MANAGEMENT OF SEIZURES
GUIDELINES FOR MANAGEMENT OF SEIZURES

DIAGNOSIS

• What is a seizure?

A seizure is the manifestation of an abnormal, paroxysmal discharge of a group of cortical neurons. This discharge may produce subjective symptoms or objective signs.

• What are the key features of a seizure?

  • Paroxysmal nature of the event.
  • Associated abnormal movements / subtle phenomena.
  • Altered responsiveness or impairment of consciousness / awareness.

• What is a convulsion?

Predominantly, an uncontrollable & involuntary contraction/relaxation or spasm of a group or groups of muscles.

• What is epilepsy?

The term epilepsy is generally used when a person has a tendency to have unprovoked, repeated seizures (minimum of two).

• Who should make the diagnosis of a seizure/epilepsy?

It must always be done by a paediatrician / paediatric neurologist because misdiagnoses are common.

DIFFERENTIAL DIAGNOSIS

Paroxysmal events, abnormal movements / subtle phenomena and states of altered responsiveness are common in infants and children. Diagnosing epilepsy in a non epileptic is more harmful than missing the diagnosis in an epileptic.

The most useful diagnostic tool is an accurate history taken from an eyewitness and/or patient.
**Mimickers of epilepsy**

1. Syncope
2. Pseudo seizures
3. Breath holding attacks (blue and pallid)
4. Cardiac (if exercise induced syncope always suspect)
   - Prolonged QT interval
   - Stokes Adams
   - Sick sinus syndrome
   - HOCM
   - Aberrant coronary artery origin
5. Benign paroxysmal vertigo
6. Shuddering
7. Self gratification or masturbation
8. Sleep disorders (sleepwalking, night terrors, nightmares, narcolepsy, cataplexy)
9. Sleep opsoconlonous/myoclonus
10. Restless leg syndrome
11. Stereotypes

<table>
<thead>
<tr>
<th>Features</th>
<th>Epileptic seizures</th>
<th>Vasovagal syncope</th>
</tr>
</thead>
<tbody>
<tr>
<td>Precipitating factors</td>
<td>Sleep deprivation, photic flicker</td>
<td>Prolonged standing, hot environment, crowded places, lack of food, unpleasant circumstances, pain</td>
</tr>
<tr>
<td>Posture</td>
<td>Any posture</td>
<td>Upright, never when walking or running</td>
</tr>
<tr>
<td>Pallor and sweating</td>
<td>Unusual</td>
<td>Typical</td>
</tr>
<tr>
<td>Onset</td>
<td>Sudden</td>
<td>Gradual</td>
</tr>
<tr>
<td>Loss of vision/hearing</td>
<td>Sudden</td>
<td>Gradual</td>
</tr>
<tr>
<td>Lateral tongue biting</td>
<td>Usual</td>
<td>Unusual</td>
</tr>
<tr>
<td>Convulsive jerks</td>
<td>Usual</td>
<td>Unusual, but may last a few seconds</td>
</tr>
<tr>
<td>Incontinence</td>
<td>Usual</td>
<td>Unusual</td>
</tr>
<tr>
<td>Unconsciousness</td>
<td>Minutes</td>
<td>Seconds</td>
</tr>
<tr>
<td>Recovery</td>
<td>Often slow</td>
<td>Rapid on supine posture</td>
</tr>
<tr>
<td>Post-ictal drowsiness</td>
<td>Usual</td>
<td>Unusual</td>
</tr>
</tbody>
</table>

- **Pseudo seizure** is the second most common cause of misdiagnosis.
  Pseudo seizures could occur when there is a history of epilepsy / family history of epilepsy or even concurrent with epilepsy and the difficulty arises when epilepsy coexists.
• **Breath Holding Attacks**
  Two types - blue and pallid. Usually self limiting and no treatment is required.

  *Blue breath holding attacks*
  • Provoked by upsetting an infant.
  • Episode starts with crying.
  • Followed by breath holding and apnoea.
  • Loss of consciousness may be associated with a few clonic jerks and bradycardia.
  • May occur repeatedly or sporadically.
  • Usually occur after 6 months of age with a peak incidence at 2 years.

  *Pallid breath holding attacks*
  • Provoked by upsetting an infant.
  • Crying prior to episode may not be apparent.
  • Become pale and bradycardic.
  • Loss of consciousness may be associated with tonic jerks.
  • Usually occur after 6 months of age with a peak incidence at 2 years.

**KEY CLINICAL FEATURES OF COMMON EPILEPSIES AND EPILEPSY SYNDROMES**

Task Force on Classification and Terminology of ILEA emphasizes the need to determine seizure type, syndromic diagnosis when applicable, and underlying aetiology and comorbidity in their new classification proposed.

ILEA (International League against Epilepsy) classification in 2001 diagnostic scheme is based on five axes:

I. Description of the seizure  
II. Seizure type  
III. Syndromic diagnosis  
IV. Aetiology  
V. Degree of impairment
Definition of key terms

**Epileptic disease:**
A pathological condition with single specific well defined aetiology which is associated with epilepsy (e.g. Tuberous sclerosis).

**Epileptic encephalopathy:**
When frequent disabling seizures often accompanied by severe epileptiform EEG abnormalities result in neurological and cognitive impairment

**Epileptic syndrome:**
Signs and symptoms ± investigations that defines a unique epilepsy condition.

**Benign epilepsy syndrome:**
A syndrome characterized by epileptic seizures that are easily treated or require no treatment and remit without sequelae.

**Reflex epilepsy syndrome:**
A syndrome in which all epileptic seizures are precipitated by sensory stimuli

**Focal seizures and syndromes:**
Replaces the partial seizures and localization related syndromes elaborate (terms of simple partial and complex partial epileptic seizures are no longer recommended).

**Idiopathic epilepsy syndrome:**
A syndrome that is only epilepsy. No structural brain lesion or any other neurological signs.

**Symptomatic epilepsy syndrome:**
Syndrome in which seizures are due to identifiable structural lesion in brain.

**Probably symptomatic epilepsy syndrome:**
Syndromes that are believed to be symptomatic but no aetiology identified.
Some examples are given below.

**Epileptic encephalopathies**

**Progressive epileptic encephalopathy - Otahara syndrome**
- Onset is within the first three months
- Tonic spasms + focal or generalized tonic clonic seizures
- 100-300 per day
- EEG burst suppression
- Imaging – malformations or porencephaly

**West syndrome**
- Triad of
  - *Epileptic spasms* (preferred over the term infantile spasms) Sudden brief jerks (5-10 sec) involving the whole body (flexion, extension or mixed)
  - *Hypsarrhythmia on EEG*
  - *Developmental delay*
- Onset 3 - 7 months
- Spasms often occurring in clusters
- More when awakening or going to sleep
- In 85-90%, underlying cause can be found.

**Severe myoclonic epilepsy of infancy – Dravet syndrome**
- Onset – less than one year
- Starts with a prolonged febrile/afebrile seizure, clonic or tonic-clonic, often unilateral, < 1 year.
- 2-4 years - segmental or massive myoclonic seizures
- Other seizure types such as atypical absences, absence status, focal seizures may occur.
- Psychomotor delay

**Lennox Gastaut syndrome**
- Onset 3-5 years.
- Seizure types – generalized tonic, atonic, and atypical absences (combination of all three).
- EEG – generalized slow spikes present (2.5 or less /sec), bursts of fast rhythms at 10-12 Hz.
- Psychomotor delay.
- In almost all underlying cause present.
- May evolve from West syndrome.
Landau Kleffner syndrome
- Onset 3-8 years.
- Acquired progressive arrest of speech.
- Severe behavioural problems
- Seizures infrequent
- Sleep EEG – CSWS – (continuous spike wave during slow wave sleep).

Benign epilepsies and epilepsy syndromes

Benign familial neonatal seizures
- Onset 2-3 days of life.
- Well baby.
- Interictal EEG normal.
- Resolves spontaneously

Benign myoclonic epilepsy of infancy
- Onset 4 months - 3 years
- Characteristic seizure – myoclonic jerks (single, repeated, subtle causing only head nods or severe causing a fall).
- EEG – generalized spikes and polyspikes

Benign partial epilepsy with centro-temporal spikes: BECTS (Benign Rolandic epilepsy)
- Very common in children; 15-20% of childhood epilepsies.
- Onset 3-12 years.
- Infrequent seizures.
- Majority nocturnal (70% during sleep, 15% awake and sleep, 15% only awake).
- Gurgling noises.
- Inability to speak.
- Hemifacial spasms, hemiclonic movements ± secondary generalization.
- EEG- characteristic focal sharp and slow single spike and waves in central and temporal areas (20% bilateral, only seen in sleep in 30%).
- Almost all remit in adolescence.
Benign occipital epilepsy

**Early onset variant: (Panayiotopoulos)**
- Onset 2-6 years.
- Infrequent.
- Majority nocturnal.
- Tonic deviation of eyes, hemiclonic movements ± secondary generalization (predominant motor ictal phenomena are compared to visual ictal phenomena).
- Ictal vomiting.
- Loss of consciousness.
- Duration minutes-hours.
- EEG- focal sharp and slow waves in occipital areas ± sharp and slow waves central and temporal areas.
- Usually remit in adolescence.

**Late onset variant (Gastaut)**
- Onset 7-9 years.
- Predominant visual ictal phenomena while awake.
  (visual hallucinations of coloured balls, field defects, visual blindness, micropsia).
- Motor seizures are of focal seizures, tonic-clonic seizures.
- Marked post ictal symptoms such as headache, nausea, vomiting.
- DD- symptomatic occipital epilepsy and migraine.
- Many will continue to adult life.

Idiopathic generalized epilepsies

**Childhood absence epilepsy (CAE)**
- Onset 4-8 years.
- Typical absences 10-20 sec.
- Marked impairment of consciousness.
- Automatisms common (Mild eyelid blinking, head nods, mild clonic movements around mouth, may occur).
- Very frequent (ten – hundreds/day).
- EEG- generalized spike and wave discharges 3 Hz.
- Usually remit in adolescence (90%).
**Juvenile absence epilepsy**
- Onset 7-16 years.
- Typical absences longer lasting than CAE.
- Less severe impairment of consciousness than in CAE.
- Less frequent than CAE (1-10/day).
- EEG - generalized spike and wave discharges 3 Hz.
- Many will continue into adult life.

**Juvenile myoclonic epilepsy**
- Onset 7-16 years.
- Triad of seizure types:
  - typical absences (in 1/3)
  - myoclonic jerks on awakening or action induced (invariable seizure type)
  - generalized tonic clonic seizures (in almost all)
- EEG - generalized polyspike and wave discharges
- Many will continue into adult life and develop generalized tonic clonic convulsions (GTCS)

**Other generalized epilepsy syndromes**
- Epilepsy with myoclonic astatic seizures (Doose syndrome)
- Epilepsy with myoclonic absences
- Eyelid myoclonia with absences (Jeavons syndrome)
- Perioral myoclonia with absences
Symptomatic and probably symptomatic epilepsies and epilepsy syndromes

**Limbic**

**Mesial temporal lobe epilepsy**
- May have prolonged febrile convulsions.
- Seizures in childhood responding well to treatment initially and returning in adolescence.
- Aura of rising epigastric sensation.
- Psychic features – fear, déjà vu.
- Autonomic changes in skin colour, pulse, blood pressure.
- Olfactory sensations, simple auditory sensations.
- Automatisms - lip smacking, chewing, swallowing, or fidgeting, vocalizations.
- Seizures may not progress beyond aura.
- Motor manifestations- motor arrest, motionless stare, tonic deviation of eyes and head, focal motor seizure, tonic or dystonic posturing.
- Language disturbances may occur.
- EEG- anterior temporal sharp waves, spikes and slow waves (may be bilateral)
- MRI- commonly hippocampal sclerosis (other causes - cortical dysplasias, hamartomas etc)

**Frontal lobe epilepsy**
*(not uncommon; under diagnosed as the EEG may be normal)*
- Seizure types
  - Focal clonic seizures.
  - Asymmetric tonic motor seizures (aversive seizure).
  - Focal motor seizures with hyperkinetic automatisms (frenetic, agitated, shouting etc).
- Mainly nocturnal.
- EEG – electrical discharges in frontal lobe may be obscured.

**Occipital lobe epilepsy**
- Visual hallucinations – coloured spots, ictal blindness.
- Abnormal eye movements.
- EEG – electrical discharges in occipital lobe.
Neocortical

**Rasmussen syndrome (Rasmussen encephalitis)**
- Onset 1-15 years.
- Strictly unilateral motor seizures at the onset.
- Progressive cerebral atrophy confined to one side initially.
- Cognitive decline.

**Reflex epilepsies and syndromes**
*Reflex epilepsy syndrome* A syndrome in which all epileptic seizures are precipitated by sensory stimuli. (Other epileptic seizures may also be precipitated by sensory stimuli but that is not considered as “reflex epilepsy”)

**Photosensitive epilepsy**
- Induced by light.
- Generalized – GTCS, absences, myoclonic (focal may occur).

### INVESTIGATIONS

**Electroencephalography (EEG)**
EEG is performed to support the diagnosis of epilepsy.

- EEG is usually done after the second epileptic seizure but in certain circumstances, as evaluated by a specialist, may be considered after first epileptic seizure.

- EEG should be done when there is uncertainty about the diagnosis of the conditions mentioned earlier in the differential diagnosis.

- EEG helps to determine the seizure type such as generalized seizures, focal seizures, myoclonic seizures etc.

- EEG helps to determine epileptic syndromes such as West syndrome, benign Rolandic epilepsy etc.

- In children presenting with the first unprovoked seizure, the epileptiform activity on the EEG may help to assess the risk of seizure recurrence.
GUIDELINES FOR MANAGEMENT OF SEIZURES

o Repeat EEG may be helpful if the diagnosis is not certain. This should be a sleep or a sleep deprived EEG (In children a sleep EEG is best achieved through sleep deprivation or use of chloral or melatonin).

o Hyperventilation and photic stimulation EEGs can be done when indicated but such activation procedures may induce a seizure.

o Video EEG may be used in patients with diagnostic difficulties. (eg: pseudo seizures, night terrors)

• **Neuro Imaging**
  Neuro Imaging should not be routinely requested when a diagnosis of epilepsy has been made.

  MRI should be the investigation of choice in epilepsy.

  **Indications for MRI**

  a) If the patient is < 2 years.
  b) If there is evidence of focal onset on history, examination and EEG (provided it is not benign focal epilepsy).
  c) In suspected neurocutaneous syndromes.
  d) If seizures persist despite treatment with first line drugs.
  e) In neurodevelopment regression.

  *(CT scan is used to identify underlying gross pathology if MRI is not available or if anaesthetic necessary for MRI.)*

• **Other Investigation (when applicable)**

  1) ECG and Holter monitoring in suspected cardiac syncope.
  2) Head Up Tilt Testing in suspected vasovagal syncope.
  3) Blood sugar, electrolytes, calcium and phosphorus to determine an underlying cause for the epilepsy.
  4) Metabolic screen

• **Neuropsychological Assessment** is necessary if

  a) Child with epilepsy has educational difficulties.
  b) MRI has identified abnormalities in cognitively important brain regions.
  c) Child is found to have memory loss or cognitive deficit or decline.
PHARMACOLOGICAL TREATMENT OF EPILEPSY

Anti-epileptic drugs (AED) should only be started once the diagnosis of epilepsy is confirmed.

Initiation of AED
- In children, should be by a Paediatrician/ Paediatric Neurologist.
- Is generally not recommended after a first unprovoked tonic-clonic seizure.
- May be considered after a first unprovoked seizure if:
  - the individual has a neurological deficit
  - a further seizure is unacceptable to the family
  - brain imaging (where indicated) shows a structural abnormality
- Children with febrile seizures, even if recurrent should not be treated with long term AED.

Choice of first AED depends on
- The seizure type/ syndrome
- The potential adverse effects
- Co-morbidity
- The availability and cost

Principles of AED Therapy
- Should use monotherapy wherever possible (x)
- Unsuccessful initial therapy, try monotherapy with another drug (x)
- If monotherapy in the maximum dose has failed, a second drug should be started. The second drug could be alternative first line.
- If the second drug reduced the seizure frequency, taper off the first and continue monotherapy with the second.
- If there is no improvement within a month, taper off either the first or the second, depending on their relative efficacy.
- If both drugs do not work, another second line drug may have to be introduced as monotherapy.
- If the response is poor consider blood levels if facilities are available.

Consider add on or combination therapy only when monotherapy has failed. Prior to initiation of combination therapy consider the following.
• Is the diagnosis correct?
• Adherence to treatment
• The appropriateness of the AED for the seizure type.
• The quality of the drug.

Long term AED therapy

• Should be planned by a specialist. (x)
• Involves adjustment of drug dosage according to the weight. (x)
• Should include discussion with the individual regarding possible side effects, rationale of treatment and what should be done if a dose is missed or during illness.
• Should involve a simplified medication regimen with clear verbal and written instructions.

Blood levels, if available, are indicated under the following circumstances

• Poor response to treatment.
• Poor compliance.
• Toxic effects.
• Management of drug interactions.

If management is straightforward, AED can be prescribed in the primary care.

Newer AED for uncontrolled epilepsy

Vigabatrin
Topiramate
Lamotrigine
Gabapentine

Levetiracetam
Oxcarbazepine
Tiagabine
Sulthiame
Stiripentol

available in Srilanka
currently not available

Indications for use of newer AED

• If older AEDs are not effective.
• Older AEDs are contraindicated.
• Poorly tolerated old AED.
• Drug interaction with old AED.
**Withdrawal of AED therapy should**

- be individualized.
- be under the guidance of the specialist.
- be considered in those who have been seizure free for at least two years.
- be done slowly (at least over 2 to 3 months)
- last longer (up to 6 months or longer) when withdrawing benzodiazepines and barbiturates.
- be abandoned if seizure recurs.
- not involve routine seizure recours.

### Drug options by type of epilepsy or epilepsy syndrome

*(This table is prepared considering the availability of the drugs currently in Sri Lanka)*

<table>
<thead>
<tr>
<th>Epilepsy syndrome</th>
<th>First option</th>
<th>Second option</th>
<th>Other drugs (under special circumstances)</th>
<th>Drugs to be avoided (may worsen seizures)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Generalized tonic-clonic seizures only</td>
<td>Sodium Valproate</td>
<td>Topiramate</td>
<td>Lamotrigine</td>
<td>Clonazepam, Phenytoin, Phenobarbital, Acetazolamide, Vigabatrin</td>
</tr>
<tr>
<td>Childhood absence epilepsy</td>
<td>Sodium Valproate</td>
<td>Topiramate</td>
<td>Lamotrigine</td>
<td>Carbamazepine, Phenytoin, Phenobarbital, Vigabatrin</td>
</tr>
<tr>
<td>Juvenile absence epilepsy</td>
<td>Sodium valproate</td>
<td>Topiramate</td>
<td>Lamotrigine</td>
<td>Carbamazepine, Phenytoin, Phenobarbital, Vigabatrin</td>
</tr>
<tr>
<td>Juvenile myoclonic epilepsy</td>
<td>Sodium Valproate</td>
<td>Carbamazepine</td>
<td>Lamotrigine</td>
<td>Carbamazepine, Phenytoin, Phenobarbital, Vigabatrin</td>
</tr>
<tr>
<td>Focal epilepsies</td>
<td>Sodium valproate</td>
<td>Lamotrigine</td>
<td>Topiramate, Carbamazepine</td>
<td>Clonazepam, Phenobarbital, Vigabatrin, Carbamazepine</td>
</tr>
<tr>
<td>Infantile spasms</td>
<td>Sodium Valproate</td>
<td>Carbamazepine</td>
<td>Lamotrigine</td>
<td>Clonazepam, Phenobarbital, Vigabatrin, Carbamazepine</td>
</tr>
<tr>
<td>Benign epilepsy with centro temporal spikes (if seizures are frequent)</td>
<td>Sodium valproate</td>
<td>Carbamazepine</td>
<td>Lamotrigine</td>
<td>Clonazepam, Phenobarbital, Vigabatrin, Carbamazepine</td>
</tr>
<tr>
<td>Severe myoclonic epilepsy of infancy</td>
<td>Sodium valproate</td>
<td>Carbamazepine</td>
<td>Lamotrigine</td>
<td>Clonazepam, Phenobarbital, Vigabatrin, Carbamazepine</td>
</tr>
<tr>
<td>Benign epilepsy with occipital paroxysms</td>
<td>Sodium valproate</td>
<td>Carbamazepine</td>
<td>Lamotrigine</td>
<td>Clonazepam, Phenobarbital, Vigabatrin, Carbamazepine</td>
</tr>
<tr>
<td>Continuous spike wave of slow sleep</td>
<td>Sodium valproate</td>
<td>Carbamazepine</td>
<td>Lamotrigine</td>
<td>Clonazepam, Phenobarbital, Vigabatrin, Carbamazepine</td>
</tr>
<tr>
<td>Lennox-Gastaut syndrome</td>
<td>Sodium Valproate</td>
<td>Carbamazepine</td>
<td>Lamotrigine, ACTH</td>
<td>Carbamazepine, Lamotrigine, Topiramate</td>
</tr>
<tr>
<td>Landau-Kleffner syndrome</td>
<td>Sodium valproate</td>
<td>Carbamazepine</td>
<td>Lamotrigine</td>
<td>Carbamazepine, Phenobarbital, Vigabatrin, Carbamazepine</td>
</tr>
<tr>
<td>Myoclonic atatic epilepsy</td>
<td>Sodium valproate</td>
<td>Carbamazepine</td>
<td>Lamotrigine</td>
<td>Carbamazepine, Phenobarbital, Vigabatrin, Carbamazepine</td>
</tr>
</tbody>
</table>

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MANAGEMENT OF AN ACUTE SEIZURE & STATUS EPILEPTICUS

Definition

Status epilepticus (SE) is defined as clinical seizure activity lasting more than 30 minutes, constituting a neurological emergency. Seizure activity may be either continuous or intermittent without the patient recovering consciousness in between.

This above definition includes convulsive as well as non-convulsive seizure disorders. What we consider here is the management of convulsive status epilepticus.

For practical purposes, the approach to the child who presents with a tonic-clonic convulsion lasting more than 5 minutes should be the same as the child who is in “established” status.

5% of children with febrile seizures and 1-5% of epilepsy patients develop status epilepticus. Overall mortality is 10-15%.

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Doses of commonly used drugs

<table>
<thead>
<tr>
<th>DRUG</th>
<th>TOTAL DAILY DOSE</th>
<th>TIMES DAILY</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium Valproate</td>
<td>10-50mg/kg</td>
<td>2</td>
<td>Monitor liver functions in children less than 3 years. Stop if vomiting, skowiness or jaundice occurs. Avoid in liver disease.</td>
</tr>
<tr>
<td>Carbamazepine</td>
<td>5-30mg/kg</td>
<td>2 - 3</td>
<td>Start with the lowest dose and increase by 2.5 to 5mg/kg/day at weekly intervals. Seek advice if a rash develops.</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>Up to 1 year</td>
<td>1 - 3mg</td>
<td>1 to 5 years 3 - 6mg 5 to 12 years 4 - 8mg</td>
</tr>
<tr>
<td>Clobazam</td>
<td>0.5 - 1mg/kg</td>
<td>2</td>
<td>Tolerance tends to develop.</td>
</tr>
<tr>
<td>Ethosuximide</td>
<td>20 - 30 mg/kg</td>
<td>2</td>
<td>Capsules of 250mg which cannot be broken</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>5 - 8 mg/kg</td>
<td>2</td>
<td>Plasma levels should be monitored if clinically indicated. Avoid administration with feeds.</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>5 - 8 mg/kg</td>
<td>2</td>
<td>Use only in refractory epilepsy because of the adverse effects on learning and behaviour.</td>
</tr>
<tr>
<td>Acetazolamide</td>
<td>10 - 30mg/kg</td>
<td>2</td>
<td>Not commonly used in children. Avoid in mild renal impairment as it may cause acidosis. May be used to enhance certain other AED drugs.</td>
</tr>
<tr>
<td>Lamotrigine</td>
<td>0.5mg/kg/day</td>
<td>2</td>
<td>Gradually increase the dose every two weeks. Can cause life threatening skin rash.</td>
</tr>
<tr>
<td>Lamotrigine for patients on valproate</td>
<td>0.15mg/kg/day for 2 weeks</td>
<td>1</td>
<td>Gradually increase to 1 - 5 mg/kg/day</td>
</tr>
<tr>
<td>Topiramate</td>
<td>1 - 3 mg/kg</td>
<td>2</td>
<td>Reduce dose in renal impairment. Glaucoma has been reported.</td>
</tr>
<tr>
<td>Vigabatrin</td>
<td>Starting dose in 40 mg/day</td>
<td>2</td>
<td>Can use larger doses in infantile spasms 150-200mg/kg/day. Greatest concern is the persistent visual field defects</td>
</tr>
<tr>
<td>Gabapentine</td>
<td>10 - 50 mg/kg/day</td>
<td>2 - 3</td>
<td>Advantageous in patients with complex medical problems as no drug interactions occur.</td>
</tr>
</tbody>
</table>
Lab Studies

Laboratory studies should be done according to the likely diagnosis based on age, history and clinical signs.

- Blood glucose using immediate bedside testing (e.g. Dextrostix)
- Serum electrolytes
- Calcium and magnesium
- Liver function tests, blood urea/creatinine
- Arterial blood gas
- Toxicology screen (always keep some blood for future tests)
- Anticonvulsant levels (if indicated by history of ingestion or existent therapy and if available)
- FBC and septic work-up
- Blood film for malarial parasites

Imaging Studies: Not essential unless clinical evidence supports a CNS lesion.

- Stabilize all children before CT scanning or other imaging studies are performed. Obtain imaging studies based on likely aetiologies.
- Cervical spine x-rays, if potential trauma
- A head CT scan is the best diagnostic imaging study, particularly if the following are suspected:
  - Haemorrhage
  - Midline shift
  - Mass lesion
- MRI is not a diagnostic tool, unless it is immediately available and the child’s cardio-respiratory status is stable.

Electroencephalogram

For unremitting status epilepticus usually performed in a critical care setting (If & when portable EEG is available)

Lumbar puncture is indicated only under following circumstances (once the patient is stable and there is no evidence of raised ICP):

- For prolonged status epilepticus of unknown etiology.
- For suspected cases of meningitis or encephalitis.

References


**TREATMENT GUIDELINE FOR AN ACUTE TONIC-CLONIC CONVULSION INCLUDING ESTABLISHED CONVULSIVE STATUS EPILEPTICUS**

- Pay attention to Airway, Breathing, Circulation
- Give high flow oxygen, suck out secretions, assist ventilation and intubate if necessary
- Decompress stomach by nasogastric tube
- Measure blood glucose and never forget to treat hypoglycaemia
- Monitor vital signs
- Immobilize the cervical spine if trauma is suspected.
- Confirm epileptic seizure.

**IMMEDIATE IV ACCESS**

1. Diazepam IV 0.3 mg/kg bolus (30-60 seconds) or lorazepam IV 0.05 - 0.1 mg/kg (if available)

   Seizure continuing at 10 minutes

   2. Diazepam IV 0.3 mg/kg bolus (30-40 seconds) or Lorazepam 0.05 to 1 mg/kg

      Seizure continuing at 10 minutes

      3. Phenytoin – 18 mg/kg, IV or IO over 20 minutes
         Or Phenobarbitone 15-20 mg/kg
         IV/IO over 10 minutes
         (use intraosseous route if still no IV access)

         Still continuing

      4. Midazolam infusion / 60-300 microgram / kg / hr
         Diazepam infusion 100-400 microgram / kg / hr
         (use intraosseous if no IV access)

         Seizure still continuing for more than 20 minutes despite infusion Call for Help (anaesthetic Team)

      5. Rapid Sequence induction of anaesthesia with Thiopentone 4 mg/kg IV/IO

**NO IV ACCESS**

1. Diazepam 0.5 mg/kg PR or buccal midazolam (0.3 mg/kg) or midazolam IM (0.15 mg/kg)

   Seizure continuing at 10 minutes

   2. Repeat Diazepam 0.5 mg/kg PR

      Seizure continuing at 10 minutes

      3. Paraldehyde 0.4 ml/kg PR (with same volume of olive oil)

      Seizure still continuing despite infusion Call for Senior Help

   4. Call for Help (anaesthetic Team)

Lorazepam was better than diazepam for reducing risk of non-cessation of seizures (RR 0.64, 95% CI 0.45 to 0.90) and had a lower risk for continuation of status epilepticus requiring a different drug or general anaesthesia (RR 0.63, 95% CI 0.45 to 0.88). The Cochrane Database of Systematic Reviews 2006 Issue 2
FEBRILE CONVULSIONS

Definition

The Commission on Epidemiology and Prognosis of the International League Against Epilepsy (1993) agreed on the following definition:

“An epileptic seizure occurring in childhood, after age 1 month, associated with a febrile illness not caused by an infection of the central nervous system (CNS), without previous neonatal seizures or a previous unprovoked seizure, and not meeting criteria for other acute symptomatic seizures”.

*This definition has replaced all other previous definitions of febrile seizures*.2,3

‘Simple’ and ‘Complex’ Febrile Convulsions

‘Complex’ febrile seizures were defined as those that had one or more of the following4:
- Duration more than 15 minutes;
- Recurrence within 24 hours;
- Focal features.

‘Simple’ febrile seizures were defined as those that did not have complex features.

Indications for admission to hospital after a febrile seizure3

- First febrile seizure.
- Age <18 months.
- Incomplete recovery after one hour.
- Any likelihood of CNS infection.
- A ‘complex’ febrile seizure.
- Fever has lasted more than 48 hours before onset of seizures.
- Home circumstances inadequate/excessive parental anxiety/parents’ inability to cope.
Investigations

- Routine blood studies are of no benefit in evaluation of the child with a febrile seizure.
- A blood glucose determination should be obtained if the child has a prolonged seizure or a prolonged period of post-ictal impairment of consciousness.
- All children who convulse with fever need not have a lumbar puncture (LP). However, it should be strongly considered:
  - Where there is a history of irritability, decreased feeding or lethargy.
  - If there are clinical signs of meningitis/encephalitis.
  - If the child is unduly drowsy or irritable or systemically ill.
  - If there is prolonged post-ictal altered consciousness or neurological deficit.
  - ‘Probably’ if the child is <18 months of age and almost certainly if the child is aged <12 months at the occurrence of the first fit.
  - After a complex convulsion.
  - After pretreatment with antibiotics.

In these situations an LP with simultaneous blood and cerebrospinal fluid (CSF) glucose levels and a blood culture should be undertaken unless there are specific contraindications.

A negative LP should not eliminate need for careful follow-up because early in the course of meningitis spinal fluid may not show pleocytosis or changes in protein and sugar content.

Ideally the decision should be taken by an experienced doctor, who may decide on clinical grounds that LP is unnecessary even in a younger child, but when in doubt the investigation should be performed. The doctor deciding not to undertake an LP should be prepared to review the decision over next 24 hrs and antibiotics should be withheld during this period.

A comatose child must be examined by an experienced doctor before LP because of the risk of coning. Brain imaging may be necessary.

- An electroencephalogram (EEG), either at time of presentation after a simple febrile seizure, or within the following month, will not predict occurrence of recurrent febrile convulsions or future afebrile seizures. Recurrent simple or complex febrile seizures also do not justify an EEG, as it is of no use in identifying a structural abnormality or in predicting...
development of epilepsy. However, an EEG may be helpful if clinical picture suggests focal pathological changes in the brain.

- Neuroimaging should be considered in children with febrile convulsions who are also found to have the following:
  - Micro/macrocephaly, a neurocutaneous syndrome or pre-existing neurological deficit.
  - Post-ictal neurological deficit persisting for more than a few hours following febrile seizure.
  - Recurrent complex febrile seizures, particularly where there is doubt whether seizures are febrile in origin.

**Treatment**

- **Management of fever**
  - The fever should be treated to promote the child’s comfort.
  - An adequate fluid intake should be ensured to prevent dehydration.
  - Physical methods to reduce the body temperature, such as fanning, tepid sponging and light clothing, are often recommended although their efficacy in preventing febrile convulsions is not established.
  - Paracetamol and ibuprofen are the recommended antipyretics.
    - Dose of paracetamol (maximum of 4 doses in 24 hours): < 3 months 60 mg, 3 months-1 year 60-120 mg, 1-5 years 120-250 mg
    - Dose of ibuprofen (3-4 times daily): 1-2 years 50 mg, 3-5 years 100 mg
  - Diclofenac sodium (rectal/oral) may also be considered for control of fever.
  - Measures such as dissolving paracetamol tablets in paracetamol syrup are completely reprehensible.
  - If paracetamol is dispensed in powdered, syrup or tablet form, the patient should be made aware that it is paracetamol to prevent duplication.
  - Parents and caregivers should be educated on the simple axillary temperature measurements

(* Note: If Dengue Fever is suspected both Ibuprofen and Diclofenac sodium should not be used.)
• *Acute management of a seizure* (refer guidelines for acute tonic-clonic convulsion)

• **Prophylaxis**
  
  ▪ There is no evidence that in the minority of children who later develop epilepsy, the prophylactic use of anticonvulsant drugs would have prevented it.

  ▪ Long-term prophylaxis with phenobarbitone, although effective, has frequent and substantial side effects. Long-term administration of sodium valproate is as effective as phenobarbitone, but may produce fatal hepatic or pancreatic dysfunction in this age group. Thus, long-term prophylaxis with phenobarbitone or sodium valproate is not recommended.

  ▪ Diazepam, given rectally or orally at the time of the fever, may prevent the recurrence of seizures and preclude the toxic effects of long-term anticonvulsant prophylaxis. However, the parent or other caregiver must be aware of the fever before the seizure occurs and as Berg indicates, recurrence is most likely when the fever has been present for less than one hour. Thus, routine use of rectal or oral diazepam for prophylaxis is not recommended. **However for children who appear to have a very low threshold for febrile convulsions with any febrile episode and particularly if the seizures are recurrent and prolonged, there is the option of using rectal diazepam in two situations:**

    ▪ As soon as the child starts fitting.
    ▪ Whenever the child is febrile and before the child starts fitting.

  ▪ Early use of oral or rectal diazepam (before the child starts fitting) may result in drowsiness and ataxia that might interfere with the carers’ and doctors’ ability to distinguish a “benign” febrile illness from a potentially more serious febrile illness, specifically meningitis/encephalitis or a structural brain lesion.

  ▪ Decision to use rectal diazepam should be based on a number of factors: the balance between potential benefits and risks, wishes and abilities of child’s carers, child’s frequency and pattern of febrile illnesses and type of febrile seizure.


**Immunisation**

- Babies having seizures with fever below the age of 4 months should be assessed by a paediatrician.

- Children who have febrile convulsions before immunization against diphtheria, pertussis and tetanus should be immunized after their parents have been instructed about the management of fever and the use of rectal diazepam or consider acellular pertussis for future immunizations.

- Measles, mumps and rubella immunization should be given as usual to children who have had febrile seizures, with advice about the management of fever to the parents. Rectal diazepam should be made available for use should a seizure occurs.

- Febrile convulsions are not a contraindication.

- JE vaccine should be given only after fit free period of 1 year.

**Prognosis**

- Parents should be told that the prognosis as regards to development and neurological status is excellent. Under exceptional situations when there is a doubt about child's current development or neurological status a Consultant Paediatrician’s opinion should be sought.

- Risk of subsequent epilepsy after a single simple febrile seizure is about 2.5%.

- With increasingly complex convulsions (three complex features) the risk rises to nearly 50% by 25 years of age but only about 1% of children with febrile convulsions are in this group.

- Risk of having further febrile convulsions is about 30% overall, increasing with younger age at first convolution and approaching 50% in children < 1 year old at time of their first seizure.

- A family history of febrile seizures (but not epilepsy) in a first degree relative is also associated with an increased risk of recurrence.

- Recurrences appear more likely in children whose initial febrile convolution occurred with a relatively low fever.
Multiple initial seizures occurring during same febrile episode also appear to be associated with an increased risk of recurrence.\textsuperscript{15}

Information for parents

- Facts and advice should be verbal and written, explaining what a febrile convulsion is, that it is common, that recurrences are unlikely, and that risk of brain damage and later epilepsy are very rare.\textsuperscript{3,16}

- They should be reassured that there is no evidence that any child has ever died as a result of a febrile convulsion.\textsuperscript{3,17}

- Advice should also include what to do if their child has a febrile illness and what to do if their child has a further febrile convulsion and specifically that arrangements should be made to give rectal diazepam or alternatively to transport the child to hospital if the tonic-clonic seizure has lasted 5-10 or more minutes and shows no signs of stopping.\textsuperscript{3}

- Information and advice sheets for parents and caregivers in Sinhala/ Tamil and English are attached.

References


Prepared by the Guidelines Committee of the Sri Lanka College of Paediatricians comprising

Dr Deepthi Samarage (Coordinator) Senior Lecturer in Paediatrics, Faculty of Medical Sciences, University of Sri Jayawardenapura, Nugegoda.

Dr G N Lucas Former Consultant Paediatrician, Lady Ridgeway Hospital, Colombo.

Dr Neomal Gunaratne Consultant Paediatrician.

Dr D H Karunatilaka Consultant Paediatrician, Lady Ridgeway Hospital, Colombo.

Dr H T Wickramasinghe Consultant Paediatrician.

Dr Nimal Katugaha Consultant Paediatrician, Sirimavo Bandaranaike Children’s Hospital, Peradeniya.

Dr Samantha Waidyanatha Consultant Paediatrician, Colombo South Teaching Hospital, Kalubowila.