STATUS EPILEPTICUS IN CHILDREN
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Department of pediatric
Plan

- Definition of status epilepticus (SE)
- Classification
- Epidemiology
- Pathophysiology
- Outcome/Prognosis
- Symptoms
- Management
Definition of status epilepticus

A condition in which epileptic activity persists for 30 minutes or more, causing a wide spectrum of clinical symptoms, and with a highly variable pathophysiological, anatomical and etiological basis.

(Shorvon, 1994)
Definition of SE

- 5 minutes or more continuous seizures or "two discrete seizures between which there is incomplete recovery of consciousness" (to avoid refractory SE),

- Aggressive early treatment is justified by recent work demonstrating a 10-fold decrease in mortality for seizures of 10-29 minutes

(Lowenstein i wsp., 1999; DeLorenzo, 1999)
Why SE should be treated quickly?

- There is increasing recognition that SE is associated with high morbidity and mortality.
- As a seizure persists, standard medications are less effective in terminating the seizure.
- This has led to shortening of the minimum seizure duration to diagnose SE from 30 minutes to 5 minutes.
Refractory SE

- SE lasting 5 to 60 min or without any improvement after treatment

- SE with no improvement after two AEDs (BZP and PHT/PB)

(Sahin i wsp., 2001; Mayer i wsp., 2002)
Classifications of SE
(Engel, 2001, 2006)

- Types of seizures
  - ILAE 2001
  - ILAE 2006

- Etiology of seizures
<table>
<thead>
<tr>
<th>Generalized SE</th>
<th>Focal SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Generalized tonic-clonic SE,</td>
<td>Epilepsia partialis continua of Kozhevnikov</td>
</tr>
<tr>
<td>Clonic SE</td>
<td>Aura continua</td>
</tr>
<tr>
<td>Absence SE</td>
<td>Limbic SE (psychomotor status)</td>
</tr>
<tr>
<td>Myoclonic SE</td>
<td>Hemiconvulsive status with hemiparesis</td>
</tr>
</tbody>
</table>
SE – traditional classification

Convulsive SE  Non-convulsive (NCSE)
Classification of SE by ILAE, 2006

(Engel, 2006)

I. **Epilepsia partialis continua (EPC):**
   A. as occurs with Rassmusen syndrome
   B. as occurs with focal lesions,
   C as a component of inborn errors of metabolism

II. **Suplementary motor area (SMA) SE**

III. **Aura continua**

IV. **Dyscognitive focal (psychomotor, complex partial):**
   A. Mesial temporal SE
   B. Neocortical SE

V. **Tonic-clonic SE,**

VI. **Absence SE:**
   A. typical and atypical ASE
   B. myoclonic ASE

VII. **Myoclonic SE**

VIII. **Tonic SE**

IX. **Subtle SE**
Classification of SE based on etiology
Classification of SE based on etiology

- Acute symptomatic,
- Remote symptomatic,
- Remote symptomatic with an acute precipitant,
- Progressive encephalopathy,
- Febrile,
- Cryptogenic,
## Classification of SE according to etiologies

<table>
<thead>
<tr>
<th>Type</th>
<th>Definition</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute symptomatic</td>
<td>SE occurring during an acute illness (an acute CNS insult)</td>
<td>Meningitis, encephalitis, electrolyte disturbances, sepsis, hypoxia, trauma, intoxication</td>
</tr>
<tr>
<td></td>
<td>(an acute encephalopathy)</td>
<td></td>
</tr>
<tr>
<td>Remote symptomatic</td>
<td>SE occurring without an acute provokedation in a patient with a prior history of a CNS insult (a chronic encephalopathy)</td>
<td>CNS malformation, previous traumatic brain injury or insult, chromosomal disorder</td>
</tr>
</tbody>
</table>
### Classification of SE according to etiologies (cont.)

<table>
<thead>
<tr>
<th>Type</th>
<th>Definition</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Remote symptomatic with an acute precipitant</td>
<td>SE occurring with a chronic encephalopathy, but with an acute provocation</td>
<td>CNS malformation or previous CNS insult with concurrent infection, hypoglycemia, hypocalcemia, or intoxication</td>
</tr>
<tr>
<td>Progressive encephalopathy</td>
<td>SE occurring with an underlying progressive CNS disorder</td>
<td>Mitochondrial disorders, CNS lipid storage diseases, amino- or organic acidopathies</td>
</tr>
</tbody>
</table>
### Classification of SE according to etiologies (cont.)

<table>
<thead>
<tr>
<th>Type</th>
<th>Definition</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Febrile</td>
<td>SE occurring when the only provocation is a febrile illness, after excluding a direct CNS infection, such as meningitis or encephalitis</td>
<td>Upper respiratory infection, sinusitis, sepsis</td>
</tr>
<tr>
<td>Cryptogenic</td>
<td>SE occurring in the absence of an acute precipitating CNS insult, systematic metabolic disturbance, or both</td>
<td>No definable cause</td>
</tr>
</tbody>
</table>
# Etiology of SE

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Children (&lt;16yrs) %</th>
<th>Adults &gt;16yrs %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever/Infection</td>
<td>35.7</td>
<td>4.6</td>
</tr>
<tr>
<td>CNS infection</td>
<td>4.8</td>
<td>1.8</td>
</tr>
<tr>
<td>Medication changes</td>
<td>19.8</td>
<td>18.9</td>
</tr>
<tr>
<td>cerebrovascular</td>
<td>3.3</td>
<td>25.2</td>
</tr>
<tr>
<td>Congenital</td>
<td>7.0</td>
<td>0.8</td>
</tr>
<tr>
<td>Unknown</td>
<td>9.3</td>
<td>8.1</td>
</tr>
</tbody>
</table>
Incidence of SE according to etiologies

(Riviello et al., 2006)
Epidemiology of SE

- Age → fundamental determinant of the epidemiology of SE.

The incidence of CSE in childhood is highest among children less than 1 y. – 51/100000/y (acute symptomatic etiology; increased propensity for seizure of immature brain; lower seizure threshold)

- Gender: M : F 1:1, 1.5:1, 2:1,

(Raspall-Chaure i wsp., 2007; Ben-Ari&Holmes, 2006)
Epidemiology of SE
(Raspall-Chaure et al., 2007)

The incidence of CSE is approximately

20/100 000/ year

Depends on: socioeconomic (developed vs. developing countries) and ethnic characteristics of population.

1 million people / year
The incidence of SE according to age

The incidence of CSE in childhood / 100,000/y

(Chin, 2006)
Epidemiology of SE

- 10-20% of children with epilepsy will have at least one episode of CSE during the course of disease (with the most occurring in the first few years of epilepsy) 
  
  (Sillanpaa & Shinar, 2002; Berg et al., 2002)

- In 12%-40% of patients with newly diagnosed epilepsy SE is the first symptom of disease
  
  (Hesdorffer i wsp., 1998)

- 62-88% of children with first episodes of CSE in population-based studies do not have prior epilepsy
  
  (DeLorenzo et al., 1996; Chin et al., 2006)
Mechanism of SE in humans
(Murdoch, 2007; Shorvon et al., 2008)

- No randomized controlled trials, systematic reviews, or meta-analyses were found in any databases searched regarding the pathophysiologic mechanism of SE in humans,

- The biochemical basis of pathophysiology remain unclear
Physiologic changes in generalized SE:

- **Early (0-30 min)**
  - Arterial hypertension
  - Cerebral venous (CV) pressure increased.
  - Brain O2 utilization increased
  - CV pCO2 high

- **Late (after 30 min)**
  - Arterial hypertension
  - CVP raised or normal
  - Brain O2 utilization decreased
  - CV pCO2 normal
Physiologic changes in generalized SE:

- **Early (0-30 min)**
  - Cerebral blood flow (CBF) increased
  - Hyperglycemia
  - Lactic acidosis

- **Late (after 30 min)**
  - CBF decreased
  - Hypoglycemia
  - Hyperpyrexia (secondary)
Systematic physiologic changes in SE
*(Shorvon et al., 2008)*

- Elevation of systematic blood pressure $\uparrow$ RR
- The risk of cerebral hypoperfusion due to inhibition of cerebral autoregulation
- Cardiac effects: $\uparrow$ HR; ECG abnormalities (ischaemia, conduction defects, prolongations of QTc interval, tachycardia)
- Acidosis $\downarrow$ pH
- Hypoxia $\downarrow$ pO2
- Hyperthermia
- Leukocytosis
- Rhabdomyolysis and renal failure
- Others: disseminated intravascular coagulation (DIC), multiorgan failure, electrolyte disturbances, AE of AEDs
Oxygen and glucose utilization in SE is greater in epileptic seizures than in any other cerebral activity. In the stage of decompensation (II) the supply of oxygen and glucose begin to fail. Cerebral cellular metabolism decreases, in late stage of SE hypometabolism and ischaemia are common. The time taken for stage I to stage II depends on factors: etiology, duration and severity of seizure activity, therapy.
CNS physiologic changes in SE
Excitotoxic cellular damage

- Activation of NMDA (glutamate receptors)
- Influx of Ca
- Mitochondrial dysfunction
- Reactive oxygen and nitrogen species
- Activation of intracellular proteineases and lipases

Excitotoxicity

The cerebral cortex, hippocampus and cerebellum are the most affected by excitotoxicity damage. Both apoptosis and necrosis occur → neuronal loss, atrophy and gliosis.
CNS physiologic changes in SE

- Intracranial pressure changes and cerebral edema, (CSE)
- Blood-brain barrier changes
- Cerebral vascular changes (hemorrhage, infarction, cortical venous thrombosis)
Symptoms of SE
Convulsive SE

- Generalized CSE has been defined as recurrent generalized convulsions without full and complete recovery of consciousness between seizures or

- As a single prolonged convulsion without the characteristic evolution of single discrete seizure

(Treiman, 2008)
Differential diagnosis

- Somatoform disorders: conversions
- Syncope

- **Psychogenic seizures**
  - video EEG
  - Occasionally, psychogenic seizures can be confused with GTCSE
  - In psychogenic seizures: evolution of behaviour during seizure, stereotyped seizures, sustained convulsive activity without pauses, opened eyes
Focal status epilepticus *(Engel, 2006)*

I. Epilepsia partialis continua (EPC):
   A. as occurs with Rasmussen syndrome
   B. as occurs with focal lesions,
   C. as a component of inborn errors of metabolism

II. Supplementary motor area (SMA) SE

III. Aura continua

IV. Dyscognitive focal (psychomotor, complex partial):
   A. Mesial temporal SE
   B. Neocortical SE
Nonconvulsive status epilepticus (NCSE)

- NCSE – condition with a prolonged state of impaired consciousness or altered sensorium associated with continuous paroxysmal activity or electrographic discharges on the EEG with no convulsive activity
Nonconvulsive status epilepticus (NCSE)

- NCSE – heterogeneous disorder with varied etiology and several subtypes,
- Unrecognized,
- Delayed in diagnosis and treatment may be associated with increasing mortality,
- No universally accepted definition.

(Maganti et al., 2008)
Absence status epilepticus (ASE) (IGE)

Clinically
- varying degree of confusion (mild or severe) and slowing of mental functions
  a/ with clonic components,
  b/ with atonic components,
  d/ tonic components,
  e/ with automatisms
  f/ with autonomic components

- Duration may range from 30 minutes to 3-4 h
Absence status epilepticus (ASE)

- Absence status epilepticus (ASE in IGE) - EEG pattern
- Absence status epilepticus of atypical absences in LGS
Nonconvulsive status epilepticus (NCSE) - EEG

- No specific clinical and radiographic findings in NCSE
- EEG – gold standard for making diagnosis of NCSE
Nonconvulsive status epilepticus (NCSE) – EEG patterns

generalized triphasic waves

generalized periodic epileptiform discharges - GPEDs
Nonconvulsive status epilepticus (NCSE) – treatment

- As quickly as possible: i.v. BZP, i.v. PHT, PB, general anesthetic
Outcome of CSE

- Comparison
  mortality vs. others complications/morbidity

- Prognostic factors:
  - Atiology
  - Age
  - Concomitant illness
  - Duration of SE
  - Treatment
How about prognosis of SE

Depends on the etiology

- 6% Mortality rate in children
- 17% Mortality rate in the first year of life, with a rate of 24% with those less than 6 months versus 9% in those between 6 to 12 months of age
- Mortality directly related to SE itself is 1 to 2%

Outcome of CSE

- **Mortality** in CSE 2-3% to 25-30% (depends on etiology, age, and coexistence of others diseases).

- Incidence of mortality in CSE:
  - 3% among children,
  - 26% in adults,
  - 60-90% in elderly

Outcome of CSE Mortality

Prognostic factor - Etiology:

- **High mortality**: anoxia, sepsis, coexistence of many illnesses

- **Intermediate mortality**: strokes, tumours, infections (CNS & general), trauma.

- **Low mortality**: cryptogenic epilepsy, withdraw of AEDs, toxic AEs, withdraw of alcohol,

(Logroscino i wsp., 2002)
Outcome of CSE Mortality

Prognostic factor - duration of SE

- Mortality increases about 30% in long term SE (especially in encephalitis)

- Duration of SE > 60 min. = mortality 32% vs. Duration of SE <60min. mortality = 2.7%

(Scholtes i wsp., 1994)
Outcome of CSE – hippocampal injury and MTS

- CSE (febrile) → MTS (35-63% of patients with MTS had history of FSE)

(Raspall-Chaure et al., 2006; Dodrill i Wilensky, 1990; Maytal i wsp., 1989)
Outcome of CSE – recurrence of SE

(Raspall-Chaure et al., 2006)

- 3-56% (based on 17 studies)
- Within 4 years – 20%, within 1-2 years 70%
- Recurrence is determined by underlying cause:
  - low for FSE and idiopathic CSE (<4%)
  - acute SE 11%; remote 44%; progressive symptomatic 67%
- The effect of age at the time of the first episode of CSE on the risk of recurrence is controversial
Outcome of CSE

In 15%-50% (depends on age, duration of CSE)

- focal neurological deficits
- cognitive impairment (minimal neuropsychological morbidity),
- behavioural problems

(Dodrill, 1990; Raspall-Chaure et al., 2006)
Outcome of status epilepticus in children treated in the intensive care unit:


- 302 cases (age 2 months to less than 18 years)

- 489 episodes of SE

- Etiology, treatment, and clinical and (EEG) features of SE and their impact on the outcome
Outcome of status epilepticus in children treated in the intensive care unit:


The outcome was classified into

- Unchanged neurologic status
- Neurologic consequences
- Lethal outcome
Outcome of status epilepticus in children treated in the intensive care unit:


- Neurologic status: unchanged in 235 children (77.8%)
- Neurologic consequences: 39 patients (12.9%)
  - younger age,
  - progressive encephalopathy,
  - duration of SE >24 h,
  - and prior epilepsy,
- Case-fatality ratio: 9.3%
  - progressive encephalopathy,
  - preexisting neurologic abnormalities
Outcome of SE in children is favorable in most of the cases. Mortality and morbidity rates are still high. Etiology and prior neurologic abnormalities were the main predictors of mortality. The main predictor of morbidity was underlying etiology.
Management: Why There is a Need to Terminate SE?

- It is unknown whether it will terminate spontaneously after a few minutes, as occurs with most seizures.
- SE is associated with high morbidity and mortality.
- As a seizure persists, standard medications are less effective in terminating the seizure.
Management

SE is a neurologic and medical emergency and requires:

- maintenance of respiration,
- general medical support,
- specific treatment to aimed both stopping electrographic and clinical seizures
Lab Studies

- **Stabilization phase:** While attending to the airway, breathing, and circulation (ABCs) and inserting an IV line, obtain laboratory studies:
  - for anticonvulsant medications levels, electrolytes, BUN/creatinine, calcium, magnesium, and CBC;
  - Serum glucose.
  - urine/serum toxicology, especially in teenagers. A lumbar puncture is commonly indicated in children with GTCSE, especially those with unexplained fever or mental status changes preceding or following the seizure episode.
Lab Studies

**Continued evaluation**: Continue evaluation after seizures are controlled.

- Basic tests recommended are liver function tests (LFTs), toxicology screen, and brain imaging.
- After an SE episode, perform a lumbar puncture for individuals with fever or other evidence of CNS infection. Convulsive status may be associated with CNS infection without typical meningeal signs.
Imaging Studies

- All patients who have histories of neurologic (including mental status) changes.
- Deficits on the neurologic examination that persist after cessation of seizures.
- Prior to lumbar puncture for patients with acute neurologic changes as evidenced by increased intracranial pressure.
Imaging Studies

- Children with complex partial seizures preceding or leading to the episode of GTCSE should have brain magnetic resonance imaging (MRI).

- Brain imaging may be unnecessary for patients who have already had MRI.
Electroencephalography

- Every patient who presents with SE needs an EEG, but do not delay treatment to wait for EEG results. When a seizure persists longer than 30-60 minutes, making immediate arrangements for an EEG is advisable.

- The EEG helps the differentiation of convulsive status from pseudoseizure (nonepileptic or psychogenic seizure).
Electroencephalography

- Patients who cannot be aroused following a seizure should have an EEG performed to rule out subclinical SE.

- EEG can confirm the seizure pattern and help indicate the most appropriate long-term treatment, if that is necessary.
SE – treatment guidelines

- Requires rapid and aggressive treatment to prevent neuronal damage, systematic complications and death.
Therapy for SE: Initial assessment

- Brief physical examination
  - Respiratory
  - Circulation
- Rapid neurological examination
  - HR and pupillary response
  - Type of status epilepticus
Early management to stabilize patient

- As in any medical emergency, attend first to the ABCs. (Place patients in the lateral decubitus position to avoid aspiration of emesis)

- Carefully monitor the patient's vital signs, including blood pressure and patient's temperature; try to establish IV access and to draw samples for laboratory tests.

- Administer IV glucose if serum glucose is low or cannot be measured. In these instances, children should receive 2 mL/kg of 25% glucose, and adults should receive 50 mL of 50% glucose, as well as 100 mg of thiamine.
## Treatment of SE – prolonged epileptic seizure – premonitory stage (out of hospital – nonmedical person)

*Kalviainn, 2007; Walker & Teach, 2006*

<table>
<thead>
<tr>
<th>Time</th>
<th>Drug treatment</th>
<th>General measures</th>
<th>Emergency investigations</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 min.</td>
<td>Diazepam 0.5mg/kg rectally</td>
<td>Airway, Breathing, Circulation, Safety</td>
<td>Glucometer</td>
</tr>
</tbody>
</table>
Treatment of SE – prolonged epileptic seizure – first stage (out of hospital – medical person)

*(Kalviainen, 2007; Walker & Teach, 2006)*

<table>
<thead>
<tr>
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<tr>
<td>5-20min</td>
<td>Lorazepam i.v. 0,1mg/kg (max. 4mg) or</td>
<td>Airway, oxygen, cardiorespiratory function and regulatory monitoring; ECG, blood pressure, SpO2</td>
<td>Glucose, Na, K, Ca, CRP, Levels of AEDs, toxicology screening, kidney and liver function tests</td>
</tr>
<tr>
<td></td>
<td>Diazepam i.v. 0,3mg/kg (max. 10mg)</td>
<td>Intravenous access; i.v.: glucose, thiamine, piridoxine</td>
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<td>Treat acidosis</td>
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Therapy for SE: Antiepileptic Drugs

Benzodiazepines

Diazepam, DZP (Valium)
- Initial dose, 0.2mg/kg
- Faster onset of action because of greater lipid solubility
- Must be followed by another AED in order to prevent further seizure activity.
Treatment of SE – if seizure continues, proceed;
Established SE, - second stage / emergency department

(Kalviainen, 2007; Walker & Teach, 2006)

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<tr>
<th>Time</th>
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</tr>
</thead>
<tbody>
<tr>
<td>20-60 min</td>
<td>Phenytoin i.v. 20mg/kg</td>
<td>■ Monitoring: Cardiorespiratory function, ECG, blood pressure, SpO2; use pressor if needed</td>
<td>■ CT scan for etiology, CSF for CNS infection, EEG for pseudostatus</td>
</tr>
<tr>
<td></td>
<td>Fosphenytoin i.v. 20mg/kg</td>
<td>■ Identify and treat medical complications</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Phenobarbital i.v. 20mg/kg</td>
<td></td>
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</tbody>
</table>
Treatemnt of SE – if seizure continues, proceed;
Refractory SE - third stage / intensive care unit

(Kalviainen, 2007; Walker i Teach, 2006)

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<th>General measures</th>
<th>Emergency investigations</th>
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</thead>
<tbody>
<tr>
<td>&gt;60min.</td>
<td>General anesthesia</td>
<td>Intensive care; ventilatory and hemodynamic treatment</td>
<td>Continuous EEG monitoring; Electrographic seizures, depth of anesthesia (bust-suppression)</td>
</tr>
<tr>
<td></td>
<td>Thiopental 3-5mg/kg bolus; Pentobarbital 10-15mg/kg</td>
<td>Increased intracranial pressure; measure and treat if signs</td>
<td>Monitor</td>
</tr>
<tr>
<td></td>
<td>Midazolam 0,1 – 0,3 mg/kg bolus (T1/2=1,5-3,5)</td>
<td>Anesthesia continued for 12-24 h after last clinical of electroencephalographic seizure; Optimaze maintenance AED treatment</td>
<td>K, Na, glucose, lactate. Levels of AEDs</td>
</tr>
</tbody>
</table>
# Treatment of SE (first line)

<table>
<thead>
<tr>
<th>SE</th>
<th>First line drugs</th>
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<tbody>
<tr>
<td>SE in newborns</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Phenobarbital iv.</td>
</tr>
<tr>
<td></td>
<td>Lorazepam iv.</td>
</tr>
<tr>
<td></td>
<td>Fosphenytoin iv.</td>
</tr>
<tr>
<td>SE in children</td>
<td>Lorazepam iv.</td>
</tr>
<tr>
<td></td>
<td>Diazepam iv.</td>
</tr>
<tr>
<td>SE of GTCS</td>
<td>Diazepam per rec.</td>
</tr>
<tr>
<td></td>
<td>Fosphenytoin iv.</td>
</tr>
<tr>
<td>SE of CPS</td>
<td>Fosphenytoin iv.</td>
</tr>
<tr>
<td>Absences SE</td>
<td>VPA iv.</td>
</tr>
</tbody>
</table>

*(Murdoch, 2007)*
Treatment of SE
(Walker & Teach, 2006, Morton & Pellock, 2006)

■ NCSE – absence SE after use BZP,

■ VPA is effective in the treatment of generalised and focal SE 20-40mg/kg, also 6mg/kg/min
Treatment of SE

- TPM - p.o., no parenteral forms
- LEV

(Towne i wsp., 2003; Bensalem i Fakhoury, 2003; Reuber i wsp., 2002; Kahriman i wsp., 2003)
## Treatment of NCSE

*(Korff & Nordli, 2007)*

<table>
<thead>
<tr>
<th>SE</th>
<th>Treatment</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absence SE</td>
<td>Clobazam per os</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>Lorazepam iv.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>VPA iv.</td>
<td></td>
</tr>
<tr>
<td>SE of simple partial seizures</td>
<td>Clobazam per os</td>
<td>Good</td>
</tr>
<tr>
<td></td>
<td>Lorazepam iv.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>VPA iv.</td>
<td></td>
</tr>
<tr>
<td>SE of simple complex seizures</td>
<td>Treatment depends on etiology</td>
<td>depends on etiology</td>
</tr>
<tr>
<td></td>
<td>Clobazam per os</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lorazepam iv.</td>
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<td></td>
<td>Fosphenytoin iv.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>VPA iv.</td>
<td></td>
</tr>
<tr>
<td>NCSE in children with learning difficulties</td>
<td>Clobazam per os</td>
<td>uncertain</td>
</tr>
<tr>
<td></td>
<td>Steroids per os</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Surgery</td>
<td></td>
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<tr>
<td>NCSE in coma</td>
<td>Lorazepam iv.</td>
<td>Poor</td>
</tr>
<tr>
<td></td>
<td>Fosphenytoin iv.</td>
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<td></td>
<td>Anesthetic drugs</td>
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</table>
**Convulsive SE Algorithm**

**Impending SE G5 minutes Out-of-hospital**
- Consider IM midazolam (0.2 mg/kg to maximum 10 mg) or buccal midazolam (0.5 mg/kg to maximum of 10 mg) or rectal diazepam (0.2Y0.5 mg/kg to maximum of 20 mg)

**Benzodiazepines**
- Lorazepam 0.1 mg/kg IV (max 5 mg) over 1 min
- Diazepam 0.2 mg/kg IV (max 10 mg) over 1 min
- Allow 5 minutes to determine whether seizure terminates.
- Give oxygen. Support airway, breathing, and circulation. Obtain IV access. Check bedside glucose.
- Continue ECG and vital sign monitoring. Continue neurologic assessments.
Convulsive SE Algorithm

Established SE 5Y10 min Repeat benzodiazepine administration.

- Do not wait for seizure termination.
- Administer fosphenytoin 30 mg/kg IV at 2Y3 mg/kg/min (max 150 mg/min)
- [or phenytoin 30 mg/kg IV at 1 mg/kg/min (max 50 mg/min)]
- If less than 2 yr, consider pyridoxine 100 mg IV push.
- Administer bolus of 2 mL/kg IV 50% glucose if hypoglycemic.
- Consider thiamine 100 mg IV first.
Convulsive SE Algorithm

Initial RSE

- If seizure continues 10 minutes after fosphenytoin infusion then patient has RSE regardless of time elapsed.
- Administer third line medication: administer phenobarbital 30 mg/kg IV at 2 mg/kg/min (max rate 60 mg/min).
- Admit to PICU. Continue to support airway, breathing, and circulation. Obtain central venous access and continuous hemodynamic monitoring through arterial line.
Convulsive SE Algorithm

Coma Induction

- If seizure continues 10 minutes after completion of phenobarbital infusion, then initiate coma with: IV midazolam 0.2 mg/kg bolus (max 10 mg) over 2 minutes and then initiate infusion at 0.1 mg/kg/hr.

- If clinical seizures persist after another 5 minutes, then administer another midazolam bolus of 0.2 mg/kg and

- increase infusion to 0.2 mg/kg/h. Repeat as needed.
Convulsive SE Algorithm

Coma Phase

- Continue pharmacologic coma for 24 hours after last seizure with EEG goal of burst-suppression.
- Continue EEG monitoring.
- Continue initial medications (total phenytoin goal level 20Y30 2g/mL, phenobarbital goal level 40Y50 2g/mL).
- Daily phenobarbital and free and total dilantin levels.
Weaning Phase

- Reduce midazolam by 0.05 mg/kg/hr every 3 hours with frequent EEG review.
- If no clinical or electrographic seizures then wean until off.
- Continue EEG for at least 24 hours after end of infusion to evaluate for recurrent electrographic seizures.
Conclusion

- Rapid recognition and treatment of SE offer the best opportunity to improve the outcome in those affected with this neurologic emergency
- Standard protocol done in advance
- Start phenytoin (Non sedative anticonvulsant) with the second dose of benzodizepine
Conclusion

- Phenobarbital is commonly used in place of phenytoin or when benzodiazepines and phenytoin fail to terminate status epilepticus.

- Respiratory depression is a common side effect of phenobarbital, often necessitating intubation.
Conclusion

- Consider Midazolam, propofol or general anesthesia if SE continues for 45 to 60 min
- EEG Monitoring if patient is anesthetized
- Cardiorespiratory and hemodynamic monitoring
- Drugs with rapid clinical effect and rapid clearance, as midazolam and propofol should be used for treating refractory SE
Thank you for attention