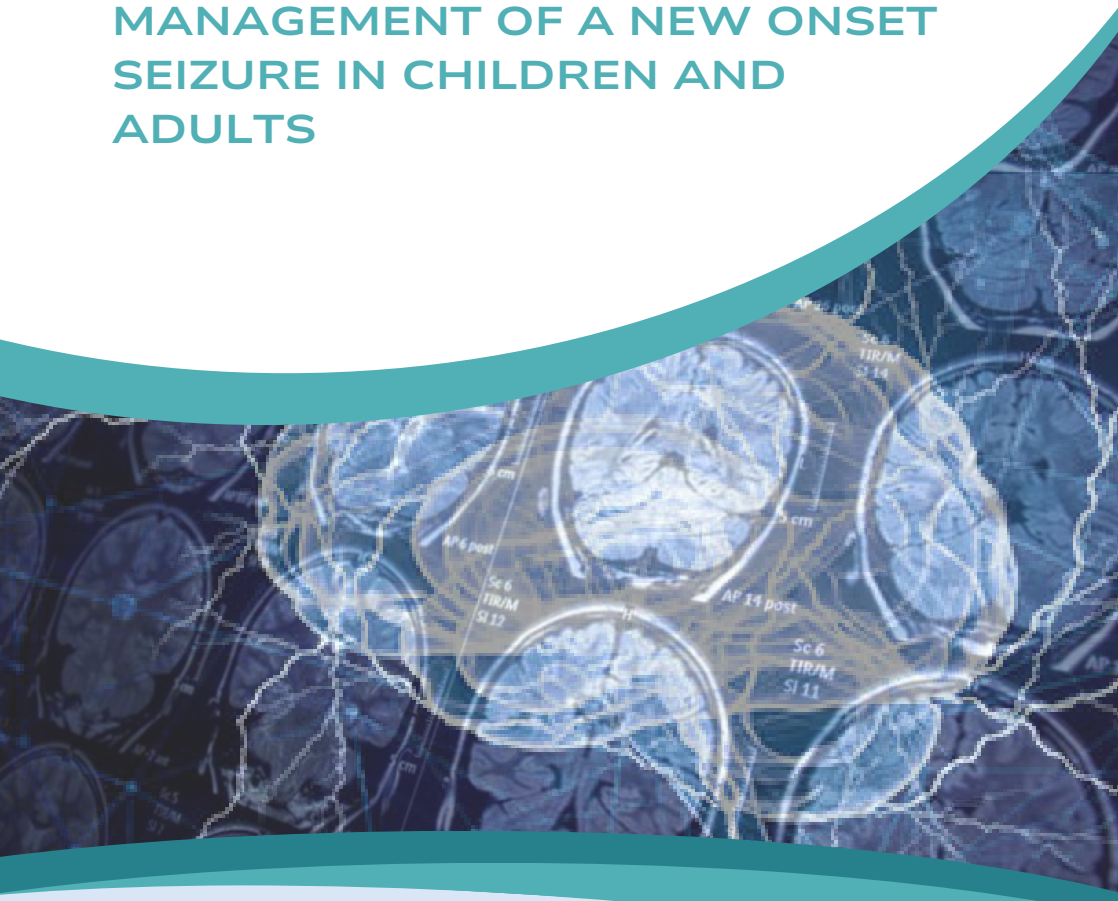
Association of Sri Lankan Neurologists

Epilepsy Association of Sri Lanka

GUIDELINE ON

MANAGEMENT OF A NEW ONSET SEIZURE IN CHILDREN AND ADULTS



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Association of Sri Lankan Neurologists

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ISBN 978-624-6537-00-5

Published by:
Association of Sri Lankan Neurologists

First Edition 2024

Copyediting, cover & book design, book formatting

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Dip. Academic English (Pearson Assured)

Message from the president

Dr Pyara Ratnayake
President
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It is with great pleasure that I write this message to the first seizure management guideline. Epilepsy is one of the commonest neurological diseases and evidence-based management of the first seizure is of importance for optimum care. The guideline takes you through the intricacies of the scientific basis of management decisions. I congratulate the authors for the elegant document which is detailed but user friendly. I thank the reviewers for their valuable inputs and the past presidents Prof Thashi Chang and Dr Darshana Sirisena for the initiative. On behalf of the Association of Sri Lankan Neurologists I hope to see this document used throughout Sri Lanka and hope that this will be the first of many such management guidelines constructed by our members.

About this Document

The guideline included in this document has been developed for use by any medical practitioner engaged in the assessment of patients presenting with suspected seizures. It addresses the approach to be taken for an adult and a child separately. This guideline is based on the best possible evidence and guidelines laid down by the International League Against Epilepsy (ILAE) and reflect the highest standard of care expected in the management of cases of suspected seizures/ epilepsy. This document was developed with the consensus of the contributors and has been endorsed by the Association of Sri Lankan Neurologists. It provides recommendations only. This guideline is up to date as of August 2023.

*Section on **First seizure evaluation and referral pathway** relates to the requirements to be fulfilled/ assessments to be done by in-hospital medical officers evaluating patients experiencing their first seizure.

Acknowledgements

We would like to thank the following individuals for their contribution in the development of this document:

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List of abbreviations

AE	Adverse Events
ASM	Antiseizure medication
BECTS	Benign Epilepsy with Centrotemporal spikes
CMV	Cytomegalovirus
CNS	Central Nervous System
CT	Computed tomography
ECG	Electrocardiogram
EEG	Electroencephalogram
FBC	Full Blood Count
GCS	Glasgow Coma Scale
HIV	Human Immunodeficiency Virus
IFCN	International Federation of Clinical Neurophy
ILAE	International League Against Epilepsy
LFT	Liver Function Tests
LP	Lumbar puncture
MAE	Myoclonic Astatic Epilepsy
MRI	Magnetic Resonance Imaging
PNES	Psychogenic Nonepileptic Seizures
SeLECTS	Self-Limited Epilepsy with Centrotempc
SeLFE	Self-Limited Focal Epilepsies of childhoo
SJS	Steven Johnson Syndrome
TEN	Toxic Epidermal Necrolysis
U&E	Urea & Electrolytes

Contents

Preface

Approximately 8% to 10% of the population will experience a seizure during their lifetime. Only about 2% to 3% go on to develop epilepsy, which is defined as the tendency to experience recurrent unprovoked seizures. The overall prevalence of epilepsy is 1% of the adult population and 0.4 – 0.8% in children.

When a patient presents with the first seizure, the question whether or not to start anti-seizure medication (ASM) is often considered. To decide on the initiation of treatment, several factors need consideration. These include confirmation of diagnosis of epilepsy, understanding risk of seizure recurrence, side effects of medication, patient circumstances including preference and cost. It is important to note that early administration of ASM reduces the risk of seizure recurrence on the short-term. However, it does not affect the prognosis for the development of epilepsy.

Clinical variables that increase the risk of seizure recurrence include a prior brain insult, an EEG with epileptiform abnormalities, a significant brain-imaging abnormality, and having a nocturnal seizure. Since the International League Against Epilepsy (ILAE) definition of epilepsy now facilitates identifying epilepsy even after a single seizure (if the risk of recurrence is determined as > 60% over a 10-year period), careful review of risk factors may enable initiation of ASM treatment in those indicated.

This guideline is written to aid any medical practitioner engaged in the assessment of patients presenting with suspected seizures to make a correct diagnosis of epilepsy and plan the appropriate initial investigations. It emphasizes the diagnostic framework described by the ILAE and discusses the role of electroencephalography and neuro-imaging in the initial evaluation of a patient with epilepsy. Finally, an outline on the choice of most appropriate therapy for a newly diagnosed patient with epilepsy is briefly discussed.

Definitions

Seizure

An epileptic seizure is a transient occurrence of signs and/or symptoms that occur due to an abnormal, excessive and synchronous neuronal activity in the brain[1]. These signs take the form of motor, sensory and autonomic manifestations occurring in isolation or in any combination. The clinical characteristics of a seizure reflect the area of the brain that is abnormally stimulated. This may include area of onset as well as the extent and pattern of propagation of the electrical activity during the seizure.

Epilepsy

Epilepsy is a disease characterized by an enduring predisposition to generate epileptic seizures. It is a disease of the brain. It is defined as fulfilment of any one of the following three conditions[2]:

1. At least two unprovoked (or reflex) seizures occurring >24 h apart

2. One unprovoked (or reflex) seizure and having a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years*

For example, if a person experiences a seizure after a period of 1 month following a stroke, that person's 10-year risk of seizure recurrence is > 60%. Therefore, in such a case even after a single seizure, such a person can be diagnosed as having epilepsy.

3. Diagnosis of an epilepsy syndrome

E.g., Self-limited epilepsy with centrotemporal spikes (SeLECTS) which was previously known as Benign epilepsy with centrotemporal spikes (BECTS). This epileptic syndrome is characterized by brief, focal aware hemifacial motor seizures with associated somatosensory symptoms, which have a tendency to evolve into bilateral tonic-clonic seizures. EEG shows high-voltage centrotemporal spikes often followed by a slow wave. A single characteristic seizure with the typical EEG would be adequate to diagnose this benign childhood epilepsy syndrome.

**Epilepsy is considered resolved in individuals who had an age-dependent epilepsy syndrome but are now past the applicable age (e.g., Self-Limited Focal Epilepsies of Childhood or (SeLFE) or those who have remained seizure-free for the last 10 years, without seizure medicines for the last 5 years.*

Acute symptomatic seizure (provoked seizure or reactive seizure)

It is a seizure that occurs in close temporal association with a documented brain insult such as seizures occurring within one week of stroke, traumatic brain injury, anoxic encephalopathy, central nervous system infection, intracranial surgery etc. In addition, acute symptomatic seizures may occur with a systemic insult such as in the presence of severe metabolic derangements documented by specific biochemical abnormalities within 24 hours such as hypoglycaemia, hyponatraemia, or hypocalcaemia or haematological abnormalities, drug or alcohol intoxication and or withdrawal or exposure to well defined epileptogenic drugs[4].

Remote symptomatic (unprovoked) seizure

A remote symptomatic seizure is defined as a seizure occurring in the presence of pre-existing brain injury that had occurred beyond the interval estimated for the occurrence of acute symptomatic seizures[4]. In other words, it is a seizure that occurs in a person with an old CNS insult, especially those known to increase the risk of developing epilepsy like stroke, traumatic brain injury, CNS infection, etc. e.g., a seizure that occurs 6 months after a traumatic brain injury or a stroke.

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Introduction to this guide

Approximately 8% to 10% of the population will experience a seizure during their lifetime. Only about 2% to 3% go on to develop epilepsy[1]. Thus, only a minority of those who experience a single seizure would have recurring seizures i.e. epilepsy (see definitions).

Epilepsy is one of the most common chronic neurological diseases in the world[1]. It affects 1% of the adult population[2] and 0.4 – 0.8% of children[2]. Eighty percent of the adults with epilepsy live in low- and middle-income countries such as Sri Lanka[1]. Men and women are equally affected. Prevalence of epilepsy is highest in childhood, particularly during the first year of life[2]. The epilepsy prevalence in Sri Lankan children is reported as 5.7 per 1000 children in the 0-16 age group[3]. There is a greater expression of genetic epilepsies during this age group. Most childhood epilepsies carry a better prognosis. Thus, it is important that children presenting with a new-onset seizure are carefully evaluated for the diagnosis of epilepsy, its cause and if possible, identification of the underlying epilepsy syndrome.

The first seizure raises the question whether or not to start ASM. Initiation of ASM should be considered carefully since epilepsy is usually a chronic disorder necessitating long-term therapy. However, certain epilepsy syndromes such as SeLECTS seen in children are self-limiting, and hence may not necessarily require long term treatment. ASMs do not alter long term remission. There is no convincing evidence that ASMs impact human epileptogenesis (the process by which a brain network that was previously normal is functionally altered toward increased seizure susceptibility) but they have an impact on ictogenesis (the processes of transition from the interictal state to a seizure). Therefore, it is the risk of recurrence that often supports or defers commencement of ASM treatment when patients present with a new onset seizure.

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Approach to first seizure

A seizure presents as a provoked or unprovoked paroxysmal event. However, not all paroxysmal events are seizures. Correct diagnosis is necessary to provide the most appropriate treatment. If the event is a seizure, the seizure type and associated clinical, electroencephalographic, and neuroimaging findings assist in determining the risk of seizure recurrence and the need to commence ASM.

A stepwise approach is often required when evaluating a patient who presents with his/her first ever seizure. This guideline focuses on the following questions that need to be answered during the evaluation:

1. Is it a seizure?
2. What is the type of seizure?
3. Is it epilepsy?
4. What kind of epilepsy is it?
5. What is the cause of the epilepsy?
6. What are the investigations needed when evaluating a new onset seizure?
7. How should this epilepsy be treated?

Is it a seizure?

The initial step in evaluation of a possible seizure is to differentiate the event from seizure mimics. Some common mimics include syncope, transient ischemic attacks, migraine with auras, paroxysmal movement disorders, sleep disorders, intracranial hypertension, and psychogenic non-epileptic seizures (PNES).[1] Specifically in children, these include complex motor tics, stereotyped behaviours, gastroesophageal reflux disease (Sandifer syndrome), breath-holding spells[2] etc. A reliable account from a witness is essential to define the event semiology, since a patient suspected of having suffered a seizure is unable to provide a reliable history, this may be due to altered consciousness during the seizure or the patient being a young child who is often unable to elaborate his/her experience. It is important to gauge information with regards to details on what occurred prior, during and after the event and in the modern-day smartphone video capturing the event would improve the diagnostic accuracy.

Table 1 highlights some of the clinical features that differentiate a generalised seizure from syncope and PNES, two common differential diagnoses for seizures seen in adults and children[1].

Table 1: Features that help to differentiate a generalised epileptic seizure from syncope and psychogenic non-epileptic seizures (children, adolescents and adults)

Clinical features suggesting a generalized seizure	Clinical features suggesting syncope	Clinical features suggesting PNES
• Cyanosis, frothing at the mouth, and the absence of pallor during the event • Ictal cry during the tonic phase • Stertorous breathing • Lateral tongue bite and posterior shoulder dislocation (high specificity) • Confusion, sleepiness, aching muscles following event	• Light headedness, diaphoresis, nausea, or diminution of hearing and vision prior to event • Onset during prolonged standing or sitting or triggered by emotion. • Rapid recovery and orientation **Tonic and myoclonic muscle activity, eye deviations, oral and limb automatisms and vocalizations may be seen during convulsive syncope. However, they are of shorter duration, on average lasting less than thirty seconds, and there is relative lack of post-event confusion.*	• Long-lasting events (at times hours) • Gradual onset or termination • Fluctuating course • Side to side (head or body) movements, compared to tonic-clonic movements pelvic thrusting*, opisthotonic posturing, stuttering, forced persistent eye closure > 50% of the duration of seizure • Crying or yelling, weeping during the event • Asynchronous jerks* **Pelvic thrusting and asynchronous jerks can occur in frontal lobe seizures as well*

**Note: urinary incontinence does not reliably distinguish seizures from syncope or PNES*

The presence of a single feature mentioned above on its own will not necessarily predict the specified diagnosis.

Table 2: Seizure mimics encountered in neonates and infants

Children are known to have a range of seizure mimics. These differ according to the age of the child. Some are more frequent in early life while others are more frequent in the older age.

Common seizure mimics in babies include jitteriness, benign sleep myoclonus, breath-holding spells, shuddering and gastroesophageal reflux[2].

Jitteriness	Benign sleep myoclonus	Gastroesophageal reflux	Shuddering
Jitteriness is the most common movement disorder in the neonatal period. This is composed of recurrent tremors in the extremities. They occur with stimulation and stop when the affected limb is held or movement with slight flexion of the extremity.	Consists of myoclonic jerks that involve limbs, trunk, or the whole body, occurring in clusters during quiet sleep. It disappears during wakefulness. Typically occurs from birth to about 6 months of life. They are brief, repeatedly occurring over 10 to 20 seconds, though may persist even up to 30 minutes.	Infants may stiffen, appear limp, look startled, or arch in response to gastro-oesophageal reflux. The spasmodic torsional dystonia with arching of the back and opisthotonic posturing is known as Sandifer syndrome.	This is a paroxysmal, brief, motor event, which usually begin in the first year of life. They manifest as shivering of the head, shoulders, and or the trunk. They recur frequently throughout the day. Nursing, eating, and or excitement may initiate the attacks.

Table 3: Seizure mimics seen in toddlers and young children

Breath-holding spells, tics, stereotypies and parasomnias are some of the seizure mimics experienced in the toddler age group and in young children[3].

Breath-holding spells	Tics	Stereotypies	Parasomnias
Classical breath holding spell is a sequence of crying followed by apnoea, cyanosis, loss of consciousness, and tonic posturing. Most commonly seen between 6 to 18 months of age but can occur even in older children.	Sudden, brief, intermittent, involuntary or semi-voluntary movements mainly in the face, neck and upper limb or sounds; child is often able to suppress tics.	Repetitive sometimes self-stimulating behaviours, or ritual movements including clapping, shaking arms, and shaking the head.	Sleep disorders that occur during the first 1/3 of night sleep.Confusional arousal, night terrors, and sleepwalking are the most common parasomnias in childhood.

Key messages

- The first step in evaluating a new onset seizure is to confirm if it is indeed a seizure by differentiating the event from seizure mimics.
- The spectrum of seizure mimics differs according to age.
- A detailed history of the index event from the patient and eyewitness is imperative for accurate diagnosis.

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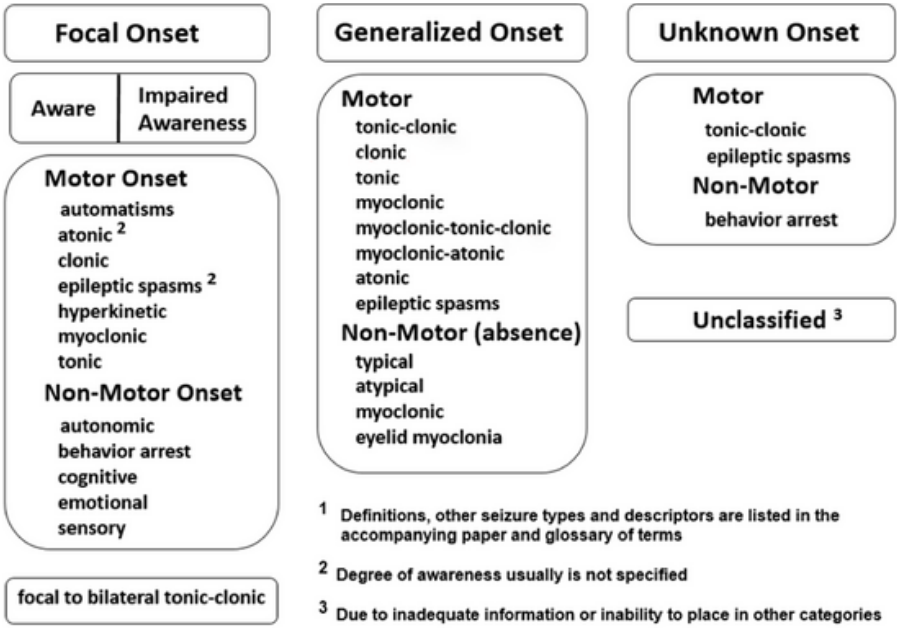
What is the type of seizure?

When the question of whether you are dealing with a seizure is answered, the next step in evaluation is understanding the type of seizure.

The ILAE has provided a classification for types of seizures, with initial categorisation into focal, generalized onset or unknown onset seizures[1,2] (see figure below on seizure classification). This classification is based on the localization of (signs/symptoms) at the onset of the seizure. The next step in the classification is understanding the different types of seizures based on semiological features. The frame work for this was outlined by the ILAE in their position paper on operational classification of seizure types and is shown in figure 1 below.

Figure 1: ILAE 2017 Classification of Seizure Types Expanded Version[1]



Descriptors of seizure types

Generalized seizures:

Generalized epileptic seizures are conceptualized as originating at some point within, and rapidly engaging, bilaterally distributed networks[3]. Such bilateral networks do not necessarily include the entire cortex. Generalized seizures can be asymmetric. All generalized seizures cause loss of awareness. Generalized-onset seizures are divided into motor and non-motor (absence) seizures. They can be classified in the following way:

(a) Motor seizures

- **Tonic-clonic seizure**
  Bilateral tonic followed by clonic convulsions.
- **Clonic**
  Symmetrical or asymmetrical jerking of the same group of muscles.
- **Tonic**
  An increase in muscle contraction, lasting from a few seconds to some minutes[4].
- **Myoclonic**
  Sudden, brief contractions of muscles resulting in dropping or spilling things and/or falls. Generalized myoclonic seizures can occur in isolation or in conjunction with tonic or atonic activity[2]
  - Myoclonic tonic-clonic
  - Myoclonic atonic (Brief jerking of limbs or trunk, followed by a limp drop). Commonly seen in Myoclonic-Astatic Epilepsy (MAE) also called Doose syndrome.
- **Atonic**
  Sudden loss of muscle tone lasting a few seconds, which can affect the head, body, arms and legs. When leg tone is lost during a generalized atonic seizure, the patient falls on the buttocks or sometimes forward onto the knees and face[2].
- **Epileptic Spasm**
  An epileptic spasm is a sudden flexion, extension or mixed flexion-extension of proximal and truncal muscles, lasting 1-2 seconds i.e., longer than a myoclonic jerk (which lasts milliseconds) but not as long as a tonic seizure (which lasts > 2 seconds). Spasms may be bilaterally symmetric, asymmetric, or unilateral, depending on whether they are generalised onset or focal onset.

(b) Non-motor Generalized seizures

- **Absence**
  A sudden interruption of activities accompanied by a blank stare with occasional deviation of the eyes lasting a few seconds to half a minute with subsequent rapid recovery. Absence seizures are considered atypical when they are associated with changes in tone that are more pronounced than in typical absence or when the onset or cessation is not abrupt.

- Typical Absence
- Atypical Absence
- Myoclonic absence
- Eyelid myoclonia

For further information with regards to the above visit: https://www.epilepsydiagnosis.org/syndrome/epilepsy-syndrome-groupoverview.html

Focal seizures:

Focal epileptic seizures are conceptualized as originating within networks limited to one hemisphere[3]. They may occur either with retained awareness or with altered awareness.

The first group, classified as focal aware corresponds to the former term of "simple partial seizure". The second group classified as impaired awareness corresponds to the former term "complex partial seizure". These thereafter should be classified into different types of seizures based on their manifestation, as outlined below. These reflect the earliest prominent sign or symptom other than awareness[2].

These are descriptors of focal seizures:

- **Focal motor onset include:** atonic (focal loss of tone), tonic (sustained focal stiffening), clonic (focal rhythmic jerking), myoclonic (irregular, brief focal jerking), or epileptic spasms (focal flexion or extension of arms and flexion of trunk)[1]. Other less obviously focal motor behaviours include hyperkinetic activity (agitated thrashing or leg pedalling movements) and automatisms (non-purposeful, stereotyped, and repetitive behaviours that commonly accompany focal impaired awareness seizures) Examples for oral automatisms include, lip smacking, chewing and swallowing and examples for manual automatisms include, picking, fumbling and patting.

- **Focal non-motor onset** seizures involve **autonomic** manifestations (gastrointestinal sensations, a sense of heat or cold, flushing, piloerection, palpitations, sexual arousal, respiratory changes), **sensory** manifestations (somatosensory, olfactory, visual, auditory, gustatory, hot–cold sense, or vestibular sensations), **emotional** manifestations (fear, anxiety, agitation, anger, paranoia, pleasure, joy, ecstasy, laughing also known as gelastic, or crying also known as dacrystic) or **cognitive** manifestations such as déjà vu (feeling as though one has lived through the present situation before), jamais vu (the experience of being unfamiliar with a person or situation that is actually very familiar), hallucinations, illusions and forced thinking[1]. These experiential manifestations of seizures correspond to the concept of an aura. Furthermore, if a person has a behavioural arrest as the

predominant aspect of the entire seizure this too is classified as a non-motor onset seizure.

If both motor and non-motor symptoms occur at the start, usually the motor symptoms dominate unless the sensory symptoms are prominent.

- **Focal to bilateral tonic-clonic seizure** corresponds to the term "secondarily generalized seizure". This is reflective of a propagation pattern of seizure activity. In the new classification, "bilateral" is used for propagation patterns of seizures and "generalized" for seizures of generalized onset.

Seizures of unknown onset:

These can be categorized as motor, including tonic–clonic, non-motor, or unclassified. The term unclassified comprises both seizures with patterns that do not fit into the other categories or seizures presenting insufficient information to allow categorization.

Key messages

- Classify the seizure/s in to either focal onset, generalised onset or unclassified onset according to the 2017 ILAE seizure classification.

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Is it epilepsy?

When confronted with a patient's first seizure, two issues have to be addressed. First is to identify whether they are provoked or unprovoked seizures. Provoked or acute symptomatic (see definitions) seizures such as alcohol withdrawal seizures or early post-traumatic seizures (in adolescents and adults) have to be identified[1]. Similarly, the commonest acute symptomatic seizure seen in childhood is a febrile seizure[2]. This is a seizure activity precipitated by fever occurring in relation to an infection which is unrelated to the central nervous system or related to any metabolic derangement complicating the underlying infection. This occurs during a specific window period of life.

Secondly, the seizure in question may not be the first seizure at all because many patients do not remember or recognize previous seizures, such as auras, automotor (seizures with automatisms) dialeptic seizures (reduced responsiveness or awareness and with subsequent at least partial amnesia of the event) in temporal lobe epilepsy or myoclonic seizures in generalized epilepsy[3]. Therefore, evidence of previous unrecognized seizures should be sought by a careful history, i.e., by asking about previous spells of unresponsiveness, myoclonic jerks, or stereotyped sensory experiences suggestive of auras. If such previous seizures do exist, even though the presentation is seemingly of a "first ever seizure", a diagnosis of epilepsy needs to be made (operational definition – criterion 1)[4] and commenced on therapy due to seizure recurrence risk of >60% during the following 10 years (which means that > 60% of individuals will experience a further seizure over the next 10 years).

If the seizure is a true single seizure the risk of recurrence needs to be gauged to decide whether the patient can still be diagnosed to have epilepsy in line with operational definition - criterion 2[4].

The risk for seizure recurrence increases in certain clinical circumstances (especially in adults). These include[5]:

1. A prior brain lesion or insult (stroke, trauma, CNS infection, cerebral palsy, and cognitive developmental disability) beyond the period of causing acute symptomatic seizures causing the seizure
2. EEG with epileptiform abnormalities6 (patients with generalized spike wave epileptiform abnormalities are more likely to have seizure recurrence, however focal slowing and focal spike and wave discharges are considered abnormal as well)

3. A significant brain-imaging abnormality which has a higher risk (> 60%) for recurrence of seizures
4. Nocturnal seizure[5]

Thus, if one or more of the above criteria are fulfilled the recurrence risk may qualify the patient to be regarded as having epilepsy (operational definition – criterion 2)[4]. A remote symptomatic cause, such as stroke, traumatic brain injury or infection can be associated with a 10-year recurrence risk of over 60%[7,8]. Please note, not all instances mentioned above qualify for a diagnosis of epilepsy and are often dependent on the identified aetiology. For example, structural lesions such as mesial temporal sclerosis or cortical malformations such as focal cortical dysplasias may not carry a risk of > 60% as there is lack of data on recurrence risk. In such cases we may refrain from diagnosing epilepsy. A patient with a single generalised seizure with an EEG having generalised epileptiform discharges will qualify for a diagnosis of epilepsy as the most likely diagnosis in such a case would be an idiopathic generalised epilepsy (operational definition - criterion 3, see below).

Clinical variables that are not consistently associated with an increased seizure recurrence risk after an unprovoked first seizure in adults include[5]:

1. Patient's age
2. Sex
3. Family history of seizures
4. Seizure type
5. Presentation with status epilepticus or multiple (2 or more) discrete seizures within 24 hours with recovery in between

The diagnosis of an epilepsy syndrome (operational definition – criterion 3)[4] depends on a cluster of common electro-clinical characteristics (i.e., age, seizure types, EEG characteristics). Such syndromes may have a typical age of seizure onset, specific seizure types and EEG characteristics, specific prognosis and often other features which when taken together allow the diagnosis of a specific epilepsy syndrome diagnosis. The following are common epilepsy syndromes:

1. Infantile Epileptic Spasms Syndrome (previously known as West syndrome)
2. Lennox-Gastaut syndrome
3. Self-limiting Epilepsy with Centro-temporal Spikes (SeLECTS)
4. Childhood absence epilepsy
5. Juvenile absence epilepsy
6. Juvenile myoclonic epilepsy
7. Epilepsy with generalized tonic clonic seizures alone

It is important to note that even after a single seizure an epilepsy syndrome could be diagnosed especially with benign childhood epilepsies (i.e., Self-limiting Epilepsy with Centro-temporal Spikes (SeLECTS) or “Rolandic” epilepsy, Panayiotopoulos syndrome (Self-Limited Focal Epilepsies of Childhood or (SeLFE)), late-onset idiopathic childhood occipital epilepsy (Gastaut type), and idiopathic photosensitive occipital lobe epilepsy of childhood). These epilepsies have a typical seizure type/ semiology, age of onset and EEG pattern which make diagnosing such a syndrome even after a single seizure possible.

Another group of epilepsies which occur when exposed to a particular trigger or stimulus are grouped as reflex epilepsy syndromes. The trigger is considered simple if the seizures occur in response to sensations such as touch, light or movement. On the other hand, they may be complex when they occur in relation to activities like writing, reading, thinking about a specific activity etc. Simple reflex seizures occur immediately on exposure whereas the complex reflex seizures may take a short while after exposure. They are mostly related to genetic inheritance of a causative gene or occur in relation to other types of epilepsies where photic sensitivity is an associated feature.

Further information on epilepsy syndromes can be found at: https://www.epilepsydiagnosis.org/syndrome/epilepsy-syndrome-groupoverview.html

Key messages

- Identify whether the new onset seizure is provoked or unprovoked.
- Confirm whether there have been previous seizures gone unrecognized (e.g., auras, myoclonic and dialeptic seizures).
- Gauge the risk of recurrence if it is a true new-onset seizure.
- Assess whether the new onset seizure qualifies as epilepsy based on criterion 2 of the ILAE diagnostic criteria.

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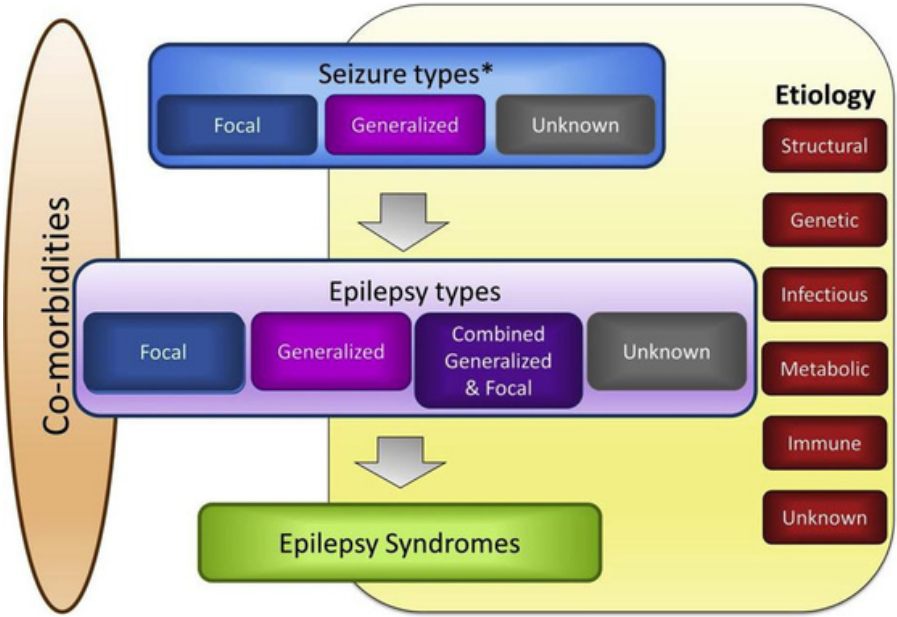
What kind of epilepsy is it?

Once epilepsy has been diagnosed it is important to classify the seizures and epilepsy, as this has implications on management.

The latest ILAE Classification in 2017 of the epilepsies is a multilevel classification. The starting point of this framework is classification of the seizure type[1]. The second level is to classify the epilepsy type into the following: focal, generalized, combined generalized and focal and unknown (in cases of inadequate data) based on the seizure type (see Figure 2 below).

Figure 2: ILAE Epilepsy classification framework[1]



Key messages

- Once epilepsy is confirmed, classify the epilepsy using the ILAE epilepsy classification framework.
- Classify the epilepsy into focal, generalized, combined or unknown epilepsies

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1. Scheffer IE, Berkovic S, Capovilla G, et al. ILAE classification of the epilepsies: Position paper of the ILAE Commission for Classification and Terminology. Epilepsia. 2017;58(4):512–521. doi:10.1111/epi.13709

What is the cause of epilepsy?

Once the diagnosis of epilepsy is established, the clinician should aim to determine the aetiology of the patient's epilepsy[1]. Six aetiologic groups have been recognized and include the following:

(1) Structural causes

Structural causes of epilepsy can be recognised via a range of brain imaging investigations. The anatomical abnormality needs to relate directly to the symptoms and signs of the seizures, since many people without epilepsy may have abnormal brain imaging. The past history may be a useful contributory factor, e.g., a previous head injury, stroke, tumour, birth injury, brain infection, etc., which may be associated with the particular type of seizures under scrutiny.

(2) Genetic causes

The concept of genetic epilepsy is that the epilepsy is, as best as understood, the direct result of a known or presumed genetic defect(s) in which seizures are the core symptom of the disorder. Examples include SCNIA mutation and Dravet syndrome (seen in early childhood).

(3) Infectious causes

An infectious aetiology refers to a patient with epilepsy, rather than with seizures occurring in the setting of acute infection such as meningitis or encephalitis. Common examples in specific regions of the world include neurocysticercosis, tuberculosis, HIV, cerebral malaria, subacute sclerosing panencephalitis, cerebral toxoplasmosis, and congenital infections such as Zika virus and cytomegalovirus. These infections sometimes have a structural correlate. For example, CNS infection with CMV after 20 weeks of gestation can cause malformations of cortical development (including polymycrogyria and schizencephaly), and intracranial calcification in the developing brain.

(4) Metabolic causes

There are a number of unusual and complicated disorders involving the production or

breakdown of natural substances in body cells that are also associated with the development of epilepsy. Examples include disorders of amino acids and organic acids, cofactor deficiency and disorders of energy homeostasis. These are often found in early infancy or childhood.

(5) Immune causes

Over the last 10 to 15 years, there has been increasing recognition that autoimmune or inflammatory conditions can cause epilepsy. When this is the underlying cause, treatment requires targeted immune therapy, in addition to anti-seizure medications.

(6) Unknown causes

Unknown is meant to be viewed neutrally and to designate that the nature of the underlying cause is as yet unknown; it may have a fundamental genetic defect at its core or it may be the consequence of a yet unrecognized disorder.

Key messages

- Classify the aetiology of the epilepsy to one of the following categories, i.e. structural, genetic, infective, metabolic, immune, and unknown.

References

1. Scheffer IE, Berkovic S, Capovilla G, et al. ILAE classification of the epilepsies: Position paper of the ILAE Commission for Classification and Terminology. Epilepsia. 2017;58(4):512–521. doi:10.1111/epi.13709.

What are the investigations needed when evaluating a new onset seizure?

(1) Brain imaging

In the setting of new onset seizures brain imaging should be considered for:

- The identification of CNS insults responsible for acute symptomatic seizures such as: intracranial haemorrhage, ischemic stroke, cortical vein thrombosis, posterior reversible encephalopathy syndrome, infectious encephalitis and meningitis, paraneoplastic and non-paraneoplastic autoimmune limbic encephalitis
  or
- The identification of underlying aetiology and prediction of risk of recurrence in remote symptomatic seizures thus aiding in the diagnosis of epilepsy.

In the second instance; an unprovoked first seizure due to remote symptomatic aetiology, imaging should be considered as part of routine evaluation in adults (N.B. imaging however is not indicated in cases where there is clear evidence of an electro-clinical epilepsy syndrome in which imaging is expected to be normal such as in idiopathic/ genetic generalized epilepsy). However, in children, it is indicated in certain circumstances only[1].

(a) Computed Tomography Scan

In adults: This is often the recommended first imaging tool used in the acute setting because it is widely available and can be performed quickly[2]. Furthermore, it is also useful in the evaluation of patients who cannot tolerate an MRI examination or who have contraindications to MRI. An abnormal neurologic examination or a focal seizure onset are predictive of an abnormal CT study[1]. Emergent CT brain scan must be considered in adults with a first seizure with the red flag signs that may suggest an acute symptomatic aetiology (figure 3 in page 28). A non-contrast CT is deemed adequate for this purpose in most cases, as IV contrast is of little benefit and does not definitively improve the sensitivity of CT for emergent findings[3].

In unprovoked new-onset seizures (non-acute setting) CT may be reasonable in

patients without access to MRI or in cases where MRI is contraindicated or in the patient who is intolerant to MRI. It can be useful in excluding pathologies that need urgent intervention (E.g.: tumours). It is important to note that, only 10% of patients have abnormalities revealed by CT imaging[4] and it is less sensitive than MRI which has a detection rate of 30%). Furthermore, the presence of a history of prior CNS insult or focal slowing or focal epileptiform abnormalities on EEG, predict an epileptogenic lesion being identified by MRI when CT is normal[5]. However, CT is more specific than MRI in the depiction of calcification, which is relevant in populations at risk of calcified lesions, such as vascular malformations, including cavernous malformations[2].

(b) Magnetic Resonance Imaging

MRI is clearly superior to CT in detection of epileptogenic abnormalities in a patient with first ever unprovoked seizure, particularly mesial temporal sclerosis and malformations of cortical development[5]. MRI detects a lesion not evident on CT in 1 of 8 patients. However, an undetected lesion in CT being a tumour is seen only in approximately 1 out of 67 patients[5]. Wherever possible, Magnetic Resonance Imaging (MRI) is the preferred method of neuroimaging in adults (due to its higher detection rate[4]) presenting with a new onset remote symptomatic seizure (unprovoked), although MRIs may not be readily available.

Neuroimaging in children: In children the need for urgent imaging may be different. Those with a clinical history and EEG findings consistent with an idiopathic genetic epilepsy (genetic generalized epilepsy), such as childhood absence epilepsy, juvenile absence epilepsy, juvenile myoclonic epilepsy, do not require brain imaging. Similarly, those presenting with self-limiting focal epilepsies of childhood do not require imaging, unless there is a doubt on the diagnosis during follow up.

Brain imaging should, however, be performed in children who have had two or more afebrile epileptic seizures and who do not have the clinical or EEG features of an idiopathic epilepsy, mentioned above. There are two questions regarding imaging; what modality of imaging is recommended and what is the timing of such imaging? Although MRI is superior to CT in demonstrating subtle brain developmental abnormalities, this choice will be influenced by availability of MRI and the need for a general anaesthetic for young children during the procedure. As for the timing, although neuroimaging abnormalities occur in up to one-third of children with a first afebrile seizure, only 2% will demonstrate a clinically significant abnormality that will influence immediate management. It is important to note that seizures are an uncommon presenting symptom of a brain tumour in children.

Therefore, routine brain imaging (CT or MRI) in the emergency department following

the first afebrile seizure in a child is not usually warranted. However, emergency neuro-imaging should be considered in a child in the following instances: emergent head CT for patients with risk factors for abnormal neuro-imaging e.g., in paediatric age groups, especially children under the age of six months, those with a focal seizure, a new focal deficit, hydrocephalus, recent cerebrospinal fluid shunt surgery, neuro-cutaneous disorders, persistent altered mental status or recent head trauma, presenting with afebrile status epilepticus, focal neurological signs that persist for several hours, history of cancer or on anticoagulation or not returning to baseline within several hours of the seizure[6].

(2) Electroencephalography (EEG)

The EEG is considered as part of the neuro-diagnostic evaluation of an adult or a child with an apparent **unprovoked first seizure** because it has value in determining the risk for seizure recurrence[1'4]. The EEG may be arranged on an elective outpatient basis, unless there is a concern for non-convulsive status epilepticus often in the acute setting in which **provoked seizures** are common. Epileptiform abnormalities on the EEG may be useful in confirming that the event was a seizure; however, an **EEG abnormality by itself is not sufficient to make a diagnosis of an epileptic seizure, nor does its absence rule out a seizure[7].** An EEG is necessary to determine many of the epilepsy syndromes[4]. The syndromic diagnosis may be helpful in determining the need for specific investigations including imaging studies, specific ASM and predicting the prognosis.

An EEG is useful in predicting the prognosis of recurrence of seizures[4]. The abnormalities are of two types: **epileptic discharges** and **focal background slowing**. An epileptic discharge can be generalized or focal. Approximately 12–50% of adults and 18–56% of children with a single unprovoked seizure have epileptiform abnormalities on EEG[8]. **Generalized epileptiform discharges** confer the **highest risk of recurrence**. Both epileptiform discharges and focal background slowing/paroxysmal slow wave events have been shown to predict the risk of recurrence of seizures, with generalized epileptic discharges offering the greatest predictive ability[9'10].

An EEG done within 24 hours of the seizure is most likely to show abnormalities. One should be aware that some abnormalities which are transient such as postictal slowing must be interpreted with caution[11]. However, transient focal slowing may be of a lateralising value.

If a standard EEG is normal, repeated standard EEGs **should not** be used in preference to sleep or sleep-deprived EEGs[9]. A sleep EEG is best achieved through sleep deprivation. A sleep EEG increases the yield of detecting epileptiform discharges in

both children and adults[9]. EEG that includes both wakefulness and sleep should be recommended as it detects a high proportion of abnormalities.

N.B: An EEG should not be performed in the case of probable syncope because of the possibility of a false-positive result[11].

(3) Other Tests

Adults: Routine screening of patients after new-onset seizures for hypoglycaemia, hypocalcaemia, uraemia, drug intoxication, and hyponatremia is recommended for patients seeking care in the emergency setting in which a higher frequency of acute symptomatic seizures occur and should be requested when clinically indicated[1,12]. Cut off values which can cause acute symptomatic seizures are: serum glucose < 36 mg/dL (2 mmol/L) or > 450 mg/dL (25 mmol/L), serum calcium < 1.2 mmol/L (5 mmol/L), creatinine > 10 mg/dL (884 µmol/L) and serum sodium < 115 mg/dL (5 mmol/L)[13].

Routine screening for the above conditions in the outpatient clinic–based setting is of limited utility[14] and thus not recommended unless there is a strong clinical indication.

Seizure-like attacks with a cardiovascular cause may be misdiagnosed as epilepsy. A 12-lead electrocardiography (ECG) should be performed in adults with a suspected seizure in cases of diagnostic uncertainty[1].

If there is persistent (cause unknown) alteration of mental status or failure to return to baseline following a seizure, or in anyone with meningeal signs and fever, a lumbar puncture (LP) should be performed. Imaging should precede the LP to exclude any cause of raised intracranial pressure in which case an LP would be contra-indicated[1].

Children: Laboratory tests should be ordered based on individual clinical circumstances[14] based on history and/or clinical findings. The minimal blood tests required include a complete blood count, serum blood glucose, electrolytes and a blood gas, though in children such disturbances remain rare[14]. In any child with features of CNS infection, or persistent altered level of consciousness, or failure to return to baseline alertness after the seizure a lumbar puncture should be performed following imaging[1].

Key messages

- Imaging should be considered as part of routine evaluation in adults with a new onset seizure.
- Wherever possible MRI is preferred. It helps to identify underlying aetiology and to predict risk of recurrence in remote symptomatic seizures.
- Routine imaging in children is not always indicated.
- An EEG is useful in predicting the risk of recurrence of seizures.
- Routine blood tests to find the metabolic aetiology of acute symptomatic seizures is indicated in the emergency setting.
- Specific cut off values to attribute seizures to a metabolic cause are defined by the ILAE.

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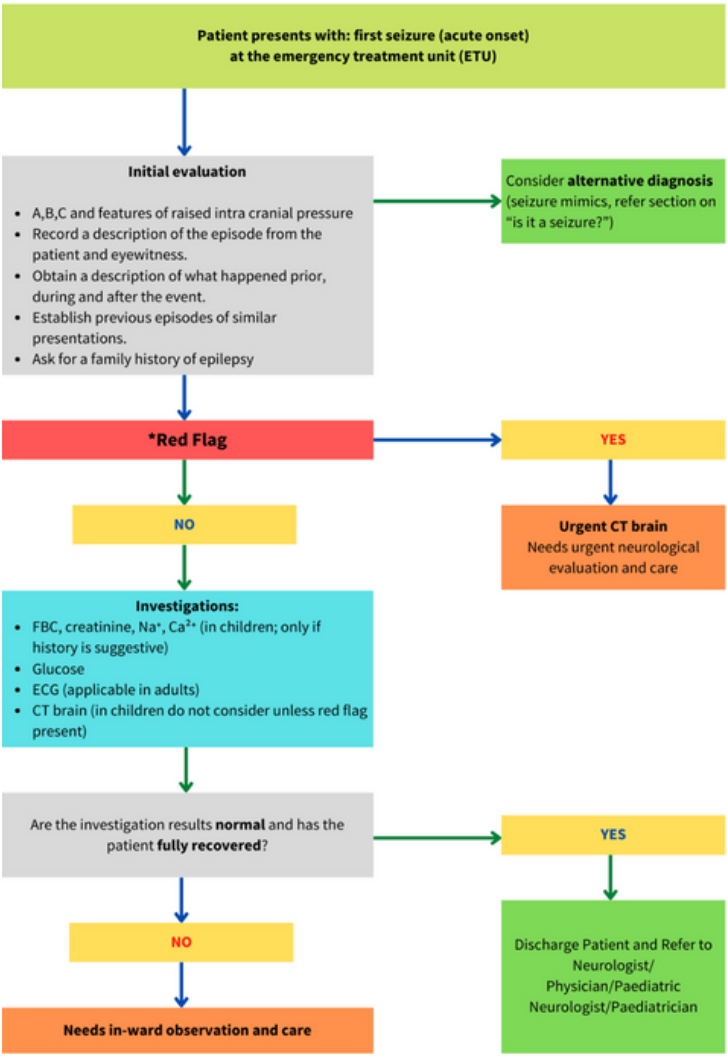
First seizure evaluation flow chart

This section is for medical professionals who would encounter patients with a new onset seizure within the hospital in the emergency setting. The following flow chart summarises the evaluation process of a suspected seizure and the step-by-step process one has to follow before the patient is referred for specialist evaluation.

Figure 3: Flow chart for evaluating first seizure presenting to the emergency setting



*Red Flags	
• GCS persistently <15 • New onset focal neurological signs • Sudden onset headache • Head injury • Signs of meningitis or encephalitis • Signs of raised intra-cranial pressure • History of malignancy	• Pregnancy/post-partum • HIV +ve patients • Patients on anti-coagulants, • Chronic alcoholic patients • Features suggestive of Pregnancy induced Hypertension.

**For paediatric patients please refer section on neuroimaging in children
*** In cases where the primary care physician/ general practitioner encounters such patients we would recommend that these patients be referred to the regional hospital in which a specialist (paediatrician/ paediatric neurologist/ physician/ neurologist) is available.

How should this epilepsy be treated?

If there are 2 or more unprovoked seizures which are > 24 hours apart then a diagnosis of epilepsy is made in line with the 1st criterion laid down by the ILAE. When considering treatment of epilepsy, a knowledge of seizure type and aetiology, suitable choice of ASM, and anticipated duration of treatment is essential. Other factors to consider include gender, age, co-morbidities, other medications the patient may be on and occupation.

The decision to treat a single new onset unprovoked seizure is based on the fulfilment of the second criterion in diagnosing epilepsy. For this, the risk of recurrence of seizures needs to be gauged.

Risk of recurrence is greatest in (adults)[1]:

1. In patients with prior brain lesion/s or insult/s (e.g., stroke, trauma and CNS infection)
2. An abnormal EEG (generalised/ focal epileptiform abnormalities and or focal slowing)
3. A significant brain imaging abnormality (features of previous stroke, trauma and CNS infection)
4. Nocturnal seizures

Since the seizure recurrence risk is highest for the aforesaid and may fulfil the second operational criterion on the diagnosis of epilepsy, we recommend considering ASM monotherapy for these patients (figure 4 – page number 45). If in case of a remote symptomatic aetiology with a risk of recurrence > 60% over the next 10 years (stroke, traumatic brain injury, infection) then initiating ASM is indicated as this would fulfil criterion 2 of the operational definition of epilepsy of the ILAE. If the aetiology is unknown or the risk of recurrence is unknown due to lack of data (E.g.: mesial temporal sclerosis, focal cortical dysplasia) the clinician may consider initiating ASM considering other factors such as occupation, risk of injury etc. Initiating ASM should be a shared decision made with the patient.

The risk of seizure recurrence after a new-onset unprovoked seizure ranges from 21 to 45%[1]. Immediate therapy with an ASM after such an unprovoked first seizure significantly reduces seizure risk on the short term (first 2 years) but is unlikely to

improve the chance of attaining sustained seizure remission in the longer term (≥3 years)[2]. ASMs currently used are effective in aborting or preventing seizures through a symptomatic effect but have no known impact on the disease process (prognosis)[3].

Therefore, we recommend a 2-year period of observation without ASM if the seizure recurrence risk is low (in patients not fulfilling the above criteria) as the highest risk of recurrence is within the first 2 years (see above). However patient preference and patient circumstances (i.e., occupation, recreational activities etc.) may need consideration and may influence the commencement of ASM monotherapy (see below).

Choice of Anti-seizure Medication (ASM)

The ideal first prescribed ASM should be efficacious, well tolerated, have long term safety with minimal interactions and easy for the clinicians to prescribe and the patients to take[4]. The cost of the ASM is another consideration. In addition, available therapeutic formulations and patient choice has a role in prescribing. There are many available ASMs that can be considered for first-line monotherapy in adults and children with epilepsy each with its advantages and limitations. It is important to note no single antiseizure medication is clearly more effective or best tolerated.

The choice of ASM is primarily based on the presumed type of epilepsy. ASM can be divided into broad-spectrum and narrow spectrum (broad spectrum agents treat both focal and generalized epilepsies, narrow spectrum agents treat one or the other). Sodium valproate, lamotrigine, topiramate and levetiracetam, are common ASMs which are considered broad spectrums. Phenytoin, carbamazepine, oxcarbazepine and lacosamide are considered narrow spectrum ASM.

Sodium valproate should remain the drug of first choice for many patients with generalised and unclassified epilepsies/ seizures[5]. However, because of known potential teratogenic effects of sodium valproate during pregnancy, it should not be considered as first line for these epilepsies in women of childbearing age[6]. Narrow spectrum agents are typically effective in patients with all forms of focal seizures regardless of state of consciousness or progression to bilateral tonic-clonic formerly known as secondary generalization. Lamotrigine, carbamazepine and oxcarbazepine are treatment options for adult patients with focal epilepsy. According to SANAD[7] which was an open study that included both children and adults, where carbamazepine was compared with topiramate, gabapentin, lamotrigine, or oxcarbazepine by randomization 1:1 in four treatment arms, lamotrigine was recommended as a first choice for focal seizures mainly based on evidence from adults. This was due to increased tolerability of the drug and resultant increase in the retention rate[8]. In older

adults, lamotrigine may be preferred over carbamazepine due to better tolerability and less drug interactions. In children carbamazepine and lamotrigine are first line[9].

Table 4. Examples of recommended ASM for specific epilepsy syndromes in children/ adolescents[10,11]

Epilepsy syndromes	Recommended ASM
Childhood absence epilepsy- If absences only	Ethosuximide (Level A*) is the drug of choice. Valproate (Level A*) is equally effective but has a higher rate of discontinuation due to adverse effects. Lamotrigine (Level C***) is less efficacious than above two.
Juvenile absence epilepsy	Sodium valproate, Lamotrigine (preferred in young females)
Juvenile myoclonic epilepsy	Sodium valproate (Level D****) and Topiramate (Level D****), Levetiracetam
Generalized tonic-clonic seizures alone	Sodium valproate (Level C*), Lamotrigine, Topiramate (Level C*), Levetiracetam
Infantile epileptic spasm syndrome	Vigabatrin (in tuberous sclerosis), steroids (oral prednisolone/ intramuscular ACTH

*Level A: Established efficacy of antiepileptic drug as initial monotherapy
**Level B: Probably effective antiepileptic drug as initial monotherapy
***Level C: Possibly effective antiepileptic drug as initial monotherapy
****Level D: Potentially effective antiepileptic drug as initial monotherapy

ASMs to be avoided or used with caution:

- Absence seizures: phenytoin, carbamazepine, oxcarbazepine, and gabapentin
- Myoclonic seizures and or atonic seizures: phenytoin. carbamazepine and phenytoin*
- Children less than 1 year of age with suspected neuro-metabolic disorders including mitochondrial disorders, sodium valproate may be avoided.

*Topiramate has not been shown to be effective against absence seizures[4]
*Lamotrigine can worsen myoclonic seizures[4]

Special considerations

The choice of ASM should account for the patient's comorbidities, other medication use, age, sex, and the cost of the medication. Older ASM such as phenytoin and carbamazepine are potent inducers of the cytochrome P450 system and sodium valproate is an inhibitor, resulting in possible drug-drug interactions. Furthermore, sodium valproate, may have adverse consequences pertaining to glucose and lipid metabolism[12].

Thus, in patients with multiple comorbidities, or those taking medications such as warfarin or chemotherapeutic agents, use of newer ASM such as levetiracetam, lamotrigine, or lacosamide with limited drug interactions is favoured whenever possible. Of the ASMs lamotrigine is preferred and is considered first line in older adults[13].

- Older adult with focal epilepsy: lamotrigine, levetiracetam, lacosamide, carbamazepine, oxcarbazepine
- Comorbid depression with focal epilepsy: lamotrigine, oxcarbazepine, lacosamide
- Comorbid depression with genetically mediated generalized epilepsy: valproate, lamotrigine
- Hepatic failure or after organ transplantation: gabapentin, levetiracetam, lacosamide
- Renal failure on hemodialysis: lamotrigine, oxcarbazepine, levetiracetam

Use of Anti-seizure medication (ASM) in females

Women with epilepsy in the childbearing age group should be referred to the neurologist for preconceptual counselling and choice of the appropriate ASM. In women of childbearing age, it is also necessary to consider the teratogenic effects and interactions with contraception when selecting ASM. Several ASMs, most notably P450 inducers (phenytoin, carbamazepine, and to a lesser degree oxcarbazepine), have been shown to increase the clearance of oral contraceptives, possibly resulting in contraceptive failure. In such an instance It is important for patients to choose alternative methods of contraception. Intrauterine devices seem to be the best method for contraception in this population. Medroxyprogesterone is another alternative.

Sodium valproate is well known for its potential teratogenic effects and also reported

to have impact on cognitive functions in the offspring. It is estimated to have a 10% risk for major congenital malformations[14,15]. E.g., neural tube defects, craniofacial defects like oral clefts, craniosynostosis, cardiovascular malformations, hypospadias, and limb malformations have been reported. Major congenital malformations also have been reported with use of phenobarbital, topiramate, phenytoin, and carbamazepine[14]. ASM medication with least teratogenic effects are levetiracetam and lamotrigine.

The prevalence of malformations due to above ASMs are 1.47% for gabapentin, 1.77% for levetiracetam, 2.39% for oxcarbazepine, 4.28% for topiramate, 4.93% for carbamazepine, 6.26% for phenytoin, 7.10% for phenobarbital, 8.49% for primidone, and 10.93% for sodium valproate. There is limited data for clobazam monotherapy.

However, it is also important to note that lamotrigine levels can fall unpredictably in pregnancy[14]. There is limited evidence of the potential teratogenicity with newer ASM such as lacosamide and oxcarbazepine. In addition to the type of ASM the dose of ASM is important as the teratogenicity is dose dependent.

Women with epilepsy have an increased frequency of menstrual disturbances compared to women without epilepsy. In women on polytherapy, the frequency of menstrual disturbances is further increased. The highest frequency of menstrual disturbances occurs in women using sodium valproate[16].

Acute symptomatic seizures

Long-term ASM is generally not indicated, based on the significantly lower recurrence risk of acute symptomatic seizures than unprovoked seizures, as already discussed above. Patients with acute symptomatic seizures should be treated with ASMs during the acute phase of the underlying disease or as long as the precipitating factor or cause is present. Data on how long to treat such patients with ASMs is lacking. A shorter duration of ASM may be indicated in persistent metabolic causes (days) while a longer duration of ASMs (weeks) are indicated in acute brain insults (e.g., traumatic brain injury, autoimmune encephalitis) with ongoing pathology[17].

Adverse Events

Patients should be aware that the risk for ASM adverse events (AE) ranges from 7% to 31% and that these AEs are predominantly mild and reversible. Clinician needs to consider this when making a decision on initiating ASM[1].

Most commonly experienced AE are, somnolence, dizziness, blurry vision, and difficulties with concentration and memory[4]. Typically, these are dose dependent

adverse effects and are most prominent during the first few days of therapy. All ASM can cause a rash, ranging from a mild erythematous maculopapular rash to severe reactions such as Stevens-Johnson syndrome or toxic epidermal necrolysis. ASM most commonly associated with development of a Steven Johnson Syndrome (SJS)/ Toxic Epidermal Necrolysis (TEN) are phenytoin, carbamazepine, and lamotrigine (this risk is further increased with the combination of valproate and lamotrigine).

The following flow chart summarizes (figure 5- treatment algorithm, page 45) decision making with regards to treatment (for the specialist). It should be noted that some patients may choose not to take medication, e.g., a young woman with focal aware seizures and who is about to start a family. In certain instances, the treating physician might be compelled to treat the first seizure even when the risk of recurrence may be low. E.g.; if there is a high risk for injury, such as patients with severe osteoporosis or bleeding diathesis. Another example would be a social situation in which a second seizure would result in loss of employment.

Lifestyle, work, personal safety, driving and responsibilities towards others should be discussed with the individual and their carers/ family when deciding whether to start medication or not.

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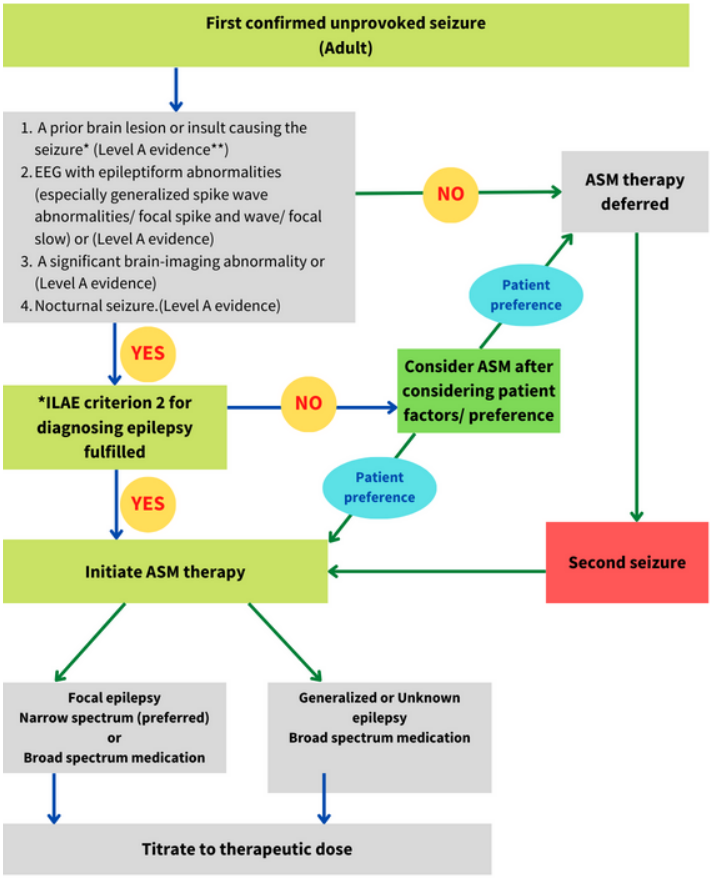
Figure 4: First adult seizure treatment algorithm for the specialist



*Patient with the following remote symptomatic aetiologies: stroke, infection, traumatic brain injury.
EEG with generalized epileptiform discharge *(criterion 3 fulfilled for epileptic syndrome)*
**Level A evidence ≥1 Class I studies or meta-analysis meeting class I criteria OR ≥2 Class II studies (Applicable for adults)

Annexure 1

This section is about advice for the patient/ family after a new-onset seizure[1]

For your safety;

- You must not drive for a period of 6 months or operate dangerous machinery.
- You should avoid any dangerous unsupervised work or leisure activities which include activities such as swimming, cycling on busy road or using ladders. Consider situations where having a seizure could have serious consequences such as standing close to train platforms or roads.
- Avoid the consumption of alcohol as much as possible (this may be a seizure trigger). It is a good idea to inform your family/friends that you have had a seizure and make sure they know what to do in case you have another seizure.

If you are diagnosed to have epilepsy following a single seizure

- Seizures can be precipitated by missing your anti-seizure medications
- Inadequate sleep and alcohol may precipitate seizures
- In photosensitive epilepsies, seizures can be triggered by TV screens and screens of other electronic devices. However, this is only if they have a flicker frequency that is known to cause photosensitivity.

First Aid during a Seizure (information for the carer)

Once a seizure starts, it will usually stop on its own after a few minutes. Follow the advices given below:

Do:

- Watch the seizure carefully and if possible, let it run its natural course.
- Keep calm and note the time the seizure starts and how long it lasts.
- Clear a space around the person, removing any sharp, hot or hard objects.
- Cushion the person's head with whatever is available.
- Loosen any tight clothing round the neck and gently remove glasses if worn.
- Turn the person onto their side into the recovery position once the movements stop (applicable only for tonic clonic seizures).
- Stay with the person, if possible, until any confusion passes.
- Call for emergency assistance (1990)—if the seizure lasts more than 5 min, if the seizure recurs shortly after the first one, if the person is injured or has trouble breathing after the seizure, if the person cannot be awakened, or if the person is aggressive.

Do not:

- Do not move the person while the seizure is ongoing unless there is an immediate danger (E.g., in a busy road, at the top of stairs, in water).
- Do not try to restrain the person.
- Do not attempt to lift the person up.
- Do not put anything between the teeth or into the mouth.
- Do not give any medication, food or drink while the seizure is happening.
- Do not leave the person until they have recovered.

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Annexure 2

This section is for EEG technologists

The following standards of recording of a routine EEG need to be adhered to[1]:

- The baseline record should contain at least 20 min of technically satisfactory recording.
- All 21 electrodes and placements recommended by the International Federation of Clinical Neurophysiology (IFCN)[2] should be used. The 10-20 System is the only one officially recommended by the IFCN.
- For standard recordings, the low-frequency filter should be no higher than 1 Hz (— 3 dB) corresponding to a time constant of at least 0.16 s. The high-frequency filter should be no lower than 70 Hz (-3 dB).
- The sensitivity of the EEG equipment for routine recording should be set in the range of 5—10 uV/ mm of pen deflection.
- Interelectrode impedances should be checked as a routine prerecording procedure. Ordinarily, electrode impedance should not exceed 5000 Ohms (5 KOhms).
- A paper speed of 3 cm/s, or digital display of 10 seconds/page, should be utilized for routine recordings.
- The recordings should include periods when the eyes are open and when they are closed.
- Hyperventilation should be used routinely unless medical or other justifiable reasons (e.g., a recent intracranial haemorrhage, significant cardiopulmonary disease, sickle cell disease or trait, or patient inability or unwillingness to cooperate) contraindicate it. It should be performed for a minimum of 3 min with continued recording for at least 1 min after cessation of over-breathing.

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