

REVIEW

Epilepsy in Children and Adolescents: What a Psychiatrist Should Keep in Mind

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ABBREVIATIONS:

EEG = Electroencephalogram
ILAE = International League Against
Epilepsy
NCSE = Non Convulsive Status
Epilepticus
CSE = Convulsive Status Epilepticus

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ABSTRACT

Epileptic seizures can be mistaken for psychiatric disorders. Complex partial seizures may present with changes in autonomic nervous system function, somatosensory auras, difficulties in speech attention or movement, stupor, intense motor behavior, posturing, aimless wandering, changes in perception of place time or persons, illusions and hallucinations. These phenomena are not usually accompanied by loss of consciousness. Temporal epilepsy exhibits symptoms, such as headache, irritability, insomnia, personality changes, auras, déjà vu - jamais vu, dreamy states, feelings of fear and strangeness, automatisms, tonic-dystonic posturing of extremities, transpiration, dizziness, faintness, tachycardia, hypoxia, disordered consciousness. The seizure may be followed by confusion, tiredness, disorientation, headache, automatisms, impaired attention, memory and learning, hyperactivity, oppositionality, panic attacks, dissociation, and obsessive-compulsive symptoms. In frontal lobe epilepsy, three types of seizures may be mistaken for psychiatric problems: hyperkinetic seizures, tonic seizures of complementary movement area, and absences. The complex and emotionally charged behavior of hyperkinetic seizures, and occasionally of tonic seizures, combined with a normal EEG, may lead to the misdiagnosis of a psychiatric disorder such as psychosis, dissociation, conversion disorder, or panic attacks. In parietal and occipital seizures, visual hallucinations and distortion of visual perception may be mistaken as psychotic symptoms. Sensory auras resemble simple hallucinations. Psychic auras are complex hallucinatory experiences that comprise autoscopic phenomena, a sense of someone's presence, déjà vu – jamais vu, fear and elation. Limbic seizures are associated with the emergence of psychiatric symptoms. Non-convulsive status epilepticus (NCSE), can last for hours, days, or weeks and presents with change in behavior and mental status that ranges from mild changes, to psychiatric manifestations, delirium or coma. Antiepileptic drugs also have an impact on mental functioning and mood. Child and adolescent psychiatrists need to be informed about epileptic semiology that imitates psychiatric disorders.

INTRODUCTION

The boundaries between neurological and psychiatric disorders are not always clear, while comorbidity is high.¹ Moreover, certain epileptic phenomena with an unusual clinical presentation can be mistaken for a psychiatric disorder.² Many Child and Adolescent Psychiatrists have observed cases with atypical clinical presentation,

either comorbid with epilepsy, or accompanied by seizures that began after the onset of the mental disorder. In such instances, a psychiatrist needs to differentiate between epileptic phenomena and psychiatric illness.

The purpose of this selective review of the literature article is to assist the diagnostic process of complex neuropsychiatric problems and facilitate communication and collaboration between neurologists and psychiatrists, emphasizing the role of the brain as a biological substrate of psychiatric events. It describes various types of epileptic phenomena that may be mistaken for psychiatric disorders.

Data were obtained from PubMed database using the terms epilepsy, temporal, frontal, parietal, occipital, limbic, status epilepticus, EEG, antiepileptic drugs, children, adolescent and psychiatric. Recent review articles were selected, as well as terminology and taxonomy articles and large studies concerning semiology, diagnosis, or other significant issues. The bibliography cited in selected articles and chapters from the books "Imitators of epilepsy, second ed." and "Pellock's Pediatric Epilepsy: Diagnosis and therapy. 4th ed." were manually searched for relevant content.

TERMINOLOGY

Epilepsy is a set of different disorders that share the common characteristic of increased susceptibility to seizures.³ Epileptic seizure is defined as a transient episode of signs and symptoms, caused by abnormal brain activity. Specific types of crises are diagnostic entities with etiological, therapeutic and prognostic implications.⁴

The most widely accepted classification system is that proposed by the ILAE (International League Against Epilepsy) which differentiates epilepsy depending on the etiology and type of seizure.⁴ The classification of epileptic phenomena of 1981 (International Classification of Epileptic Seizures) and 1989 (International Classification of Epilepsies, Epileptic Syndromes and Related Seizure Disorders) is based on terms used before current neuroimaging, genetics, and molecular biology practices.⁵ Advanced diagnostic technology resulted in changes to the existing terminology and the way epilepsy is conceptualized.^{4,6,7} In 2001, the ILAE Task Force of Classification and Terminology published a report on semiology in epilepsy. This descriptive and phenomenological glossary does not imply or require knowledge of ictal pathophysiology or etiology.⁷ It is particularly helpful to psychiatrists in terms of describing clinical phenomena and improving communication with neurologists. The most recent classification of seizure types is that of ILAE, 2016. This new classification does not represent a fundamental change, but allows greater flexibility and transparency in naming seizure types.

The diagnostic scheme proposed by ILAE, includes five axes: Axis 1: Description of ictal phenomenology, Axis 2: Di-

agnosis of specific seizure types, Axis 3: Diagnosis of a specific syndrome, Axis 4: Etiology, Axis 5: Assessment of impairment

In this article, older terminology is used, as found and referenced in the existing literature. The term "focal" defines seizures that remain confined to a single cerebral hemisphere.⁵ The term "generalized" describes seizures involving both cerebral hemispheres.⁵ The first clinical signs of the seizure are key to identifying the focus of initial activation.⁸ The causes of abnormal discharges may be static or progressive. Epilepsies are categorized as genetic (idiopathic), structural-metabolic (symptomatic) and of unknown origin (cryptogenic).^{5,9}

Diagnosis is principally clinical: it requires clinical examination, accurate descriptions of the event (both from the patient and from witnesses) and a history of past seizures, risk factors (family history of epilepsy, history of head trauma, brain damage, febrile convulsions) and neurological, psychiatric or cardiological disorders. Recording and neuroimaging techniques are also important.

DIAGNOSTIC VALUE OF EEG

The electroencephalogram (EEG) is a key tool in the diagnosis and classification of epileptic seizures and syndromes.⁶ However, a single EEG can neither confirm nor exclude epilepsy.¹⁰

The sensitivity of the EEG (the extent to which it can identify true positive cases) depends on the strategy used. Typical EEG of 30' duration using standard activation procedures (hyperventilation, intermittent light stimulation) can reveal epileptiform discharges in 50% of adults with newly diagnosed epilepsy.¹⁰ Sequential EEGs, in conjunction with prolonged recordings during sleep, increase the yield to 92%, while continuous EEG-recording is even more effective. High performance is also achieved with simultaneous video and EEG recordings (video-EEG).¹⁰

The prevalence of epileptiform EEG abnormalities in healthy subjects is less than 1%. It is higher in patients with cerebral dysfunction, reaching 17% in patients with congenital or perinatal brain lesions, 8% in those with brain tumors, 6% in cases of intellectual disabilities and 2.6% in psychiatric patients.¹⁰ The epileptiform activity recorded between seizures is not a reliable indicator for the classification of seizure type.¹⁰

SEIZURE TYPES

TEMPORAL LOBE SEIZURES

Temporal lobe seizures represent the most common type of focal epilepsy in adults (30%), but are less common in children (8% of childhood epilepsies). Phenomenology is also different, especially at younger ages.¹²

In adults they are characterized by the presence of epi-

gastric aura, automatisms, posturing, activity interruption, and alteration of mental status.^{12,13} Seizures may be accompanied by psychiatric manifestations.¹¹ Experiential phenomena may occur, with perceptual, mnemonic and emotional characteristics.¹⁴ Auras usually precede seizure manifestations by five to ten minutes. Automatisms consist of involuntary, coordinated, repetitive and stereotypical motor activity; they are commonly accompanied by an altered level of consciousness, and are followed by amnesia of the event.¹¹ Such preictal activity may continue stereotypically during the seizure.⁷

In children younger than three years of age, temporal lobe seizures present a clinical picture of generalized epilepsy. Automatisms are rare and simpler. Children aged 3-6 years may experience dystonic postures, turning of the head, eyes or mouth deviation, complex automatisms such as lip smacking, staring, gaze fixing, and applause (hand clapping). In children older than 6 years, the clinical picture resembles that of adults. Prodromal symptoms, including headache, irritability, insomnia, personality changes, or a feeling of imminent evil, can occur hours or days before the onset of the seizure.¹³ Auras, described as olfactory, gustatory, auditory, visual and somatosensory alterations, are more common in older children. Other phenomena, such as *déjà vu*, *jamais vu*, dream-like situations, and feelings of paradox or fear, may also be observed.^{8,13} Automatisms are expressed as oral-alimentary movements, such as lip-smacking, chewing and swallowing, searching or collectible hand gestures, fidgeting, undressing, or purposeless movements. Vocalization is common. Speech may be reduced, or completely cease. There may be tonic or dystonic posture of a limb. Simultaneous loss of consciousness is associated with bilateral involvement of limbic structures.¹³

Temporal epilepsy may manifest with abdominal pain (lasting 10 to 15 min) and vomiting, accompanied by headache, impaired consciousness, dizziness, sweating, fainting and temporary loss of vision.¹³ Tachycardia during the crisis is reported in 98% of cases of childhood epilepsy. Hypoxia is common (50% of children have saturation <90% and occasionally < 60%).¹³ In 6% of cases, hyperkinetic seizures are present, expressed with tonic or dystonic limb posture, violent vocalizations, complex movements of the limbs, trunk rotation and pelvic thrust.¹⁵ There may be signs of post-ictal confusion, fatigue, disorientation, headache and continuing automatisms.

Cognitive functions are often affected, especially reading, memory and learning. Temporal epilepsy in children often coexists with developmental delay, attention deficit and hyperactivity, oppositional behavior, obsessive compulsive symptoms, dissociative events, fugue, amnesia and panic attacks.^{9,16} Many studies report increased psychiatric symptoms in patients with temporal lobe epilepsy.³ Participation of the limbic system appears to play an important role in the occurrence of psychiatric disorders.

Temporal epilepsy may be of unknown etiology or attributed to congenital or acquired causes including hippocampal

sclerosis, infection, tumors or vascular malformations. It can be triggered by environmental factors such as fever, alcohol, or drugs.^{13,17} Neuroimaging studies show abnormal findings in half of the cases. Intracranial stereo-electroencephalography recordings reveal prolonged epileptic discharges in the hippocampus and other medial-temporal structures, even in cases of normal EEG findings.²⁰

FRONTAL LOBE SEIZURES

Frontal lobe epilepsy is the second most common type of focal epilepsy in both adults and children.^{21,22} In children, 75% of cases are of unknown etiology and 40% are resistant to treatment.²² Frontal lobes regulate important functions, such as mobility, urination and defecation control, ocular movements, attention, working memory, organization, planning, scheduling, selection of targets and behavior regulation, activity initiation and control, response inhibition, motivated behavior and social skills.²² Hence, frontal seizures can affect both cognitive skills and behavior. Changes in mood, vigilance, motor activity and problems of attention and social interaction are common.²² Behavioral disturbance may be the only seizure manifestation and may also appear after the seizure or between two seizures.²² There have been reported cases of interictal psychosis with delusional thinking, paranoia, depression, and brief stereotypical events of excitation.²²

Frontal lobe seizures are brief, rarely lasting more than one to two minutes. They occur in clusters and are often sleep-related.²³ The incidence of status epilepticus is higher in both convulsive and non-convulsive frontal lobe seizures. Seizures are usually preceded by an aura. Many patients report somatosensory aura symptoms, localized in the chest or epigastrium, diffuse sensations in the head, dizziness, numbness, tightness, heat, shortness of breath and palpitations.²³⁻²⁵ Complex auditory hallucinations, visual hallucinations, aggressive behavior and feelings of fear have also been documented.^{26,27,31} Seizures arising from the orbitofrontal cortex can present with an unpleasant olfactory aura. Autonomic nervous system involvement produces symptoms related to the cardiovascular system (tachycardia, bradycardia, arrhythmia, asystole), respiratory system (hyperventilation, apnea, dyspnea, wheezing), gastrointestinal system (epigastric aura, spitting, vomiting, defecation), urogenital system (urge to urinate, sexual/orgasmic aura, sexual automatisms) the skin (piloerection, pale skin, redness) and eyes (miosis, mydriasis).²³ Other symptoms may include intense vocalizations or difficulty in verbal communication.

Three types of frontal lobe seizures are commonly confused with psychiatric problems: paradoxical hyperkinetic seizures, tonic seizures of the supplementary motor area and frontal lobe absences (spike-wave stupor).²³⁻²⁵

a) Paradoxical hyperkinetic seizures

These seizures are characterized by motor excitation accompanied by intense emotional behavior.²⁵ They tend to

occur during sleep, but may also occur during wakefulness. Consciousness is usually preserved. They are often preceded by an aura (fear, somatosensory phenomena, or undefined feelings).²⁸ Typical features include repetitive movements, bizarre gestures and automatisms. The movements are wide in range, involve trunk and proximal limb segments, and are stereotypically repeated in every seizure attack.^{8,28}

Patients jump out of bed, make circular limb movements, run, kick, knock objects, and thrust body parts.^{23,28} Violent or rhythmic movements of the pelvis and legs, and rhythmic manipulations of the groin and genitals, can suggest sexual activity or exhibitionism.⁸ Although awake, the patients cannot control their motor behavior. Motor behavior is often accompanied by vocal outbursts (screaming, laughing, growling, barking, verbal profanities) and urinary incontinence. Visual and auditory hallucinations or delusions have also been reported.²⁵ A particular type of hyperkinetic seizure is that of meaningless wandering, accompanied by irritability, depression and dizziness.²⁹ Ictal and interictal EEG may be uninformative, especially when seizures originate from the deep-seated cortex.

b) Tonic seizures

Tonic seizures usually begin in childhood. Clinical manifestations do not differ between children and adults. They occur at night, usually in clusters, at a frequency of up to 100 seizures a night.²³ In 50% of cases they are preceded by an aura, which is generally somatosensory, i.e. pulling, numbness, heaviness and tingling sensation.²⁵ They are characterized by an abrupt onset of bilateral asymmetric tonic limb posturing; for example, one arm is extended while the other is abducted and flexed in a “fencer” posture. Equally common is bilateral symmetrical tonic posturing of the arms or legs.^{23,25,28} The seizure lasts for 15 to 40 seconds and consciousness remains clear. A progressively lower level of consciousness and reduced memory is observed in seizures of longer duration. In a quarter of cases, the initial tonic phase is followed by movements of the trunk, pelvis and legs.^{25,28} Speech arrest is common and, one third of cases are accompanied by explosive vocal utterances.^{23,25,30} Usually, the focus lies in the supplementary sensorimotor area, which is located in the medial surface of the frontal lobes.²¹

c) Absences

Absence seizures are a type of generalized epilepsy. They present as staring spells and can persist, lasting minutes, hours, or even days.^{23,25} Patients show variable responsiveness: they can be completely unresponsive, in a trance-like state, move slowly, seem confused, disoriented, and exhibit automatisms.^{23,31} Absences often result in generalized convulsions triggered by hyperventilation.^{23,31} The EEG is always abnormal.

Diagnostic assessment of frontal lobe seizures

The exact description of the seizure is of paramount importance for diagnosis. The skilled clinician can identify

frontal lobe epilepsy from the characteristic clinical picture.³²

A large part of the frontal lobes is inaccessible to simple EEG. Case series report seizure EEG's without abnormalities in 33-36% of patients.²⁴ A single EEG detects abnormalities in only 29%-55% of patients.²⁴ Consequently, a normal EEG does not necessarily exclude the diagnosis of epilepsy.²¹ Also, a normal EEG, combined with the preservation of consciousness, may lead to incorrect diagnosis of a “psychogenic” seizure or psychiatric disorder. Epileptiform discharges between seizures occur in 60%-80% of cases.²⁸

The examination of choice is video-EEG. Any odd, stereotypically repeated behavior should be further investigated with 24-hour video recording and simultaneous EEG. Magnetoencephalography can also prove helpful. High-resolution magnetic resonance imaging (T3-weighted MRI) is the imaging method of choice, although it does not always reveal pathological findings. Other imaging methods (FDG-PET, SPECT) are performed only in the case of brain surgery.²⁴

Complex and emotionally colored behavior, coupled with absent EEG findings, often leads to the diagnosis of a psychiatric disorder (e.g. psychogenic non-epileptic seizure, dissociative episode, conversion disorder, obsessive compulsive disorder, or panic attacks).^{24,32}

Parasomnias, particularly sleep terrors, should not be confused with nocturnal frontal lobe epilepsy.^{24,32,33} Appropriate diagnostic tools include video-EEG monitoring and EEG-polysomnography.

PARIETAL LOBE SEIZURES

Parietal lobe functions are related to body sensations, the formation of body image, integration of sensory input and body relation to its surroundings, language, planned movements (e.g. writing) and mathematic skills.³⁴

Epilepsy of the parietal lobe is infrequent, accounting for just 5% of all epilepsies. Because of the extensive connections of parietal lobes with other brain regions, the identification of the initial focus is difficult with a single scalp-EEG.³⁵ Moreover, seizures are often electrically silent. EEG between seizures may be normal or show intermittent bradyrhythmic characteristics.³⁶ Video-EEG monitoring is a useful diagnostic tool.

The onset of seizures has been reported to occur before 7 years of age in 21-47% of cases.³⁷ The duration of seizures ranges from seconds to a few minutes. In children, the frequency of seizures can reach 100/day.³⁷

Seizures present as sensations. All sensory modalities may be represented. Somatosensory auras are common, characterized by numbness, pain, warmth, tingling or electricity sensations. More rarely, a genital sensation or even orgasm can occur. There may be vertigo, a sense of posture deformation or of a body part's absence, or a feeling of inability to move. Realistic visual hallucinations and distortion of visual perception (objects appear too large, too small, too near or to be moving) are common. There may be difficulty in speech, comprehen-

sion, reading, or math skills. Auras of emotional type are also frequent. Kinetic symptoms are common and include tonic, tonic-clonic, or myoclonic seizures and oral automatisms.³⁷

Compared with adults, children aged 3 months to 7 years have more frequent and shorter seizures, expressed with subtle signs such as smile, redness, head-nodding and changes in behavior.³⁷

OCCIPITAL LOBE SEIZURES

Occipital seizures arise from the occipital lobe and spread to other brain regions. The pattern of seizures is mainly focal, without impairment of awareness. Symptoms are mainly visual and include visual hallucinations, darkening of the visual field, scotoma, hemianopsia, blurred vision and diplopia.³⁸ Head-turning, tonic gaze deviation, vertigo, nystagmus and eyelid blinking are observed.³⁸ Vomiting and complex automatisms (psychomotor and psychoparetic episodes) are due to secondary spread to other brain structures.

Visual hallucinations are simple or complex. Simple visual hallucinations are elementary optical phenomena, such as flashes or lights, either steady or moving along the visual field, expressed contralateral to the seizure site and extending throughout the visual field.^{38,39} Complex visual hallucinations include scenes of varying complexity (static or crossing the visual field). They are caused by seizure activity in the visual associative fields and adjacent cortical areas.^{38,39} Objects may be deformed in size (makropsia or mikropsia), shape (metamorphopsia), tilt (plagiopsia), straightness of lines (dysmorphopsia), color and distance, or the person perceives a mirror image of himself.

Such episodes last from minutes to hours. A short duration indicates an epileptic event, while a prolonged duration denotes a post-epileptic phenomenon.

Owing to the rapid propagation of the discharge to temporal, parietal and frontal areas, the ictal EEG does not usually reveal the initial focus.³⁹ Careful analysis of the initial symptoms helps identify the initial focus.

Occipital lobe epilepsy is associated with benign idiopathic epilepsies, perinatal injuries, developmental deformities, tumor and vascular lesions, infectious diseases, metabolic encephalopathies and epileptic syndromes.^{38,39} Differential diagnosis should include migraine.

AURAS

Auras are special sensory or psychic phenomena caused by seizure activity. They are classified by symptom type: somatosensory, visual, auditory, olfactory, gustatory, with autonomic symptoms, epigastric or psychic.⁸ Somatosensory auras are defined as localized sensory phenomena (e.g. numbness, tingling). Visual auras involve simple or complex visual hallucinations.⁸ Complex visual hallucinations and delusions arise from the temporoparietal associative cortex and often coexist with psychic auras.⁸ Auditory auras are simple auditory

hallucinations. Olfactory auras are olfactory hallucinations, usually unpleasant and often caused by a tumor of the amygdala or hippocampus.⁴¹ Gustatory auras are characterized by an unpleasant taste and originate from the insular cortex.⁸ Auras arising from limbic structures are often referred to as “indescribable” or as having a fearsome quality.

Abdominal auras are associated with temporal, frontal, or insular epilepsy.⁸ Symptoms include nausea and feelings of tension, weight, tightness, pain, swirl movements or pressure in the abdomen, gas, tingling, knot, electrical discharge, emptiness and hunger, heat, steep descent in an elevator, vibration, flutter, heartburn.⁸ Orgasmic auras are associated with right parietal lobe seizures.⁴²

Psychic auras are complex hallucinatory and delusional experiences.⁸ They are characterized by autoscopic phenomena (viewing double parts of oneself and out-of-body experiences), a feeling of someone’s presence, déjà vu / jamais vu, fear, elation and defective body perception.^{8,43}

NON-CONVULSIVE STATUS EPILEPTICUS

Status epilepticus has been defined as “a condition characterized by an epileptic seizure that is sufficiently prolonged, or repeated at such brief intervals, that recovery between attacks does not occur”. Non-convulsive status epilepticus (NCSE), like convulsive status epilepticus (CSE), is continuous or intermittent seizure activity, without normalization between seizures, of >30-minute duration.^{6,46,47} The onset of NCSE can be either gradual or sudden. An episode can last for hours, days, or weeks. The end of seizure activity is usually followed by sudden clarification of consciousness in the case of absences, or by a prolonged postictal state accompanied by depression, in the case of complex partial seizures.⁴⁷

Its characteristic feature is the change in behavior and mental status that ranges from mild changes, to psychiatric manifestations, delirium or coma. The clinical picture comprises decreased attention, mild disorientation or confusion, prolonged confusional states, mood disturbances, psychotic states, cortical blindness, speech disturbance (verbal perseveration, speech arrest, muteness, aphasia, echolalia), confabulation, bizarre behavior (e.g. laughing, dancing, singing inappropriately), autonomic disturbances (belching, borborygmus, flatulence) and sensory phenomena.⁴⁷ Motor activity is usually normal or focal jerks, twitching of eyelids, clumsiness, apraxia and automatisms may be observed.⁴⁷

NCSE does not represent a single disease entity, but covers a range of epilepsy cases with different etiologies, prognosis and treatment.^{46,48} It is often misdiagnosed as a psychiatric disorder.^{19,47} A detailed history-taking is necessary for diagnosis. EEG investigation is necessary whenever clinical signs raise suspicion for NCSE.

LIMBIC SYSTEM AND AUTONOMIC SEIZURES

The limbic system has a prominent role in the initiation

and persistence of “psychomotor” seizures and the occurrence of psychiatric symptoms.^{3,20,40} Hallucinations, emotional dyscontrol, aggressive reactions and changes in higher cognitive functions may be caused by limbic epileptic discharges. These behavioral and mental changes can be either short-lived or long-lasting. When the focus of the epileptic discharges is located in subcortical structures, the seizure can be undetectable by EEG. “Autonomic seizures” are episodes of autonomic arousal which can be demonstrated objectively (e.g. with EEG).⁴⁴ In 2007, an international consortium proposed the following definitions: “Autonomic seizure is an epileptic seizure characterized by an altered autonomic function of any type at seizure onset, or in which manifestations consistent with altered autonomic function are prominent even if not present at seizure onset”.^{49,50} “Autonomic status epilepticus is an autonomic seizure which lasts more than 30 min, or a series of such seizures over a 30-min period, without full recovery between seizures”.^{49,50}

Autonomic seizures can be the main seizure or the result of a secondary spread of discharge from another brain area. Symptoms from the cardiovascular system (e.g. tachycardia, heartburn), respiratory system (e.g. hyperventilation, bradypnoea, dyspnea, cyanosis, ictal cough), digestive system (e.g. nausea, vomiting, hiccups, salivation), genitourinary system (e.g. urge to urinate) are observed, along with changes to the skin (sweating, flushing, pallor, piloerection) and eyes (miosis, mydriasis).^{8,44,45}

The clinical picture depends on the initial focus and the secondary spread of discharge (e.g. auditory hallucinations associated with discharges in Heschl helix).⁴⁰ Discharges from the insula can be expressed as autonomic seizures.⁴⁹ Temporal lobe seizures often manifest with autonomic seizure symptomatology.⁴⁵

The main clinical signs are described in a study which has used stereotaxic depth recordings in medically intractable epileptics. The signs are referred to in order of diminished frequency as follows: Gastric -epigastric sensations, twilight situations, fugue, olfactory and gustatory hallucinations, experiences of “*déjà-vu*”, “*déjà-vécu*”, activity interruption, nausea, vertigo, oral automatism, dysphasia, speech arrest, tonic/clonic kinetic phenomena, cephalic aura, interictal mnemonic deficits, paroxysms of laughter, intense signs and symptoms of Autonomous NS, breathing arrest, visual hallucinations, somatosensory hallucinations, fear, aggressive outbursts, forced thinking, auditory hallucinations.⁴⁰ This study highlights the special role of mesiobasal limbic structures, i.e., amygdala and hippocampus, and its connections to hypothalamic and frontobasal-cingulate areas. The authors concluded that besides the short-lived ictal aberrations of mental state and emotional sphere (“psychical seizures”) also some of the more prolonged behavior and personality changes might be attributed to narrowly confined limbic seizure discharges or to a “limbic dyscontrol syndrome”.

EFFECTS OF ANTIEPILEPTIC DRUGS

Antiepileptic drugs can have adverse effects on the mental and cognitive state. Their pharmacokinetic properties, clinical efficacy and side effects vary considerably, depending on age, genetic factors, and factors affecting liver and kidney function.⁵¹

The risk of cognitive dysfunction increases with rapid titration of drug, higher doses, elevated drug levels in the blood, or co-administration of different anticonvulsants.⁵² The effects on cognitive functions are reversible with dose reduction or drug discontinuation.⁵² Cognitive domains commonly affected include attention and psychomotor speed.^{52,53} Phenytoin, valproate and carbamazepine have a negative impact on attention and memory, although they are less often associated with adverse effects on cognitive functions than bromides, benzodiazepines and phenobarbital.⁵² Newer anticonvulsants have a more favorable cognitive profile, with the exception of topiramate and zonisamide.⁵² Topiramate causes mental slowness, difficulty in recalling words and attention problems.⁵³

Data for the psychoactive effects of anticonvulsants in epilepsy patients are limited. Clinical data and small case series indicate that valproate, carbamazepine, lamotrigine, and possibly oxcarbazepin, have stabilizing properties of emotion.⁵⁴ Lamotrigine possibly has antidepressant properties, while gabapentin, pregabalin and tiagabine may possess anxiolytic properties.⁵⁴ Barbiturates, topiramate and phenytoin are associated with the onset of depression.⁵⁴ Symptoms of depression and anxiety can be exacerbated by levetiracetam.⁵⁴ Psychotic symptoms have been reported as side effects of most antiepileptic drugs, particularly levetiracetam, topiramate and zonisamide.^{54,55} The psychotic manifestations may follow the discontinuation of antiepileptic therapy. Clobazam can produce behavioral changes, aggression and sleep disorders.³¹ Phenobarbital and benzodiazepines may cause distractibility and impulsivity.⁵³ Benzodiazepines in minors can trigger an activation syndrome, characterized by disinhibition, irritability, aggression and hyperactivity.⁵⁴

Antiepileptic drugs are also associated with increased risk of suicidality (completed suicide, suicide attempt, and suicidal ideation) relative to placebo in randomized placebo-controlled trials.^{55,56}

CONCLUSION

Some manifestations of epileptic discharges can be mistaken for symptoms of psychiatric disorders. Behaviors, mood states, perceptions, sensations and cognitive dysfunction are common clinical manifestations of many types of epileptic phenomena. However, in clinical practice “pure” cases are not the rule. Many Child and Adolescent Psychiatrists have

observed cases with atypical clinical presentation and unusual symptoms, either comorbid with epilepsy, or accompanied by seizures that began after the onset of the mental disorder. On the other hand, neurologists may consider such ambiguous symptoms as a psychiatric disorder.

The aim of this paper is to describe signs and symptoms of various types of epileptic seizures and does not examine the equally important area of the psychiatric comorbidity observed in children and adolescents with epilepsy.

A thorough developmental history is a necessary part of every psychiatric examination of children and adolescents. A body systems review across development, provides valuable information for the detection of possible etiopathological parameters. A physical examination and appropriate laboratory examinations should also be performed when indicated. Many children (and adults) do not spontaneously refer to signs and symptoms, so they should be asked directly, in the form of closed questions (eg. did you ever had problems with your vision? Do you see things that others do not see? You say that you see circles, are they static, or moving? How? Do they have color? etc.). For such an enquiry, child and adolescent psychiatrists should have a good knowledge of the manifestations of seizure phenomena so as to avoid misdiagnosis.

The diagnosis of epilepsy is mainly clinical and good history-taking is of paramount importance. The exact description of seizure semiology, the triggering factors and sequence of events is a useful tool for the diagnosis and detection of initial focus. The episodic, paroxysmal and stereotypical appearance, or the atypicality of psychiatric symptoms or behaviors can raise the suspicion of a seizure disorder.

A single surface EEG can neither confirm nor exclude epilepsy. Serial EEG's during wakefulness in combination with prolonged sleep recordings or video-EEG recordings, increase the diagnostic accuracy. Video-EEG is the gold standard in the diagnosis of complex behaviors and psychic phenomena related to epileptic discharges.

The relationship between epilepsy and psychiatric symptoms is a fertile ground for the study and understanding of brain dysfunction. It would undoubtedly help child and adolescent psychiatrists to acquire a broader insight into seizure activity manifested as a psychiatric disorder. Collaboration between psychiatrists and neurologists is vital and in the best interest of the patient.

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