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## Summary

Parietal lobe epilepsy (PLE) and Occipital lobe epilepsy (OLE) are related to a variety of sensory, visual, vertiginous or visuo-spatial symptoms with different degrees of complexity. The seizure symptoms are of varying localizing and lateralizing value; seizure discharges may spread rapidly and perceived symptoms may reflect secondary recruitment rather than the primary site of seizure onset, making localization of the epileptogenic zone more difficult. We present a pediatric patient with parieto-occipital epilepsy, who was evaluated in our epilepsy presurgical evaluation unit and had surgical intervention with a favorable outcome. Further on, the clinical manifestations, etiology, diagnostic and treatment procedures, as well as the differential diagnosis of different surgical and non-surgical situations in parieto-occipital epilepsy are discussed. When surgical therapy is the treatment of choice, the prognosis is better if multimodal imaging and co-registration is employed and if surgery is carried out at a younger age. Careful work-up allows determining an idiopathic or symptomatic origin and also addresses rare congenital syndromes or medical conditions which are often related to seizures from the posterior lobes.

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**Key words:** Epilepsy, parietooccipital, surgical, non-surgical

## Parieto-okzipitale Epilepsien: chirurgische vs nicht-chirurgische Syndrome

Parietallappen-Epilepsie (PLE) und Okzipitallappen-Epilepsie (OLE) sind mit einer Vielzahl von sensorischen, visuellen, vertiginösen und visuo-spatialen Symptomen verschiedenster Komplexität assoziiert. Die Anfalls-symptome unterscheiden sich in ihrem lokalisierenden und lateralisierenden Wert; es ist jedoch zu beachten, dass sich Anfallsentladungen schnell ausbreiten können und wahrgenommene Symptome können schon ei-

ne sekundäre Rekrutierung statt Entladungen aus dem primären Fokus darstellen, was die Lokalisierung der epileptogenen Zone schwierig macht. Wir stellen den Fall eines pädiatrischen Patienten mit parieto-okzipitaler Epilepsie dar, der in unserer prächirurgischen Abteilung evaluiert wurde und mit sehr gutem Resultat chirurgisch behandelt wurde. Klinische Manifestationen, Ätiologie, Diagnostik, Behandlungsstrategien in parieto-okzipitalen Epilepsien werden diskutiert. Falls die chirurgische Therapie gewählt wird, ist das Resultat im Allgemeinen besser, wenn multimodales Imaging und Ko-registrierung der bildgebenden Verfahren angewandt werden, und wenn die Operation in einem jungen Alter durchgeführt wird. Eine sorgfältige Evaluation erlaubt die Differenzierung zwischen einem idiopathischen und symptomatischen Ursprung und sollte auch seltene kongenitale Syndrome oder internistische Affektionen beeinhaltend, die oft mit Anfällen der posterioren Hirnareale vergesellschaftet sind.

**Schlüsselwörter:** Epilepsie, parieto-okzipital, chirurgisch, nicht-chirurgisch

## Epilepsies pariéto-occipitales : conditions chirurgicales vs non-chirurgicales

Les épilepsies du lobe pariétal (ELP) et du lobe occipital (ELO) sont liées à une variété de symptômes sensitifs, visuels, vertigineux ou visuo-spatiaux avec différents degrés de complexité. Les symptômes ont une valeur localisatrice et de latéralisation variables; les décharges épileptiques peuvent se propager rapidement et les symptômes perçus reflètent peut-être le recrutement secondaire plutôt que le site principal du début des crises, ce qui rend la localisation de la zone épileptogène plus difficile. Nous présentons une enfant

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souffrant d'épilepsie pariéto-occipitale, qui a été évaluée dans notre unité d'évaluation pré-chirurgicale de l'épilepsie et a bénéficié d'une intervention chirurgicale avec un résultat favorable. Les manifestations cliniques, étiologie, diagnostic et traitement, ainsi que le diagnostic différentiel des différentes situations chirurgicales et non-chirurgicales dans l'épilepsie pariéto-occipitale sont discutés. Lorsque la chirurgie est le traitement de choix, le pronostic est meilleur si l'imagerie multimodale et la reconstruction sont employées et si la chirurgie est réalisée à un âge plus jeune. Une analyse rigoureuse permet de déterminer une origine idiopathique ou symptomatique et de rechercher des rares syndromes ou pathologies congénitales ou des conditions médicales qui sont souvent liés à des crises des lobes pariétaux ou occipitaux.

**Mots clés :** Epilepsie, pariéto-occipital, chirurgical, non-chirurgical

## Introduction

Posterior cortex epilepsies (PCEs) encompass a group of epilepsies originating from the occipital, parietal, or occipital border of the temporal lobe, or from any combination of these regions [1]. No clear anatomic or neurophysiological distinctions are apparent between these cortical areas and the epileptogenic regions are not always limited to the anatomical borders of the parietal or occipital lobes, hence epilepsies originating from them are better analyzed and understood when grouped together. The parietal and occipital lobe epilepsies are included among the localization related epilepsies and epileptic syndromes in the 1989 ILAE classification. Both epilepsies are usually characterized by simple partial and secondary generalized seizures. Ictal discharges starting from the posterior areas tend to spread quickly to other cortical regions, leading to clinical semiology characteristic of other lobes, possibly more related to seizure diffusion than to seizure origin [2]. Thus the precise diagnosis of PCEs and their adequate therapeutic management require an optimized combination of clinical, electrophysiological, and radiologic studies.

Since seizure disorders emanating from parietal or occipital lobes are less frequent and the localization of the epileptogenic zone is more difficult, surgical treatment for epileptic seizures arising from the posterior cortex is less common than for seizures arising from temporal or frontal regions [3]. With the introduction of functional neuroimaging (PET, SPECT, high resolution MRI) and the combined processing of these investigative modalities, more patients with intractable epilepsy are operated with a better seizure free outcome and decreased neurological morbidity [4].

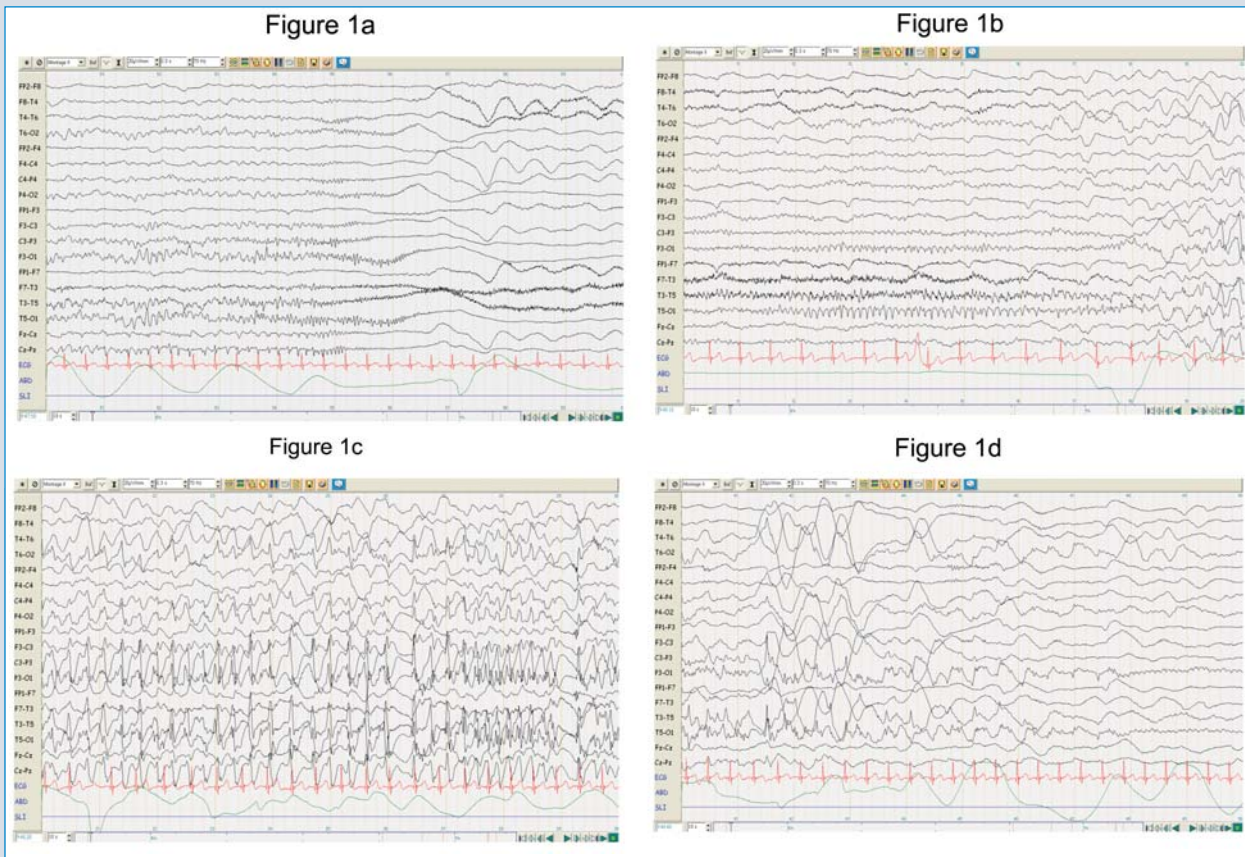
We present a child with parieto-occipital epilepsy, who was evaluated in our epilepsy presurgical evalua-

tion unit and was operated with a favorable outcome. Further on, the clinical manifestations, etiology, diagnosis and treatment, as well as the differential diagnosis of different surgical and non-surgical situations in parieto-occipital epilepsy are discussed.

## Case

This boy was born prematurely at 31 4/7 GW by emergency cesarean section for placental detachment and fetal distress. His Apgar score was 1/9/9, and umbilical cord venous pH was 6.99. His neurological and skin examination was normal and the initial neurological evolution was favorable. The cerebral MRI performed at 2 weeks and at 2 months was unremarkable. At 3 months, seizures appeared, and manifested as fixed gaze and repeated rhythmic bilateral eyelid jerks. Occasional limb myoclonias were also reported by the parents. His initial EEG revealed slow background activity, and frequent spike and polyspike discharges predominant in the posterior regions, of higher amplitude on the left side. Valproic acid treatment was started. Vigabatrin was added soon after because of refractory daily seizures. This modification was followed by transient clinical and EEG improvement. The seizure frequency increased at 19 months, and the patient presented with up to 70 convulsions per day without recovering full consciousness. The seizures were characterized by a combination of tonic posturing of the left arm and leg, eye deviation to either side alternatively, eyelid myoclonias, nystagmus to the right and bradycardia (70/minute). On EEG, these events were correlated with low-voltage rhythmical spikes in the left posterior temporal-occipital region, followed by a generalized flattening of the background activity, and high-amplitude spike-waves in the left posterior region (**figure 1**). On one occasion, the onset was characterized as left frontal attenuation and low voltage fast activity, with rapid diffusion to the contralateral hemisphere, and followed by rhythmical spike-waves in the left temporal-occipital region. The interictal EEG showed left occipital spikes and slow waves. During this period, various additional treatments were tried including lamotrigine, phenobarbital, phenytoin, clobazam, pyridoxine, carbamazepine, topiramate and levetiracetam. A repeat MRI was performed at 19 months, and revealed ill-defined left parietal-occipital sulci on FLAIR, inversion recovery and T2 sequences. A PET-scan revealed hypo metabolism in the same region. Seizures were controlled under topiramate monotherapy with no single event being observed during 11 months.

Refractory breakthrough seizures were again observed at the age of 31 months, and the pre-surgical work-up was completed. The interictal (**figure 2**) and ictal EEG confirmed the previously observed abnormalities in the left posterior region. The repeat cerebral MRI revealed extensive cortical dysplasia in the left parietal-



**Figure 1:** 1a - Ictal EEG, left parieto-occipital onset, generalized attenuation, 1b - seizure continuation, left temporal posterior rhythmic spikes, 1c - seizure continuation, left posterior spikes of higher amplitude and decreasing frequency, 1d - seizure termination

occipital region (figure 3). The interictal SPECT-scan showed hypoperfusion in the temporal-parietal-occipital region (figure 4). The ictal SPECT-scan showed moderate hyperactivity in the right frontal lobe and more intense hyperperfusion in the left parietal-occipital region, as well as in the left posterior mesial and lateral temporal lobe (figure 5). A median occipital corticectomy and superior parietal circonvolution ablation was performed at 34 months, without complications. A few focal seizures were observed until the 4th post-operative day, and a single seizure was observed during a febrile illness 3 months later, under oxcarbazepine monotherapy. This medication was weaned later without seizure recurrence. On follow-up visit, at 3.5 years, left hemianopsia was reported by the parents, and the convergent strabismus, noted from the 8<sup>th</sup> month of life, was still present. Although slightly delayed, particularly in the tasks requiring visual attention, the boy is in constant progress in all domains with the help of physical and educational therapies. Integration in the normal school system is planned.

### Clinical manifestations

*Parietal lobe epilepsies* (PLE) manifest with seizures originating from a primary epileptic focus within the parietal lobe. It may start at any age and both sexes are equally affected. These seizures are mainly simple focal events that manifest with subjective symptoms, such as somatosensory, somatic illusions, vertiginous, visual illusions or complex visual hallucinations, receptive or conductive linguistic disturbances, in the order of prevalence. These clinical seizure manifestations are usually related to the epileptogenic location, anterior or posterior, of the dominant or the non-dominant parietal lobe. Onset with sensorimotor foci is usually associated with anterior parietal lobe foci whereas more complex symptomatology emanates from the posterior regions. Some patients experience more than one type of seizure. Somatosensory seizures are by far the most common type with various types of paresthetic, dysesthetic and painful sensations. Tingling is one of the characteristic described symptoms, and pain, sometimes excruciating, is experienced by 25 % of patients with somatosensory seizures [5, 6, 7]. When lateralized the painful symptoms are contralateral to the side of seizure origin. The symptoms may remain confined to the region of origin in 40 % of cases or march in a manner similar to a



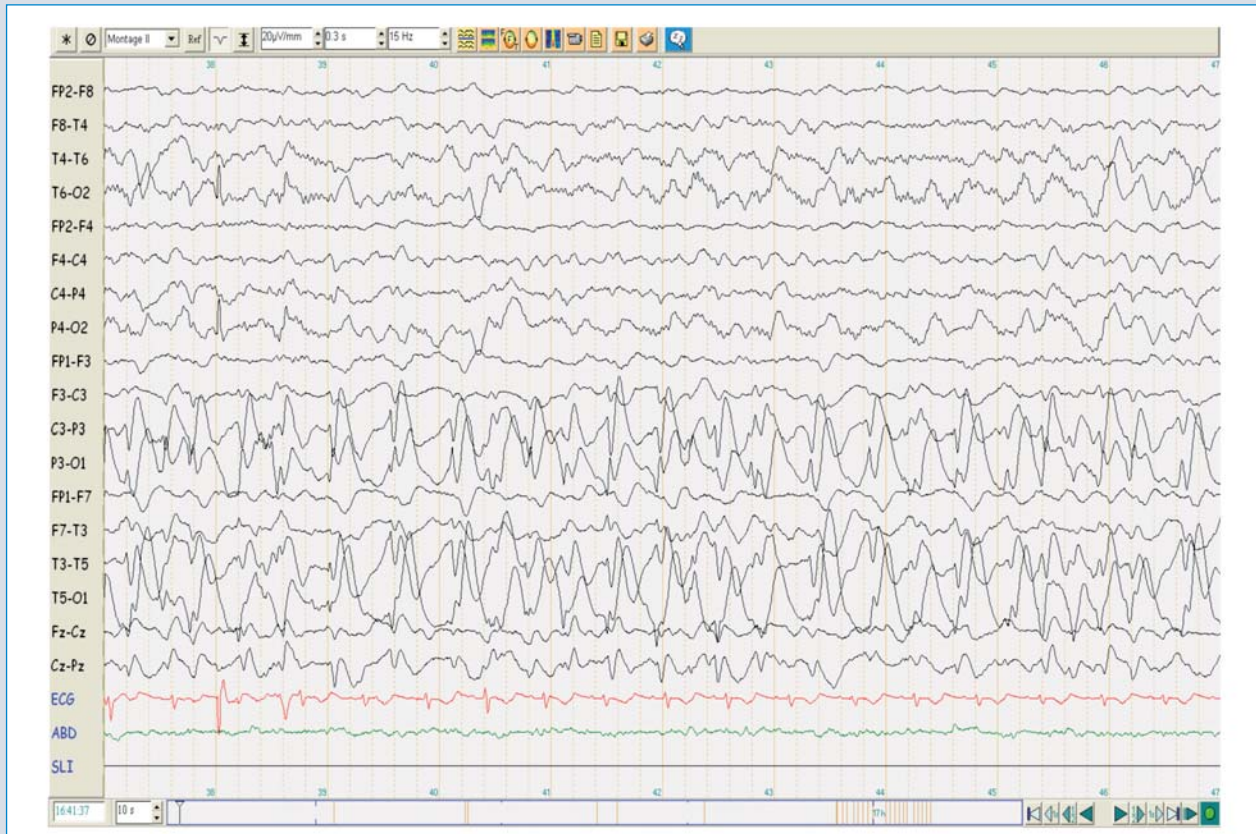


Figure 2: Interictal EEG, left parietal-occipital and posterior temporal spike-waves

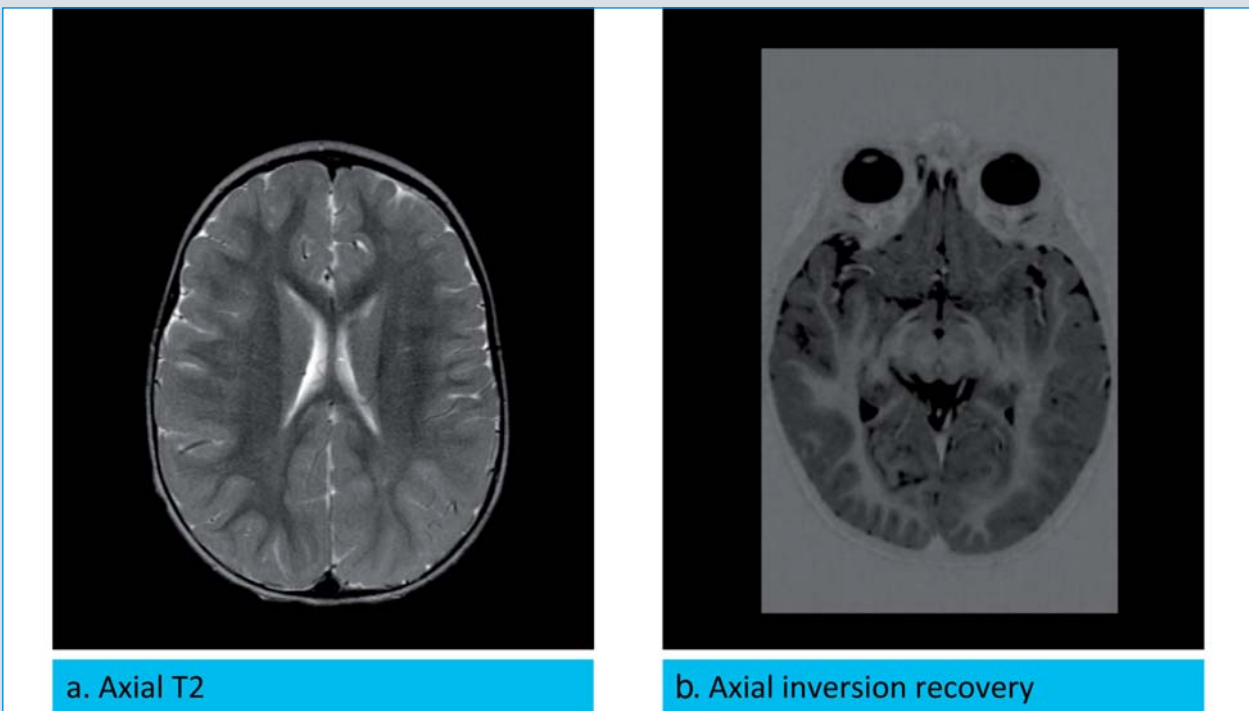


Figure 3: MRI showing left posterior cortical dysplasia, a. Axial T2, b. Axial inversion recovery

Jacksonian motor seizure. Unilateral somatosensory seizures are usually contralateral to the epileptogenic zone.

Disturbances of body image and somatic illusions are the second most common ictal symptom of PLE. They include illusions of distorted posture, limb position or movement, a feeling that an extremity or a body

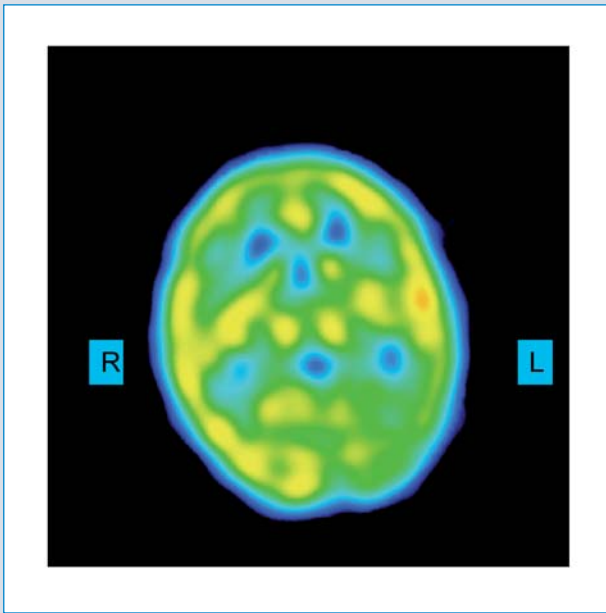


Figure 4: Intercictal SPECT, left parietal-occipital and posterior temporal hypoperfusion

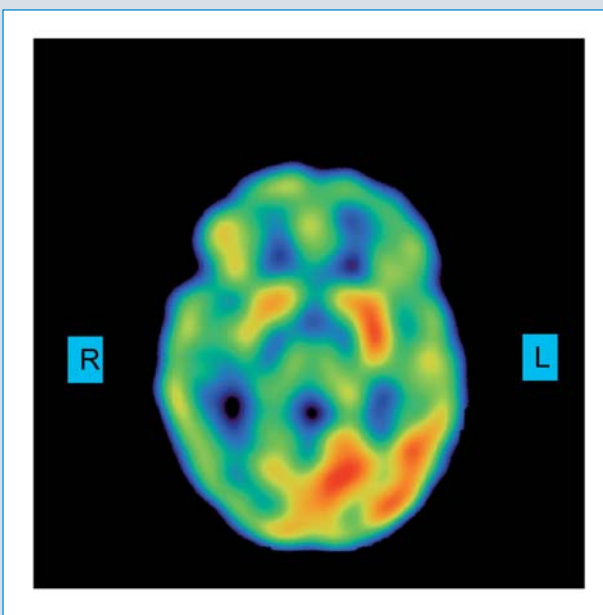


Figure 5: Ictal SPECT, left parietal-occipital and posterior temporal hyperperfusion

part is alien or absent, dissociations and misperceptions of location and body part identity. Most patients have paresthesia associated with these illusions. Ictal somatic illusions reflect seizure discharges in the inferior parietal lobule and superior part of the post central gyrus of the non-dominant hemisphere. Somatoagnosia occurs frequently with the dysfunction of the nondominant cerebral hemisphere. Ictal limb agnosia and phantom limb sensations probably originate in the posterior parietal region. Neglect [8] is more commonly associated with the right than the left inferior parietal lobe. Il-

lusions of movement are typical of parietal lobe seizures while the sensation of a desire to move emanates from the precentral gyrus.

Vertigo and other vertiginous sensations or illusion of rotatory body movement are reported in about 10 % of parietal lobe seizures and are elicited predominantly from the temporal-parietal border. Visual illusions and complex formed visual hallucinations occur in about 12 %. Dominant parietal lobe seizures can be associated with a variety of language disturbances, alexia with agraphia, and significant calculation defects while non dominant parietal-occipital-temporal seizure activity usually results in spatial disturbances.

Seizures spreading to extraparietal regions produce unilateral focal clonic convulsions, head and eye deviation, tonic posturing of usually one extremity and automatisms. The duration of these seizures may vary from a few seconds to 1-2 minutes. Prolonged isolated sensory auras without any motor manifestations have been reported [9] and may be even misdiagnosed as nonepileptogenic psychogenic seizures, transient ischemic attacks or migraine with aura [10]. Postictal manifestations are rare, although Todd's paralysis and dysphasia have been reported [9]. Seizures may be provoked by movements of the affected part of the body or other somatosensory stimuli [11]. Commonly, genuine seizures are only suspected when they progress to motor symptoms or impairment of consciousness. Ictal pain and sensory epilepsy partialis continua are also unlikely to be initially diagnosed as epileptic seizures.

*Occipital lobe epilepsies (OLE):* Occipital epilepsies account for about 5-10 % of all epilepsies. Occipital seizures originate from an epileptic occipital focus that arises spontaneously or is triggered by external visual stimuli. Idiopathic epilepsy usually starts in childhood; symptomatic occipital seizures may start at any age and at any stage during or after the course of the underlying causative disorder.

The ictal clinical symptoms of occipital lobe epilepsies are subjective, objective or both, the cardinal symptoms being visual and oculomotor. Visual subjective symptoms include elementary and less often complex visual hallucinations, blindness, visual illusions, palinopsia, sensory hallucinations of ocular movements, and ocular subjective symptoms such as ocular pain. Ictal objective oculomotor symptoms are tonic deviation of the eyes (pursuit like), oculoclonic movements or nystagmus and repetitive eyelid closure or eyelid fluttering. Some of the ictal manifestations such as elementary visual hallucinations are generated in the primary visual cortex; others such as visual illusions emanate from the neighborhood of the occipital lobe, i.e. occipital-parietal and occipital-temporal regions.

Seizures may spread from the occipital to the other more anterior regions of the brain generating symptoms from the temporal, parietal and frontal lobes and secondarily generalized convulsions. Infracalcarine occipital foci propagate to the temporal lobe causing com-

plex focal seizures while supracalcarine foci tend to propagate to the parietal and frontal areas giving rise to predominantly motor seizures.

Elementary visual hallucinations are the most common, characteristic and well defined ictal symptoms of occipital lobe seizures. They are usually the first and the only ictal symptom during a seizure and may progress to other occipital and extraoccipital manifestations. The predominant patterns in the ictal elementary hallucinations are colored, usually multicolored (bright red, yellow, blue and green) and circular flickering patterns. Shapes are mainly circular, spots, circles or balls. Individual elements are multiple and rarely single. The components increase in number, size or both with progression of the seizure. Their location at onset is usually unilateral, mainly in the temporal visual hemi-field. The side of the unilateral elementary visual hallucinations is contralateral to that of the epileptogenic focus. Ictally, vision may be obscured only in the area occupied by the visual hallucinations. Blurring of vision at onset with or without visual hallucination may be a mild form of an occipital visual seizure. Visual seizures develop rapidly within seconds and they are usually brief, lasting from a few seconds to 1-3 min, rarely longer. Very often, they are longer prior to secondary generalization. They occur often in multiple clusters, daily or weekly, and several may occur in a day and are usually diurnal. Ictal symptoms of elementary visual hallucinations are stereotyped, in all aspects other than duration of seizures.

These may remain as the only seizure manifestation, or may progress to other occipital or non occipital seizure manifestations (other ictal symptoms like complex visual hallucinations, oculoclonic seizures, tonic deviation of the eyes, eyelid fluttering or repetitive eye closures, impairment of consciousness, experiential phenomena, hemianesthesia, and unilateral or generalized convulsions). On other occasions they progress to extraoccipital seizure manifestations by spread to temporal, frontal or parietal regions.

Complex visual hallucinations, visual illusions and other symptoms from more anterior ictal spreading, mainly occur in progression and may terminate in hemiconvulsions or generalized convulsions. The complex visual hallucinations may take the form of persons, animals, objects, figures or scenes. They may be familiar or unfamiliar, friendly or frightening, and may appear in a small or large area of a hemifield, or in the centre or whole of the visual field, may be static or move horizontally. In patients with visual field defects due to structural brain lesions complex structural hallucinations appear in the defective visual field. Complex visual hallucinations of occipital seizures do not have the emotional and complex character of temporal lobe seizures. Complex visual hallucinations including ictal autoscopia [12] probably originate from the occipital-parietal and occipital-temporal junctions.

Visual illusions include false percepts of real external images, with distorted images (metamorphopsia),

changes in perceptions of object size (micropsia or macropsia), distortions in shape, color (achromatopsia, monochromopsia), distortion of direction or speed and even changes in spatial interpretation affecting stereoscopic vision. Ictal visual illusions may occupy part or whole of the visual field and are more likely to be associated with symptomatic than with idiopathic occipital seizures. Palinopsia (persistence of visual stimulus after the exciting stimulus is removed) is another interesting form of visual illusion associated with right posterior parietal-temporal lesions. Sensory hallucinations of ocular movements may be seen but are rare.

Ictal blindness (amaurosis) may follow the visual hallucinations and progress to other epileptic symptoms, but may occur as a starting or the only ictal seizure manifestation with abrupt onset. The duration of ictal blindness is usually longer than ictal visual hallucinations, occasionally blindness may last for hours or days – status epilepticus amauroticus [13, 14]. Ictal blindness and less frequently ictal hemianopia occur in one third of patients with symptomatic and two thirds of patients with idiopathic occipital epilepsy.

Tonic deviation of the eyes, often followed by ipsilateral turning of the head is the most common non visual symptom of occipital seizures. This usually follows visual symptoms but may also occur from the seizure onset. The epileptogenic focus is almost always contralateral to the movement of the head and the eyes if consciousness is preserved. Ictal nystagmus is mainly horizontal; the quick phase of the nystagmus is opposite to the epileptic focus, and in the same direction of the head and eye deviation. Repetitive eyelid closure, fluttering and blinking is another interesting ictal clinical symptom of symptomatic and idiopathic occipital epilepsy, usually occurring after the phase of visual hallucinations, at a stage when consciousness is impaired and heralds impending secondarily generalized convulsions.

Ictal or postictal headache is frequently associated with occipital seizures. Postictal headache often indistinguishable from migraine is far more common in occipital than in any other focal epilepsy and occurs in more than 50 % of cases [15].

## Etiology

The etiology of parietal epilepsy syndromes is diverse and includes symptomatic, probably symptomatic and idiopathic causes. Lesions include malformations of cortical development (focal cortical dysplasia), polymicrogyria, subcortical band heterotopia, neoplasms (DNETs, astrocytomas, gliomas), vascular malformations and Sturge Weber syndrome, or other causes like trauma and encephalitis. Malformations of cortical development are a common cause, and are being increasingly recognized with MRI as it is the case in all focal symptomatic and probably symptomatic epilepsies. In



symptomatic occipital epilepsy, lesions may be congenital, residual or progressive resulting from vascular, neoplastic, metabolic, hereditary, inflammatory, parasitic, systemic diseases or infectious. Diverse epileptic conditions with onset in childhood and early adolescence have been reported in patients with symptomatic or asymptomatic celiac disease [16]. These include also severe epilepsies such as Lennox Gastaut syndrome, myoclonic epilepsies with ataxia and more commonly symptomatic occipital epilepsy. Seizures may be the first manifestation of a devastating course as in Lafora disease or mitochondrial disorders. There is a selective vulnerability of occipital lobes in eclamptic or non-eclamptic hypertensive encephalopathy or toxic-metabolic origin (e.g. antibiotics like imipenem).

Recognized benign epilepsy syndromes include be-

Occipital or diffuse interictal EEG spikes indicate cortical hyperexcitability, with some predominance over the occipital lobes (figure 6). The prognosis of PS is excellent with remission within 1-3 years from the first seizure. The prognosis of GS is less certain, given that remission is rare. Migraine with visual aura, acephalgic and basilar migraine can be a differential diagnosis for occipital epilepsies of PS-type.

### Diagnosis and Treatment

Neurological examination is usually normal in patients with non-lesional PLE. Sensory deficits such as impaired two point discrimination or astereognosia or mild limb atrophy and inferior quadrantic visual field



Figure 6: Occipital sharp waves in Panayiotopoulos Syndrome

nign epilepsy with occipital spikes as described by – the more frequent – Panayiotopoulos syndrome (PS) and the idiopathic childhood occipital epilepsy of Gastaut's type (GS). Gastaut's idiopathic childhood epilepsy manifests mainly with visual seizures and the EEG shows occipital spikes, which disappear with eye opening. In contrast, the electro-clinical characteristics of PS are characterized by tonic eye deviation and vomiting, and the typical ictal clinical features (behavioural changes, ictal vomiting, unilateral deviation of the eyes) reflect mainly the involvement of the autonomic network that expands over multiple cortical and subcortical areas in both hemispheres in a vast majority of children with PS.

defects should be looked for, as well as disturbances of written language, aphasia, spatial orientation and right left disorientation. Mild contralateral weakness may be seen in patients with tumors.

EEG is essential in the diagnosis, while recognizing certain limitations. Surface interictal EEG in PLE may be normal, non specific or even misleading. In symptomatic patients, localized slow waves may be the only interictal abnormality. Epileptiform abnormalities, if they occur, may appear in areas other than parietal regions, involving frontal, temporal or occipital electrodes. Ictal EEG may be normal in 80 % of simple focal sensory seizures. Ictal onset may be distant from the area of pre-

dominant interictal spikes and ictal EEG patterns may be difficult to interpret particularly when seizures become rapidly generalized. Thus, the diagnosis of PLE on EEG alone is difficult and may be misleading. Post ictal EEG may have some localizing value when focal slow wave attenuation of background activity or spike activation occurs.

In OLE, in symptomatic cases, the background EEG is usually abnormal with posterior lateralized slowing. There may be occipital photosensitivity response. Surface ictal EEG, irrespective of cause, usually manifests with paroxysmal fast activity, fast spiking or both, localized in the occipital regions with occasional gradual anterior spreading and generalization with irregular spike wave discharges but also monomorphic spike and wave activity. Brief occipital flattening may be seen before the fast rhythmic pattern.

Brain imaging with high resolution MRI is useful to detect unsuspected residual or progressive lesions, tumors, vascular malformations and malformations of cortical development. In order to search for the underlying cause in symptomatic occipital epilepsies, hematology and biochemistry screening for metabolic disorders, molecular DNA analysis or even skin or other tissue biopsy may be indicated.

The *treatment* of focal epilepsies of any cause begins first with antiepileptic drugs; when this fails, neurosurgical options are considered and are beneficial in symptomatic and probably symptomatic epilepsies. Drug treatment in parieto-occipital epilepsies is similar to that for any type of focal seizures. In intractable cases, neurosurgery after proper presurgical evaluation and proper selection of patients is associated with a large proportion of patients achieving seizure freedom or reduction in seizure frequency.

*Presurgical evaluation:* The epileptogenic zone located in parieto-occipital areas can be assessed by both invasive and noninvasive evaluation. A detailed clinical history described by the patient, family or an observer should be obtained in order to search for clues indicating a posterior cortex origin. The spreading pathway of the seizures varies greatly. Ictal scalp recordings do not always show a seizure originating from the posterior cortex because of the rapid propagation to other lobes or contralateral lobes. Diffuse or bilateral distribution of interictal spikes may be observed in posterior cortex epilepsies and spikes may be observed in frontal rather than the posterior part of the brain. Apart from the MRI, functional brain imaging with PET and SPECT provides valuable noninvasive information for epileptogenic zones involving extra-temporal locations. However none of the imaging techniques (structural or functional) is reliable by itself for localizing the seizure focus. In patients with diagnostic modalities giving inconclusive or discordant results, intracranial EEG monitoring can play an important role in epileptogenic zone localization.

## Prognosis of epilepsy surgery

In children, surgery for extratemporal lobe epilepsy represents approximately 50 % of procedures and has been shown successful in one-half to two-thirds of the cases [17, 18]. Seizure freedom was reported in 63 % of patients operated for extratemporal epilepsy during a mean follow-up period of 5 years [19]. Parietal-occipital resections comprised 10-15 % of all surgical treatments in our centre. Regarding the surgical outcome, 80 % of patients achieved a seizure free status, suggesting the value of appropriate surgical treatment in patients with pharmaco-resistant posterior cortex epilepsies.

The likelihood of the surgical outcome being favorable is greater when the diagnostic modalities show concordance in the epileptogenic zone localized [20, 21, 22]. The concordance of the diagnostic modalities also depends on the underlying pathology. FCD lesions are intrinsically epileptogenic, while the epileptogenic zone is diffuse or less defined by scalp EEG in polymicrogyria and subcortical heterotopia and may be localized at some distance from the major structural anomaly. Therefore the results of the various diagnostic modalities cannot be expected to be perfectly concordant for the different pathologies. Other variables associated with a favorable outcome include the presence of MRI abnormalities and completeness of resection of the epileptogenic zone. The correlation between the completeness of the epileptogenic zone resection and surgical outcome in several studies show that a presurgical protocol allowing a precise definition of the area of resection gives better results in posterior epilepsies [23]. The operative outcome in 80 consecutive adult patients with lesional PLEs who underwent resective surgery for intractable epilepsy between 1991 and 2006 retrospectively evaluated by El Sharkawy et al. [24] shows seizure freedom rates of up to 47 % up to 10 years, with low risk of permanent neurological complications. While incomplete resection on a postoperative MRI predicts seizure relapse in a short-term follow up, the presence of interictal discharges in a 6 month postoperative EEG predicts seizure relapse on a longterm followup. Factors predicting a good short term outcome were childhood onset of epilepsy, short epilepsy duration, ipsilateral spikes, visual aura, and presence of a well circumscribed lesion in a preoperative MRI.

Finally, concerning the *neuropsychological outcome*, several studies have reported that surgical treatment improves cognitive prognosis in children with epilepsy [25], however very few studies have measured the outcome after surgery in parietal and occipital epilepsies [26]. In our experience the vast of majority of patients benefit from surgical intervention. However, some case studies report distinct, new difficulties after surgery in the posterior lobe. Jambaque et al. reported visual aperceptive agnosia with severe face recognition impairment, executive function deficits, and autistic symptoms in a 13-year-old girl who had undergone a right



occipital lobectomy for cortical dysplasia at age 7 years [27]. Remarkably, her academic abilities in spelling, reading, and arithmetic were higher than her functional intelligence level. Cohen et al. demonstrated brain reorganization of the visual word form area toward the right hemisphere in a young epileptic child operated at the age of 4 years for a Sturge-Weber syndrome in the left occipito-temporal lobe [28]. Sinclair et al. reviewed the epileptic outcomes of nine children with parietal resections and six with occipital resections [29]. Cognitive outcome was not addressed; all occipital lobe patients showed visual field losses.

Focusing on the possibility of generalized impairments, Gleissner et al. assessed 15 children with parietal lobe epilepsy [30]. One year after surgery, these children were seizure-free with postoperative improvements in attention and behavior, but they showed a decrease in performance intelligence quotient (IQ). Occipital-temporal areas are involved in color, object, and face recognition, reading and other functions. In both children and adults, the impact of a lesion in the occipital and parietal lobes can result in visual agnosia, neglect, visuospatial disorientation, alexia, sensorial extinction, and Gerstmann syndrome [31]. Lippe et al. assessed the long-term cognitive outcome (3-7 years post surgery) of occipito-parietal epilepsy in five children who showed early onset due to Taylor-type focal cortical dysplasia [32]. Neurosurgical resection involved the occipital lobe and temporal or parietal lobe junctions, and neuropsychological assessment specifically targeted the cognitive functions of the posterior regions of the brain. Although recovery for visual perceptual cognition was more limited than for verbal functions, long-term neuropsychological outcome in these 5 children showed that early surgery for epilepsy offers the possibility of optimizing cognitive outcomes in children with posterior intractable epilepsies.

## Conclusion

PLE and OLE are epilepsies maybe difficult to diagnose in some patients and are related to a variety of sensory, visual, vertiginous, visuo-spatial symptoms with varying degree of complexity. OLE is not always benign. Careful work-up allows determining an idiopathic or symptomatic origin, provided that the research goes beyond EEG and MRI (if necessary) and also addresses rare congenital syndromes or medical conditions, which are often related to seizures from the posterior lobes. If surgical therapy is the treatment of choice, the prognosis is better if multimodal imaging and co-registration is readily employed and if surgery is carried out at a younger age.

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