REVIEW
Neurology Series Editor, William J. Mullally, MD

THE AMERICAN JOURNAL of MEDICINE®

Epilepsy: A Clinical Overview

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ABSTRACT

The diagnosis and treatment of seizures and epilepsy is a common task of the physician. Approximately 1 in 10 people will have a seizure during their lifetime. Epilepsy is the tendency to have unprovoked seizures. Epilepsy is the fourth most common neurological disorder and affects 1 in 26 people in the United States and 65 million people worldwide. Evaluation of a patient presenting with a seizure involves excluding an underlying neurologic or medical condition, classifying the seizure type and determining if the patient has epilepsy. Proper treatment requires accurate diagnosis of the epilepsy type and syndrome and use of a medication that is effective and without adverse effects. Most patients can achieve complete seizure control with medication, but if medication is unsuccessful, surgical treatment can be an option. Special situations in the care of people with epilepsy include status epilepticus, women with epilepsy, the older adult, and safety issues.
 • *The American Journal of Medicine (2021) 134:840–847*

KEYWORDS: Epilepsy; Diagnosis; Seizure; Treatment

The word epilepsy is derived from the Greek word *epilepsia* meaning to seize or attack. Seizures and epilepsy are common disorders and have been documented since the earliest recordings of humans. Initially, epilepsy was thought to be a spiritual disease and the oldest detailed account of epilepsy was in 2000 BC.[1] It was not until the fifth century BC that it was described as a brain disease (Hippocrates' *The Sacred Disease*). In most cultures, epilepsy has continued to be stigmatized and it is only in the past few decades that there has been more organized effort to counter the stigma, secrecy, and discrimination often associated with epilepsy.[2]

Epilepsy is the tendency to have recurrent nonprovoked seizures. A seizure is a brief, excessive discharge of electrical activity in the brain that transiently alters behavior. Neurons communicate through chemical and electrical signals and form networks with other neurons. In most seizures, a relatively small number of abnormal neurons cause changes in other neighboring or networked neurons. During a seizure there is a progressive recruitment of other neurons in the network resulting in a pattern of hypersynchrony. This abnormal propagation occurs due to insufficient inhibition, excessive excitation, or both within the neuronal network. This abnormal neuronal hypersynchrony can be congenital or develop at any time during life.

Epilepsy is defined as a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures, and by the neurobiological, cognitive, psychological, and social consequences of this condition.[3] This definition emphasizes that epilepsy consists not only of the neurobiology of seizures but also of the associated neuropsychosocial comorbidites. The most recent definition of epilepsy requires the occurrence of at least 1 epileptic seizure. After a single seizure, epilepsy can be diagnosed if the seizure was unprovoked (eg, unrelated to drugs, alcohol, hyponatremia, or glucose abnormality) and the patient has a >60% chance of having another unprovoked seizure. Epilepsy is fully defined as follows:

Epilepsy is a disease of the brain characterized by any of the following conditions: 1) At least 2 unprovoked (or reflex) seizures occurring >24 hours apart; 2) 1 unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after 2 unprovoked seizures, occurring over the next 10 years; 3) diagnosis of an epilepsy syndrome.[4]

Epilepsy affects more than 3.4 million people in the United States (1.2% of the US population) and more than

Funding: None.
Conflict of Interest: None.
Authorship: The author is solely responsible for the content of this manuscript.

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0002-9343/© 2021 Published by Elsevier Inc.
https://doi.org/10.1016/j.amjmed.2021.01.038

65 million people worldwide. The lifetime prevalence rate is 3%. It affects people of every age and background, but most commonly starts before the age of 1 year and increases again after the age of 50, with the highest incidence after the age of 75.[5] In the United States, approximately 1 in 26 people will develop epilepsy at some point in their lifetime. The annual cost of epilepsy in the United States is over 12.5 billion dollars.[6] Only 44% of American adults with epilepsy reported having their seizures controlled.[7] However, approximately 70% of patients may become seizure-free with appropriate treatment. The remaining 30% of patients have drug resistant epilepsy, which is the failure of adequate trials of 2 tolerated, appropriately chosen and used antiepileptic drug (AED) schedules to achieve sustained seizure freedom.[8]

CLINICAL SIGNIFICANCE

- Epilepsy is the fourth most common neurologic disease and affects 1 in 26 people in the United States during their lifetime.
- With proper diagnosis and treatment, approximately 60%-70% of people with epilepsy can achieve seizure freedom.
- Treatment options are increasing and include new medications, surgical techniques, and medical devices.
- Epilepsy can be deadly and is associated with an increased risk of depression, accidents, and death.

CLASSIFICATION OF SEIZURES AND EPILEPSY

There are distinct types of epileptic seizures. The classification of seizure type has important consequences for determining the etiology, best treatment, and overall prognosis. Seizures are classified based on the appearance of the seizure and parts of the cerebral cortex involved in the seizure. The 2017 classification system for seizures and epilepsy builds on the original classification system but emphasizes updated terms with the goal of being more understandable (Figure).[9, 10]

The first step in classification of seizures is to identify whether the seizures are focal in onset (involving a localized network of neurons) or generalized in onset (rapidly engaging a bilaterally distributed neuronal network). Focal seizures are due to a small group of neurons that have enhanced excitability and the ability to occasionally spread that activity to neighboring regions and thereby cause a seizure. A seizure can begin in any lobe of the brain, but the most common lobe is the temporal lobe, particularly the mesial temporal lobe containing the amygdala and hippocampus. The clinical manifestations of a focal seizures will depend on the normal function of the region of cortex involved in the seizure (Table 1).

During a focal seizure, there may be preservation of consciousness and full awareness throughout the seizure. In the older nomenclature, this type of seizure was referred to as a "simple partial seizure" or "aura." In the 2017 classification system, this type of seizure is more precisely termed "a focal aware seizure." During a focal aware seizure, the patient is alert and able to respond and remembers the seizure. This type of seizure is synonymous with an aura. Focal seizures can either progress to altered awareness or begin with altered awareness, during which time the patient has altered responsiveness and memory. This type of seizure is called a focal seizure with impaired awareness, and in the older classification system was called a complex partial seizure. Automatisms are common during focal seizures with impaired awareness and can include the eyes (eg, blinking), mouth (eg, lip smacking and chewing), hands (eg, fumbling and picking), vocalizations (eg, grunts and repetition of words or phrases), or more complex acts (eg, walking and attempting to use a cellphone). This type of seizure generally lasts from 30 seconds to 2 minutes and is followed by a brief period of confusion and fatigue. A focal seizure can spread to involve networks in both cerebral hemispheres leading to tonic-clonic movements. This process is called focal to bilateral tonic-clonic. This term distinguishes convulsions that begin focally and spread, focal to bilateral tonic-clonic, from primary generalized tonic-clonic seizures, which have a different mechanism and etiology.

Primary generalized seizures involve bilaterally distributed networks at onset. The different seizure types include myoclonic, absence, atonic, tonic, clonic, and tonic-clonic seizures. Myoclonic seizures are very quick shock-like jerks of a muscle or group of muscles and typically involve the upper extremities without altered level of awareness. In contrast, the other forms of generalized seizures involve loss or alteration in consciousness. Absence seizures consist of brief episodes (2-15 seconds) of sudden onset and offset staring, impaired responsiveness, and eye fluttering, and do not have a post-ictal confusion (in contrast to focal seizures with impaired awareness). Tonic and atonic seizures consist of sudden changes in muscle tone lasting seconds and often lead to falls. Tonic-clonic seizures are commonly referred to as "convulsions" or "grand-mal seizures." They begin with loss of consciousness. The tonic phase can lead to falls and an epileptic cry caused by air forced through contracted vocal folds. The clonic phase consists of jerking of the upper and lower extremities with cyanosis, tongue biting, and foaming at the mouth and incontinence. After a tonic-clonic seizure, the patient is lethargic and confused, and may become agitated as consciousness is regained.

Some forms of epilepsy can be classified as an epilepsy syndrome. Epilepsy syndromes are types of epilepsy in which a clear demographic, seizure type(s), electroencephalography (EEG) pattern, and prognosis are known. Increasingly, the etiology is also known, and it is often genetic. Examples of epilepsy syndromes include infantile spasms (West syndrome), Lennox-Gastaut syndrome, childhood absence epilepsy, and juvenile myoclonic epilepsy.

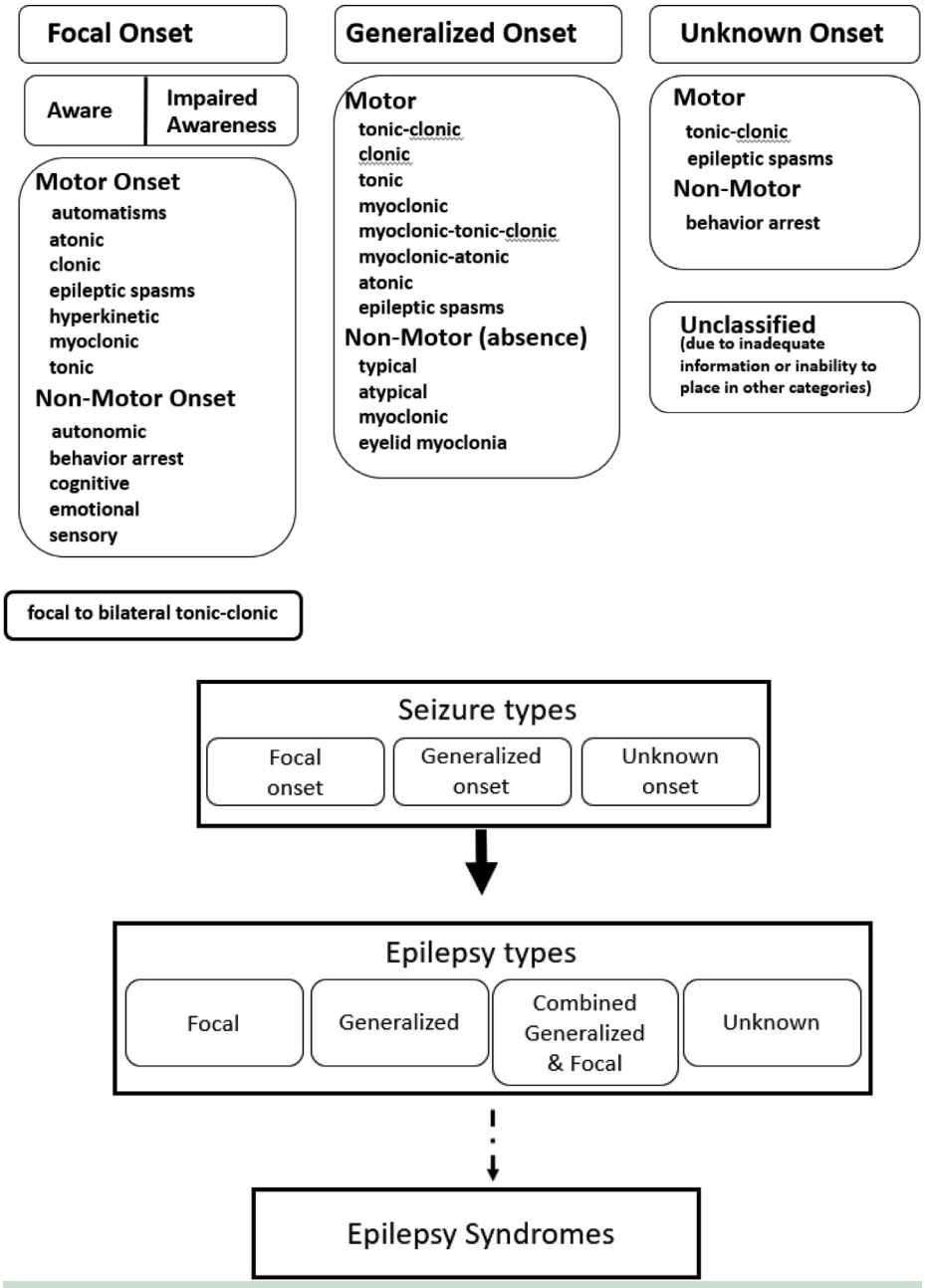

Figure 2017 International League Against Epilepsy Seizure and Epilepsy Classification Systems.

ETIOLOGY

Seizures have different causes of which epilepsy is one. A diagnosis of epilepsy requires unprovoked seizures. Epilepsy also has a wide variety of etiologies. The most likely cause depends on the age of onset (Table 2). Often despite extensive diagnostic testing, the etiology cannot be determined.

DIAGNOSIS

A diagnosis of epilepsy requires the occurrence of at least 1 unprovoked seizure and a >60% likelihood that the patient will have another unprovoked seizure. The likelihood of having a subsequent seizure can be determined by etiology and EEG. However, the initial step in diagnosis is to determine if the event was in fact a seizure and to explore the possibility that the seizure was provoked. Convulsive syncope and psychogenic nonepileptic seizures can appear like epileptic seizures and are often mistaken for epileptic seizures. Other seizure mimickers are listed in Table 3. It is important to also consider whether the seizure was provoked. Illicit and prescription drugs, alcohol withdrawal, and glucose and electrolyte abnormalities are common reasons for provoked seizures (Table 4). The International

Table 1 Seizure Semiology

Localization	Signs and Symptoms
Mesial temporal (amygdala, hippocampus)	Déjà vu, jamais vu, depersonalization, derealization, olfactory hallucinations, oral and manual automatisms, behavioral arrest, sweating, pallor, piloerection, epigastric sensations, anxiety, fear
Temporal lobe	Auditory hallucinations, aphasia (left)
Frontal lobe	Contralateral tonic and/or clonic movements, contralateral gaze deviation, contralateral versive head movement, bizarre-appearing hypermotor movements
Parietal lobe	Contralateral tingling and other somatosensory phenomena
Occipital	Visual hallucinations (simple [colors, shapes]), more complex visual hallucinations (temporo-occipital), vision loss

League Against Epilepsy has an educational website for the diagnosis of epilepsy, which illustrates seizure types and provides video and EEG examples.[11]

The diagnosis of epilepsy is made by history, examination, neuroimaging, and EEG. Risk factors for the development of epilepsy include, complications of pregnancy and childbirth, history of febrile seizures, family history of epilepsy, developmental delay or autism, history of traumatic brain injury or brain infection or other structural lesion of the cerebral cortex, and dementia. Physical examination for causes of provoked seizures and epilepsy includes blood pressure and other vital signs, skin exam for stigmata of systemic or neurologic disease (eg, café au lait spots seen with neurofibromatosis or adenoma sebaceum seen with tuberous sclerosis), extremity examination for smaller limbs or a smaller thumbnail that can indicate early life injury to the contralateral cerebral cortex, physical examination for signs of cancer or infection, and a full neurologic examination assessing for cognitive dysfunction and focal abnormalities.

Table 2 Common Causes of Epilepsy

Age Group	Common Causes
Infants	Perinatal hypoxia, metabolic disorders, intracranial hemorrhage, genetic disorders, developmental and congenital maldevelopment
Children	Perinatal anoxia, injury at birth or later, infection, vascular, metabolic, cortical malformations, genetic disorders
Teens and young adults	Trauma, infection, genetic disorders, brain tumor, congenital
Adults	stroke
Older adults (over 60 years)	Stroke, dementia, brain tumor, infection, trauma

Table 3 Differential Diagnosis of Seizures and Seizure Mimickers

Seizure
Syncope (convulsive)
Psychiatric disturbance (psychogenic non-epileptic seizure)
Migraine
Cerebral ischemia (transient cerebral ischemia)
Movement disorder
Sleep disorder
Metabolic disturbance

Electroencephalography is a necessary component to the evaluation of epilepsy. The procedure can help determine whether the seizure was focal or generalized in onset. If the seizure was focal, EEG can also help determine the location of the seizure onset. After a single seizure, an epileptiform EEG can lead to a diagnosis of epilepsy and initiation of treatment.[12] Ambulatory EEG monitoring has increased sensitivity and can be performed at home for several days at a time.[13] In general, interictal EEG sensitivity is only 20%-55% with only about a third of initial EEGs being abnormal in a patient who is later diagnosed with epilepsy.[14, 15] Sensitivity improves to 80%-90% if EEGs are repeated over time.[16]

Neuroimaging is important in the evaluation of seizures and epilepsy. In nonacute settings, magnetic resonance imaging (MRI) is the preferred imaging modality. Magnetic resonance imaging can assess for the etiology of the seizure and help determine the risk of recurrence after a first-time seizure. Neuroimaging reveals the cause of a first-time seizure approximately 28% of the time and is most sensitive in patients with focal seizures.[17] Neuroimaging can reveal mesial temporal sclerosis, neurocystercercosis, brain tumors, and some neurodevelopmental abnormalities such as cortical dysplasia and other structural abnormalities.

TREATMENT OF EPILEPSY

Currently available AEDs and their key features are summarized in Table 4. The goal of selecting an AED regimen is to find a medication that is fully efficacious and to which the patient has no side effects. Medications vary in cost, drug-drug interactions and teratogenicity. Antiepileptic drugs are divided into narrow-spectrum and broad-spectrum agents. Narrow-spectrum AEDs only work for specific types of seizures and may be ineffective or worsen other types of seizures. Narrow-spectrum AEDs include carbamazepine, oxcarbazepine, gabapentin, pregabalin,

Table 4 Antiepileptic Drugs*

Antiepileptic Drug	Advantages	Disadvantages
Broad Spectrum	Effective in both focal and generalized epilepsies	
Breviracetam	IV formulation available; easy titration	Somnolence; nausea; dizziness; psychiatric side effects
Clobazam	FDA approved for treatment of seizures associated with Lennox-Gastaut syndrome	Behavioral side effects; excessive salivation
Lacosamide	IV formulation available	Can lead to syncope and cardiac arrythmias; avoid in AV block
Lamotrigine	Once daily formulation available; favorable data for pregnancy; mood stabilizer; mild antidepressant properties; generally very well tolerated	Slow titration with drug-drug interactions; rash; headache; lowered levels occur with pregnancy and hormonal contraception due to increased metabolism
Levetiracetam	IV and once daily formulations available; favorable data for pregnancy; no drug-drug interactions; rapid titration	Irritability, behavioral problems, depression
Perampanel	Dosed once daily; different mechanism of action (blocks glutamate activity at AMPA receptor)	Hostility and aggression; weight gain; rarely, homicidal ideation
Phenobarbital	Oldest AED in use; commonly available; inexpensive; long half-life; IV formulation	Increased risk of bone loss; sedation; depression; hematologic toxicity
Primidone	Effective for treatment of essential tremor	Metabolized to phenobarbital; same disadvantages as phenobarbital
Topiramate	Once daily formulation available; FDA approved for prevention of migraines; weight loss	Anorexia, metabolic acidosis, oligohidrosis, nephrolithiasis, language and cognitive impairment; acute angle glaucoma; teratogenic
Valproate	Once daily formulation available; very effective; FDA approved for prevention of migraines; mood stabilizer	Highly teratogenic; dose-related tremor, hair loss, weight gain, hepatotoxicity; pancreatitis, thrombocytopenia; hyperammonemia; increased risk of bone loss
Zonisamide	Once daily dosing	Nephrolithiasis, anorexia, rash (may cross react with sulfa), oligohidrosis
Narrow Spectrum	May be more effective in select syndromes or for a specific seizure type	May not prevent or even worsen other seizure types
Carbamazepine	IV formulation available; inexpensive; mood stabilizer	Increased risk of bone loss and vascular disease; rash; weight gain; hyponatremia; bone marrow suppression; hepatotoxicity; severe dermatologic reactions in patients of Asian descent who have the human leukocyte antigen-B*1502 allele
Cenobamate	Once daily dosing; may be more efficacious than other AEDs in focal epilepsy	New agent; slow titration due to risk of drug reaction with eosinophilia and systemic symptoms (DRESS); somnolence, dizziness, cognitive impairment, visual changes
Cannabidiol	FDA approved for treatment of seizures associated with Lennox-Gastaut syndrome, Dravet syndrome, and tuberous sclerosis complex	Only available in oral solution; obtain liver function tests prior to starting; can cause hepatotoxicity; somnolence; diarrhea; nausea; anorexia; fatigue; sleep disorder
Eslicarbazepine	Once daily dosing	Less risk of hyponatremia compared with carbamazepine and oxcarbazepine
Ethosuximide	Best choice for childhood absence epilepsy	Narrow spectrum - absence seizures only
Gabapentin	Well tolerated; treats neuropathic pain; easy to titrate; no drug interactions	Less efficacious compared with other AEDs; weight gain; peripheral edema
Oxcarbazepine	Once daily dosing available	Hyponatremia; severe dermatologic reactions in patients of Asian descent who have the human leukocyte antigen-B*1502 allele
Phenytoin	Commonly available; IV formulation; once daily dosing	Rash; gingival hyperplasia, hirsutism; hepatotoxicity; lupus like reactions; aplastic anemia; increased risk osteoporosis; multiple drug-drug interactions; nonlinear pharmacokinetics make dosing challenging; glucose control challenging with DM; severe dermatologic reactions in patients of Asian descent who have the human leukocyte antigen-B*1502 allele

Table 4 (*Continued*)

Antiepileptic Drug	Advantages	Disadvantages
Pregabalin	Well tolerated; easy titration; treats neuropathic pain	Peripheral edema, weight gain, potential abuse
Rufinamide	FDA approved for treatment of seizures associated with Lennox-Gastaut syndrome	GI side effects; minimally effective for focal seizures
Tiagabine	Different mechanism of action (inhibits GABA uptake into presynaptic neurons)	Associated with new-onset seizures and status epilepticus in patients without epilepsy
Vigabarin	Effective for infantile spasms associated with tuberous sclerosis complex	Permanent visual field deficits

AED = antiepileptic drug; AMPA = α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid; AV = atrioventricular block; FDA = Food and Drug Administration; GABA = gamma-aminobutyric acid; GI = gastrointestinal; IV = intravenous.

*Although there is evidence to support the use of these medications as indicated in this table, the medication may not be indicated for this use by the United States Food and Drug Administration.

tiagabine, and eslicarbazepine. These AEDs can exacerbate myoclonic and absence seizures.[18] Broad-spectrum AEDs have some efficacy for a wide range of seizures (focal, absence, generalized tonic-clonic, and myoclonic). The newer AEDs generally have fewer side effects than the older agents. Potential side effects common among all AEDs include fatigue, gastrointestinal side effects, mood changes and cognitive side effects. The older generation AEDs have more side effects and they are associated with an increased rate of fractures due to osteopenia.[19] Bone density screening is recommended for anyone who has taken 1 of the following agents for 5 years or longer: phenytoin, phenobarbital, primidone, valproate, carbamazepine.[20, 21]

WOMEN WITH EPILEPSY

Many women with epilepsy have increased seizures associated with their menstrual cycle (catamenial epilepsy). Women with epilepsy also have a higher rate of anovulatory cycles and polycystic ovarian syndrome. Folate supplementation, 1 mg daily, is recommended for all women of childbearing potential as it decreases the risk of neural tube defects. Some AEDs reduce the efficacy of hormonal contraception (see Table 5). Estrogen containing hormonal contraception reduces the level of lamotrigine, placing women who take lamotrigine at risk of breakthrough seizures with the addition of hormonal contraception unless the dose is appropriately increased. Pregnancy will also lower the level of lamotrigine. Long-acting reversible contraceptives (progestin implant) or intrauterine devices are recommended for women with epilepsy on medications that decrease efficacy of oral contraceptives.[22, 23] The two AEDs with the most safety data for women during pregnancy are levetiracetam and lamotrigine and currently are the preferred agents for use during pregnancy. The agents with the highest known teratogenicity are valproic acid, phenobarbital, and topiramate and they should be avoided if possible and women counseled accordingly.[24]

Table 5 Common Causes of Provoked Seizures

Metabolic (hyponatremia, hypoglycemia, hyperthyroidism, nonketotic hyperglycemia, hypocalcemia, hypomagnesemia, renal failure, porphyria)
Medications (benzodiazepine withdrawal, barbiturate withdrawal, phenothiazines, bupropion, tramadol)
Substance abuse (alcohol withdrawal, cocaine, amphetamine, phencyclidine, methylenedioxymethamphetamine ["Ecstasy"])
Acute neurologic insults (within 1 week of injury)
Eclampsia

ELDERLY

The lifetime incidence of developing epilepsy follows a J-shaped curve with rates increasing dramaticallly at age 60 and older. The metabolism of AEDs slows down, and a given dose of an AED results in a higher serum level. A low dose of an AED with no or limited drug-drug interactions is preferred. One randomized controlled study of AED efficacy and tolerability in the elderly compared carbamazepine, gabapentin, and lamotrigine. Lamotrigine was the most efficacious and best tolerated of the three. The target dose in that study was 50 mg twice daily, titrating up as needed.[25] Levetiracetam has also been compared with carbamazepine and lamotrigine and has been shown to be efficacious and well tolerated. Levetiracetam has the advantage of having no drug-drug interactions.[26]

STATUS EPILEPTICUS

Status epilepticus is a life-threatening neurologic emergency, and is defined as a state of continuous seizures without interval recovery. The most recent definition of status epilepticus recognizes 2 points in time. For convulsive seizures, the first timepoint at 5 minutes is when the patient should be clinically diagnosed and treated for status epilepticus and the second timepoint at 30 minutes is when neuronal damage is likely to occur.[27] Status epilepticus is treated with benzodiazepines and AEDs delivered intravenously. Refractory status epilepticus requires intubation for airway protection, continuous EEG monitoring, and use of an anesthetic agent such as midazolam, propofol, ketamine or pentobarbital. The prognosis of status epilepticus is primarily determined by the etiology of the status epilepticus.

SURGICAL MANAGEMENT OF EPILEPSY

All patients with drug-resistant epilepsy should receive evaluation for possible surgical treatment. There is level-1 evidence suggesting that for patients who are candidates for temporal lobectomy, epilepsy surgery is superior to medical therapy. At 1 year, 58% of surgically treated patients were seizure-free versus only 8% in the medically treated group.[28] The most common surgical approach is open resection of the epileptogenic cerebral cortex, but MRI-guided laser ablation is increasingly being used and has the advantage of requiring only a burr hole rather than a craniotomy.[29] Other surgical methods include responsive neurostimulation (eg, NeuroPace), vagal nerve stimulation, thalamic stimulation, and corpus callosotomy.

DIET TREATMENT OF EPILEPSY

The ketogenic diet is the most established diet for the treatment of epilepsy. This diet is high in fat and low in carbohydrates and is challenging for many people to continue but can be successful in the prevention of seizures. The low-glycemic index diet is also being used in the treatment of epilepsy and it better tolerated.[30]

SAFETY ISSUES

People with epilepsy have an increased risk of injuries and accidents, particularly drowning, burns, poisoning, adverse effects of medication, and traumatic brain injury.[31] The risk of sudden unexpected death in epilepsy is approximately 1 per 1000 adults a year and 1 per 4500 children a year. The risk factors for sudden unexpected death in epilepsy include convulsive seizures and uncontrolled seizures.

Counseling regarding safety issues is a vital component of the treatment plan. It is temporarily illegal to drive after a seizure. Driving is an important safety topic.[32] The role of the physician in both reporting and determining the amount of time seizure free before driving varies from state to state, from physician discretion to 3 months to more than a year. The law of each state can be found at epilepsy.com/driving-laws.

DEPRESSION

There is a bidirectional relationship between epilepsy and depression. Patients with major depressive disorder have a 4- to 7-fold increased risk of an unprovoked seizure.[33] Patients with epilepsy have a higher rate of depression, with about 30% of people with DRE developing clinically significant depression.[34] There is an increased risk of suicide in people with epilepsy, particularly at time of diagnosis and in individuals with a history of depression. Treatment with selective serotonin and norepinephrine reuptake inhibitors (SSRIs and SNRIs) is considered safe in people with epilepsy when used at therapeutic doses.[35]

WITHDRAWING MEDICATIONS

Once patients have been seizure-free for a certain period of time (minimum 2 years, typically 4 to 5 years in adults), it is important to reassess whether or not medications can be withdrawn through a tapering process. Abrupt cessation is unsafe and the longer the patient has been seizure-free, the better their prognosis for successful withdrawal of AED. The seizure recurrence rate is on average 34%,[36] but varies depending on the history of the individual patient.[37] Recent nomograms have been published as tools to estimate the risk of seizure recurrence after AED withdrawal.[38] Important negative predictors for seizure freedom include longer duration of epilepsy, an abnormal neurologic examination, an abnormal EEG, lesion on MRI of the brain, and certain epilepsy syndromes that are known to increase the risk of recurrence (eg, juvenile myoclonic epilepsy).

SUMMARY

Epilepsy is one of the most common disorders encountered in both the inpatient and outpatient settings and impact people of all ages and backgrounds. A knowledge of the types of seizures and manifestations of seizures, diagnostic work-up, and treatment options is of critical importance to physicians. The overall goal of care is to precisely diagnose the type and cause of seizure before calling it epilepsy and then after diagnosis of epilepsy provide treatment that prevents seizures, tailoring this treatment to prevent side effects and maximize quality of life. High-quality care of patients with epilepsy also includes screening for and treating depression, and referring any patient with uncontrolled seizures to an accredited epilepsy center.

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