## Status epilepticus

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#### **Abstract**

Status epilepticus is defined as epileptic activity that continues for longer than 5-30 minutes, either as a single seizure or as recurrent seizures without inter-ictal return of consciousness. The seizure activity is usually classified as partial or generalized. Most episodes of status develop without a prior history of epilepsy. This fact is of major clinical relevance as the emergency management of all patients with status epilepticus must include a search for underlying causes. Although status epilepticus is an uncommon admission diagnosis among patients admitted to a general intensive care unit, a sound knowledge of the causes and treatment of this medical emergency is essential for prompt and effective management. Convulsive seizure activity causes marked systemic disturbances and neuronal injury proportional to the duration of seizure activity. Non-convulsive status epilepticus should be suspected in patients with impaired consciousness without obvious cause and can be definitely diagnosed only with electroencephalography monitoring. An intravenous bolus dose of a benzodiazepine (diazepam or lorazepam) should be used initially to terminate seizures. After this, an appropriate antiepileptic agent (phenytoin) should be started and any systemic disturbances treated. Patients with status epilepticus refractory to the above treatment measures should have general anaesthesia induced and maintained with infusions of an agent with  $\gamma$ -aminobutyric acid (GABA)-ergic activity (thiopentone, propofol or midazolam), aiming for electro-encephalographic burst suppression for 24 hours in the first instance, but longer if necessary.

**Keywords** aetiology; intensive care; management; non-convulsive seizures; status epilepticus

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Among patients admitted to an intensive care unit (ICU), status epilepticus (SE) is a rare admission diagnosis (<1%). However, 12% of patients admitted to an ICU with a primary non-neurological diagnosis will experience neurological complications, of which the second most common are seizures or SE.

SE is variably defined as longer than 5–30 minutes of persistent, generalized, convulsive seizure activity or two or more discrete seizures between which there is incomplete recovery of consciousness, or as persistent seizure activity after sequential administration of appropriate doses of appropriate first- and second-line antiepileptic drugs.

Generalized tonic–clonic status is a medical emergency. The probability of neuronal damage is proportional to the seizure duration and the overall mortality rate is ~20%. The occurrence of non-convulsive status epilepticus in critically ill patients is being increasingly recognized with reported incidences between 8% and 34%. Young children and the elderly are at greater risk of developing SE, especially if they have a previous history of SE.

#### **Classification and diagnosis**

SE can be classified as partial or generalized, depending on whether the seizure discharges arise from a specific focus or not. Generalized seizures can take the form of tonic-clonic, isolated tonic or clonic, or myoclonic seizures. Convulsive tonicclonic status should not pose diagnostic difficulty. Up to 15% of patients with SE remain in non-convulsive status epilepticus (NCSE) after apparently successful termination of convulsive activity. NCSE should be suspected in patients with impaired consciousness without obvious cause. Some patients will have subtle physical signs such as finger movements, lip smacking or eyelid twitching. Definitive diagnosis of NCSE requires electroencephalography (EEG); ideally the duration of monitoring should be > 24 hours, as monitoring for shorter periods will fail to detect NCSE in 20% of patients. The EEG findings in NCSE are variable, ranging from electrical activity identical to convulsive SE to generalized triphasic discharges or periodic lateralized epileptiform discharges (PLED) alternating with equivocal patterns.

### **Pathophysiology**

SE is caused by a cerebral disturbance leading to a failure of mechanisms which would normally abort a single seizure. This generally involves either ineffective inhibition (γ-aminobutyric acid (GABA)-ergic transmission) or excessive excitation (glutamatergic transmission). The early 'compensated' stage of SE is characterized by increased cerebral blood flow and cerebral metabolic rate of oxygen consumption secondary to increased demand; however, the later 'uncompensated' stage reflects the loss of cerebral autoregulation, onset of cerebral oedema and subsequent cell death. While most neuronal damage can be attributed to systemic stresses secondary to seizure activity (e.g. hyperpyrexia, hypoxia, hypoglycaemia), it is recognized that seizure activity itself is injurious to neurones. It is postulated that excessive glutamate release leads to intracellular calcium accumulation with subsequent neuronal dysfunction (excitotoxicity) and death. Certain neuronal populations, such as those of the hippocampus, are particularly vulnerable to seizure-induced neuronal damage and this is directly proportional to the duration and intensity of electrographic seizure activity.

Although NCSE is known to be associated with poor response to treatment and a poor outcome, it is uncertain whether the seizure activity *per se* is deleterious or simply a sign of adverse pathology. Outcome studies of NCSE are often limited by lack of long-term follow-up, lack of comprehensive clinical evaluation and difficulty controlling for the confounding effect of co-morbidities.

#### Investigations and management

The management of status epilepticus should be directed towards the following goals:

 immediate and continued stabilization of vital signs/cardiorespiratory function when seizure duration exceeds 5 minutes

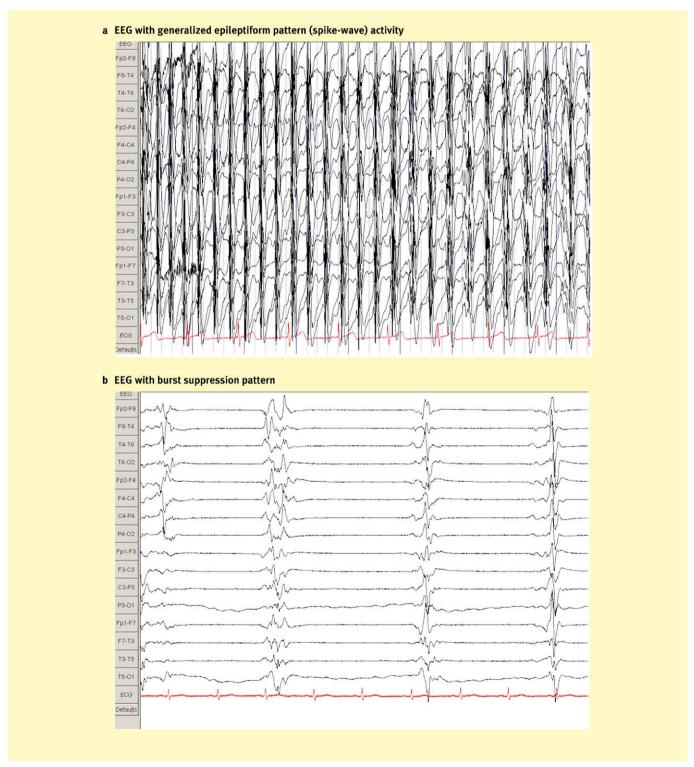


Figure 1

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