



Seizure classification key to epilepsy management

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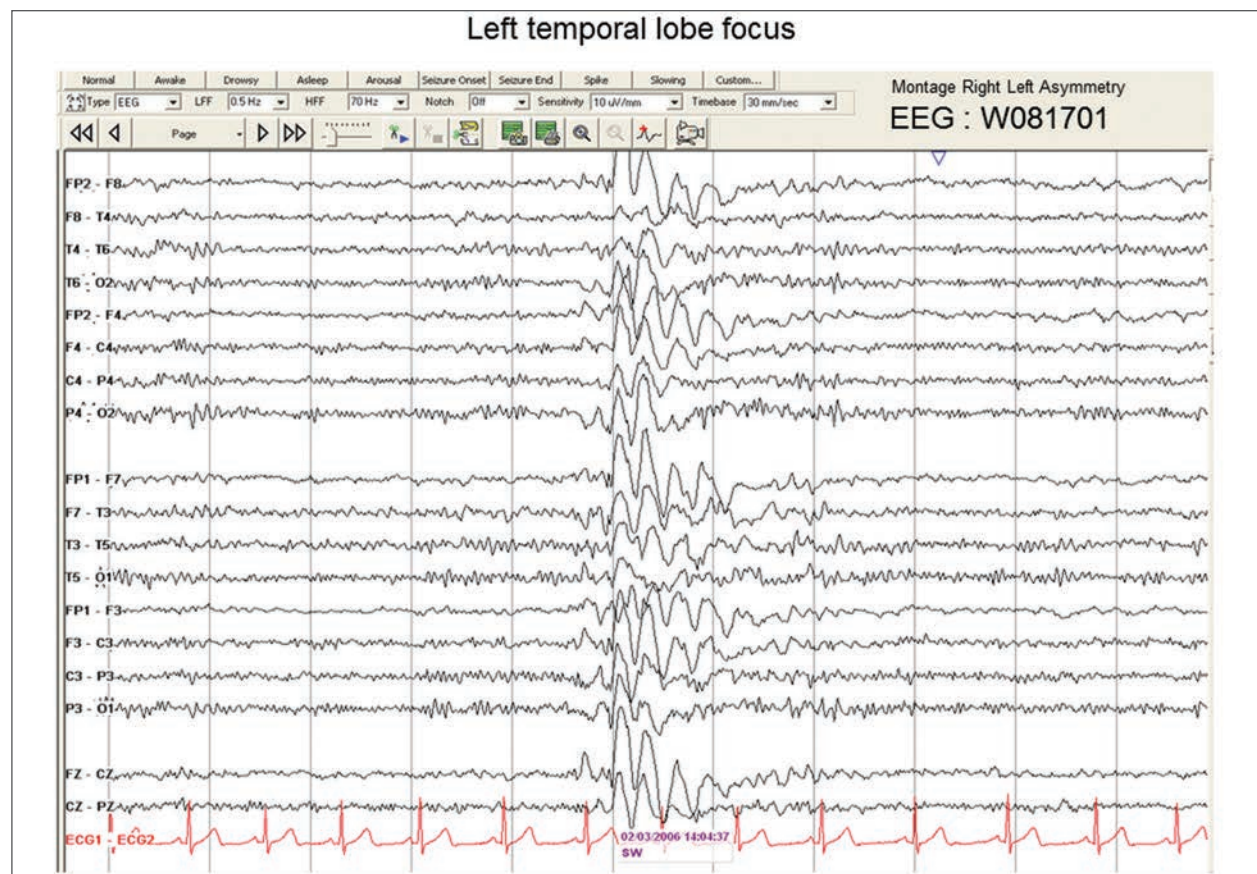


FIGURE 1
EEG showing
generalised spike
and wave activity
typical of a genetic
generalised epilepsy

How should
epilepsy be
diagnosed?



**EPILEPSY IS A COMMON
CONDITION, WITH AN
ANNUAL INCIDENCE OF
5 PER 10,000 PER YEAR**

and prevalence of 7.5 per 1,000.¹

The management of epilepsy often involves a range of medical professionals, including neurologists, epilepsy specialist nurses, GPs, learning disability teams, and emergency, general and geriatric medicine physicians.¹ Hence, an up to date working knowledge of epilepsy among all healthcare professionals is essential in order to deliver optimal patient care.

The diagnosis of epilepsy carries significant implications for physical, psychosocial and financial wellbeing as

How should
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be classified?

well as a small but significant increased risk of mortality.³ An incorrect diagnosis of epilepsy can be harmful, and it is, therefore, vital that every effort is made to reach a correct diagnosis. Evidence indicates that misdiagnosis is significantly more common among nonspecialists.^{2,4,5}

It is well recognised that there is an exhaustive list of epilepsy mimics that

**‘An incorrect
diagnosis
of epilepsy
can be harmful’**

What are the
treatment
options?

can result in misdiagnosis and expose patients to unnecessary antiepileptic drugs (AEDs).² Moreover, epilepsy itself is not a single entity, but comprises a range of seizure types and epilepsy syndromes.

Making an accurate assessment of seizure type and classification is important, both for treatment and prognostication.^{1,3}

There have been a number of recent advances in the field of epilepsy and SIGN has recently published updated guidance (SIGN 143).³

The terminology, see box 1, p14, and classification of seizures and epilepsy has changed over the years, see below.⁶

CLASSIFICATION

The most widely used classification system for epilepsy is that published by the International League Against Epilepsy (ILAE).^{3,7,8} The most recent ILAE classification update was in 2010.⁹

Classification largely depends on two characteristics:^{1,3,9}

- Site of seizure onset determines whether a seizure is:
 - Generalised (i.e. simultaneous

involvement of both hemispheres)
— Focal (i.e. starting from one area of the brain)

- Presumed aetiology
 - Structural-metabolic (an underlying cause is present, previously referred to as symptomatic, see box 1, below)
 - Epilepsy of unknown origin (presumed underlying cause but not identified, previously referred to as cryptogenic)
 - Genetic (previously idiopathic)

Classification is frequently overlooked but is key in epilepsy management,¹ and the importance of this is highlighted in the updated SIGN guideline.³

Distinguishing between a genetic generalised epilepsy and a focal epilepsy is vital, as this influences investigation, treatment and prognosis. In focal epilepsies, identifying the underlying cause is important.³

Box 1**Seizure terminology⁶**

- **Epileptic seizure** results from abnormal, repetitive and synchronised electrical discharges from a group of neurones within the brain. The observed clinical manifestations will depend on the area of the brain where the seizure is occurring
- **Unprovoked single seizure** is used to describe a single seizure or cluster of seizures occurring within 24 hours without an underlying condition or precipitating cause
- **Acute symptomatic or provoked seizure** is an immediate response to any kind of brain insult. Examples include a metabolic or toxic cause (such as alcohol withdrawal or low sodium levels), CNS infection or brain trauma
- **Epilepsy** is defined as a tendency to recurrent, unprovoked seizures. It is usually diagnosed when a patient has had two or more unprovoked seizures
- **Epileptic syndromes** are groups of different types of epilepsy that comprise a characteristic set of seizure types
- **Seizure semiology** is the description of a seizure and includes the symptoms and signs noted directly before, during and after the event
- **Status epilepticus** describes a single seizure with a duration longer than 30 minutes or a cluster of seizures that last longer than 30 minutes with no recovery to baseline in between events

Focal epilepsies

The focal epilepsies are so called because seizures start in a small area of the brain; the location of this abnormal area dictates the clinical features of the attack.

Focal seizures are usually described as simple partial (seizures in which awareness is retained) or complex partial (awareness is impaired).

The new ILAE 2010 classification has replaced these descriptions by the single term focal seizure; if impairment of consciousness clearly occurs, the adjective *dyscognitive* (i.e. focal *dyscognitive seizure*) can be applied.

Initial symptoms: Some patients with focal epilepsy lose awareness early in a seizure, and may report no warning or initial symptoms. However, some initial symptoms are common, and depend on the site of seizure onset.

Common clinical symptoms include:⁶

- Motor – spasms, jerking (frontal onset)
- Sensory – tingling or burning (parietal onset)
- Autonomic – rising abdominal sensation, palpitations (temporal onset)
- Psychic – altered perception of size or distance, dysphasia, altered memory (temporal onset)

Observed behavioural changes:

If consciousness is lost or impaired, automatic behaviours (automatisms) may be seen and described by witnesses.

Simple automatisms include lip smacking, chewing, fidgeting or plucking at clothes; occasionally more complex behaviours including undressing or wandering.⁶

Automatisms are most common in seizures arising from the temporal lobes, although may occur in focal seizures arising from other areas.

Focal seizures can spread to involve both hemispheres, and develop into a generalised tonic-clonic seizure (i.e. secondary generalisation).

A summary of features of focal seizures, subdivided by the site of seizure onset, is given in table 1, left.^{1,2,6}

Table 1**Types of focal seizure depending on site of seizure activity^{1,2,6}**

Seizure type	Features
Temporal lobe	Subjective symptoms: Déjà vu, fear, rising abdominal sensation Observed automatisms: Fidgeting, lip smacking, chewing
Frontal lobe	Wide variety of presentations Rapid onset and offset Some awareness may be retained Bilateral and bizarre automatisms (e.g. cycling movements of the legs) Often tonic postures and focal limb jerking Secondary generalisation common
Parietal lobe	Relatively rare Typically contralateral sensory symptoms (tingling, burning)
Occipital lobe	Contralateral visual hallucinations e.g. coloured shapes or formed objects Ipsilateral eye deviation to the side of the focus

Genetic (idiopathic) generalised epilepsies

Prior to the ILAE 2010 classification, these epilepsies (characterised by onset in diffuse, bilateral networks of neurones) were called idiopathic generalised epilepsies. This term remains in widespread clinical use, although the 2010 system has changed this to genetic generalised epilepsies.

'The diagnosis of epilepsy should be made by an epilepsy specialist ideally in a dedicated first seizure or epilepsy clinic'

There are several clues in the history that point towards a genetic generalised epilepsy. Individuals typically report no warning at all of seizures, and have usually one or more of three seizure types: tonic-clonic seizures; myoclonic jerks (brief electric shock like jerks of the limbs); or absences (brief staring spells,

usually lasting a few seconds only). There are subtypes of genetic generalised epilepsy (syndromes), the most common being juvenile myoclonic epilepsy and childhood absence epilepsy.

Specific features suggesting a genetic generalised epilepsy:^{3,10}

- Childhood or teenage onset
- Seizures triggered by sleep deprivation and alcohol
- Early morning tonic-clonic seizures or myoclonic jerks
- Brief absence seizures
- Photostimulation response on EEG
- Generalised three per second spike and wave or polyspike and wave on the EEG, see figure 1, p13.

DIAGNOSIS

SIGN states that the diagnosis of epilepsy should be made by an epilepsy specialist, ideally in the setting of a dedicated first seizure or epilepsy clinic.³

The differential diagnosis of patients presenting with episodic altered states of consciousness is exhaustive and full discussion is beyond the scope of this article. A list of epilepsy mimics is shown in table 2, below.² In many cases, the key challenge is differentiating between seizure, syncope and psychogenic

attack.^{1,2,3} Distinguishing features of each are highlighted in table 3, p16.^{1,2,3,6}

The diagnosis of epilepsy relies primarily on the history. Investigations can support the diagnosis but cannot make it in isolation,^{1,3} and negative investigation findings are common in epilepsy. Given that awareness is often impaired in attacks, a detailed witness account is crucial.^{2,3,6} It is important to establish what happened before, during and after the attack, from both the patient and the witness.^{3,6} Table 4, p17, highlights some of the important questions to cover.^{12,6}

'Awareness is often impaired in attacks, a detailed witness account is crucial'

In cases where there is an established history of epilepsy, it is good practice to go over the history again asking questions as for a new patient, especially if the patient has treatment-resistant seizures.

The diagnosis of epilepsy is often incorrect, potentially in up to 20% of cases,² so should be revisited if seizures are not responding to treatment.

Additional questions to be considered in such situations include:⁶

- When was the diagnosis of epilepsy made?
- Who made the diagnosis?
- What investigations were performed and what were the results?
- What treatments have been tried and at what dose?
- How did they affect the seizures?
- Were there any side effects?

'The diagnosis of epilepsy is incorrect in up to 20% of cases, so should be revisited if seizures are not responding to treatment'

DIAGNOSTIC INVESTIGATIONS

Investigations cannot replace an adequate history in the diagnosis of epilepsy, but can aid in clarifying the diagnosis and refining the epilepsy syndrome. Several modalities can be used, and the key techniques are discussed here.

Table 2

Epilepsy mimics^{1,2,6}

Syncope	
<i>Reflex</i>	Vasovagal, (also certain specific triggers including: micturition, swallow, cough)
<i>Cardiac</i>	Arrhythmia (e.g. long QT syndrome)
<i>Orthostatic</i>	Autonomic failure
Psychogenic	Panic disorder Dissociative
Sleep disorders	Parasomnias
Episodic symptoms of structural brain disease	Multiple sclerosis Tumour
Vascular	Migraine TIA
Hypoglycaemia	Behavioural disturbance
Movement disorder	Paroxysmal kinesigenic dystonia/dyskinesia
Hydrocephalus	Colloid cyst
Drop attacks	Postural instability Psychogenic

EEG

The EEG has its place in the diagnosis and classification of epilepsy.^{1,3,11} However, it has limitations and can sometimes cloud, rather than clarify, the diagnostic picture.

Many patients with epilepsy have a normal EEG between attacks, and so a normal test neither confirms nor refutes the diagnosis. In contrast, non-specific EEG abnormalities are common in people without epilepsy (especially the elderly, patients with migraine and those on psychotropic medication).³

Epileptiform abnormalities (changes associated strongly with epilepsy) occur in approximately 0.5% of healthy young adults and so, although suggestive, are not unequivocal evidence of epilepsy.³

The EEG is, therefore, best seen

as a supportive tool in diagnosis and classification, but one that is not routinely indicated, and best requested and interpreted by an epilepsy specialist.

In difficult cases, ambulatory EEG or video telemetry (combined EEG and video monitoring) can be used with the aim of obtaining an ictal EEG (i.e. EEG changes during an attack itself).

An ictal EEG is far more likely than an interictal EEG to give definitive diagnostic information, but is only really practically possible to obtain in individuals having frequent attacks.^{1,3,6}

Hand-held video

In difficult cases (for example when distinguishing epileptic from non-epileptic attacks), video recording of attacks can provide useful information

to clinicians experienced in diagnosing epilepsy. Based on anecdotal evidence, SIGN has suggested the use of hand-held videos such as smart phones to record attacks. Patient consent must be obtained before taping attacks.³

Brain imaging

Neuroimaging is an important part of the evaluation of patients with epilepsy. Advances in imaging techniques have resulted in structural lesions being identified in some people previously deemed to have epilepsy of unknown cause.⁶ Imaging identifies lesions in 21-37% of patients with epilepsy.³ The sensitivity and specificity of MRI is much higher than CT, and MRI is the recommended modality of structural imaging in epilepsy.^{1,3}

Table 3**Distinguishing features between seizure, syncope and dissociative attack^{1,2,3,6}**

	Seizure	Dissociative	Syncope
Predisposing factors	Birth trauma Febrile convulsion CNS infection Head injury Family history Stroke	History of childhood abuse/psychological trauma History of other medically unexplained symptoms	Family history Medication e.g. antihypertensives Cardiac disease Young
Triggers	Sleep deprivation Alcohol	Sometimes in situations causing psychological distress	Postural trigger Medical procedures Neck movement Heat Standing Exercise Micturition
Aura/prodrome	Positive neurological symptoms: Déjà vu, altered taste/smell, sensation, focal jerking	Dissociative out of body sensations Panic symptoms	Palpitations Lightheadedness Visual symptoms Breathlessness Distal tingling Sweaty, clammy, hot
The attack	Movements: Tonic (stiffening) Rhythmic jerking Automatic movements Cyanosis Eyes open	Flailing/thrashing Side to side head movement Prolonged motionless stare Eyes closed Resisting eye opening Rapid breathing Prolonged attack (> 5 minutes) Treatment resistance to multiple AEDs	Pallor Stiffening or jerks (brief < 1 min) Brief loss of consciousness Rapid orientation Incontinence
Post attack	Slow recovery: Drowsy, confused, amnesia Seizure markers: Lateral tongue biting, muscle pain, headache	Rapid recovery Sometimes upset, tearful	Rapid recovery but sometimes prolonged fatigue

Table 4**Important questions when taking a patient and witness history⁶**

The patient	The eye witness
● When did the episodes start? ● How many types do you think occur? ● How many have you had? ● Any triggers? ● How do you feel immediately before the episode? ● How long does it last? ● Do you recall anything about the episode? ● What is your next memory after the episode? ● How long did it take to feel back to normal? ● Did you bite your tongue or urinate? ● Did your muscles hurt or was there a headache afterwards? ● What medications do you take? ● Is there a family history? ● Is there a history of head trauma, CNS infection, birth trauma or substance abuse? ● Do you drive?	● How many episodes have you witnessed? ● Do they all look the same? ● What is the first thing you notice? ● What happens after that? ● Do they retain awareness? ● Do their lips go blue? ● Eyes open or closed? ● Are they stiff/rigid? ● Is there jerking? Is it synchronous? ● How long does it last? ● How long before the patient is back to normal?

Brain imaging will be undertaken in most people with epilepsy, but is not routinely required in those with a definite diagnosis of genetic generalised epilepsy (as this is not associated with an increased prevalence of brain lesions.³)

ECG

An ECG should be carried out in all patients presenting with altered consciousness, particularly in the elderly where disorders of cardiac rhythm can mimic epilepsy. If necessary, prolonged ECG recording may be required.^{1,3}

MANAGEMENT

The ultimate goal of epilepsy management is to achieve seizure freedom without adverse drug side effects. This is achievable in most cases.¹ In addition to pharmacological therapy and surgical intervention, it is also important to consider a variety of lifestyle influences on seizure control and how the diagnosis impacts on quality of life.³

The following recommendations have been made by SIGN.³

Starting AED therapy

The decision to prescribe AEDs is significant, as it commits the patient to prolonged treatment with a significant risk of adverse effects. It is therefore essential that every effort is made to ensure the diagnosis is correct before starting treatment. If there is uncertainty, a period of observation is reasonable as this will often clarify the diagnosis and

guide the need for treatment.^{1,3}

The decision to start treatment should be shared between the epilepsy specialist and the patient.³

Generally, medication should not be started following a single seizure except in specific circumstances or in cases where the risk of recurrence is high.^{1,3}

- Individuals wishing to avoid recurrent seizures e.g. drivers
- The patient has a structural cerebral disorder
- The EEG shows unequivocal epileptic discharges
- History of previous myoclonus, absence or focal seizure prior to the presenting tonic-clonic seizure.

'The ultimate goal of epilepsy management is to achieve seizure freedom without adverse drug side effects'

Choice of AED monotherapy

Monotherapy is the preferred treatment strategy in newly diagnosed epilepsy.¹ The drug of choice will be strongly influenced by the epilepsy classification, side-effect profiles, medication history, gender and the patient's wishes.^{1,3} All AEDs have an effect on focal epilepsies

but evidence-based treatment of generalised epilepsies is restricted to sodium valproate, lamotrigine and topiramate.¹

Dosing for each AED should start low and be increased slowly to the maximum tolerated or recommended dose as set out by the BNF.³ The first-line recommended drug for focal epilepsies is lamotrigine which is as effective and better tolerated than carbamazepine, topiramate and oxcarbazepine.³

Options for genetic generalised epilepsy include valproate, lamotrigine or topiramate with valproate remaining the recommended first-line choice.^{1,3} However, in certain circumstances (e.g. women of childbearing age) an alternative such as lamotrigine or levetiracetam is often used in order to reduce the risk of teratogenicity.³

Epilepsy resistant to AED therapy

This is defined as a patient who has failed to achieve sustained seizure freedom despite two trials of AED treatment, given at adequate doses, either as monotherapy or combination therapy.^{3,13} However, before this conclusion is reached, it is important to ask the following questions:³

- Is the diagnosis of epilepsy correct?
- Was the choice of AED appropriate for the epilepsy syndrome?
- Is the patient compliant with their medication?
- Is there concurrent drug or alcohol misuse?
- Is there an underlying structural or metabolic cause?



key points

SELECTED BY

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The diagnosis of epilepsy carries significant implications

for physical, psychosocial and financial wellbeing as well as a small but significant increased risk of mortality. The diagnosis is often incorrect, potentially in up to 20% of cases, so should be revisited if seizures are not responding to treatment. Evidence indicates that misdiagnosis is significantly more common among nonspecialists. SIGN recommends that the diagnosis of epilepsy should be made by an epilepsy specialist, ideally in the setting of a dedicated first seizure or epilepsy clinic. An incorrect diagnosis of epilepsy can be harmful. There is an exhaustive list of epilepsy mimics that can result in misdiagnosis and expose patients to unnecessary treatment with antiepileptic drugs.

Diagnosis relies primarily on the history. Investigations

can support the diagnosis but cannot make it in isolation, and negative investigation findings are common in epilepsy. Brain imaging will be undertaken in most patients with epilepsy, but is not routinely required in those with a definite diagnosis of genetic generalised epilepsy. The EEG has limitations and can sometimes cloud rather than clarify the diagnostic picture.

Epilepsy classification largely depends on two

characteristics. The site of seizure onset determines whether a seizure is generalised with simultaneous involvement of both hemispheres, or focal starting from one area of the brain. The presumed aetiologies may be structural-metabolic (previously referred to as symptomatic), epilepsy of unknown origin (previously cryptogenic), and genetic (previously idiopathic). Distinguishing between a genetic generalised epilepsy and a focal epilepsy is vital as this influences investigation, treatment and prognosis.

The focal epilepsies are so called because seizures

start in a small area of the brain; the location of this abnormal area dictates the clinical features of the attack. If impairment of consciousness clearly occurs, the adjective dyscognitive (i.e. focal dyscognitive seizure) can be used. Focal seizures can spread to involve both hemispheres, and develop into a generalised tonic-clonic seizure. There are usually one or more of three seizure types in genetic generalised epilepsies: tonic-clonic seizures, myoclonic jerks, or absences.

Generally medication should not be started following

a single seizure except in specific circumstances or in cases where the risk of recurrence is high. The first-line recommended drug for focal epilepsies is lamotrigine. Options for genetic generalised epilepsy include valproate, lamotrigine or topiramate. Most antiepileptic drugs are potentially teratogenic. Sodium valproate is associated with a higher rate of fetal malformations, and should not be prescribed in women of childbearing potential unless other alternatives are ineffective.

Combination therapy is often needed in treatment-resistant epilepsy. The goal is to minimise seizures (ideally achieving seizure freedom), using the minimum number of drugs, usually two or three.³

Carbamazepine, gabapentin, lacosamide, lamotrigine, levetiracetam, perampanel, pregabalin, topiramate, sodium valproate and zonisamide are all suitable adjuvant therapies for the treatment of focal epilepsy.³

Lamotrigine, levetiracetam, ethosuximide, sodium valproate and topiramate can be used in the adjunctive treatment of generalised epilepsy.³

Before commencing any AED, patients need to be warned of the potential side effects both in the acute and chronic setting. They need to be provided with clear instructions about seeking urgent medical attention should they develop symptoms such as rash, sedation, bruising or vomiting, especially within the first few weeks of treatment.³

Longer-term side effects such as osteoporosis and increased risk of clinical fracture should also be explained and the patient given dietary and lifestyle advice in relation to this.³

Blood monitoring

Routine blood monitoring of AED serum concentrations is not necessary although it can be useful in the following circumstances:^{1,3}

- Adjustment of phenytoin dose
- To establish drug compliance
- Assessment of suspected toxicity
- If drug metabolism could potentially change such as during pregnancy
- Unexplained loss of seizure control

Surgery

In true cases of drug resistant epilepsy, despite a variety of trials of combination therapies appropriate for the epilepsy syndrome, neurosurgical intervention needs to be considered as soon as possible as younger patients have better outcomes.³

Surgery is divided into curative and palliative. The aim of curative surgery (e.g. anterior and medial temporal lobe resection) is complete seizure freedom and approximately 70% of patients will achieve this.^{3,14} Palliative surgery applies to those who are not suitable for resective surgery (e.g. vagus nerve stimulation and callosotomy) and the aim is to reduce seizure activity.

In both groups, there is potential for improved seizure control, reduction in AED treatment and enhanced quality of life.³

WOMEN WITH EPILEPSY

One key area of concern is the teratogenicity of AEDs. Most AEDs are potentially teratogenic, although sodium valproate is associated with a higher rate of fetal malformations, and should not be prescribed in women of childbearing potential unless other alternatives are ineffective.

Women of childbearing age who have epilepsy need careful counselling on contraception and pregnancy as well as general epilepsy management. Patients who are better informed on this topic tend to have safer pregnancies in relation to their epilepsy and to use more reliable contraception.³

Some of the key recommendations from SIGN include:³

- To reduce the chance of contraception failure, those who take any combined hormonal contraception or progesterone-only pill should not be prescribed an AED that induces hepatic enzymes
- Patients should receive pre-pregnancy counselling at diagnosis and at regular intervals throughout their management
- Women should be informed that sodium valproate is associated with a higher rate of teratogenicity compared with other AEDs
- Patients should be well informed about pregnancy- and epilepsy-related issues such as the importance of smoking cessation
- Women should have their epilepsy reviewed by a specialist team before conception and seizure control optimised

This topic is extensive and we recommend reading the section 'Epilepsy and women's health' in the SIGN guideline for further details.

MORTALITY IN EPILEPSY

Premature death in those with epilepsy compared with the general population has been widely reported in the literature,^{3,15} including drowning, road traffic accidents, and suicide.³

Studies have reported a three-fold increase in suicide risk compared with the general population, and vigilance for signs of depression is important.^{3,16}

Sudden unexplained death in epilepsy (SUDEP) has attracted widespread attention in recent years. It is defined as 'sudden, unexpected, unwitnessed, non-traumatic, non-drowning death of a person with epilepsy, with or without a seizure, excluding documented status epilepticus and in whom post mortem examination does not reveal a structural or toxicological cause of death.'^{3,17}

Fortunately it is uncommon, affecting

1 in 1,000 people with epilepsy per year, but the predominant risk factor is poor seizure control, especially generalised tonic-clonic seizures at night.³ It is now explicitly recommended that the small risk of SUDEP be discussed with the patient, at an appropriate time and by an appropriate healthcare professional. Drug adherence should be strongly encouraged.³

'The small risk of sudden unexplained death should be discussed with the patient'

CONCLUSION

Epilepsy is a complex group of disorders, and presents a range of diagnostic and management challenges. Patients with epilepsy present to a broad spectrum of professionals, from GPs to general physicians, epilepsy nurses and neurologists, and can experience a range of comorbidities. Up to date evidence-based guidelines have recently been published by SIGN to assist these professionals in management decisions.

Good communication, between doctor and patient, and between healthcare professionals involved in the care of patients with epilepsy, is essential.

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Useful information

Epilepsy Action
www.epilepsy.org.uk

SIGN
SIGN 143. Diagnosis and management of epilepsy in adults
www.sign.ac.uk

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