EPILEPSY AND RELATED PSYCHIATRIC CONDITIONS
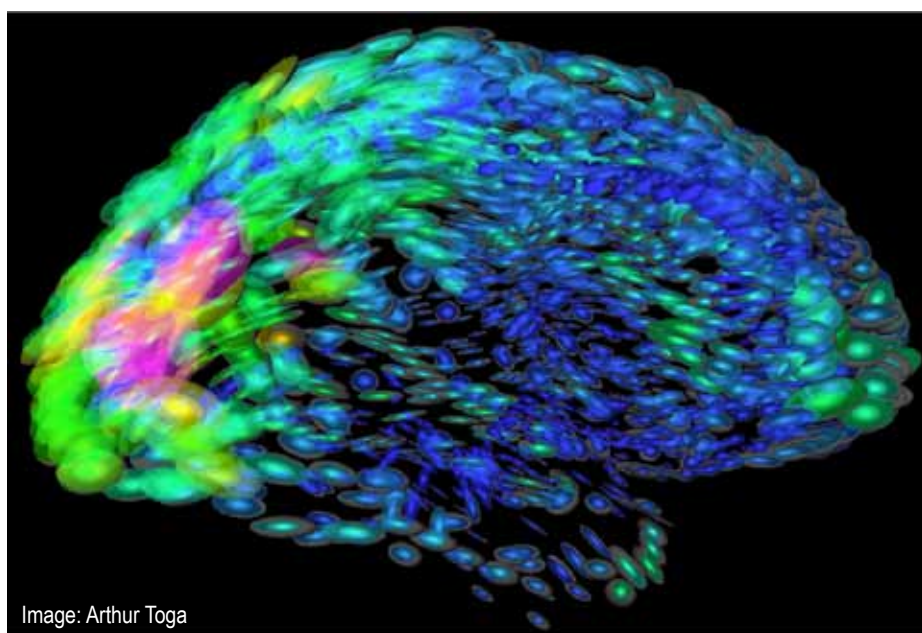
Eduardo Barragán Pérez

Image: Arthur Toga

Eduardo Barragán Pérez
MD, MSc

Pediatric neurologist, Hospital
Infantil de México, Federico
Gómez, México & Professor
of pediatric neurology,
Universidad Nacional
Autónoma de México, México

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Epilepsy (from the Ancient Greek ἐπιληψία (epilēpsía): *seizure*) is a group of chronic neurological disorders characterized by seizures, which are the result of abnormal, excessive or hypersynchronous neuronal activity in the brain (Engel, 2006). Epilepsies can be classified by the:

- Etiology (e.g., idiopathic, secondary – see side box)
- Characteristics of the seizures, such as absence, myoclonic, clonic, tonic, tonic-clonic, and atonic (Blume et al, 2001).
- Location in the brain where seizures originate:
    - Partial or focal onset seizures: simple partial (consciousness is not impaired) or complex partial (psychomotor seizure). Partial seizures may generalize (secondary generalized)
    - Generalized seizures
    - Frontal, temporal lobe
- Medical syndromes of which they are a manifestation (e.g., juvenile myoclonic epilepsy, Lennox-Gastaut syndrome)
- Event, if any, that triggers the seizures, such as reading or music.

Epilepsy is a worldwide problem that affects between 2% and 3% of the population, 75% of the cases begin before adolescence. Epilepsy can be caused by genetic, structural, metabolic or unknown factors. Among the structural factors, the most common causes in developing countries are infectious and parasitic diseases (especially neurocysticercosis), perinatal brain damage, vascular disease, and head trauma – all preventable (Barragan, 2004). The prognosis of epilepsy depends on the etiology of the illness as well as on early and sustained treatment. It is estimated that up to 70% of people with epilepsy can live normal lives if they receive proper care.

Epilepsy accounts for 0.5% of the global burden of disease, measured in disability adjusted life years (DALYs), with 80% of that burden corresponding to the developing countries; this illness accounts for 0.7% of the regional (Latin-American) burden of disease. There are no significant gender differences and the highest burden (2.8%) is found in the 5-14 age group.

The global incidence, prevalence, and mortality of epilepsy are not uniform, depending on several factors. Developing and developed countries have geographic, economic and social differences. The prevalence and incidence of epilepsy are higher in developing countries than in developed ones. However, within developing countries – even given the high incidence of epilepsy – the prevalence is relatively low, which may be due to high mortality of people with epilepsy. Prognosis in developing countries seems similar to that in developed ones. Because phenobarbital, carbamazepine and phenytoin are available and inexpensive, they are the drugs most often used in developing countries but they produce more psychiatric side effects. The most cost-effective way to decrease the treatment gap in developing countries would be to deliver epilepsy services through primary health care. All these issues have an impact on the existence of higher psychiatric comorbidity in developing countries.

EPILEPSY AND PSYCHIATRIC DISORDER

Mood, anxiety, psychosis, attention deficit hyperactivity disorder (ADHD) and autism are relatively frequent comorbidities of epilepsy. Traditionally, these

Seizure types

Idiopathic (primary)

- Seizures start during childhood or adolescence
- Genetic origen
- Good respond to pharmacological treatment
- Good prognosis,
- No brain damage

Symptomatic (secondary)

- Seizures start at any age
- Multiple etiology
- Uncertain pharmacological response
- Variable prognosis
- Usually brain damage

Click on the picture to view a short video explaining epilepsy and its types (10:42)

Table I.2.1   Prevalence of selected psychiatric disorders in young people suffering from epilepsy and in the general population

	Epilepsy sufferers (%)	General Population (%)
Depression	11-60	12-15
Anxiety	19-45	2.5-6.5
Psychosis	2-8	0.5-0.7
ADHD	25-30	2-10

The modern medical era of epilepsy began with three English neurologists: John Hughlings Jackson (pictured), Russell Reynolds, and Sir William Richard Gowers. In a seminal study, Jackson (1835-1911) defined a seizure as "an occasional, an excessive, and a disorderly discharge of nerve tissue on muscles." He also recognized that seizures can alter consciousness, sensation and behavior. Two independent teams of chemists created phenobarbital, which was marketed in 1912 by Bayer under the brand name of Luminal. Phenobarbital is the oldest antiepileptic drug and is still in clinical use.

psychiatric conditions were considered to be complications of the seizure disorder but a bidirectional relationship has now been demonstrated (Gaitatzis et al, 2004). Therefore, not only are patients with epilepsy at greater risk of developing these psychiatric disorders but patients with mood, attention deficit disorders and autism have a significantly greater risk of developing epilepsy also (Barragán & Hernandez, 2005). These bidirectional relationships suggest the existence of common pathogenic mechanisms operating in epilepsy and the major psychiatric disorders. An alternative explanation is that both epilepsy and psychiatric illness are the result of an underlying structural brain abnormality. Thus, identification of the underlying pathogenic mechanisms may shed light on the neurobiological bases of these disorders.

The existence of comorbid psychiatric disorders has a significative impact on the treatment of epilepsy (Kessler et al, 1994; Bijl et al, 1998). Epidemiological studies show that psychiatric disorders are more prevalent among people with epilepsy than in the general population (Davies et al, 2003; Devisnky, 2003) as shown in Table I.2.1. Prevalence rates in studies using ICD codes from administrative data (Bredkjaer, 1997; Gaitatzis, 2004) are highly variable due to unreliability of the recordings. Studies using structured interviews find even higher rates. However, prevalence of psychiatric conditions in clinic-based studies may be higher than in the general population because samples are biased toward individuals seeking medical attention (i.e., sicker). In summary, psychiatric comorbidity in epilepsy is high regardless of the ascertainment method used.

The presence of comorbid disorders also impacts on the response to antiepileptic drugs, particularly due to side effects, and on the quality of life of these patients. For example, a study by Tellez-Zenteno et al (2007) found that one third of patients with epilepsy had depression or anxiety, one in four had suicidal ideas and almost half had problems with attention or cognition.

Significance of the association between epilepsy and psychiatric disorder

There are several explanations for this association:
1. The comorbid psychiatric disorder is the result of an increase in psychosocial problems (e.g., stigma, impairment) associated with epilepsy
2. Repeated seizures actually increase the vulnerability for psychiatric disorder
3. Psychiatric disorder increases vulnerability for epilepsy

4. Both epilepsy and psychiatric conditions are caused by a brain abnormality

Epidemiologic studies suggest that a previous history of depression may increase the risk of epilepsy (four to seven fold), while the presence of epilepsy increases the risk of developing depression (five to 25 fold) (Kanner, 2005). This data suggest a bi-directional relationship between the two diseases. Other psychiatric disorders such as ADHD increase the risk of epilepsy 3.7 fold. A study in Wisconsin including 53 children with newly diagnosed idiopathic epilepsy using structured interviews showed that one quarter had a depressive disorder before the onset of the seizures, one quarter an anxiety disorder and one quarter ADHD.

Psychiatric comorbidity

Depression

Using DSM-IV criteria, the lifetime prevalence of depression ranges from 12% to 16% and 1-year prevalence is about 5%. Mood disorders are the most common psychiatric conditions found in people with epilepsy. For example, Grabowska-Gryzb et al (2006) found a prevalence of depression of 49.5% in 203 patients with intractable epilepsy. Rates are highest in populations with intractable epilepsy (40%-60%) but are still high in people with epilepsy in the general population (about 20%). The timely identification and treatment of depression in epilepsy is increasingly recognized as an area requiring attention (Davies et al, 2003).

Anxiety

Lifetime prevalence of anxiety disorders in the general population ranges from 2% to 5% (Hunt et al, 2002). In people with epilepsy, prevalence ranges from 11% to 15%. Tellez-Zenteno et al (2005) reported a lifetime prevalence of 13% in a Canadian general population study in adolescents using structured psychiatric interviews. Anxiety disorders seem to be more common in patients with intractable epilepsy. Regrettably, depression and anxiety do not seem to lessen in patients with intractable epilepsy who underwent surgery for epilepsy.

Psychosis

The prevalence of psychosis in the general population ranges between 1% and 2% (Johns & van Os, 2001). Psychotic symptoms in people with epilepsy may be ictal (they occur during the seizure, e.g., complex partial seizure), postictal (after having a seizure), or chronic interictal (Table I.2.2). Prevalence of interictal psychosis in non-selected epilepsy population studies varies from 3.1% to 9%. They are more frequent in adolescents but children can also show these symptoms,

Hans Berger, a German psychiatrist, discovered that it was possible to record the electric currents generated by neurons in the brain without opening the skull, and to record them on a strip of paper, which Berger named as the electroencephalogram (EEG). This discovery opened the door for a quantum leap on epilepsy diagnosis and research.

Table I.2.2   Subcategories of epileptic psychoses

- **Postictal psychosis:** psychosis that follows immediately after one or multiple seizures (mostly complex partial or secondarily generalized), occurring within one week of the last seizure
- **Acute interictal psychosis:** psychosis that develops when seizures have ceased or reduced significantly in frequency (alternative psychosis) or when seizures are unrelated to a recent increase in seizure activity
- **Chronic epileptic psychosis:** a psychotic state lasting more than six months in patients with epilepsy.

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especially children with partial complex seizures. In studies of patients with temporal lobe or refractory epilepsy, prevalence ranges from 10% to 19%, which is almost double that found in non-selected epilepsy populations (Taylor, 1972). In children, it is important to highlight that some antiepileptic drugs can trigger psychotic symptoms (e.g., topiramate, levetiracetam and phenobarbital).

By contrast, the overwhelming majority of individuals with epilepsy never experience psychotic episodes, which leads to the long disputed question of whether there is a relationship between epilepsy and psychosis (Tadokoro et al, 2007). In cases of interictal psychosis, mostly in temporal lobe epilepsy, there is a long interval (greater than 10 years since the first seizure) until the onset of psychosis. Some have suggested the link between epilepsy and psychosis is non-specific and that psychosis in patients with epilepsy occurs not as a result of the epileptic activity itself but indirectly as a sequel to non-specific damage to vulnerable parts of the brain. This contrasts with Landot's (1953) view of psychosis as the result of a paradoxical normalization of the electroencephalogram in patients with epilepsy.

ADHD

Comorbidity with ADHD is common – it occurs in as many as 30% to 50% of patients – and may cause serious social and academic impairment (Barragán at al, 2005). The association is independent of epilepsy syndrome, seizure type, age of epilepsy onset and etiology of seizures. Pharmacologic intervention in patients with epilepsy and ADHD is particularly challenging due to the potential risk for the exacerbation of seizures by the ADHD medication. Treatment of ADHD has traditionally relied on the use of psychostimulants such as methylphenidate. Unfortunately, side effects like the possibility of increasing seizure threshold and the number of seizures limit stimulant use. Atomoxetine has shown to be effective for ADHD symptoms, does not worsen seizures and is generally well tolerated. The main side effects of atomoxetine are decreased appetite, headaches, nausea and weight changes (Barrágan & Hernández, 2005). There are no interactions between antiepileptic drugs and ADHD medications.

SYNDROMES, SEIZURES, DRUGS AND MOOD

There has been considerable debate – still unresolved – as to the association between a particular type of epilepsy and depression. People with epilepsy resulting from lesions in the temporal lobe are more likely to have intractable seizures, and they are also more likely to take a larger variety of medications than those with temporal epilepsy without lesions. Overall, studies have shown that patients with temporal lobe epilepsy are more prone to depression, particularly those with temporal lobe epilepsy who had mesial temporal sclerosis. In general, there is agreement that patients with complex partial seizures (the more frequent type of seizures in temporal lobe epilepsy) are more likely to have a depressive disorder.

Antiepileptic drugs and depression

Interest on the role of antiepileptic drugs in precipitating depression has grown following the introduction of the new antiepileptic compounds (Mula & Sandeer, 2007), resulting in a revival of the concept of *forced normalization*. The antiepileptic drugs more often associated with this effect seem to be those which act at the benzodiazepine-GABA receptor complex (e.g., tiagabine, topiramate, vigabatrin, carbamazepine and valproate). It is not rare to see mood changes

Do antiepileptic drugs exacerbate or even induce ADHD symptoms?

- Barbiturates and specifically phenobarbital may induce hyperactivity especially in toddlers
- Valproate may induce irritability in newly diagnosed children
- Lamotrigine may induce hyperactivity in the developmentally disabled
- Levetiracetam may induce hostility
- Topiramate may induce both slowing and hyperactivity.

Click on the picture to view a short video about how epilepsy is diagnosed (4:30)

Forced normalization

A hypothesized phenomenon by which the sudden cessation of seizures in people with intractable epilepsy would be followed by the development of psychiatric symptoms – usually psychotic, but depression is also reported.
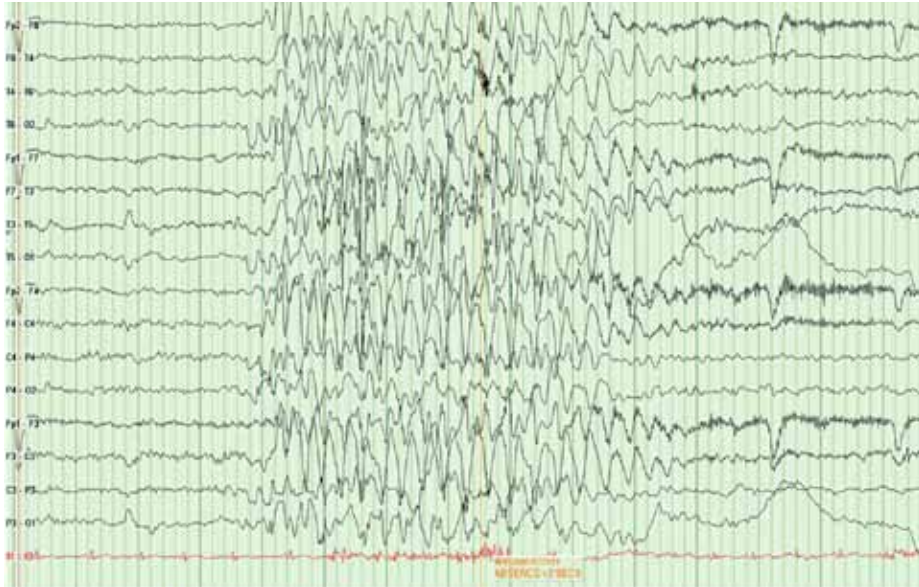
EEG recording of an absence lasting 3 seconds

in children when they take these medications, even at low doses, especially in those with temporal lobe epilepsy. Anecdotal evidence (case reports) suggest that levetiracetam can often cause mood symptoms (depression, irritability) as well as disruptive behavior, which can be ameliorated with a combination of vitamin B6 and B12. There may also be interactions between antidepressant medications and antiepileptic drugs. This largely depends on liver metabolism, thus it is important to know where medications are metabolized and whether they induce or inhibit the metabolism of other medications (see Table I.2.3). Antidepressants may

Antiepileptic medications

Older drugs

- Benzodiacepines
- Carbamazepine
- Clobazam
- Phenobarbital
- Phenytoin
- Primidone
- Valproate

Newer drugs

- Felbamate
- Gabapentin
- Lamotrigine
- Levetiracetam
- Oxcarbazepine
- Pregabalin
- Topiramate
- Vigabatrin
- Zonizamide

Table I.2.3	Interactions of antiepileptic drugs with liver enzymes		
Antiepileptic Drug	Induction	Inhibition	Enzymes
Carbamazepine	✓ (95%)		CYP3A4
Phenobarbital	✓ (75%)		
Phenytoin	✓ (90%)		CYP2C19; CYP2C9
Primidone	✓ (50%)		
Felbamate	✓ (50%)		CYP3A4
Lamotrigine	✓ (90%)		UGT (weak)
Oxcarbazepine	✓ (45%)		CYP3A4
Topiramate	✓ (40%)		CYP3A4
Felbamate		✓	CYP2C19
Oxcarbazepine		✓	CYP2C19 (weak)
Topiramate		✓	CYP2C19
valproate		✓	CYP2C19, UGT
Gabapentin			Without hepatic interaction
Levetiracetam			
Pregabalin			
Topiramate (<200 mg/day)			
Vigabatrin			

> **Table I.2.4   Common psychiatric medications and their effect on seizures.**
>
> Increase the risk of seizures:
> - High risk: bupropion, clomipramine, chlorpromazine, clozapine
> - Intermediate risk: tricyclic antidepressants, venlafaxine, thioridazine, olanzapine, quetiapine
> - Low risk: fluoxetine, sertraline, paroxetine, trazodone, haloperidol, risperidone.

increase the risk of seizures (Mula et al, 2004). Table I.2.4 lists common psychiatric medications and their effect on seizures.

BEHAVIOR PROBLEMS

Epidemiologic studies have consistently shown that children with epilepsy have higher rates of behavior problems than children with other chronic physical conditions. It is not clear when these problems begin but it is commonly assumed that they start after epilepsy is diagnosed. The reasons for these behavior problems are not well understood either. Presumed causes include:

- The effect of the seizures themselves
- Effects of medication
- Poor child and family adaptation to the illness, and
- Neurologic dysfunction that brings about both seizures and behavior problems.

With few exceptions, studies investigating behavior problems have been conducted on children who had suffered from epilepsy for many years. This makes it difficult to separate preexisting causes of behavior problems from those related to living with chronic epilepsy.

Hoare and Kerley (1991) compared psychiatric disturbance across several small groups of children. Children with new-onset epilepsy were diagnosed and treatment was initiated within three months before data collection. They found that 24% of the children either had behavior problems in the clinical range or were at risk for such; children with new-onset epilepsy had higher mean total behavior problems scores than children who had no further seizures. They hypothesized that central nervous system dysfunction might be a causal factor in both seizures and behavior problems.

If high rates of behavior problems before the first recognized seizure were to be found, then behavior problems could not be attributed to side effects of medication or to a negative emotional response to epilepsy, such as concerns about stigma. The percentage of children with previously unrecognized seizures who had behavioral problems in the clinical range (34.2%) is similar to the rate of psychiatric disturbance found in children with a brain disorder (including seizures) in the Isle of Wight study (34.3%). This finding of higher rates of behavior problems at the time of onset of seizures in children who had unrecognized epilepsy is consistent with Aicardi and Ohtahara's (2002) hypothesis that epilepsy can be a pervasive condition in children.

Thus, emotional and behavioral disturbances are common among children with epilepsy and can cause serious social and academic impairment with long-term effects. Management of the behavioral problem of these children is similar

Types of aggravation of seizures
- Re-occurrence of seizures in previously controlled patients
- Increase in the frequency or severity of pre-existing seizures
- Emergence of a new type of seizure or status epilepticus.

to that of children without epilepsy (e.g., psychoeducation, parent management training, CBT and medication). Pharmacologic intervention in patients with epilepsy and disruptive behaviors is particularly challenging due to the potential risk for exacerbation of seizures by the medication. So far, very few data exist on the efficacy and safety of typical and atypical neuroleptic medications in epileptic children. Moreover, despite the apparent consensus among physicians, no conclusive data exist on whether commonly used psychotropic drugs such as SSRIs and tricyclic antidepressants worsen seizures (see Table I.2.4), but they need to be used with caution. Antipsychotics, such as risperidone and olanzapine, and methylphenidate can be safely used in patients with epilepsy.

Most of the available information on the use of neuroleptic medications in epileptic children comes from older reports using first generation neuroleptics, such as thioridazine and haloperidol. Although effective in reducing disruptive behaviors, nowadays these drugs are prescribed less frequently because of adverse effects, both neurological and cardiovascular (thioridazine, for example, is no longer available in some countries). Second generation neuroleptic drugs such as risperidone and olanzapine cause fewer extrapyramidal symptoms. Risperidone, and sometimes olanzapine, has been shown to be very efficacious in diminishing behavioral disturbance in developmentally disabled children in the short term, in those with autism and pervasive developmental disorder as well as in patients with epilepsy, without an increase in the number of seizures (Barragán et al, 2005).

Click on the picture to access the NICE guidance on the diagnosis and management of the epilepsies in adults and children.

SLEEP AND EPILEPSY

Since Aristotle and Hippocrates noted the occurrence of epileptic seizures during sleep, the relationship between sleep and epilepsy has intrigued physicians and researchers. Sleep is an example of a physiologic state capable of modulating seizures. The influence of sleep on epilepsy is supported by the observation that in specific epileptic syndromes seizures occur exclusively or primarily during non-rapid eye movement (NREM) sleep. In almost all epileptic syndromes, interictal epileptiform discharges are more prevalent during NREM sleep and less prevalent during rapid eye movement (REM) sleep.

The proportion of patients who have seizures that occur exclusively or predominantly during sleep ranges from 7.5% to 45% in several series studying sleep-related epilepsy. This wide variation may reflect differences among patient populations, with seizures more likely to occur during sleep in certain epileptic syndromes. Frontal lobe seizures are more common during sleep and temporal lobe seizures more common during wakefulness (Commission on Classification and Terminology of the International League Against Epilepsy, 1989). Herman and coworkers (2000) analyzed 613 seizures in 133 patients with partial seizures who underwent video-EEG monitoring finding that 43% began during sleep, the majority during stages 1 and 2 NREM sleep and none during REM sleep.

As the clinical manifestations of *nocturnal frontal lobe seizures* often include prominent tonic or motor manifestations, they are more likely to be noticed by the patient or family than complex partial seizures of temporal lobe origin – complex partial seizures that begin focally and impair consciousness are the predominant seizure type in temporal lobe epilepsy. However, the brevity, the minimal or lack of postictal confusion, the apparently psychogenic features (such as kicking, thrashing and vocalizations), and the frequently normal interictal and ictal recordings may
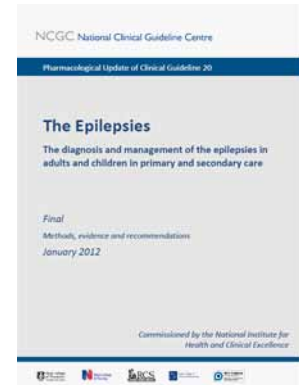
complicate diagnosis. Nocturnal seizures may suggest diagnoses of sleep terrors, REM sleep behavior disorder, psychogenic spells, or nocturnal paroxysmal dystonia.

Seizures originating in the sensorimotor area may be mistaken for psychogenic spells because of thrashing behavior, preservation of consciousness, absence of postictal confusion, and absence of interictal or ictal EEG activity. Diagnostic features supporting sensorimotor area seizures include (1) short duration (less than 30 seconds to a minute), (2) stereotyped nature, (3) tendency to occur predominantly or exclusively during sleep, and (4) tonic contraction of the arms in abduction. Psychogenic spells usually are of longer duration (one to several minutes), are nonstereotypic, and occur when the patient is awake or drowsy.

Nocturnal paroxysmal dystonia. This syndrome, initially termed hypnogenic paroxysmal dystonia and subsequently, nocturnal paroxysmal dystonia, is characterized by brief (15-45 seconds) stereotyped motor attacks consisting of dystonic posturing, ballistic or choreic dyskinesias and vocalizations during NREM sleep without clear ictal or interictal EEG changes.

Differential diagnosis

The differentiation of nocturnal seizures from non-epileptic spells during sleep can be challenging. First, in partial seizures occurring during wakefulness, patients may report postictal confusion or recall the beginning of a seizure (aura) that precedes loss of consciousness. These elements of the history support the diagnosis of epilepsy and frequently are absent in seizures occurring during sleep. Second, nocturnal events may not be observed properly. Bed partners may not be present or, if present, may not be fully awake and coherent. Complex partial seizures of temporal lobe origin in particular may lack vigorous motor activity and may fail to wake the bed partner. Third, a variety of sleep disorders are characterized by vigorous movements and behaviors that mimic seizures. Finally, certain types of seizures, particularly those of frontal lobe origin, are manifested by bizarre movements suggestive of a psychiatric disorder, including kicking, thrashing, and vocalizations. These epilepsies may be associated with normal ictal and interictal EEGs and normal imaging studies, making definitive diagnosis difficult.

Arousal disorders

NREM arousal disorders include a spectrum of confusional arousals, somnambulism (sleepwalking), and night terrors. These three disorders share the following features:

- They usually arise from NREM sleep stages 3 or 4 and, therefore, occur preferentially in the first third of the sleep cycle
- They are more common in childhood, and
- There is often a positive family history, suggesting a genetic component.

Broughton (1968) contrasted *confusional arousals* – characterized by body movement, autonomic activation, mental confusion and disorientation, and fragmentary recall of dreams – with the nightmares of REM sleep (in which subjects became lucid almost immediately and usually recall dreaming).

Somnambulism is a related NREM arousal disorder in which patients may wander out of the bedroom or house during confusional episodes. *Night terrors* begin with an intense scream followed by vigorous motor activity. Children often

are inconsolable and completely amnestic for the event. The subject appears to be awake but is unable to perceive the environment. If mental activity preceding the event is recalled, the images are simple (e.g., face, animal, or fire) compared with the complex plots of REM nightmares. Patients often report an oppressive feeling, such as being locked up in a tomb, or having rocks piled on their chests. Intense autonomic activation results in diaphoresis, mydriasis, tachycardia, hypertension, and tachypnea. In contrast to seizures, NREM arousal disorders are less stereotyped and commonly occur in the first third of the night. Patients with REM-sleep behavior disorders often present with vigorous motor activity during sleep and may injure themselves or their bed partners.

Sleep-related movement disorders

Movement disorders occurring during sleep that may resemble seizures include periodic limb movements, sleep-onset myoclonus, bruxism, and rhythmic movement disorder.

- *Periodic limb movements* in sleep may result in vigorous kicking or thrashing. A history of restless legs syndrome is commonly elicited. In contrast to seizures, periodic limb movements occur at periodic intervals (usually every 20 to 40 seconds) and involve a characteristic flexion of the leg, although the upper extremities occasionally may be involved.
- *Sleep-onset myoclonus*, also known as sleep starts, sleep jerks, or hypnic jerks, is a normal physiologic event occurring at the transition from wakefulness to sleep, often associated with sensory phenomena, including a sensation of falling. In contrast to myoclonic seizures, sleep-onset myoclonus is limited to the onset of sleep.
- *Bruxism*, stereotyped teeth grinding resembling the rhythmic jaw movements of epilepsy, may lead to excessive tooth wear, which does not occur in epilepsy.
- *Rhythmic movement disorder*, also known as head banging or body rocking, can occur during any sleep stage. It is manifested in a variety of ways, including recurrent banging of the head while the patient is prone or rocking of the body back and forth while on hands and knees. Vocalizations may accompany the repetitive movements. Rhythmic movement disorder can occur at any age, although it is more common in children than adults and is associated with mental retardation. Although complex partial seizures, particularly those of frontal lobe origin, may include similar behaviors, bilateral body rocking is more characteristic of rhythmic movement disorder. Body rocking may also occur in psychogenic seizures.

Psychiatric symptoms

Psychiatric symptoms during sleep that resemble seizures include panic attacks, post-traumatic stress disorder symptoms and psychogenic seizures. Some patients who have panic disorder present exclusively or predominantly with panic attacks that cause multiple abrupt awakenings. Symptoms on awakening include apprehension and autonomic arousal with palpitations, dizziness and trembling. In contrast with REM sleep nightmares, dreams are not recalled. In contrast with night terrors, which arise during deep NREM sleep, sleep panic attacks usually occur in the transition from NREM stages 2 to 3. Although a history of daytime panic attacks can be useful diagnostically, panic attacks may occur exclusively during sleep. An abrupt return to consciousness and autonomic arousal is more characteristic of panic disorder than of seizures, although these features may occur

in seizures. Simple partial seizures of parietal lobe origin may manifest occasionally as panic symptoms.

In post-traumatic stress disorder, repetitive rocking or head banging may occur, and the characteristic nightmares or flashbacks may arise at any stage of sleep. In contrast to seizures, patients often recall the traumatic experience. Psychogenic seizures may occur while the patient *appears to be* asleep. Diagnosis of these non-epileptic events is supported by the presence of a well-organized posterior alpha rhythm immediately before the onset of clinical changes despite the patient appearing to be asleep and the lack of ictal or postictal EEG changes. Provocative testing with suggestion may be helpful in confirming the diagnosis of psychogenic seizures.

QUALITY OF LIFE IN PEDIATRIC PATIENTS WITH EPILEPSY

One Latin-American study including more than 200 patients between six and 18 years of age showed that the quality of life of these patients was fair (López-Rojas et al, 2010). Almost half of the patients felt stigmatized. Income, the number of antiepileptic drugs and their cost, school performance and stigma were the factors which influenced quality of life. This is similar to results in other regions of the world (Devinsky & Penry, 1993).

The type of seizure is important. The principal neurocognitive difference between the two types of focal epilepsy (frontal *vs* temporal) is IQ (mean IQ of 82 in frontal epilepsy and 97 in temporal epilepsy), with more impact in the working memory and visuospatial performance in children with frontal epilepsy. Patients with temporal lobe epilepsy have more problems with attention and long term memory tasks (Barragán et al, 2006).

ADHD, cognitive decline and academic performance is one of the principal mental health problems worldwide in childhood epilepsy. In another Latin-American study, important differences between countries were noted, especially in the severity of ADHD, academic performance (lower in Central American countries) and better response to treatment in countries with a better education system.

Click on the picture to view a short video about living with epilepsy (4:21).

TREATMENT

Most low income countries have access to the four basic antiepileptic drugs (phenobarbital, phenytoin, carbamazepine, and valproate) but only at secondary and tertiary levels of care. Ensuring supply of these four medications is essential, taking into account that seizures can be controlled with monotherapy regimens in up to 70% of cases. In 1990, the WHO determined that the average cost of treatment (with phenobarbital) could be as low as US$ 5 per patient per year (World Health Organization, 2006). Public health services in most of these countries do not have access to the new-generation antiepileptic drugs, useful in treatment resistant cases but more expensive.

In the integrated management of people with epilepsy, consideration of psychosocial factors is essential and includes education for self-management; that is, measures and behaviors that people with epilepsy should adopt and maintain to control their illness. In some cases, complementary and alternative medicine

can also be helpful; including, for example, natural remedies, vitamins, relaxation techniques, a healthy diet, religious or cultural activities, and social support. Such measures are acceptable as long as the patient continues taking the basic drug therapy, which treats not only the seizures but also the psychiatric comorbidities.

Rehabilitation

The objective of rehabilitation is to improve the quality of life of people with epilepsy and help them integrate into society and work. The interventions selected will depend on the complexity of each case. Most people with epilepsy enjoy substantial autonomy and only a minority suffers from severe forms of the illness. This group includes persons with disabilities and people who are institutionalized or highly dependent on their families, in whom developing social and occupational skills is essential.

As already highlighted, there is a high proportion of psychiatric conditions comorbid with epilepsy, often unrecognized and therefore not treated properly. These disorders include depression, anxiety and psychosis, as well as cognitive and personality changes. Epilepsy associated with psychosis or dementia is often confined to psychiatric hospitals or social welfare institutions.

REFERENCES

Aicardi J, Ohtahara S (2002). [*Epilepsies séveres du nouveau-né avec tracé de "suppression-burst"*]. In Roger J, Bureau M, Dravet CH et al (eds) [*Les syndromes épileptiques de L'enfant et de L'adolescent*] 3rd ed. London: John Libbey & Co; pp33-44.

Barragan E (2004). [*Daño Neurológico y Epilepsia: Estrategia y Herramientas Multidisciplinarias de Apoyo*] 2nd ed. México DF: Editorial Linae.

Barragán E, Cruz E, Urrutia M et al (2006). Neurocognitve differences between frontal and temporal seizures in pediatric epilepsy. *Epilepsia*, 47(sup4):206.

Barragán E, Cruz E, Urrutia M et al (2006). Prevalence of attention deficit disorder in paediatric epilepsy patients in mexico. *Epilepsia*, 47(sup4):179.

Barragán Pérez E, Garza Peña A, Benavides Guerrero O (2005). [Eficacia y seguridad de la risperidona en el tratamiento agudo delas conductas disruptivas en pacientes pediátricos con epilepsia]. *Boletín médico del Hospital Infantil de México,* 62:421-427.

Barragán E, Hernández J (2005). Effycacy of atomoxetine treatment in children with ADHD and epilepsy. *Epilepsia*, 46 (sup6): 718.

Barragán E, Hernandez J (2005). ADHD and epilepsy, EEG and comorbidity. *Epilepsia*, 46:421.

Bijl RV, Ravelli A, van Zessenet G (1998). Prevalence of psychiatric disorder in the general population: results of the Netherlands Mental Health Survey and Incidence Study (NEMESIS). *Social Psychiatry & Psychiatric Epidemiology*, 33:587-595.

Bredkjaer SR, Mortensen PB, Parnas J (1997). Epilepsy and non-organic non-affective psychosis. National epidemiologic study. *British Journal of Psychiatry*, 172:235-238.

Blume WT, Lûders HO, Mizrahi E et al (2001). Glossay of descriptive terminology for ictal semiology: Report of the ILAE Task Force on Classification and Terminology. *Epilepsia,* 42:1212-1218.

Broughton RJ (1968). Sleep disorders: disorders of arousal? *Science*,159:1070-1078.

Commission on Classification and Terminology of the International League Against Epilepsy (1989). Proposal for revised classifi cation of epilepsies and epileptic syndromes. *Epilepsia*, 30:389-399.

Davies S, Heyman I, Goodman R (2003). A population survey of mental health problems in children with epilepsy. *Developmental Medicine and Child Neurology*, 45:292-295,

Devisnky O (2003). Psychiatric comorbidity in patients with epilepsy:implications for diagnosis and treatment. *Epilepsy & Behavior*, 4 (sup 4):S2-10.

Devinsky O, Penry JK (1993). Quality of life in epilepsy: the clinician´s view. *Epilepsia*, 34(sup4):S4-7.

Engel J (2006). Report of the ILAE Classification Core Group. *Epilepsia*, 47:1558-1568.

Gaitatzis A, Carroll K, Majeed A et al (2004). The epidemiology of the comorbidity of epilepsy in the general population. *Epilepsia*, 45:1613-1622.

Gaitatzis A, Trimble MR, Sander JW (2004). The psychiatric comorbidity of epilepsy. Acta Neurologica Scandinavica, 110:207-220.

Grabowska-Gryzb A, Jedrzejcak J, Naganska, et al (2006). Risk factor for depression in patients with epilepsy. *Epilepsy & Behavior*, 8: 411-417.

Herman BP, Seidenberg M, Bell M (2000). Psychiatric comorbidity in chronic epilepsy: identification, consequences and treatment of major depression. *Epilepsia,* 41(sup2):S31-41.

Hoare P, Kerley S (1991). Psychosocial adjustment of children with chronic epilepsy and their families. *Developmental Medicine & Child Neurology*, 33:201-215

Hunt C, Issakidis C, Andrews G (2002). DSM-IV generalized anxiety disorder in the Australian National Survey. *Social Psychiatry and Psychiatric Epidemiology*, 35:531-538.

Johns LC, van Os J (2001). The continuity of psychotic experiences in the general population. *Clinical Psychology Review*, 21:1125-1141.

Kanner AM (2005). Depression in epilepsy: a frequently neglected multifaceted disorder. *Epilepsy & Behavior*, 4 (sup 4):s11-9.

Kessler RC, McGonagle KA, Zhao S et al (1994). Lifetime and 12 month prevalence of DSM-III-R psychiatric disorders in US. *Archives of General Psychiatry*, 51:8-19.

Landolt H (1953). *Einige klinisch-elektroencephalographische Korrelationen bei epileptischen Dämmerzuständen. Neververarszt* , 24: 479.

López-Rojas V, Barragán-Pérez EJ, Hernández-Hernández M et al (2010). [Calidad de vida en pacientes adolescentes con epilepsia en México]. *Revista Médica Hondureña,* 78:78-82.

Mula M, Sandeer J (2007). Negative effects of antiepileptic drugs on mood in patients with epilepsy. *Drug Safety*, 30:555-567.

Mula M, Monaco F,Trimble MR (2004). Use of psychotropic drug in patient with epilepsy: interactions and seizure risk. *Expert Review of Neurotherapeutics*, 4:953-964.

Qin P, Xu H, Laursen T, et al. Risk for schizophrenia and psychosis among patients with epilepsy: population based cohort study. *British Medical Journal*, 331:23.

Taylor DC (1972). Mental state and temporal lobe epilepsy. *Epilepsia*, 13:727-765.

Tadokoro Y, Oshima T, Shimizu H et al (2007). Interictal psychoses in comparasion with schizophrenia, a prospective study. *Epilepsia,* 48:2345-2351.

Tellez-Zenteno J, Dhar R, Hernandez-Ronquillo L et al (2007). Long term outcomes in epilepsy surgery: antiepileptic drugs, mortality, cognitive and psychosocial aspects. *Brain*,130:334-345.

Tellez Zenteno JF, Wiebe S, Pattern SB (2005). Psychiatric comorbidity in epilepsy: a population based analysis. *Epilepsia*, 46:264-265.

World Health Organization (2006). *Trastornos Neurológicos: Desafíos Para la Salud Pública.* Ginebra, Suiza: World Health Organization.