

Early seizure propagation from the occipital lobe to medial temporal structures and its surgical implication

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ABSTRACT – Intracranial EEG documentation of seizure propagation from the occipital lobe to medial temporal structures is relatively rare. We retrospectively analyzed intracranial EEG recorded with electrodes implanted in the medial temporal lobe in patients who underwent occipital lobe surgery. Four patients with occipital lesions, who underwent intracranial EEG monitoring with intracerebral electrodes implanted in the medial temporal lobe prior to occipital lobe surgery, were studied. Subdural electrodes were placed over the occipital lobe and adjacent areas. Intracerebral electrodes were implanted into bilateral hippocampi and the amygdala in three patients, and in the hippocampus and amygdala ipsilateral to the lesion in one. In light of the intracranial EEG findings, the occipital lobe was resected but the medial temporal lobe was spared in all patients. The follow-up period ranged from six to 16 years, and seizure outcome was Engel Class I in all patients. Sixty six seizures were analyzed. The majority of the seizures originated from the occipital lobe. In complex partial seizures, ictal discharges propagated to the medial temporal lobe. No seizures originating from the temporal lobe were documented. In some seizures, the ictal-onset zone could not be identified. In these seizures, very early propagation to the medial temporal lobe was observed. Interictal spikes were recorded in the medial temporal lobe in all cases. Intracranial EEG revealed very early involvement of the medial temporal lobe in some seizures. Seizure control was achieved without resection of the medial temporal structures.

Key words: occipital lobe epilepsy, medial temporal lobe, intracranial EEG, seizure spread, epilepsy surgery

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Surgery is increasingly considered in patients with occipital lobe epilepsy, and several prognostic factors for surgery have been reported (Boesebeck *et al.* 2002, Lee *et al.* 2005; Dalmagro *et al.* 2005). Subclassification of occipital lobe epilepsy by comparing ictal semiology, neurological examination

and scalp EEG was also attempted (Blume *et al.* 2005). However, the surgical strategy for occipital lobe epilepsy has not yet been completely established.

Abundant multisynaptic projections from the occipital lobe to lateral and mesial temporal structures have been

Table 1. Clinical characteristics.

Patient	Age at onset (years)	Age at surgery (years)	EEG		Seizure Semiology		Preoperative visual field
			Interictal	Ictal	Subjective	Objective	
1	15	28	Lt O, Lt T	Lt T	elementary visual	versive	Lower quadrantanopsia
2	9	17	Lt pT-O	Lt pT-O	cephalic	versive, arm clonic, GTC	Normal
3	6	17	Rt O	Rt O	elementary visual, ocular sensation	blinking, oral automatism, versive, arm clonic, GTC	Upper quadrantanopsia
4	18	41	Lt T	Lt T	visual illusion	oral, manual automatisms	NA

Lt: left; Rt: right; O: occipital; T: temporal; pT: posterior temporal; GTC: generalized tonic-clonic; NA: not available.

demonstrated (Jones and Powell 1970, Turner *et al.* 1980), and it is well known that occipital-originating seizures may readily spread to the temporal lobe (Ludwig and Ajmone Marsan 1975, Olivier *et al.* 1982; Williamson *et al.* 1992, Salanova *et al.* 1992, Palmini *et al.* 1993). However, detailed descriptions of ictal discharge propagation in occipital lobe epilepsy are relatively rare, and its relevance for surgical strategy has not been clarified. To elucidate this issue, we retrospectively analyzed four patients who underwent intracranial EEG with additional depth electrodes implanted in the medial temporal lobe prior to occipital lobe epilepsy surgery.

Patients and methods

Of the 17 patients who received intracranial EEG monitoring prior to the occipital lobe surgery, four patients (three males and one female) had also intracerebral electrodes implanted in the medial temporal lobe structures including the hippocampus and the amygdala (*table 1*). Age-at-onset ranged from six to 18 (mean 12) years, and age at surgery from 17 to 41 (mean 25.8) years. The history included perinatal disorder (threatened premature labor, forceps delivery, birth weight 2050 g) in Case 1, simple febrile convulsion in Case 3, and no remarkable finding in the other two cases. All patients experienced auras. Among the three patients with visual auras, two had elementary visual auras of seeing light or colors, while the third had visual illusions. The fourth patient (Case 2) had cephalic aura. This patient had reported visual aura during the early period of her illness. Objective seizure symptoms included aversion of the head and eyes to the contralateral side of the suspected epileptogenic zone in three cases, rapid bilateral blinking in one, oral automatisms in two, and manual automatisms in one.

Interictal scalp EEG demonstrated spikes in the occipital region or the posterior temporal region in three cases, and spikes in the anterior temporal region in one case. Ictal scalp EEG showed ictal discharges in the occipital region or from the occipital to posterior temporal region in two

cases, and ictal discharges in the temporal region in the other two cases. Preoperative computed tomography (CT) or magnetic resonance imaging (MRI) demonstrated a structural lesion in the occipital lobe in all patients (*figure 1*). The preoperative visual field was evaluated in three patients using a Goldman perimeter. Quadrantanopsia was found in two cases and normal visual field in one. For the intracranial EEG, subdural electrodes were placed over the occipital lobe and adjacent cortical areas ipsilateral to the lesion in all cases. In addition, intracerebral electrodes were implanted using a stereotactic system into bilateral hippocampi and the amygdala in three patients, and in the hippocampus and amygdala ipsilateral to the lesion in one patient.

As a result of the intracranial EEG findings, the occipital lobe was resected but the medial temporal structures were spared in all patients. Total resection of the occipital lobe including the lesion was performed in two patients, and resection of the lateral cortex of the occipital lobe including the lesion was carried out in the two remaining patients (*table 2*). The histopathological diagnosis was of focal cortical dysplasia in two cases, cephalocele in one, and infarct in one. Hemianopsia was found in three patients after surgery. The follow-up period ranged from 6 to 16 (mean 12.3) years. The seizure outcome was Engel Class I-a, b, c and d. In Case 2 (Class I-b), visual auras developed from the third year following surgery.

The intracranial EEG recordings were reviewed for ictal-onset zones, spread of spontaneous seizures, and the presence or absence of interictal spikes in the medial temporal lobe structures.

Results

A total of 83 seizures were captured on intracranial EEG recordings of the four patients. Of these, 66 seizures with ictal EEGs that included the medial temporal lobe structures in the montage were analyzed. The sites of seizure-onset are shown in *table 3*. The majority of the seizures originated from the occipital lobe. In complex partial

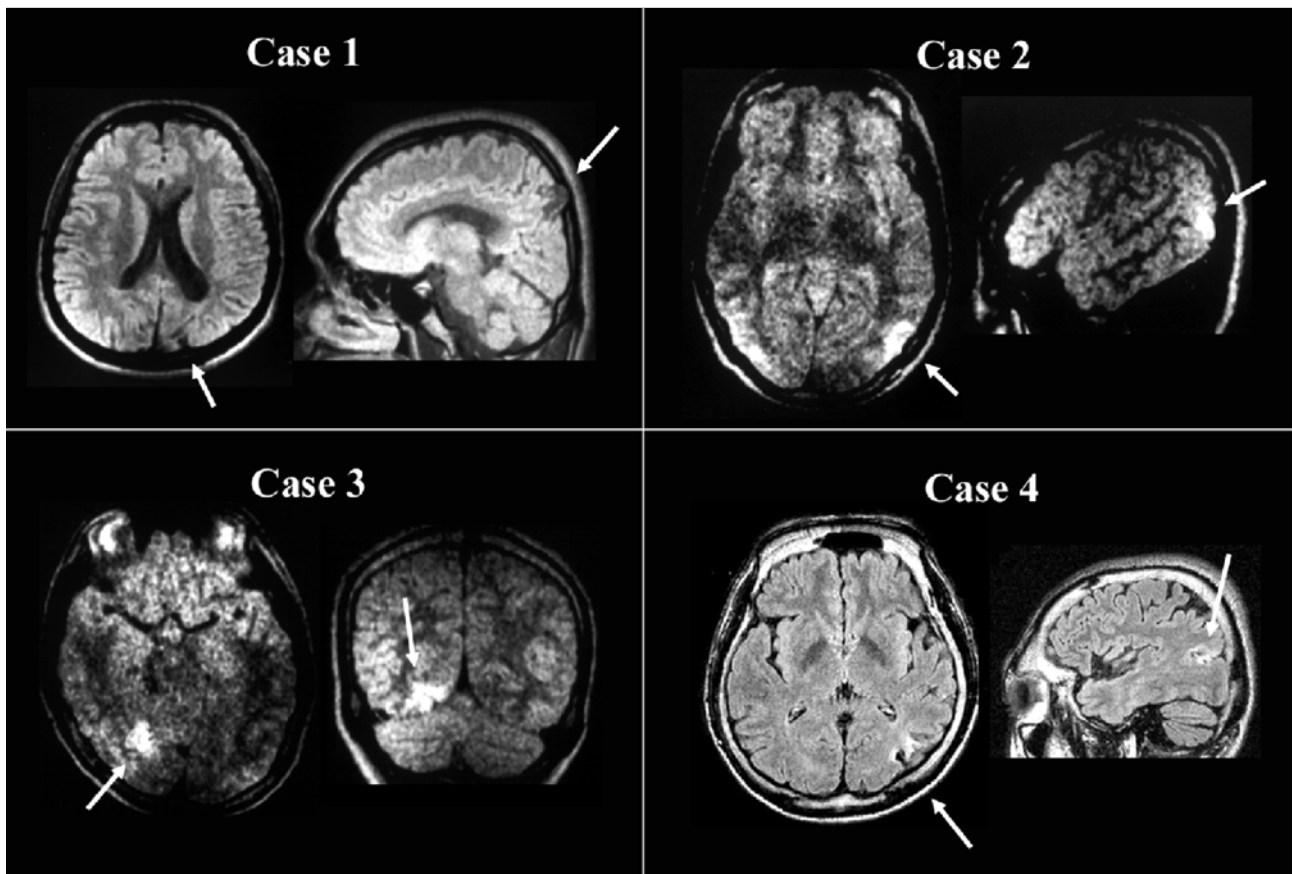


Figure 1. MRI studies showing structural lesions in four patients. Arrows indicate lesions. Upper left: long spin-echo (SE) MRI with a 0.3-T scanner showing a hypointense area in the left parieto-occipital region. Upper right: long SE MRI showing a hyperintense lesion in the left lateral occipital lobe. Lower left: long SE MRI showing a hyperintense lesion in the right inferomedial occipital lobe. Lower right: FLAIR MRI with a 1.5 scanner showing a hyperintense lesion in the left lateral occipital lobe.

seizures, the ictal discharges propagated to the medial temporal lobe. No seizure originating from the temporal lobe was observed.

In two patients (Cases 2 and 4), the ictal discharges apparently originated from the occipital lobe in all the seizures analyzed. Case 2, with a left lateral occipital lesion manifested both simple and complex partial seizures. In simple partial seizures characterized by a strange feeling at the back of the eye, the ictal discharges were confined to the left occipital lobe. In complex partial seizures, ictal dis-

charges initially started from the left lateral and basal occipital lobe. The patient became unresponsive when the discharges propagated to bilateral medial temporal structures. Ictal discharges also spread to the contralateral occipital lobe (*figure 2*).

In Case 3 with a right inferomedial occipital lesion, EEG onset in the right occipital lobe was observed in 21 seizures. During the elementary visual aura, ictal discharges were confined to the right occipital lobe. When the seizure started with an aura of feeling the eyes pulled to the

Table 2. Surgery, pathology, and seizure outcome.

Patient	Surgery	Pathology	Postoperative visual field	Follow-up (years)	Outcome (Engel)
1	Occipital lobectomy	Cephalocele	Hemianopsia	16	I-d
2	Lateral occipital corticectomy	Cortical dysplasia	Normal	14	I-b
3	Occipital lobectomy	Cortical dysplasia	Hemianopsia	13	I-c
4	Lateral occipital corticectomy	Infarction	Hemianopsia	6	I-a

Table 3. Intracranial EEG findings.

Patient	Number of seizures	Site of seizure-onset	
		Occipital	Unknown
1	9	8	1*
2	20	20	0
3	29	21	8*
4	8	8	0

* Ictal onset zone could not be identified precisely in these seizures.

left followed by impaired consciousness and adersion to the left, ictal discharges started from the occipital lobe and propagated to medial temporal structures. However, in

eight complex partial seizures, ictal EEG revealed initial widespread electrodecrement, followed by fast activity predominantly in the ipsilateral medial temporal lobe. At the same time, rhythmic activity of lower frequency appeared in the medial occipital area (figure 3). No auras were reported in these complex partial seizures. No differences in objective clinical symptoms were observed between the two types of EEG seizures.

In Case 1, eight seizures originated from the occipital lobe. However, in one secondarily generalized seizure, the initial change in ictal EEG was low voltage fast activity in the contralateral temporal base. Then, early involvement of bilateral medial temporal lobes was observed (figure 4).

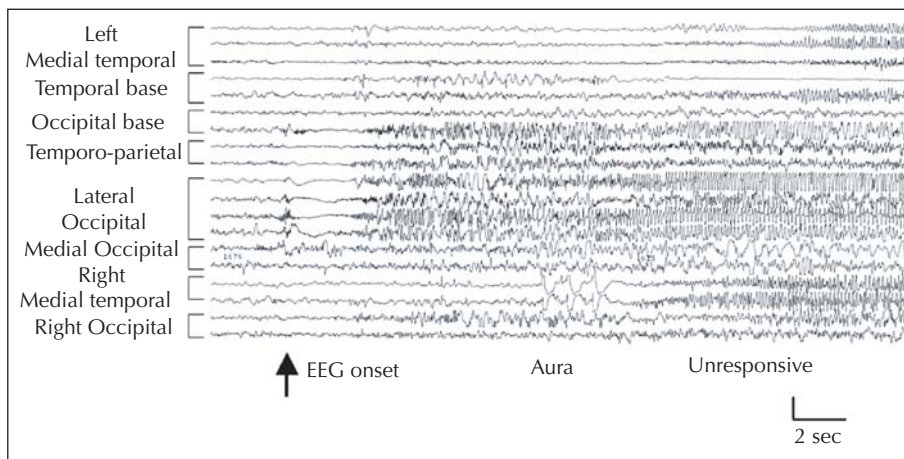


Figure 2. Ictal EEG of a complex partial seizure in Case 2 showing EEG-onset in the left lateral and basal occipital lobe and then spreading to the bilateral medial temporal lobes. When ictal discharges spread to bilateral medial temporal lobes, the patient became unresponsive. Ictal discharges also spread to the contralateral occipital lobe.

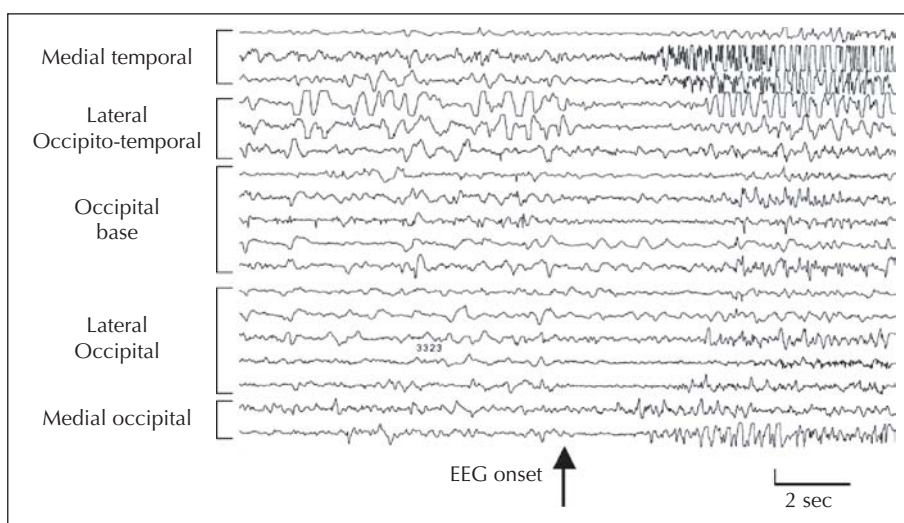


Figure 3. Ictal EEG of a complex partial seizure in Patient 3 showing initial widespread electrodecrement (arrow denotes EEG onset). About two seconds later, fast activity in the right medial temporal lobe was observed. At the same time, a rhythmic activity of slower frequency appeared in the medial occipital area. Then, semi-rhythmic activity appeared in the lateral occipito-temporal, basal occipital, and lateral occipital areas.

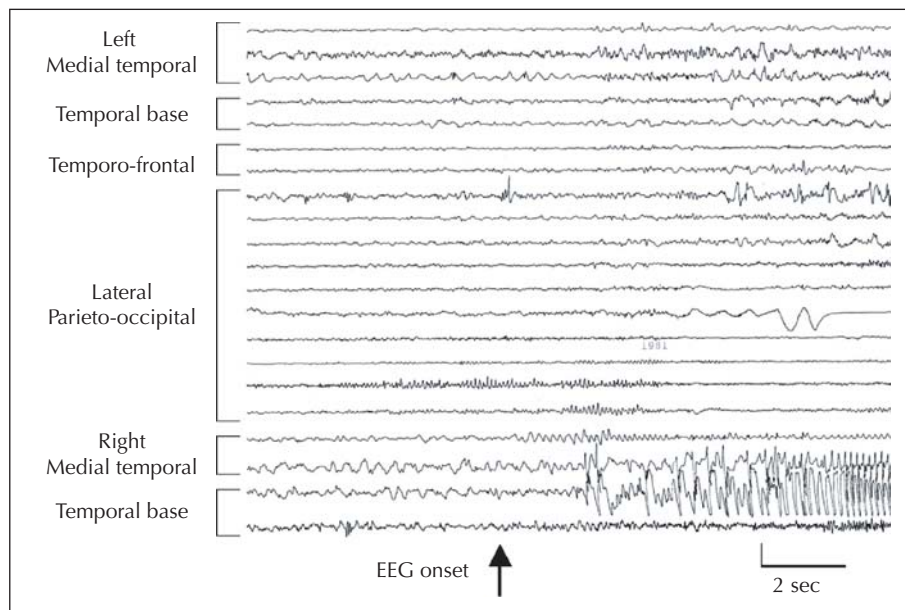


Figure 4. Ictal EEG of a secondarily generalized seizure showing low voltage fast activity in the right basal temporal area as the first EEG change (arrow denotes EEG onset). Two to three seconds later, ictal discharges appeared in the bilateral medial temporal lobes.

Interictal spikes in the medial temporal lobe structures were observed in all four patients. In Case 4, the discharges were more marked on the contralateral side than the lesion side.

Discussion

It is known that occipital seizures may spread rapidly to parietal and sensorimotor cortex, or more commonly propagate to the temporal lobe to produce clinical symptoms (Ludwig and Ajmone-Marsan 1975). Intracranial EEG studies have demonstrated that seizures originating in the occipital lobe spread readily to the medial temporal lobe (Olivier *et al.* 1982, Williamson *et al.* 1992, Salanova *et al.* 1992, Palmini *et al.* 1993). The inferior longitudinal fasciculus is considered to be a possible pathway of spread (Olivier *et al.* 1982). However, the implications of these findings for a surgical strategy, namely, whether to include the medial temporal structures in the resection, have not been clarified. Several studies reported the results of temporal resection sparing the occipital epileptogenic zone in such cases. However seizure outcome was unfavorable (Fish *et al.* 1991, Palmini *et al.* 1993). In our cases, most of the seizures originated in the occipital lobe. In complex partial seizures, ictal discharges propagated to the medial temporal lobe. In eight seizures of Case 3, and one secondarily generalized seizure of Case 1, the actual ictal-onset zone could not be identified, possibly due to sampling error. Nevertheless, it is clear that medial temporal structures were very rapidly involved in these seizures. The variable patterns of involvement of medial temporal

structures may implicate variable patterns of seizure-onset and spread. Medial temporal resection is unnecessary to relieve seizure even in cases with very early involvement of medial temporal structures during seizures. Interictal spikes in the medial temporal structures cannot be used as indicators of epileptogenesis.

Williamson *et al.* (1992) reported twenty-five patients with occipital lobe epilepsy. Seventeen patients underwent intracranial recording, and fifteen patients had temporal lobe-type seizures during some or all of their attacks. In 12, occipital to medial temporal seizure spread was demonstrated. No seizures of temporal lobe origin were recorded. Although the number of patients in our series is small, no seizures originating in the temporal lobe were observed in any of our patients with occipital lesions. In occipital lobe epilepsy with structural lesions, it may not be essential to implant electrodes in the medial temporal lobe. □

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