

## STATUS EPILEPTICUS IN CHILDREN ANAESTHESIA TUTORIAL OF THE WEEK 248

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### QUESTIONS

Before continuing, try to answer the following questions. The answers can be found at the end of the article, together with an explanation.

1. The following are recognised causes of status epilepticus (SE):
  - a. Hepatic encephalopathy
  - b. Lead poisoning
  - c. Hyperkalaemia
  - d. Anticonvulsant overdose
  
2. The following are true or false
  - a. The majority of patients presenting with SE have a prior history of a neurological disorder
  - b. Following an episode of SE, 35% of children will develop epilepsy
  - c. The mortality rate from SE in industrialised countries is 20%
  - d. Non-convulsive status epilepticus almost always occurs in children with known epilepsy
  
3. Complications associated with status epilepticus are:
  - a. Acute tubular necrosis
  - b. Disseminated intravascular coagulation
  - c. Acute pancreatitis
  - d. Tachyarrhythmias

### INTRODUCTION

Status epilepticus (SE) is the most common neurological emergency in children, with an incidence of approximately 20 events per 100 000 children per year in the developed world, and if inappropriately or under-treated can lead to significant morbidity and death. Treatment is most effective in the early stages of the convulsion, so prompt and appropriate management is essential.

### DEFINITION AND CLASSIFICATION

Status epilepticus is classified as either convulsive or non-convulsive. Generalised convulsive status epilepticus (CSE) is defined as a generalised seizure lasting 30 minutes or longer (continuous CSE), or repeated tonic-clonic convulsions occurring over a 30 minute period without recovery to normal neurological state between seizures (intermittent CSE). However, more recent definitions suggest that continuous seizure activity lasting more than five minutes very rarely stops spontaneously.

Non-convulsive status (NCSE) is diagnosed with electroencephalography (EEG) and should be considered in the child with unexplained alterations in conscious level.

Though CSE is associated with a higher mortality and morbidity than NCSE, both require prompt and effective treatment.

## AETIOLOGY

The aetiology of CSE varies with age and is the main factor that determines outcome. The incidence is highest in children under one year of age and decreases with increasing age. There is a lower seizure threshold in the immature brain and an increased incidence of acute causes of seizure in infants, which may explain the higher frequency of CSE in the very young previously neurologically normal child. The single most common cause of CSE is an acute febrile illness.

In established epilepsy, the most common causes of CSE are anticonvulsant withdrawal, intercurrent illness or metabolic disturbance. Less than 15% of children presenting with status epilepticus will have a history of epilepsy.

### *Common Causes*

Fever. Common between 6 months and 5 years of age. 8% of febrile convulsions are prolonged

Cerebral hypoxia

CNS infections. Note - CSE is not a common presenting feature of acute infection

Metabolic abnormalities including hypoglycaemia, hyponatraemia, hypocalcaemia and hepatic encephalopathy

Epilepsy and anticonvulsant withdrawal

### *Other causes*

Trauma

Poisoning

Systemic hypertension

Pseudoepilepsy

On presentation, a brief, directed history should be taken, concentrating on the time and nature of the seizure activity, inter-current illnesses or fever, any history of head injury and possible exposure to toxins. If there is a background of a neurological disorder, details should be sought.

## MANAGEMENT

It is important to assess and stabilise the airway, breathing and circulation (ABC) concurrently with treatment of the seizure. The child may require intubation and ventilation if they develop airway or respiratory compromise at any stage of the treatment algorithm. All patients should be given high flow oxygen via facemask and intravenous access should be secured as soon as possible. Intraosseous (IO) access should be used if intravenous access cannot be established quickly in the young child. Most anticonvulsants can be administered by the IO route. It is essential to check blood glucose to exclude hypoglycaemia as a cause. IV fluids should be administered according to clinical signs. Several fluid boluses (20ml/kg of 0.9% sodium chloride) may be required in the septic patient.

Irrespective of the aetiology of CSE, treatment priorities in any child presenting with a seizure lasting more than 5 minutes are listed below.

### *Priorities of treatment in CSE:*

1. Terminate seizure activity as soon as possible
2. Maintain cardiorespiratory function
3. Prevent injury
4. Avoid secondary medical complications

### **Emergency drug treatment**

Hypoglycaemia is treated with 2ml/kg 10% dextrose

#### **1<sup>st</sup> stage (0-10 minutes)**

Lorazepam 0.1mg/kg IV given over 30-60 seconds

Diazepam 0.5mg/kg PR or midazolam 0.5mg/kg buccal if no IV access

#### **2<sup>nd</sup> stage (10-20 minutes)**

Lorazepam 0.1mg/kg IV repeated

Paraldehyde 0.4mg/kg PR if still no IV access

#### **3<sup>rd</sup> stage (>15 minutes)**

Call for senior help

Phenytoin 18mg/kg IV or IO over 20 minutes or

Fosphenytoin 15-20mg/kg IV or IO over 20 minutes or

Phenobarbitone 20mg/kg IV over 10 minutes if already on phenytoin

Give paraldehyde 0.4mg/kg PR if not already given

If the seizure is persisting despite these measures, a rapid sequence induction with either thiopentone or propofol should be performed and the child transferred to an intensive care unit.

If sepsis is suspected, high dose broad spectrum antibiotics should be given, ideally after blood cultures have been taken. For cases of suspected encephalitis, an antiviral should also be commenced. Where aetiology is uncertain, cefotaxime, acyclovir, and erythromycin are recommended.

### **Investigations**

#### **Blood tests**

Arterial blood gases, blood glucose, renal and liver function, calcium and magnesium, full blood count (including platelets), clotting screen and drug levels of normal anticonvulsant medications. Other tests as appropriate may include blood cultures and toxicology.

#### **Radiology**

Chest X-ray to assess for aspiration or infection

Brain imaging: CT or MRI scan as indicated by focal neurology or signs of raised ICP.

#### **Other**

EEG

Lumbar puncture; if indicated and provided there is no evidence of raised intracranial pressure

### **Refractory convulsive status epilepticus**

There is no universally agreed definition of refractory status epilepticus. It has been variously defined as seizure activity that is unresponsive to an adequate dose of a benzodiazepine and/or to 2<sup>nd</sup> line treatment (phenytoin or phenobarbitone) or which has lasted 60-90 minutes from initiation of treatment. It occurs in 10-40% of patients who present with CSE. Obviously, these children are at much greater risk of developing complications (see below). Refractory CSE requires induction of general anaesthesia in the ITU with EEG monitored to burst suppression.

Anaesthesia is provided by midazolam (loading dose 0.15-0.5mg/kg followed by infusion up to maximum of 30mcg/kg/min) or thiopental (loading dose 4-8mg/kg followed by infusion of up to maximum of 6mg/kg/min).

Whilst anaesthetised, long acting anticonvulsant therapy (e.g. phenytoin, levetiracetam, phenobarbital, sodium valproate etc) is administered whilst monitoring blood levels.

### **Non-convulsive status epilepticus**

This is much less common than convulsive status epilepticus, and may involve complex partial or absence seizure patterns. Treatment should be instigated as for CSE, and care taken to maintain the

child's usual anti-epileptic medication. The diagnosis of NCSE can be difficult, and is dependant on EEG. Expert advice should be sought. Long-term sequelae from NCSE are less severe than those seen in CSE.

## **COMPLICATIONS**

Prolonged seizures are associated with cerebral hypoxia, hypoglycaemia, hypercarbia and a progressive lactic and respiratory acidosis. Neuronal destruction occurs when cerebral metabolic requirements cannot be met by the available oxygen, glucose and metabolic substrates. Massive sympathetic discharge occurs leading to many of the complications listed below.

### **Cerebral**

- Hypoxic/metabolic cerebral damage
- Excitotoxic cerebral damage (seizure related)
- Cerebral oedema and raised intracranial pressure
- Cerebral venous thrombosis
- Cerebral haemorrhage and infarction

### **Cardiorespiratory and autonomic**

- Hypo- or hypertension
- Cardiac failure
- Tachy or bradyarrhythmias
- Respiratory failure
- Pulmonary oedema, hypertension, aspiration, pneumonia
- Hyperpyrexia
- Hypersecretion, tracheobronchial obstruction

### **Metabolic and systemic**

- Dehydration
- Electrolyte disturbances (especially hypoglycaemia, hyponatraemia and hypokalaemia)
- Acute renal failure (acute tubular necrosis)
- Acute hepatic failure
- Acute pancreatitis
- Disseminated intravascular coagulation
- Multiorgan failure
- Rhabdomyolysis
- Fractures or joint dislocations

## **OUTCOME**

The outcome from status epilepticus has improved considerably in the last 30 years. The most important factor that determines outcome is aetiology, with age and duration of SE also contributing. In one systematic review the mortality rate for CSE was found to be 2.7-5.2%, though the children requiring admission to ICU had a higher mortality (5-8%). Mortality rates are higher in children with CNS causes of SE such as encephalitis or head trauma, compared with febrile SE from a non-CNS infection.

Idiopathic or febrile SE in normal children is usually an isolated event, with a recurrence rate of 3-4%. There is a higher recurrence rate in the child with a CNS cause or an underlying neurological disorder (11-44%). Long-term complications include secondary epilepsy, behavioural problems and neurological deficits.

It is not clear whether subsequent epilepsy occurs as a result of the SE, or as a result of the brain insult that caused the SE. Overall the risk of developing epilepsy after an episode of SE is approximately 35%, but this risk is much higher in those patients with a previous neurological disorder, or in those patients whose cause of SE was a primary CNS infection or trauma.

## SUMMARY

- Status epilepticus is a medical emergency, and early aggressive treatment is more likely to be successful in terminating the episode
- Morbidity and mortality increase with increasing seizure duration in SE
- Guidelines for the treatment of SE should be available for all personnel involved with the care of children, from pre-hospital care to intensive care

## ANSWERS TO QUESTIONS

- 1      a True  
       b True  
       c False  
       d False. SE is associated with anticonvulsant withdrawal
- 2      a False; the majority of patients presenting with SE have no prior history of epilepsy or any neurological disorder  
       b True  
       c False; the mortality rate in industrialised countries is approximately 4%, though this is higher in resource poor countries  
       d True
- 3      all True

## WEBLINKS

<http://www.bpna.org.uk/audit/Status%20Epilepticus.PDF>  
[http://criticall.org/webconcepteurcontent63/000023720000/upload/pdf/Pediatric\\_Status\\_Epilepticus\\_CPG.pdf](http://criticall.org/webconcepteurcontent63/000023720000/upload/pdf/Pediatric_Status_Epilepticus_CPG.pdf)

## REFERENCES and FURTHER READING

Epilepsy: the diagnosis and management of epilepsy in children and adults; NICE guideline; March 2004

Appleton R, Macleod S, Martland T, Drug management for acute tonic-clonic convulsions including convulsive status epilepticus in children; *Cochrane Database of Systematic Reviews 2008*, Issue 3.

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