recording, in order to rule out another possible and often misdiagnosed condition, like psychogenic nonepileptic seizures or nonepileptic events [20].

Other important exams to identify the etiology of SE are glycemia, urea, creatinine, electrolyte imbalances (sodium, potassium, calcium, phosphorus, magnesium), arterial gasometry, complete blood count, aspartate and alanine aminotransferase, gamma-GT, alkaline phosphatase, coagulation tests, antiepileptic drug levels (in patients with known epilepsy, if taking any), blood and urine screening with tests (culture and antibiogram), and toxicological analysis. In specific cases, consider obtaining a virus screening panel, blood culture, antiperoxidase, antithyroglobulin, and screening for inborn errors of metabolism.

For suspected central nervous system (CNS) infection, a lumbar puncture is recommended for cerebrospinal fluid (CSF) sampling, besides pressure measurement. The analysis of CSF should include cell count, glucose, protein, lactate, Gram stain, microbiologic serologies, bacterial and fungal cultures plus polymerase chain reaction (PCR) for enterovirus and herpes simplex virus types 1 and 2. Consider evaluating protein electrophoresis, CSF-exclusive oligoclonal bands, additional viral PCR, and autoantibodies for diagnosis of autoimmune encephalitis.

## 31.7 Differential Diagnosis

Any neurological disease with alteration of consciousness or torpor, such as toxic or metabolic encephalopathies, infections of the central nervous system, and stroke, may be distinguishable from nonconvulsive SE. In a patient with impaired consciousness, certain EEG patterns suggest metabolic, toxic, or infectious encephalopathy; drug intoxication; or focal brain injury (Figs. 31.1 and 31.2).

Among various epileptic or nonepileptic paroxysmal disorders that mimic SE, the psychogenic crises are probably the most frequent. Nonepileptic psychogenic seizures (NEPS) are a diagnostic finding often misdiagnosed and treated as epileptic seizures. The clinical manifestations can be very similar to the epileptic seizures and, therefore, have a variable semiology, with prolonged duration or recurrence within short intervals. It is estimated that about 20% of patients referred to epilepsy centers have psychogenic seizures [20]. However, it should not be forgotten that an epileptic patient may also have psychogenic seizures [20].

EEG recording during NEPS seizure is normal, with movement artifacts, but without any epileptiform discharges.

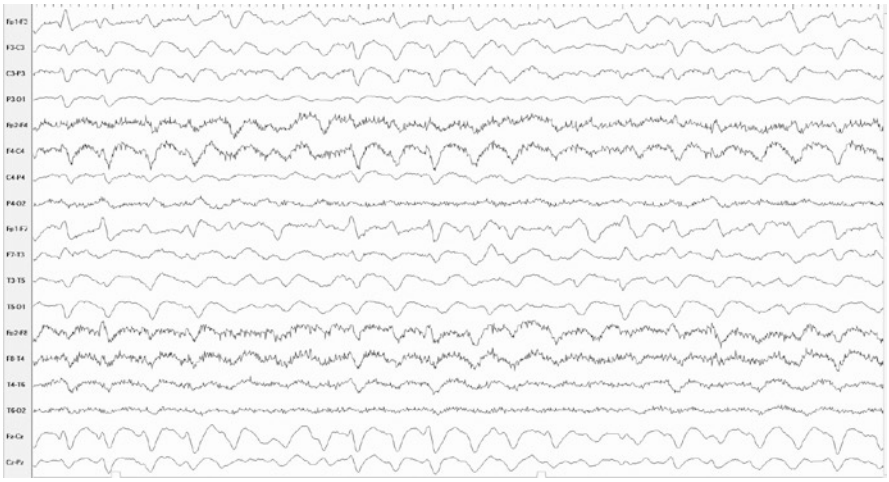

**Fig. 31.1** Male, 49 yrs. Stupor and liver failure. EEG showing generalized triphasic waves, which suggests that the stupor condition is metabolic

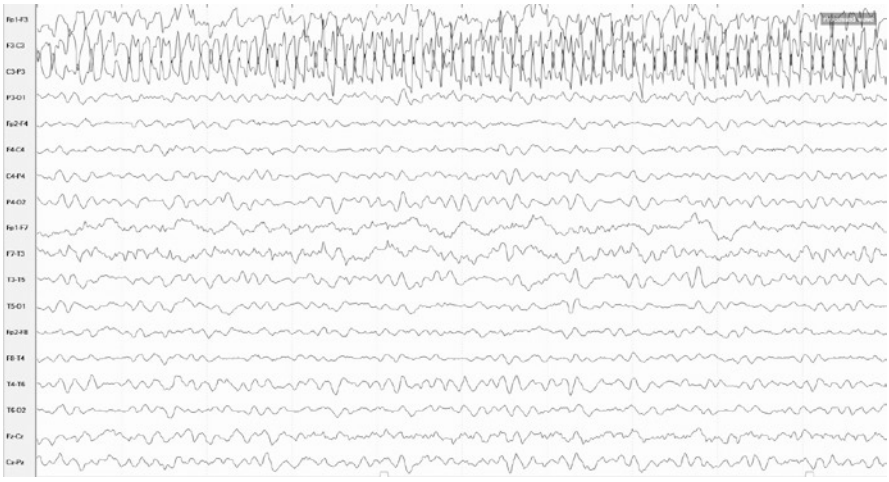

**Fig. 31.2** Female, 52 yrs. Brain trauma with subdural hematoma on the left (frontal region). Patient is comatose and occasionally showing subtle jerks on her face, at right side. EEG shows continuous discharges in the central left region, suggesting SE with a pattern of continuous discharges

## 31.8 Treatment

All treatment protocols include a staged approach to treatment, with different drugs used in early (stage I), established (stage II), refractory (stage III), and super refractory SE (stage IV) (Fig. 31.1), and also emphasize the prompt recognition and treatment of persisting seizure activity at each stage, aiming to reduce morbidity,

mortality, and long-term consequences of the status epilepticus (beyond t2). The most recent reviews focus on the pharmacotherapy of the status, but the general measures for neurological emergencies, as well as a thorough search for the causes, are equally important [21, 22].

Status epilepticus is a medical emergency with the potential for significant morbidity and mortality. It requires immediate treatment and an individualized management plan, based on the specific patient needs, combined with antiseizure drugs [21, 22].

## 31.9 Initial Care

The initial treatment strategy for SE includes simultaneous assessment and management of ABCs (airway, breathing, and circulation), identification and correction of life-threatening causes, and initiation of seizure abortive drug treatment. In near half of the cases, there is an acute etiology that is potentially treatable [21, 22].

A supportive treatment should be provided, and the patients should remain in bed with bars or lateral protection to avoid falls or head trauma. During the clonic phase, a Guedel's cannula should be inserted between the teeth, preventing bites and lacerations of the tongue. They must be constantly aspirated to avoid aspiration and pneumonia. The vital signs and the temperature should be frequently monitored. The airways must be kept clear to ensure proper ventilation. Whenever necessary, orotracheal intubation and oxygenation should be performed to prevent hypoxia.

## 31.10 Antiseizure Drugs

A staged approach using operational definitions for diagnosis has been advocated. Consider initiating most appropriate therapy for each stage [21, 22] (Fig. 31.3).

### *31.10.1 Stage I or Pre-SE (Epileptic Seizures Lasting >10 min and < 30 min)*

*Intravenous diazepam (0.2 to 0.3 mg/kg).* It can be repeated if the seizures persist (up to two doses, with a 5 to 10 minutes interval between doses). Maximum infusion rate: 1 mg/kg/min (risk of respiratory depression).

Diazepam can be replaced by *intravenous Midazolam (0.2 to 0.3 mg/kg).* Maximum infusion rate: 4 mg/min.

If seizures are continuous for 20 minutes, first-line antiseizure drugs can be started.

*Alternatives for a difficult venous access:*

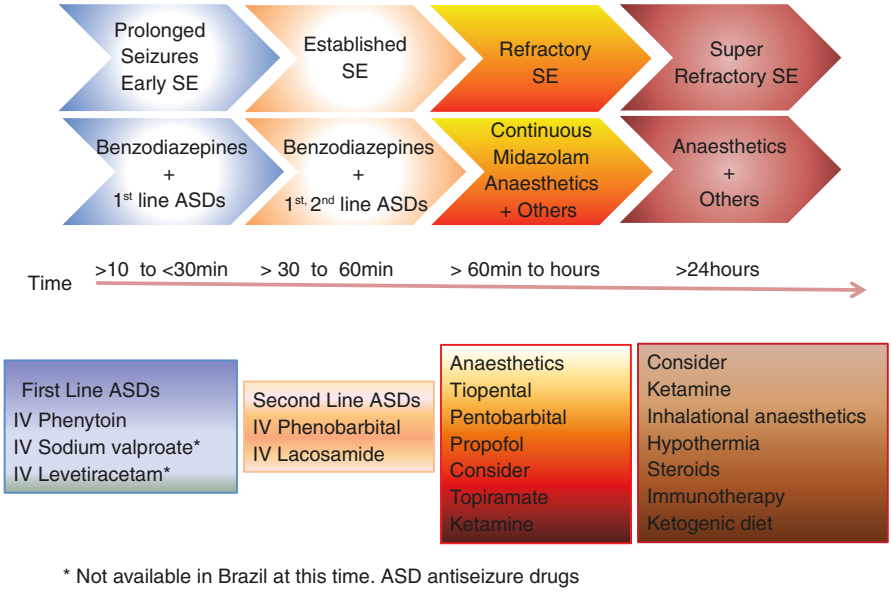


**Fig. 31.3** Status epilepticus. Guidelines for treatment

*Intranasal, buccal, or intramuscular midazolam (0.2 to 0.5 mg/kg).* You can use the intravenous solution.
*Rectal diazepam (0.5 to 0.75 mg/kg).*

### *31.10.2 Stage II or Established SE (Epileptic Seizures Lasting >30 min and < 60 min)*

If a benzodiazepine has not been administered yet, it should be done at least once, while preparing phenytoin.

*Intravenous phenytoin (bolus of 18 to 20 mg/kg)* (from 10 to 20 mg/Kg). Infusion rate: 1 mg/kg/min; maximum 50 mg/min). An additional 10 mg/kg can be given if crises persist, 20 minutes after the initial dose. Dilute it with saline solution or distilled water, and use equipment with filter, because it precipitates.

*Alternatives to phenytoin:*

*Phenobarbital (10 to 20 mg/Kg).* Especially in young children. For neonatal SE or febrile SE, phenobarbital should be the first option.
*Lacosamide (7 to 10 mg/kg).* Consider using *intravenous pyridoxine (100 mg)* in infants up to 18 months of age.
Intravenous levetiracetam and valproic acid are good options but they are not available in Brazil.

### 31.10.3 Stage III or Refractory SE (Epileptic Seizures Lasting Longer than 60 Minutes)

Start coma-inducing drugs. The patient should be at the intensive care unit (ICU), intubated and mechanically ventilated, on complete hemodynamic support, and under continuous EEG (cEEG).

**First option**
*Intravenous Midazolam (bolus of* 0,2 mg/kg). It can be repeated every 5 to 10 minutes, up to 2 mg/kg (total), and the infusion is started at 0.05 to 0.4 mg/kg/h.

**Second option**
*Intravenous pentobarbital* 5 mg/kg. The loading dose can be repeated to burst the suppression effect (suppression interval of 20–30 s); start infusion at 0.5 mg/kg/h and titrate up to 3 to 5 mg/kg/h.

For both drugs, if there is a need to increase the dose, it is preferable to give an additional bolus rather than increasing infusion rate.

**Third option**
Intravenous propofol (bolus of3–5 mg/kg). The loading dose is followed by infusion at 5–10 mg/kg/h.

Coma-inducing drugs should be titrated until cessation of seizures (both clinical and electrographic) or a certain degree of suppression of cerebral activity, as assessed on cEEG. The drug-induced coma should be continued for at least 24–48 hours after the seizures have ceased. Gradually tapper coma-inducing drugs, and keep the patient on one or two antiseizure drugs, intravenously, at therapeutic levels.

### 31.10.4 Stage IV or Super Refractory SE—Epileptic Seizures Lasting More Than 24 Hours Despite the Use of Anesthetic Drugs

It is necessary to continue investigating the underlying etiology, seeking for unusual causes of SE, and, if possible, target the treatment to a specific etiology.

Maintain two (no more than three) antiseizure drugs at therapeutic levels, avoiding frequent changes [23].

*Options:* phenytoin, phenobarbital, levetiracetam, sodium valproate, topiramate, lacosamide.

*Phenobarbital* (you can choose to maintain high dose phenobarbital-induced coma).

*Ketamine.* Give a loading dose (0.5 to0,45 mg/kg), followed by continuous infusion up to 5 mg/kg/h.

Alternative therapies can be used in refractory and super refractory SE, when SE have not stopped despite adequate treatment at therapeutic levels with lidocaine (bolus of 1 to 2 mg/kg as loading dose, followed by a maintenance dose of 1.5 to 3.5 mg/kg/h in adults, or 6 mg/kg/h in children), halothane, and isoflurane anesthetics (requires the presence of an anesthesiologist, and an inhalation is often impracticable due to the duration of SE (hours or days). In children, consider using pyridoxine, pyridoxal-5-phosphate, folinic acid, and biotin.

In specific and very refractory cases, a neurosurgical resection of epileptogenic focus, ketogenic diet, vagus nerve stimulator, immunomodulation (immunoglobulin, methylprednisolone, plasmapheresis), hypothermia, repetitive transcranial magnetic stimulation, and electroconvulsive therapy can be used.

## 31.11 Complications

The main systemic complications are apnea, hypotension, hypoxia, hyperkalemia, pulmonary hypertension, and rhabdomyolysis [24].

Rhabdomyolysis causes intense release of proteins such as myoglobin, and may lead to acute tubular necrosis. Leukocytosis is a common finding, even in the absence of infection. Also, a mild CSF pleocytosis may occur. Autonomic symptoms such as vomiting, loss of fluids and electrolytes, fecal incontinence, urinary incontinence, increased salivation, sweating, and increased tracheobronchial secretion may be part of the clinical manifestations. An aspiration pneumonia is a common complication.

The metabolic acidosis can be severe and should be immediately corrected with sodium bicarbonate, but it must always be kept in mind that administration of sodium bicarbonate represents an additional load of sodium that may eventually worsen cerebral and pulmonary edemas. Hypotension can further aggravate the clinical situation and should be corrected with vasopressor drugs, if necessary. Antiarrhythmic drugs may be needed, and it is recommended to monitor the ECG for 24 hours after SE has been controlled. The hyperthermia eventually associated with convulsive SE can also be an aggravating factor (it may contribute to increase the brain injury) and should be treated with antipyretics and hypothermia, whenever necessary. The hypoglycemia that may appear at a later stage of SE should be approached very carefully, and should only be routinely corrected when very intense. There is evidence that the hyperglycemia at late-stage SE may lead to a higher degree of brain injury, and that a mild hypoglycemia would even function as a neuroprotective mechanism. Airway clearance should be provided and an adequate ventilatory support ensured. In case of aspiration pneumonia, broad-spectrum antibiotics should be prescribed. Several factors may compromise renal function including myoglobinuria, hypoxia, and hypotension. In the early stages of renal failure, dopamine and mannitol may be useful. Electrolytes and renal function should be continuously monitored. Cerebral edema may occur secondarily to structural damage or simply due to the presence

of prolonged seizures. There are no clinical evidences of the efficacy of steroids or mannitol in SE, but the administration of mannitol or methylprednisolone for 24 hours could be considered in those cases with imaging studies showing significant brain edema.

## 31.12 Pearls/Tip

Status epilepticus has heterogeneous clinical presentations, and the nonconvulsive SE especially represents a diagnosis challenge. EEG is essential for diagnosis in such cases. Patients with intellectual deficiency, psychiatric diseases, and especially those with critical illness in the ICU are potential candidates for diagnostic delays due to their underlying conditions. All emergency rooms and ICU should a have a written protocol for rapid diagnosis of SE, correction of the underlying etiology, treatment, and attention to potential complications.

Despite the efforts of specialists and epileptology researchers to precisely define and classify SE, additional work is still needed to delineate an optimal management, as well as to improve outcomes.

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