# Guidelines

# For The Management Of

# Epilepsy


**By**

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**Clinical Standards & Guidelines**

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Epilepsy can be defined as recurrent, usually unprovoked epileptic seizures that result from excessive synchronous and abnormal firing patterns of the cerebral cortical neurons. An epileptic seizure is defined as a paroxysmal stereotyped disturbance of consciousness, motor function, sensation, emotion, behaviour or perception that on clinical grounds result from cortical neuronal discharge.
Therefore, epileptic seizures may be viewed as the symptoms of the disease, epilepsy. Epileptic seizures are classified on the basis of their clinical features alone whereas the classification of epilepsies and epileptic syndromes is based on electroclinical criteria described by the International Classification of Epilepsies and Epileptic syndromes

1. Any patient having a first seizure should be seen as soon as possible by a specialist in the management of the epilepsies to ensure precise and early diagnosis and initiation of therapy as appropriate to their needs.
2. Patient presenting with an attack, a physical examination should be carried out involving their cardiac, neurological, mental status, and should include a developmental assessment for children.
3. A detailed history should be taken from eyewitness to the attack, where possible, to determine whether or not an epileptic seizure is likely to have occurred. Video recording of the event will help the clinical decision as to whether an epileptic seizure has occurred.
4. Neuropsychological assessment should be considered in whom it is important to evaluate learning disabilities and cognitive dysfunction, particularly in regard to language and memory. It is indicated in:

*Patient with epilepsy is having educational or occupational difficulties.
*Patient complains of memory or other cognitive deficits and/or cognitive decline.
*MRI has identified abnormalities in cognitively important brain regions.

**Investigation:**

The main objectives of investigating patients with epilepsy are to:

1. Clarify the diagnosis of epilepsy and non-epileptic attacks.
2. Determine the nature of the seizure types and epilepsy syndrome.
3. Identify the laterality and localization of seizure onset (partial seizures).
4. Identify the aetiology of the epilepsy.
5. Identify concomitant problems, both neurological and general.
6. Monitor the progression of the condition, and the consequences of the epilepsy and its treatment.

These objectives are achieved through the electroencephalogram (EEG), neuroimaging, neuropsychological assessment and other investigations (including blood tests and muscle biopsy).

**EEG**: except for adult patients with a clear metabolic or structural abnormality on brain imaging, **all** patients with epilepsy will require an EEG. The aim of the EEG is to clarify, rather than confirm, the diagnosis of epilepsy.
*An EEG should be performed only to support a diagnosis of epilepsy in adults in whom the clinical history suggests that the seizure is likely to be epileptic in origin.

*It should be performed after the second epileptic seizure but may in certain circumstances, as evaluated by the specialist, be considered after a first epileptic seizure.
*An EEG may be used to help determine seizure type and epilepsy syndrome in patient in whom epilepsy is suspected & can be used to assess the risk of seizure recurrence.
*The EEG should not be used in isolation to make a diagnosis of epilepsy *NOR* be used to exclude a diagnosis of epilepsy in a patient in whom the clinical presentation supports a diagnosis of a non-epileptic event.
*When a standard EEG has not contributed to diagnosis or classification, a sleep EEG should be performed which is best achieved through sleep deprivation or the use of melatonin.

**MRI**: should be the imaging investigation of choice & is particularly important in:
*Partial or focal seizures based on the history and/or EEG.
*Fixed or progressive neurological or psychological deficit.
*Onset of generalised seizures before the age of 2year and after 20 years.
*Acutely, after significant head trauma.
*Difficulty obtaining seizure control with AED.
*Loss of seizure control or status epilepticus, if this does not have a clear explanation such as omission of medication.

Imaging may be ***postponed*** in the following circumstances:
*There is a clear provoking factor, such as alcohol withdrawal.
*Children with benign focal epilepsy with centrotemporal spikes.
*Pregnant women with no acute problems, such as a possible intracerebral haemorrhage or infection.

**At the initial evaluation**; the following investigations should be considered to exclude other diagnoses, and to determine an underlying cause of epilepsy:
* Blood glucose, renal profile, liver profile, serum electrolyte.
*A 12-lead ECG with/out CXR & Echo study should be performed in adults with suspected epilepsy & in young people in cases of diagnostic uncertainty.
*For selected cases serum or CSF studies including VDRL, HIV serology, connective tissue screening & investigations for inborn errors of metabolism. These investigations must be individualised and ordered judiciously.

During **subsequent follow up**, the decision to *repeat or perform other investigations* must be individualized
1. Repeat EEG and neuroimaging if there is a need to reassess the cause of the epilepsy or suspicion that there is progression of the underlying disease; in children it is important to periodically assess cognitive function as its decline may be subtle but often greatly influences prognosis.
2. Repeat biochemical, haematological & clotting profiles to detect adverse effects of AED treatment, especially before surgery & to evaluate adverse events that have already developed.

3. In patients taking enzyme-inducing AEDs (Table 1); repeat the full blood count, liver, renal functions and electrolyte especially serum calcium every 1-2 years.
4. In patients on valproate, repeat full blood count more frequently or before surgical procedures as valproate may cause acute hematological toxicities, especially in children, including rare reports of myelodysplasia and acute leukemia-like syndrome.
5. Monitoring of serum AED concentrations is preferred.
6. *Asymptomatic minor abnormalities* in test results are not necessarily an indication for changes in medication

**Management:**

1.All patients with epilesy should have a comprehensive care plan that is agreed by the person, family and healthcare professionals which include lifestyle issues as well as medical issues.
2. Treatment with AED therapy is generally recommended after a second epileptic seizure. However; it should be considered after a first unprovoked seizure if:

- The patient has a neurological deficit
- The EEG shows unequivocal epileptic activity
- The risk of having a further seizure unacceptable
- Brain imaging shows a structural abnormality.
- The seizures are associated with a previous absence and/or myoclonic seizures

3. The AED (anti-epileptic drug) treatment strategy should be individualized according to the seizure type, epilepsy syndrome, co-medication, co-morbidity and the preferences of the person and their family as appropriate
4. Start with a single first line AED after deciding on the type of seizure(s) and the epilepsy syndrome; beginning at a low dose and increase gradually over 2 to 3 weeks.
5. Review the patient within a month to assess compliance, side effects and seizure control.
Review every 6-8 weeks. If the seizures are not controlled and there are no side effects, increase the dose appropriately. In about 60-70% of patients, these steps are sufficient to achieve good seizure control. If the AED fails to control seizures:
* Review the diagnosis and seizure pattern.
* Review compliance.
* Ensure that the maximum tolerated dosage has been used.
6. If the first AED has failed because of adverse effects or continued seizures despite maximum tolerated dose, a second drug should be started (which may be an alternative first-line or second-line drug) and built up to an adequate or maximum tolerated dose without tapering the first. If the patient has a good response to the second AED then the first drug should be tapered off slowly. Consider long-term two-drug therapy if monotherapy has not achieved remission or good seizure control.
If the second drug is unhelpful, either the first or second drug may be tapered, depending on relative efficacy, side effects and how well the drugs are tolerated before starting another drug.

7. If the first add-on AED is ineffective, or produces undesirable side effects, withdraw it slowly, and simultaneously replace it with a second add-on AED from the remaining choices. This process can be repeated for other possible add-on AEDs.
If the seizures are still not adequately controlled on two AEDs, some patients may benefit from an additional third AED.
8. Combination therapy (adjunctive or 'add-on' therapy) should only be considered when attempts at monotherapy with AEDs have not resulted in seizure freedom. If trials of combination therapy do not bring about worthwhile benefits, treatment should revert to the regimen (monotherapy or combination therapy) that has proved most acceptable to in terms of providing the best balance between effectiveness in reducing seizure frequency and tolerability of side effects.
9. If using carbamazepine, offer controlled-release carbamazepine preparations.
When prescribing sodium valproate to women, discuss the possible risk of malformation and neurodevelopmental impairments in an unborn child, particularly with high doses of this AED or when using as part of polytherapy.
Consider the risk–benefit ratio when using vigabatrin because of the risk of an irreversible effect on visual fields.
10. Seizures due to alcohol withdrawal or other metabolic or drug-related causes or sleep deprivation should not be treated with AEDs. Treatment should be considered only if there are recurrences suggestive of epilepsy.
11. All patients developing seizures within a week of head injury should be treated, but AED withdrawal should subsequently be considered.
12. If seizures continue **beyond 2-3 years**, the patient is considered to have chronic epilepsy (accounting for about 10-20% of all epileptic patients), and the following management steps must be taken:

a. Review the diagnosis and aetiology - history, EEG, neuroimaging, etc.
b. The possibility of NEAD must be considered.
c. Re-classify the epilepsy (seizure type{s} and syndrome).
d. Review compliance.
e. Review drug history - which AEDs have or have not been useful in the past, which have not been tried, drug and blood levels of previous therapy.
f. Set a treatment plan - sequence of drug changes, serum level monitoring.
g. Recognise limitations of therapy; patients with intractable epilepsy must be able to accept their disability and continue with life. There are limits to the effectiveness of AEDs available and it is important to create a balance between seizure frequencies, side effects from AEDs, and quality of life.
h. Evaluate for a possible progressive structural lesion, especially if the patient has partial seizures, and surgery may be considered.
i. Consider surgical therapy

**Decision to withdraw AEDs**

1.The decision to continue or withdraw medication should be taken by the specialist, family, and the patient who have been seizure free for at least 2 years and after a full discussion of the risks and benefits of withdrawal.

2. When AED treatment is being discontinued, it should be carried out slowly (at least 2–3 months) and one drug should be withdrawn at a time.
3. Particular care should be taken when withdrawing benzodiazepines and barbiturates (may take up to 6 months or longer) because of the possibility of drug-related withdrawal symptoms and/or seizure recurrence
4. There should be a failsafe plan whereby if seizures recur, the last dose reduction is reversed and medical advice is sought.
5. When freedom from seizures has been achieved for a period of at least 2 years, drug withdrawal may be considered. Exceptions occur in certain epilepsy syndromes e.g. JME, which has a high relapse rate.
6. No guarantee of seizure freedom can ever be given when a drug is withdrawn. There is a 40- 50% risk of relapse within the Ist year of cessation. The risk of relapse is higher in patients:
*16 years of age.
*Whose age at seizure onset was < 3, or > 30 years.
*With tonic-clonic (primary or secondary) or myoclonic seizures.
*With partial onset seizures.
*With seizures needing > 1 AED for good control at the time of discontinuation.
*With an abnormal EEG-the EEG is not helpful in predicting seizure recurrence, although a normal EEG is reassuring
*With a past history of status epilepticus.
*With a history of afebrile or atypical febrile seizures in childhood.
*Experiencing one or more seizures after the start of treatment.
*With a short duration of seizure-freedom.
*Whose duration of treatment exceeds 10 years.
*With a known aetiology of seizures (symptomatic epilepsy) and associated neurological handicap.
*With a fast rate of drug withdrawal
7. Patients in whom seizure recurrence is less likely include:
- Those who have been seizure-free for ≥ five years or at least between 3-5 years.
- Those with benign Rolandic and familial neonatal convulsions.

**Driving and epilepsy**:
conditions that may allow for safe driving include:

- Well-controlled epilepsy and the patient is on treatment.
- Seizure freedom for at least 1 year; off or on treatment.
- Purely nocturnal seizures.

## Epilepsy in women

**Catamenial epilepsy** is exacerbation of seizures occur immediately before or during menses because of the proconvulsant effect of oestrogen. Intermittent clobazam or acetazolamide given during the menstrual period may alleviate catamenial exacerbation of seizures.

**Oral contraceptive pills failure** is increased with AEDs that induce hepatic microsomal enzymes EIAED so be advised about additional contraceptive measures. If a woman wishes to rely on the OCP alone, she should be prescribed a preparation containing at least 50 pg of oestradiol. If breakthrough bleeding occurs, the dose of oestrogen should be increased to 75-100 pg per day, and tricycling (taking three packs without a break) should be considered. The progesterone-only pill is not recommended as a reliable contraceptive in women taking enzyme-inducing AEDs. Intramuscular Depo-Provera at a dose of 150 mg should be given at shorter intervals (every 10 weeks instead of 12 weeks) if the patient is on enzyme-inducing AEDs. There is no evidence that hormonal contraception adversely affects seizure control, except for those patients treated with lamotrigine whose metabolism is significantly increased by OCP.

If emergency contraception is required for women taking enzyme inducing AEDs, the dose of levonorgestrel should be increased to 1.5 mg and 750 µg 12 hrs apart.

**Pregnancy** there is little evidence that seizures adversely affect pregnancy other than increasing the risk of trauma on the developing foetus. Pregnancy does not increase the risk of developing new epileptic seizures for the first time.

For eclampsia, evidence is in favour of magnesium sulphate as compared to standard AEDs in terms of lower rates of recurrent seizures, pneumonia and ventilation, and lower mortality as well as being safer for the baby.

Women on AEDs should be monitored throughout pregnancy to detect foetal malformations by Serum alpha-fetoprotein in maternal blood & ultrasound

**Labour** the risk of seizures is greatest during the delivery period; 1-2% of epileptic women suffer a GTCS during labour. The patient's regular AEDs must be continued through labour, via a nasogastric tube or intravenously, if necessary.

As pain, emotional stress and hyperventilation may increase the risk of seizures; epidural anaesthesia should be considered early during labour. If frequent GTCS or complex partial seizures do occur during labour, a caesarean section is indicated.

An elective caesarean section is also recommended if frequent GTCS or complex partial seizures occur during the last weeks of pregnancy; the treatment of the seizure itself should proceed in the usual manner**.**

**Infant** there is insufficient evidence to support an increased risk of bleeding in infants born to mothers on AEDs (particularly hepatic enzyme-inducing drugs). However, the precautionary measure of giving 20 mg/day of oral vitamin $K_1$ in the last month of pregnancy, and/or their newborns 1 mg of vitamin $K_1$ intramuscularly at birth should be practiced till proven otherwise. If there is evidence of bleeding in the newborn, intravenous fresh frozen plasma should be given.

**Breastfeeding** all women with epilepsy should be encouraged to breastfeed, which is safe in the majority of cases. As most AEDs are secreted in breast milk in small quantities, the infant may become sedated (in about 5-10%). Under these circumstances, bottle-feeding can be used to supplement breastfeeding.

**Status Epilepticus SE**
defined as a one continuous unremitting seizure lasting more than 5 minutes or a series of seizures without recovery of full consciousness or without an interictal return to the baseline clinical state.

1. **General measures 1st stage (0−10 minutes)**
Secure airway and resuscitate
Administer oxygen
Assess cardiorespiratory function
Establish intravenous access

**2. 2nd stage (0−30 minutes)**
Institute regular monitoring
Emergency AED therapy ▼
Emergency investigations ▼▼
Treat acidosis if severe
Administer glucose (50 ml of 50% solution) and/or 1.V thiamine (250 mg) if any suggestion of alcohol abuse or impaired nutrition

**3rd stage (0−60 minutes) Established status**
Establish aetiology
Alert anaesthetist and ICU
Identify and treat medical complications
Pressor therapy when appropriate

**4th stage (30−90 minutes) Refractory status**
Establish ICU care and EEG monitoring
Initiate intracranial pressure monitoring where appropriate
Initiate long-term, maintenance AED therapy

**Emergency AED therapy ▲**
**Early status**
Diazepam I.V 0.15 mg/kg( typically 5-10 mg) repeated once after 15 min OR
Lorazepam I.V 0.1 mg/kg (usually a 4 mg bolus, repeated once after 10−20 minutes; rate not critical).
Give usual AED medication if already on treatment.
For sustained control or if seizures continue, treat as below.

**Established status**
Phenytoin infusion at a dose of 15–18 mg/kg at a rate of 50 mg/minute OR
Fosphenytoin infusion at a dose of 15−20 mg phenytoin equivalents (PE)/kg at a rate of 50–100 mg PE/minute AND/OR
Phenobarbital bolus of 10–15 mg/kg at a rate of 100 mg/minute followed by infusion at 1-10 mg/kg/hr OR
Sodium valproate 15-25 mg/kg over 30 min followed by infusion at 1 mg/kg/hr for 6 hrs

**Refractory status**

General anaesthesia, with one of:

Propofol (1–2 mg/kg bolus, then 2–10 mg/kg/hour) titrated to effect

Midazolam (0.1–0.2 mg/kg bolus, then 0.05–0.5 mg/kg/hour) titrated to effect

Thiopental sodium (3–5 mg/kg bolus, then 3–5 mg/kg/hour) titrated to effect; after 2–3 days infusion rate needs reduction as fat stores are saturated

1. In the above scheme, the refractory stage (general anaesthesia) is reached 60-90 minutes after the initial therapy.
2. AED therapy must be given in parallel with emergency treatment. The choice of drug depends on previous therapy, the type of epilepsy, and the clinical setting.
3. Any pre-existing AED therapy should be continued at full dose, and any recent reductions reversed.
4. If phenytoin or phenobarbital has been used in emergency treatment, maintenance doses can be continued orally or intravenously guided by serum level monitoring. Other maintenance AEDs can be started also, with oral loading doses.
5. Care needs to be taken with nasogastric feeds, which can interfere with the absorption of some AEDs.
6. Once the patient has been free of seizures for 12−24 hours, then the anaesthetic should be slowly tapered.

**Emergency investigations ▲▲**

Blood gases, glucose, renal, liver function & electrolytes

Full blood count (including platelets), blood clotting,

AED drug levels; 5 ml of serum and 50 ml of urine samples should be saved for future analysis, including toxicology, especially if the cause of the convulsive status epilepticus is uncertain.

Chest radiograph to evaluate possibility of aspiration

Other investigations depend on the clinical circumstances and may include brain imaging, lumbar puncture.

| | |
|---|---|
| AED | Antiepileptic drugs |
| EIAED | Enzyme-inducing AEDs |
| GCSE | Generalised Convulsive Status Epilepticus |
| GTCS | Generalised tonic-clonic seizures |
| JME | Junctional myoclonic epilepsy |
| NEAD | Non-epileptic attack disorder |
| OCP | Oral contraceptive pill |
| SE | Status epilepticus |

**References:**


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**Table - 1**

| Hepatic enzyme inducing AEDs | Non-hepatic enzyme inducing AEDs |
|---|---|
| Carbamazepine | Acetazolamide |
| Oxcarbazepine | Benzodiazepines |
| Phenobarbital | Ethosuximide |
| Phenytoin | Gabapentin |
| Primidone | Lamotrigine |
| Topiramate | Levetiracetam |
| | Tiagabine |
| | Valproate |
| | Vigabatrin |

**Suggested choice of AEDs by seizure types**

| Seizure type | First-line drugs | Second-line drugs | Other options | Drugs to be avoided |
|---|---|---|---|---|
| Primary Generalised Tonic-clonic | Sodium valproate<br>Carbamazepine<br>Phenytoin<br>Lamotrigine<br>Topiramate | Clobazam Levetiracetam<br>Oxcarbazepine | Primidone<br>Clonazepam<br>Phenobarbital | |
| Absence | Ethosuximide<br>Sodium valproate<br>Lamotrigine | Clobazam Clonazepam<br>Topiramate | | Carbamazepine<br>Gabapentin<br>Pregabalin<br>Oxcarbazepine |
| Myoclonic | Sodium valproate<br>Levetiracetam | Clobazam Clonazepam<br>Piracetam Topiramate | Lamotrigine | Carbamazepine<br>Gabapentin<br>Pregabalin<br>Oxcarbazepine |
| Tonic | Sodium valproate<br>Lamotrigine | Clobazam Clonazepam<br>Topiramate<br>Levetiracetam | Primidone<br>Phenobarbital<br>Phenytoin | Carbamazepine<br>Oxcarbazepine |
| Atonic | Sodium valproate<br>Lamotrigine | Clobazam Clonazepam<br>Levetiracetam<br>Topiramate | Phenobarbital<br>Primidone | Carbamazepine<br>Oxcarbazepine<br>Phenytoin |
| Focal with/without secondary generalisation | Carbamazepine<br>Phenytoin<br>Sodium valproate<br>Lamotrigine<br>Oxcarbazepine<br>Topiramate<br>Levetiracetam | Clobazam Gabapentin<br>Pregabalin | Clonazepam<br>Phenobarbital<br>Primidone | |

**Suggested choice of AEDs by epilepsy syndromes**

| Epilepsy syndrome | First-line drugs | Second-line drugs | Other drugs | Drugs to be avoided |
|---|---|---|---|---|
| Childhood or juvenile absence epilepsy | Ethosuximide<br>Sodium valproate<br>Lamotrigine | Levetiracetam<br>Topiramate | | Carbamazepine<br>Gabapentin<br>Pregabalin<br>Oxcarbazepine<br>Phenytoin |
| Juvenile myoclonic epilepsy | Sodium valproate | Levetiracetam<br>Lamotrigine<br>Clobazam<br>Clonazepam<br>Topiramate | | Carbamazepine<br>Gabapentin<br>Pregabalin<br>Oxcarbazepine<br>Phenytoin |
| Infantile spasms (IS) | ACTH/Steroids<br>Vigabatrin** (first line for IS with tuberous sclerosis) | Clobazam<br>Clonazepam<br>Sodium valproate<br>Topiramate | Nitrazepam | Carbamazepine<br>Oxcarbazepine |
| Benign epilepsy with centrotemporal spikes or occipital paroxysms | Carbamazepine<br>Lamotrigine<br>Oxcarbazepine<br>Sodium valproate | Levetiracetam<br>Topiramate | | |
| Severe myoclonic epilepsy of infancy | Clobazam<br>Clonazepam<br>Sodium valproate<br>Topiramate | Levetiracetam | Phenobarbital | Carbamazepine<br>Lamotrigine<br>Oxcarbazepine |
| Lennox-Gastaut syndrome | Lamotrigine<br>Sodium valproate<br>Topiramate | Clobazam<br>Clonazepam<br>Ethosuximide<br>Levetiracetam | | Carbamazepine<br>Oxcarbazepine |
| Landau-Kleffner syndrome | Lamotrigine<br>Sodium valproate<br>Steroids | Levetiracetam<br>Topiramate | | Carbamazepine<br>Oxcarbazepine |
| Myoclonic astatic epilepsy | Clobazam<br>Clonazepam<br>Sodium valproate<br>Topiramate | Lamotrigine<br>Levetiracetam | | Carbamazepine<br>Oxcarbazepine |

Dosages of commonly used AEDs

| AED | Usual daily dose | No. of doses/day |
|---|---|---|
| Carbamazepine | Initial: 100 mg at night (adults); 5 mg/kg (children). Maintenance: 400-1600 mg (adults); 10-20 mg/kg (children). | 2-3 |
| Clonazepam | Initial: 0.25mg (adults); 0.02 mg/kg (children). Maintenance: 0.5-4 mg (adults); 0.1-0.2 mg/kg (children). | 2-3 |
| Ethosuximide | Initial: 250 mg (adults); 10 mg/kg (children). Maintenance: 750-2000 mg (adults); 20-40 mg/kg (children). | 2-3 |
| Gabapentin | Initial: 300 mg (adults); 10 mg/kg (children). Maintenance: 900-3600 (adults); 30-60 mg/kg (children). | 2-3 |
| Lamotrigine | Initial: 25 mg (adults); 0.15 mg/kg (with valproate), 0.6 mg/kg (without valproate) (children). Maintenance: 100-200 mg (adults); 1-5 mg/kg (with valproate), 5-15 mg/kg (without valproate) (children). Adjunctive therapy with valproate: gradual increment in the dose over one month (adults). | 1-2 |
| Levetiracetam | Initial: 500 mg. Maintenance: 1000-3000 mg; 20-50 mg/kg (children). | 2 |
| Oxcarbazepine | Initial: 600 mg (adults); 10mg /kg (children) Maintenance: 1200-2400 mg (adults); 20-40 mg/kg (children) | 2 |
| Phenobarbitone | Initial: 30 mg. Maintenance: 30-180 mg (adults); 3-5mg/kg (children). | 1-2 |
| Phenytoin | Initial: 200-300 mg. Maintenance: 300-400 mg (adults); 5 mg/kg (children) | 1 |
| Topiramate | Initial: 25-50 mg (adults), 0.5-1 mg/kg (children). Maintenance: 200-400 mg (adults); 3-9 mg/kg (children). | 2 |
| Valproate | Initial: 400-600 mg (adults); 10-20 mg/kg (children). Maintenance: 400-2500 mg (adults); 20-40 mg/kg (children under 20 kg); 20-30 mg/kg (children over 20 kg). | 2 |

Common and important side effects of AEDs

| AED | Side effects (acute and chronic) |
|---|---|
| Carbamazepine | Drowsiness, fatigue, dizziness, ataxia, diplopia, blurring of vision, rash and other skin reactions, leucopaenia, and hyponatraemia. |
| Clonazepam | Sedation, drowsiness, ataxia, and blurring of vision. |
| Gabapentin | Drowsiness, dizziness, ataxia, headache, and myoclonus. |
| Lamotrigine | Rash (sometimes severe), dizziness, and somnolence. |
| Levetiracetam | Somnolence, asthenia, dizziness, and headache. |
| Phenobarbitone | Sedation, ataxia, dizziness, and hyperactivity in children. |
| Phenytoin | Ataxia, dizziness, lethargy, sedation, gingival hypertrophy, and hirsutism. |
| Topiramate | Dizziness, ataxia, paraesthesiae, tremor, somnolence, cognitive dysfunction, emotional lability, word- finding difficulties, nephrolithiasis, open angle glaucoma, hypohidrosis (children), and weight loss. |
| Valproate | Drowsiness, tremor, hair thinning and hair loss, menstrual irregularities, weight gain, and thrombocytopaenia. |