

Networks in Posterior Cortex Epilepsies



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KEYWORDS

• Occipital • Parietal • Seizure propagation • Intracranial EEG • Epilepsy

KEY POINTS

- Posterior cortex epilepsies summarize epilepsies with seizures deriving from the occipital, parietal and posterior temporal lobe.
- Seizure semiology and EEG findings in these epilepsies are often falsely localizing due to fast ictal spread.
- A profound understanding of network connection between the occipital and parietal structures and other brain regions is essential for successful surgical treatment of these epilepsies.

A DEFINITION AND ANATOMIC OVERVIEW

In contrast to other more common epilepsy syndromes, such as frontal or temporal lobe epilepsy the definition of epilepsies originating from the posterior parts of the brain is less precise. This is partially because seizures are less well localized in these brain regions as well as the fact that the epileptogenic zone might not necessarily respect the lobar boundaries. For this reason epilepsies originating from the parietal, occipital and posterior part of the temporal lobe are often referred to as posterior cortex epilepsies.¹ Overall it is discussed that posterior cortex epilepsies comprise less than 10% of all refractory epilepsies. In the group of patients undergoing presurgical investigation the percentage of posterior cortex and occipital lobe epilepsies varies between 2% and 20%, with pediatric epilepsy centers having substantially higher occurrence than adult centers.¹⁻³

Refractory posterior cortex epilepsies pose a special challenge to epileptologists, as the seizure semiology often is the result of fast ictal

propagation and might mimic frontal or temporal lobe semiology. For this reason a profound understanding of propagation pathways and epileptic networks is especially crucial in this group of patients.

CLASSIFICATION OF POSTERIOR CORTEX EPILEPSIES

Posterior cortex epilepsies have been divided into different subtypes in larger series of patients, because involved anatomic structures or by separating different typical pathways of seizure propagation. Most surgical series differentiate between isolated occipital lobe epilepsy, isolated parietal lobe epilepsy and combined types, such as occipito-parietal, occipito-temporal, and occipito-parieto-temporal depending on the size of surgery which is performed.⁴⁻⁶ Even if nomenclature is variable the key principle of these studies is to determine the percentage of epilepsies having an epileptogenic area beyond the border of 1 lobe and whether this finding has positive or negative

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impact on postsurgical seizure freedom. In the reported series, between 12% and 35% of patients received surgeries limited to only 1 lobe/location; multilobar resections were more common especially in pediatrics.^{3,5,6}

A second way of classifying posterior lobe epilepsies is based on understanding typical propagation pathways as discovered using intracranial depth electrode recordings. Craciun and colleagues⁷ differentiate between patients with ventral propagation to the temporal/mesiotemporal structures and patients with dorsal propagation to the frontal and parietal brain structures. This differentiation improves the understanding of some ictal phenomena. Occipital lobe symptoms were much more frequent in the dorsal propagation group, whereas typical temporal lobe symptoms were nearly exclusively seen in the ventral propagation group. However, some semiological findings were seen in both groups and therefore remain less well understood and unspecific.

Francione and coworkers² propose a different separation for their database of 208 patients, of whom 113 received intracranial stereoelectroencephalography (SEEG) investigations. Depending on results from noninvasive presurgical investigations the group differentiates 3 different patterns of possible intracranial electrode placement. Patient are investigated according to the posterior *pattern* if the epileptic network seems to be limited to the superior parietal lobule, intraparietal sulcus, angular and supramarginal gyri, lingual gyrus, cuneus, pericalcarine cortex, fusiform gyrus, and the posterior cingulate cortex. SEEG is planned after a *temporoposterior pattern* if in addition to the posterior pattern the mesial temporal structures, posterior insula, basal temporal region, and the inferior, middle, and superior temporal gyri might be involved. A *centroposterior pattern* of implantation is planned if the postcentral sulcus, precentral gyrus, operculum, inferior frontal gyrus, cingulate cortex, and posterior insula have to be explored in addition to the posterior *pattern*. Even if these patterns reflect typically performed SEEG investigation the *centroposterior pattern* is conceptually designed to investigate the *dorsal* propagation pathway described above and the *temporoposterior* pattern will cover propagation following the *ventral* pathway. Interestingly, in the cohort reported by Francione and colleagues, patients undergoing the posterior or centroposterior investigation are more likely to have a focal resection within 1 lobe and consequently to gain seizure freedom than patients with a temporoposterior implantation pattern.

Overall the different classification and concepts for subdividing posterior cortex epilepsies found in

the literature underline an important point. Epilepsies from this brain region are less well defined and often cross lobar boundaries. Key areas which might be involved in seizure onset and propagation are summarized in **Fig. 1**.

ANATOMIC CONSIDERATIONS AND FUNCTIONAL CONNECTIONS

On the mesial surface the occipital lobe is separated from temporal and parietal areas by the parieto-occipital sulcus. The calcarine fissure on the mesial surface separates the cuneus (O6) from lingual gyrus (O5). The lingual gyrus connects anteriorly to the parahippocampal gyrus. Directly underneath the surface of the occipital lobe, the superior, middle, and inferior occipital gyri fuse posteriorly to form the occipital pole.^{8,9} The primary visual cortex, an important structure to consider in epilepsy surgery, covers the mesial aspects of the occipital lobe in the upper and lower lips of the calcarine sulcus. The primary visual cortex is connected via the geniculocalcarine fibers to the lateral geniculate nucleus. In addition the superior retinal fields are connected via fibers to the superior part of the calcarine sulcus. The inferior retinal field fibers in contrast pass through the temporal lobe before reaching the primary visual fields. Although the knowledge of fibers from primary visual fields is essential for epilepsy surgery to avoid loss of function, they seem less relevant for seizure propagation. Knowledge about the connections from the visual association areas, however, clearly explain observed propagation patterns.

The visual association cortices are located in the lateral and superior occipital lobe as well as the posterior parietal areas. They comprise many tasks supportive to visual function, such as recognition, interpretation, and perception of color, depth, and motion. Long association bundles connect these regions with prefrontal, motor, insular, sensory, and temporal areas.⁸ Frontal lobes and visual association areas are directly connected via the superior longitudinal and inferior fronto-occipital bundles.¹⁰ Moreover, primary and secondary visual cortices are connected with each other via interhemispheric connections. The latter is the explanation for the often falsely lateralizing or widespread scalp EEG findings in occipital lobe epilepsies.^{3,5}

As described above, occipital lobe gyri directly merge into posterior temporal areas on mesial aspects of the brain. The parieto-occipital fissure creates a visible boundary between occipital and parietal lobe on the mesial surface. On the lateral aspects of the brain however the superior parietal

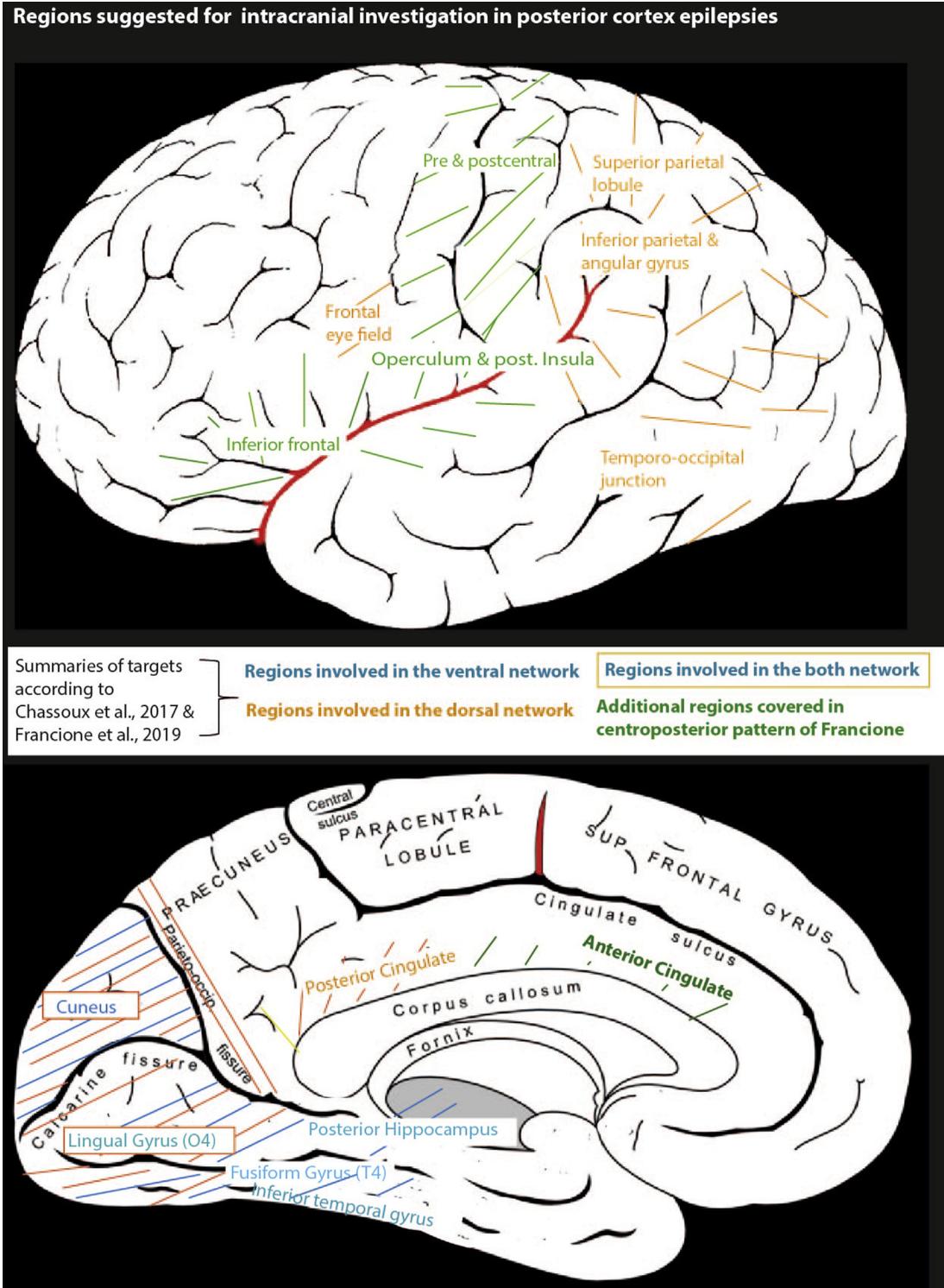


Fig. 1. Regions suggested for intracranial investigation in posterior cortex epilepsies.

lobule (P1) is directly connected with the superior occipital gyrus. Similarly the angular gyrus (p2) merges into the middle occipital gyrus and the inferior temporal gyrus directly merges into the

inferior occipital gyrus, with their separation only marked by the preoccipital notch. This continuity explains why so many lesions are not limited to 1 lobe but comprise the posterior quadrant.

Functionally, the postcentral gyrus serving primary sensory function is the best understood part of the parietal lobe. Like the occipital lobe the parietal lobe has long association bundles connecting somatic sensory areas with the frontal and visual association cortex.¹⁰ The sensory cortex is also closely linked to primary motor areas via the subcortical extrapyramidal pathway. Moreover, the corpus callosum directly connects the sensory areas on both hemispheres with each other.

Functions of the superior parietal lobe are less well understood and less likely to be recognized in typical seizure semiology. This region harboring the sensory association areas is well contacted to the ipsilateral cingulate, superior temporal gyrus, postcentral gyrus, and occipital region. This allows fast spread of ictal activity and most seizure generated here are most likely only symptomatic when spread has occurred.

Whether insular regions can be part of posterior cortex epilepsies is a matter of debate, with some groups investigating the posterior insula as part of intracranial investigations.³ The arcuate fasciculus borders the dorsal aspects of the insula and the inferior occipito-frontal fasciculus is close to the inferior insular border.

CLINICAL SPECIFICS OF POSTERIOR LOBE EPILEPSIES

Cause

The cause of posterior cortex epilepsies is similar to other refractory epilepsies. Most common structural lesions include focal cortical dysplasia and malformations of cortical development, as well as benign tumors and ischemic or gliotic changes.^{2,3,5} In all reported studies, lesional cases comprised more than 70% of patients, which might also represent a selection bias, as the posterior quadrant sometimes might not be even considered in patients with temporal or frontal semiology and in the absence of a lesion. Also many lesions (45%–68%) have unclear borders on MRI or span more than 1 lobe.^{2,11} The latter might explain the high percentage of intracranial EEG recordings of more than 60% in most adult studies despite evidence of a clearly epileptic lesion.²

Seizure Semiology

Posterior cortex epilepsies are the chameleons of epilepsies as seizure semiology can mimic an onset from many other brain regions. This is because of 2 facts, first the lack of some brain region in the posterior cortex to elicit objective visible positive seizure phenomena. Second, as described above, the extensive connectivity of

the posterior brain to other brain areas via fast propagation pathways. Although certain localizing phenomena, such as elementary visual auras and ocular-motor signs are considered specific for the occipital lobe, other auras, such as vertigo are less specific. Moreover, many patients only experience seizure phenomena like manual automatisms or tonic posturing that are usually not attributed to the posterior cortex. Even in confirmed occipital lobe epilepsy visual phenomena are reported in a little as 50% of adults.¹² **Table 1** summarizes ictal phenomena of some larger recent studies. In most studies, visual phenomena or auras are seen in less than 50% of cases. Many symptoms may suggest some posterior cortex involvement but rarely are specific enough to allow surgical planning without further investigations. In this table frontal and temporal seizure phenomena are separated, as has been suggested by some investigator.⁷ The separation reflects the 2 most common propagation phenomena from the posterior cortex and summarized seizure semiology usually associated with frontal and temporal lobe seizures respectively. In the presented studies typical temporal phenomena observed in posterior cortex epilepsies were manual and oral automatisms, loss of consciousness, as well as typical auras, such as epigastric or déjà vu. Frontal phenomena were usually motor manifestations, such as tonic posturing, contralateral cloni and hypermotor behavior.^{3,7,11}

The high connectedness of the posterior cortex might also explain some other phenomena which are described across studies. Most patients have a seizure onset at a young age with an average age of onset around 6 years even in the adult patient series.^{2,5} In patients with multilobar seizure onset, the average age was even younger: between 1 and 2 years. A relatively large proportion of patients present with epileptic spasm: up to 50% of those with occipital lobe plus epilepsy.³ Spasms persist beyond the first year and until surgery in 8% to 12% of patients.^{4,7,11} Moreover, patients present with unusually high seizures counts, with around 50% of adults presenting with daily seizures.² The occurrence of status epilepticus in 10% to 30% of patients also highlights the high seizure propensity in this group.

Results from Other Noninvasive Presurgical Investigations

Regarding seizure semiology the interictal and ictal EEG findings in posterior lobe epilepsy are far from clear and uniform. Some cases with focal lateral lesions might show unilateral posterior interictal epileptic discharges; most, however,

Study	Population & Patient No.	Daily Seizures	Elementary Visual	Visual Complex or Loss	Nonvisual Aura	Temporal Type	Frontal Type	Complex Partial Seizure	Spasms	Generalized Tonic-Clonic Seizure	Status
Jobst et al, ¹² 2000	Adult, 14	na	28.6%	21.4%	64%	57%	36%	100%	na	93%	na
Boesebeck et al, ⁶ 2002	Adult, 42	na	28.6	26.2	33%	na	na	78.6	na	80.1	na
Ibrahim et al, ⁴ 2011	Ped, 41	na	27%		22%	na	na	na	14%	5%	na
Liava et al, ³ 2013	Ped, 62	63%	17.8%	11.3%	25.8%	54.8%	30.6%	73%	26%	18%	11%
Ramantani et al, ⁵ 2017	Ped, 50	74%	na	na	na	na	na	na	na	56%	10%
Francione et al, ² 2019	Mixed 208, Ped & Adult	51%	30.8%		53.8%	46.7%	46.7%	na	4.8%	30%	9%
Sierra-Marcos et al, ¹¹ 2017	Ped 55	63,6	32.2%		21.8%	49.1%	na	na	14.5%	18.2%	na
Craciun et al, ⁷ 2018	Ped 20	Na	20%	25%	na	30%	20%	na	40%	20%	na

In all studies visual auras as the hallmark phenomenon are seen in less than 50% of patients and in the adult series nonvisual auras, such as vertigo or epigastric are more common than visual auras of any kind. Temporal and frontal type refer to seizures that would clinically be classified as having typical frontal or temporal semiology. Please note that Jobst et al., and Craciun et al., report primary occipital lobe only, whereas the other studies report posterior cortex epilepsies. Also note that there is some overlap in patients between Liava et al and Francione et al.

Abbreviation: na, information not available.

show multifocal, bilateral or even falsely localizing activity. Although focal unilateral EEG activity is considered prognostically relevant for seizure free surgical outcomes, the contrary is not the case for more widespread interictal activity. **Table 2** summarizes findings about the localization of scalp EEG findings and prognostic relevance from some recent studies. It is not surprising that ictal EEG activity is often visible over areas of ictal propagation and therefore might also be falsely localizing.^{3,7} Regarding all epilepsy types, ictal rhythms that occur only late within the clinical seizure should be considered carefully, especially in patients with other conflicting results.

As mentioned above positive MRI findings are likely to be the most common reason for considering a posterior epilepsy onset even in a context of temporal or frontal lobe semiology. Another flag is PET hypometabolism extending to posterior areas. This is especially the case in nonlesional temporal lobe epilepsy.⁷

Neuropsychological testing is often used in the presurgical evaluation to confirm or shed doubt on a localization hypothesis. This is complicated in posterior lobe epilepsies because of 2 reasons, first many parietal lobe functions are difficult to test and rather unspecific.¹³ Second many patients with early onset and frequent seizures have low baseline functioning overall.¹¹ The clearest focal abnormality is probably a preexisting visual field defect, which occurs in 10% to 36% of patients and is highly indicative of structural changes in occipital areas.^{3,5,7}

SURGICAL OUTCOME

Early reports on occipital lobe or posterior cortex epilepsies are rather discouraging in regard to the postsurgical seizure outcome. Especially in the context of high risks for visual functional deficits that might be caused by surgery. Boesebeck and colleagues⁶ summarize surgical studies before the year 2000 with variable results and with seizure freedom rates of between 25% and 90% of patients. Most studies, however, reported rates of around 50% and below.^{1,14–16}

In the 208 patients reported from Italy, 70% became seizure free after surgery.² Interestingly this was less frequently the case in the group investigated with SEEG than in the group without (80% vs 61%) indicating that the latter most likely had more complicated network constellations. Recently, reports were published with purely pediatric populations with comparably good outcomes, ranging from 65% to 85% of patients being seizure free after surgery.^{3,5,11} This is especially promising considering the disease severity

and early onset of posterior cortex epilepsies. **Table 3** summarizes the outcomes and variables considered relevant for outcome from selected studies. In summary, studies looked at varying factors that might influence outcome and there is conflicting evidence for many variables. This includes the effect of age of onset of epilepsy, duration of epilepsy, cause, and location of resection. Commenting on the cause is also difficult because many series only include patients with structural epilepsy. The negative correlation between the use of intracranial EEG and outcome is discussed to be mostly related to the fact that patients who underwent intracranial EEG had more challenging presentation of their epilepsy in regard to identifying the epileptogenic zone.

SPECIFICS IN PEDIATRICS

A substantial percentage of posterior cortex epilepsies occur in the pediatric population. This is reflected by the early onset in life, the high amount of seizures, and the likelihood to develop epileptic spasms. It has been hypothesized that this phenomenon is also related to the distribution of physiologic maturation during brain development, the posterior cortex being one of the first parts of the brain that is supposed to show gray–white matter differentiation and maturation, developing most of its key function within the first years of life.^{17,18} In the case of posterior lesions this maturation may be delayed and effect widespread networks early on.

A distinct pediatric problem is also the inability of patients to report subjective seizure phenomena. Craciun and coworkers⁷ suggest that many of the investigated patients had a period of behavioral arrest before the first objective seizure symptoms and that this might reflect a possible visual phenomenon that cannot be reported by the patients. In a patient without structural abnormality, the false localization of scalp EEG, which is more common in children combined with the inability to report auras may lead to a false localization of the epileptogenic areas (see **Table 2**). For pediatric epileptologists it is therefore especially important to be aware of the highly variable nature of seizure semiology in posterior cortex epilepsies. It is crucial to identify red flags, such as contradictory findings in the presurgical investigation, high daily seizure counts, a history of spasm and preexisting visual problems. The latter might actually help to support an occipital lobe hypothesis because 16% to 30% of pediatric patients showed preexisting visual field defects.^{3,5} In possible candidates for posterior cortex epilepsies, intracranial investigations might be helpful and it will be a

Table 2
Summary of whether interictal or ictal electroencephalography was believed to be localizing, lateralizing, or rather widespread or even falsely lateralizing

Study	Interictal Localizing	Interictal Scalp Laterlizing	Interictal False Localizing/ Laterlizing	Ictal Localizing	Ictal Laterlizing	Ictal False Laterlizing/ Localizing	% of Intracranial EEG	Interictal SEEG Localizing	Ictal SEEG Localizing
Jobst et al, ¹² 2000	43%	93%	7%	43%	93%	7%	100%	na	na
Boesebeck et al, ⁶ 2002	66%	29%	5%	na	59.5%	10%	0%	na	na
Ibrahim ^b et al, ⁴ 2011	71%	12%	21%	76%	4%	20%	56.1%	na	na
Liava et al, ³ 2013	35.5%	98%	19.3% (localizing)	37.5	32%	25%	38.7%	46%	80%
Ramantani et al, ⁵ 2017	34%	66%		54%	46%		26%	na	na
Francione et al, ² 2019	16% (40%) ^a	18%	11%	28% (50%) ^a	9%	5.5%	55.8	na	na
Sierra-Marcos et al, ¹¹ 2017	na	na	20%	63%	83.3%	16.7%	36.4	na	na
Craciun et al, ⁷ 2018	na	na	na	20%	80%	20%	100%	na	na

Even if EEG in posterior epilepsies is generally seen as less reliable, the percentage of patients with widespread or false localization of EEG activity is generally lower than the ones with correct localization. Overall misleading EEGs seem to be more common in the pediatric series.

Please note that authors Jobst and Liava count all localizing findings also as lateralizing findings while the author groups classify either EEGs as lateralizing or localizing. Ramantani et al, only differentiate regions versus nonregional EEG changes. Francione et al, differentiates between localizing and regional.

Abbreviation: na, information not available.

^a Number in brackets refers to regional. In Ibrahim et al EEG correlation is given only for seizure free patients.

^b 27 patients.

Table 3
Summary of the correlations found between clinical characteristics of patients with posterior cortex epilepsies and postsurgical outcome

Study	% Seizure Free	Age at Onset	Epilepsy Duration	Cause	Scalp EEG	Intracranial EEG	Resection size/ Completeness (Epileptogenic Zone)	Resection Site	Other
Jobst et al, ¹² 2000	50%	na	Na	No	Na	na	No	Yes	
Boesebeck et al, ⁶ 2002	45	na	Na	All lesional Tumor > malformations of cortical development	No (only postoperative)	na	No	No	Lateralizing aura > nonlateralizing aura
Ibrahim et al, ⁴ 2011	68% ^a	No	Yes	No	No	na	No	No	Contralateral magnetoencephalography dipole as negative predictor
Liava et al, ³ 2013	85.5%	Yes	No	Structural > nonlesional	na	Yes	Yes	No	SEEG was negative predictor
Ramantani et al, ⁵ 2017	70%	No	Yes	No	No	na	No	Parietal < occipital Left < right hemisphere	
Francione et al, ² 2019	69.7%	na	na	No	na	Yes	na	Yes	SEEG as negative predictor for outcome
Sierra-Marcos et al, ¹¹ 2017	62%	No	No	All structural	Postoperative interictal epileptiform discharge	na	No	No	No. of antiepileptic drugs and high seizure frequency as negative predictor
Craciun et al, ⁷ 2018	68.7%	No	No	All structural	na	na	No	Yes	Patient with frontal propagation > temporal propagation Extensive PET hypometabolism, as negative predictor

Completeness of resection refers to a complete removal of the assumed epileptogenic zone as defined by available diagnostics. Two studies suggested that the necessity of intracranial EEG is negatively correlated with outcome.

Yes, correlation between variable and outcome; na, information not available; no, no correlation.

^a Engel class 1 and 2 reported together.

question for the future whether the increased use of intracranial EEG in children in recent years¹⁹ will lead to more posterior resections even in children with nonlesional epilepsy.

CONSEQUENCES FOR INTRACRANIAL ELECTROENCEPHALOGRAPHY

As with all intracranial evaluations the key concepts of these investigations have to be reconsidered in the face of posterior cortex epilepsies. Invasive EEG studies can be used to delineate epileptic areas in the absence of lesional tissues, in cases with unclear lesional borders or multiple lesions. Alternatively they should be considered whenever the noninvasive presurgical investigation produces contradictory findings regarding localization of epileptic tissue. In the context of posterior lobe epilepsies, these might be large undefined lesions in the posterior part of the brain or a lesion in the occipital area with a seizure semiology pointing toward temporal, parietal or frontal epilepsies.² Some studies have demonstrated success in nonlesional posterior lobe epilepsies even if these are underrepresented in the literature.

A second valid reason from implanting patients with posterior cortex epilepsy is functional mapping and the aim of preserving visual, sensory or language function. In these situations, it is important to be aware of preexisting deficits. In contrast to language representation, however, visual fields do not switch location even in patients with longstanding early epilepsies and the same is true for most sensory areas. Functional imaging and tractography might therefore be a less invasive and reliable way to assist in surgical planning.^{20,21}

Before planning an intracranial depth investigation to delineate the epileptic zone in posterior cortex epilepsies, such as in all invasive EEG procedures, the predominant question and hypothesis addressed in the investigation has to be clear. More commonly than in other epilepsies pathways of propagation which might explain semiology have to be covered with electrodes to differentiate between the epileptogenic and symptomatic zones.²² For this task the 3 implantation schemes, posterior, centroposterior and temporoposterior are helpful to revise possible structures that might be involved in possible ways of spread. However, the implantation patterns can always only reflect the hypothesis rising from the noninvasive presurgical investigation and do not necessarily correlate with the results of the investigation. In cases in which scalp EEG was widespread or nonlateralizing, bilateral coverage may be necessary.²³

In posterior cortex epilepsies intracranial investigation should not only help to shed light on the

seizure onset and spread but also delineate the extent of the epileptogenic zone as precisely as possible.¹⁴ This is especially crucial in the occipital lobe and visual cortex, as the postoperative visual deficits can have large impact on the patient's daily life. Therefore, it is absolutely crucial that electrodes cover the mesial as well as lateral aspects of the occipital lobe. Moreover electrode placement should allow differentiating between activities arising from above and below the calcarine and parieto-occipital fissure, respectively.^{2,3,5} Providing full coverage of the occipital lobe can be challenging. Posterior-anterior-oriented electrodes entered directly from the back of the head should definitely be avoided as this is unpleasant for patients and might cause electrode breakage.²³ Fig. 1 is a schematic of possible implantation patterns as suggested by Chassoux and colleagues²³ and Francione and colleagues.²

The availability of intracranial EEG methods varies largely across geographic locations. Some publications analyzed the usefulness of subcortical grid recordings in the occipital lobe and were successful in delineating the epileptogenic zone.^{12,24} Although subcortical electrodes might be easier to use when aiming to stimulate for functional mapping in the posterior temporal language areas, they cannot localize epileptic areas located within deeper structures of the brain. This may pose a challenge when considering a seizure onset zone in the occipital pole or occipital mesial structures. Subdural electrodes also might be limited when investigating larger networks of propagation, such as aiming for frontal eye fields in a posterior cortex epilepsy.²⁴

Independent of the chosen method, epileptologists will be blind to all brain areas not explored during the intracranial investigation. The use of simultaneous scalp and intracranial recordings might help to better understand patterns of propagations in areas not covered by intracranial electrodes and avoid misinterpretations.²⁵ The fact that surgical outcome after intracranial EEG investigations decrease rather than increase the likelihood to become seizure free should raise awareness that posterior cortex epilepsies are complex and epileptologists should be aware of the limitations of all investigations in the context of making surgical decisions. Methods of automatic analysis in the future may become more widely accessible to help differentiate between seizure onset and spread.²⁶ These methods might be especially helpful in posterior lobe epilepsies to identify patients in whom the seizure onset has been missed during the investigation or propagation phenomena are hard to understand.

SUMMARY

Posterior cortex epilepsies are less common and more diverse than those deriving from temporal or frontal brain structures. When talking about network structures these epilepsies are a prime example with seizures mimicking onset from other brain regions due to fast ictal propagation. This poses a challenge for identifying posterior cortex epilepsies as they are often overlooked if no structural lesion is present. Seizures often start early in life, are frequent, and are devastating. Successful treatment often requires network understanding and extensive diagnostic investigations to provide best possible seizure and functional outcome.

DISCLOSURE

The author has nothing to disclose.

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