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Distinctive epileptogenic networks for parietal operculum seizures

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ABSTRACT

Objective: The present study investigated the electroclinical features and epileptogenic networks of parietal operculum seizures (POS) by using stereoelectroencephalography (SEEG) intracerebral recordings.
Methods: Comprehensive presurgical evaluation data of seven patients with drug-resistant epilepsy with POS were analyzed retrospectively. Stereoelectroencephalography-recorded seizures were processed visually and quantitatively by using epileptogenicity mapping (EM), which has been proposed to ergonomically quantify the epileptogenicity of brain structures with a neuroimaging approach.
Results: Six patients reported initial somatosensory or viscerosensitive symptoms. Ictal clinical signs comprised frequently nocturnal hypermotor seizures and contralateral focal motor seizures, including tonic, tonic–clonic, or dystonic seizures of the face and limbs. Interictal and ictal scalp EEG provided information regarding lateralization in the majority of patients, but the discharges were widely distributed over perisylvian or "rolandic-like" regions and the vertex. Furthermore, two subgroups of epileptogenic network organization were identified within POS by SEEG, visually and quantitatively, using an EM approach: group 1 (mesial frontal/cingulate networks) was observed in three patients who mainly exhibited hypermotor seizures; group 2 (perisylvian networks) was observed in four patients who mainly exhibited contralateral focal motor seizures.
Conclusion: This study indicated that POS could be characterized by initial specific somatosensory sensations, followed by either frequently nocturnal hypermotor seizures or contralateral focal motor seizures. The distinctive seizure semiology depended on the organization of two primary epileptogenic networks.
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1. Introduction

The human parietal operculum (OP) consists of four distinct cytoarchitectonic areas (OP 1–4) that are involved in somatosensory, pain, and vestibular processing [1–4]. In contrast to the well-investigated OP cytoarchitecture, few studies have described the anatomoclinical correlations in seizures originating from the OP; these were often roughly regrouped as parietal lobe seizures or insular-opercular seizures [5–7].

As the secondary somatosensory cortex (SII) is located on the OP, it is not surprising that parietal operculum seizures (POS) manifest somatosensory signs such as anesthesia, pain, and burning sensations. However, those somatosensory signs can also originate from the primary somatosensory cortex (SI) located in the parietal lobe and insular cortex. In addition, hypermotor seizures, which primarily suggest frontal seizure onset [8–11], have also been reported in the parietal lobe including the OP and insular-opercular regions [5–7,12]. Therefore, it is necessary to investigate the electroclinical features of seizures originating from the OP and to distinguish them from seizures originating from other regions, including the frontal lobe, other parietal lobe regions, and the insular lobe.

Furthermore, the underlying mechanism of semiology in patients with POS is unclear. The emergence of seizure semiology may rely on epileptic network organization that includes the epileptogenic zone and propagation networks [13,14]. Nonhuman primate studies and functional imaging studies in humans have demonstrated that the insular cortex is densely connected to sensorimotor regions, including the supplementary motor area (SMA)/pre-SMA and cingulate cortex [15–

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17]. It is possible that a similar anatomic and functional network exists in the OP. When epileptic discharges are restricted to the OP region, only somatosensory signs are present. However, when the discharges propagate to other connected cortical regions, such as the primary motor cortex, SMA, and the cingulate cortex, additional elementary motor seizures and complex motor seizures are present. The occurrence of different semiologies in POS reflects the spread of seizures to different cerebral regions that may constitute distinct epileptogenic networks.

In addition, depth electrode implantation is required in many cases, particularly when magnetic resonance imaging (MRI) is negative or demonstrates large extended lesions. The stereoelectroencephalography (SEEG) investigations offer a unique opportunity to precisely study the electroclinical features and epileptogenic networks of POS.

Epileptogenicity mapping (EM) has been proposed to quantify ictal high-frequency oscillations (iHFOs) at seizure onset by adopting the framework of statistical parametric mapping (SPM) software as a neuroimaging approach [18]. This tool quantifies changes in iHFOs, as recorded by SEEG, between seizure onset and a preictal baseline period for each intracranial contact. The epileptogenicity map has been proven to exhibit good reproducibility and sensitivity and has deepened our understanding of epileptogenic brain networks underlying various types of focal seizures [19–21].

This study aimed to characterize the electroclinical features of POS and to determine whether different networks of POS, associated with different seizure semiologies, could be identified visually and quantitatively by using EM.

2. Methods

2.1. Patient selection and presurgical evaluation

The data of patients with drug-resistant focal epilepsy, who were evaluated for resective surgery at our center from July 2013 to June 2017, were retrospectively reviewed to select patients who met the following criteria: (i) SEEG recording included the OP cortex; (ii) the seizure onset zone predominantly involved in the OP cortex, as demonstrated by invasive SEEG recording; and (iii) patients underwent surgical resection that included parts of the OP cortex. This study was approved by the Tsinghua University Yuquan Hospital Human Ethics Review Committee (No. 2016005), and informed consent forms were signed by the patients or their guardians.

All patients had a comprehensive evaluation, including detailed history and neurological examination, neuropsychological testing, 3-T MRI, long-term scalp video-electroencephalography (video-EEG), and SEEG recordings. The SEEG explorations were performed by using intracerebral multiple contact electrodes (HKHS Healthcare Co., Ltd., Beijing, China; 8–16 contacts, length: 2 mm, diameter: 0.8 mm, 1.5 mm apart) that were placed following the guidelines of the Leksell Stereotactic System (Elekta, Stockholm, Sweden). The arrangement of electrodes was tailored according to a predefined localization hypothesis, based on noninvasive findings. After implantation, computerized tomography (CT) scans were performed, and images were coregistered with preimplantation MRI to accurately check the anatomical location of each contact along the electrode trajectory by using SinoPlan software (Sino Precision Medical Technology Co., Ltd., Beijing, China). For multimodality image coregistration, 6 degrees of freedom, cubic linear interpolation, and normalized mutual information algorithms were used. Signals were recorded on a 256-channel Nihon Kohden digital EEG machine (NeuroWorkbench, Nihon Kohden, China). The signals were recorded with a bandpass filter between 0.08 and 600 Hz, at a sampling rate of 2000 Hz. The data were acquired by using a referential montage, with the reference electrode chosen in the white matter. For data analysis, we used a bipolar montage between adjacent contacts of the same electrode to improve sensitivity to local activity.

2.2. SEEG signal analysis

2.2.1. SEEG recordings and visual analysis

Electrode arrangement, tailored according to each patient's requirements, typically covered the perisylvian structures (the insular and opercular cortices), lateral and mesial frontocentral cortices, parietal cortex, and (occasionally) temporal cortex. The OP cortex was sampled by using the transopercular approach, which employed stereotactic trajectories either orthogonal to the midsagittal plane or with different degrees of obliquities; the internal contacts sampled the insular cortex, and the external contacts covered the supra- or infrasylvian opercular structures.

All seizures were visually analyzed to determine the seizure onset zone (i.e., the exact timing of the first relevant electrical changes that occurred prior to the clinical onset of the seizure). The seizure onset zone was defined from SEEG recordings by using different patterns: (i) low-voltage fast activity over 20 Hz and (ii) recruiting fast discharge of spikes or polyspikes [18]. Furthermore, propagation structures were determined based on the emergence of fast activities or recruiting discharges of spikes or polyspikes that followed the first electrical changes of the seizure onset zone.

2.2.2. EM

The EM approach was first proposed in 2011 [18]. It has good reproducibility and sensitivity and has proven to be helpful in enhanced comprehension of epileptogenic networks. Specifically, the origin of time for each seizure was determined by using visual analyses at the time of suspected seizure onset. Baseline recordings were chosen as periods of at least 20 s without strong artifacts or epileptic activity, during the 5 min preceding each seizure. The placement of seizure onset did not consider isolated or repetitive spikes at lower frequencies, which might have preceded or began with few seizures.

The raw SEEG signals were transformed into the time–frequency domain around seizure onset, typically by using a Morlet wavelet transform, which yielded a measure of SEEG power P (f, t), at time t and frequency f, as output for each electrode. Mean power at seizure onset was compared with mean power obtained from the baseline period. To capture temporal changes of the frequency distribution of power, overlapping epochs of data were successively selected from the time–frequency plane, whose epoch duration could be adapted to the expected duration of bursts of SEEG oscillations. In this study, the epoch duration was 3 s, with a time step of 2 s. The band of interest in high frequency was selected based on visual analysis of power transformations; it varied among patients (average, 80–120 Hz; lowest, 30–60 Hz; highest, 100–180 Hz). Log transformation was applied to obtain a distribution of data more closely resembling a normal distribution; images of ictal power were produced by using local spatial projections of SEEG values on each patient's cortical surface and smoothing with an isotropic Gaussian kernel, 3 mm in width. Statistical analyses were then performed in SPM12 software (www.fil.ion.ucl.ac.uk/spm) on images of log-power. Differences in the log-power of rapid discharges between seizure and baseline were obtained by using a standard two-sample *t*-test on the images of log-power [22], which determined epileptogenicity index values. When several seizures were available for the same patient, the map of epileptogenicity was computed for the group of seizures. The epileptogenicity index was defined as the *t*-value of the differences in smoothed log-power between seizure and baseline. Statistical significance was directly obtained with the associated family-wise error corrected p-value (<0.05). A secondary analysis was performed at the group level by averaging EM among patients [21]. To allow group analyses, all images were normalized to the MNI space by using nonlinear normalization, as proposed in SPM software [23]. This transformation was computed by using the T1 MRI of every patient and was applied to the electrode coordinates taken from the coregistered CT, in order to obtain the electrode coordinates in the MNI space.

Table 1
Main clinical features of the patients.

Patient	Sex	Age at SEEG (years)	Age at onset (years)	Aura	Main objective features	IID[a]	ID[a]	SOZ	Propagation network
1 (Gr2)	M	11	8	Abdominal discomfort, chest distress	Right facial tonic and right versive	C3, P3, T3, T5	C3, P3, T3, T5	OP	ALG, OT
2 (Gr1)	F	27	17	Left electric paresthesia	Hypermotor (wave, twisting)	C4, P4, T4, T6, Cz, Pz,	C4, P4, T4, T6, Cz, Pz,	OP	ACC, ALG, PCL
3 (Gr2)	M	20	11	Palpitation, left hand anesthesia	Left arm tonic	F4, C4, T4	Right hemisphere	OP	SMG, OF, PSG
4 (Gr2)	F	4	3	Left hand pain	Left facial and hand tonic	F4, C4, Fz, F8, T4	C4, T4	OP PLG	ALG, PSG
5 (Gr2)	F	37	10	Left arm anesthesia	Salivation, left hand dystonia	F4, C4, Fz, Cz, T4	No lateralization	OP	PSG, PLG
6 (Gr1)	M	12	4	No	Hypermotor (rocking)	C3, Cz, T3	F3, C3, Fz, F8, T4	OP	MCC, cs, MFG
7 (Gr1)	M	10	1.5	Whole body burning sensation	Hypermotor (kick, rotation)	C3, P3, Cz, F7, T3	Fz, Cz	OP	SMA, PCL ACC

F, female; M, male; IID, interictal discharges; ID, ictal discharges; SOZ, seizure onset zone; OP, parietal operculum; OF, frontal operculum; OT, temporal operculum; SMG, supramarginal gyrus; PCL, paracentral lobule; PSG, posterior short insular gyrus; ALG, anterior long insular gyrus; PLG, posterior long insular gyrus; SMA, supplementary motor area; ACC, anterior cingulate gyrus; cs, cingulated sulcus; Gr1 and Gr2: two subgroups of epileptogenic network organization.
[a] The 10–20 international system of electrode placement.

3. Results

Among 153 consecutive SEEG procedures performed in our center, seven patients were selected on the basis of the above criteria. There were four males and three females, with age at onset that ranged from 4 months to 17 years, and epilepsy duration that ranged from 1 year to 27 years. Magnetic resonance imaging disclosed features of focal cortical dysplasia in two patients (patients 2 and 4), prenatal stroke in one (patient 3), and tuberous sclerosis complex with inferior parietal tubers in two (patients 6 and 7); MRI was negative for two patients (patients 1 and 5). The OP was involved for all five MRI-positive patients. All patients had undergone resections that included the OP and parts of the posterior insular cortex. Table 1 summarizes the main clinical features of all included patients, and Table 2 summarizes the main features of surgery, histology, and postoperative follow-up.

3.1. Ictal clinical features

3.1.1. Subjective manifestations

Most of the studied patients, with the exception of patient 6, reported initial specific subjective manifestations. Somatosensory manifestations were reported in five patients; these were described as electrical paresthesia, anesthesia, pain, and burning sensation. The cutaneous distribution was restricted to the hand or arm, whereas the burning sensation was distributed throughout the body. Viscerosensitive symptoms were reported in one patient, including abdominal discomfort and chest distress.

3.1.2. Objective manifestations

Three patients exhibited seizures mainly comprising hypermotor seizures, whereas the other four patients mainly exhibited contralateral focal motor seizures, including tonic, tonic–clonic, or dystonic seizures of the face and limbs. Secondary bilateral asymmetric tonic seizures and generalized tonic–clonic seizures were solely recorded in patients 1 and 4 respectively. Tachycardia, as well as hyperventilation and hypersalivation, was observed during seizures in patients 3 and 5, respectively. In six patients, awareness and contact with the environment were fully preserved during seizures. In addition, all patients had a high-seizure frequency (several per day), and seizures were nocturnal in six patients.

3.2. Electrophysiological investigations

3.2.1. Interictal and ictal scalp EEG

Interictal EEG showed informative epileptic discharges, including sharp spikes, spike and wave, and polyspikes, during both wakefulness and sleep in all patients. In most patients, interictal and ictal EEG abnormalities provided lateralization information but were widely distributed over perisylvian, frontocentral, central-parietal, and temporal regions. Given that this derivation mimicked benign rolandic epilepsy with centrotemporal spikes (BECTs), we designated these regions as “rolandic-like”. Moreover, the vertex was often involved (Table 1).

3.2.2. Visual analysis and electrical cortical stimulation

The seizure onset zone of each patient was visually localized in the OP. Two groups of main early propagation networks were identified. Group 1 involved mesial frontal/cingulate network structures, including the anterior cingulate gyrus, middle cingulate gyrus, cingulate sulcus, and SMA; these were observed in patients 2, 6, and 7 (Fig. 1). Group 2 involved perisylvian structures, including the frontal operculum, temporal operculum, posterior short insular gyrus, anterior long insular gyrus, posterior long insular gyrus, and supramarginal gyrus; these were observed in patients 1, 3, 4, and 5 (Fig. 2).

In addition, electrical cortical stimulation confirmed that habitual aura or seizures were elicited in six patients when the OP underwent high-frequency stimulation (50 Hz, pulse width: 0.3 ms, intensity: 0.5–3 mA). Somatosensory responses, such as anesthesia, were also elicited by stimulating the OP, posterior insular cortex, and postcentral gyrus. However, pain and temperature responses were only obtained in the OP and posterior insular cortex. Moreover, skin territories that

Table 2
Main features of surgery, histology, and postoperative follow-up.

Patient	Side	Surgery	Histology	Engel's class	Follow-up (months)
1	L	OP and pINS resection	FCDIIa	IA	42
2	R	OP resection	FCDIIb	IA	40
3	R	SMG, OP, and pINS resection	Encephalomalacia	IA	24
4	R	OP and pINS resection	FCDIIa	IA	10
5	R	OP, OT, and pINS resection	FCDIb	IIA	11
6	L	OP and tuber resection	TSC	IA	15
7	L	OP and tuber resection	TSC	IA	13

L, left; R, right; OP, parietal operculum; OT, temporal operculum; SMG, supramarginal gyrus; pINS: posterior insular; FCD, focal cortical dysplasia; TSC, tuberous sclerosis complex.

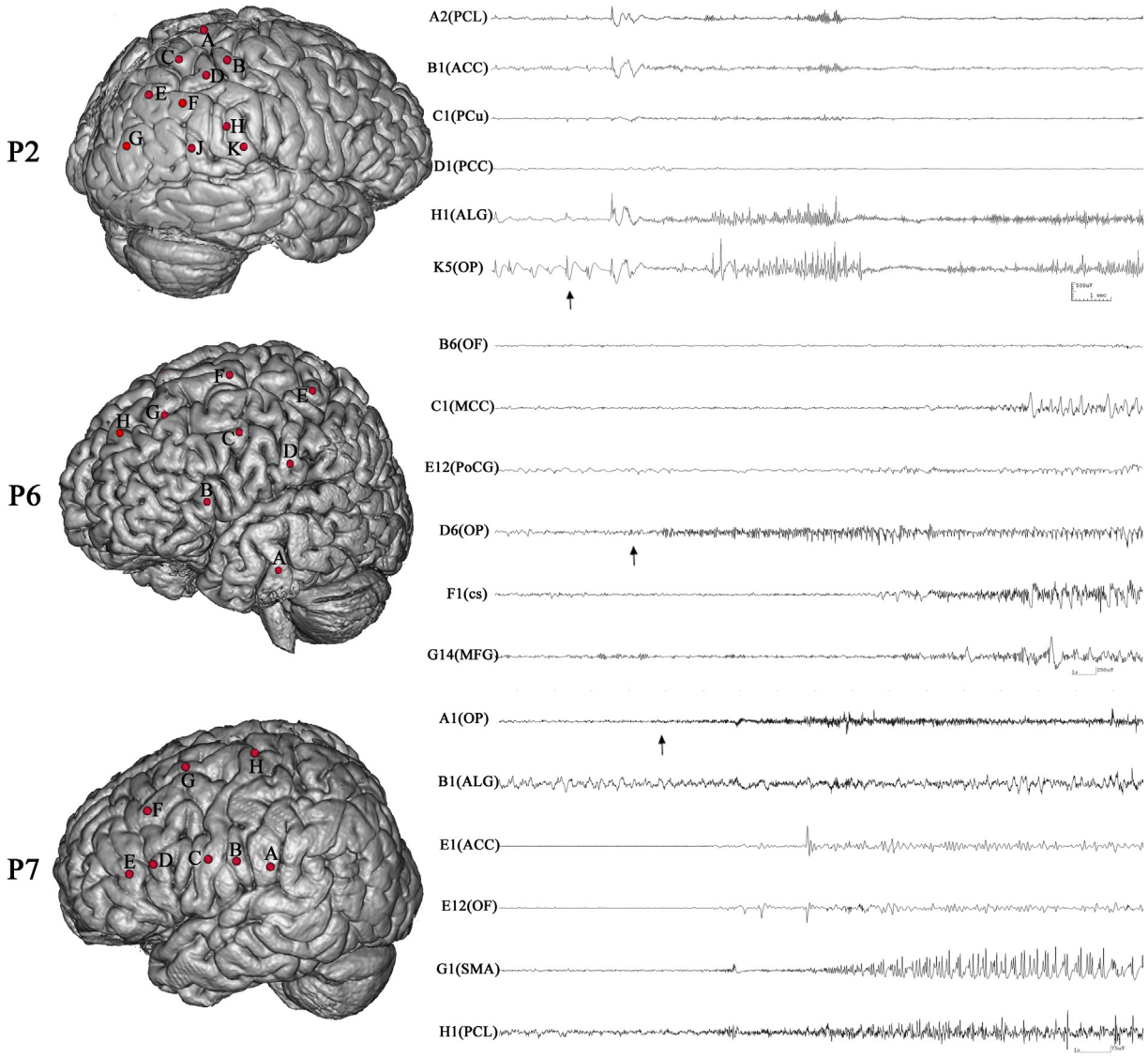

Fig. 1. SEEG recordings from group 1 demonstrate that the propagation network involved mesial frontal/cingulate structures (the left is SEEG implantation scheme with electrode labels, and the right is SEEG recording from the contact of each electrode corresponding to a brain region; arrows indicate seizure onset). OP, parietal operculum; OF, frontal operculum; SMG, PCL, paracentral lobule; ALG, anterior long insular gyrus; SMA, supplementary motor area; ACC, anterior cingulate gyrus; PCu, precuneus; MCC, middle cingulate gyrus; PoCG, postcentral gyrus; cs, cingulated sulcus; MFG, middle frontal gyrus.

involved somatosensory responses obtained after stimulation of the OP and insula varied from restricted regions (such as a hand or a foot) to large areas (half of the body). Somatosensory responses obtained after postcentral gyrus stimulation were restricted (such as a foot, the tongue, or a thumb) and sometimes manifested as a marching phenomenon. Table 3 summarizes the results of electrical cortical stimulation for each patient.

3.2.3. EM

In agreement with the visual analysis, EM at seizure onset revealed that the maximum epileptogenicity index was located in the OP. Furthermore, for the patients in group 1, EM also disclosed a relatively high-epileptogenicity index in mesial frontal-central structures, including the paracentral lobule, anterior cingulate gyrus, and SMA (Fig. 3). In contrast, for the patients in group 2, EM disclosed high-epileptogenicity indexes locally in perisylvian structures, including the frontal operculum, temporal operculum, posterior short insular gyrus, anterior long insular gyrus, posterior long insular gyrus, and supramarginal gyrus (Fig. 4). These results, which quantified the epileptogenicity of brain structures by adopting a neuroimaging approach, confirmed two subgroups of epileptogenic network organization in POS. A secondary analysis was performed at the group level by averaging EM among the patients of each group (Fig. 5). The result clearly showed distinctive network organization.

4. Discussion

4.1. Electroclinical features of POS

In the literature, most POS have been sporadically reported as parietal or insular-operculum seizures [5,6]. The present study is the first to report a cohort of seven patients with POS confirmed by SEEG, and to summarize their electroclinical features and epileptogenic network organization.

Somatosensory manifestations were the most frequently subjective symptom, observed in five patients. They were described as electrical paresthesia, anesthesia, pain, and burning sensation, which reflects the role of the OP in basic and higher order somatosensory processing [24]. In terms of electrical cortical stimulation, habitual aura or seizures were elicited in six patients when the OP underwent high-frequency

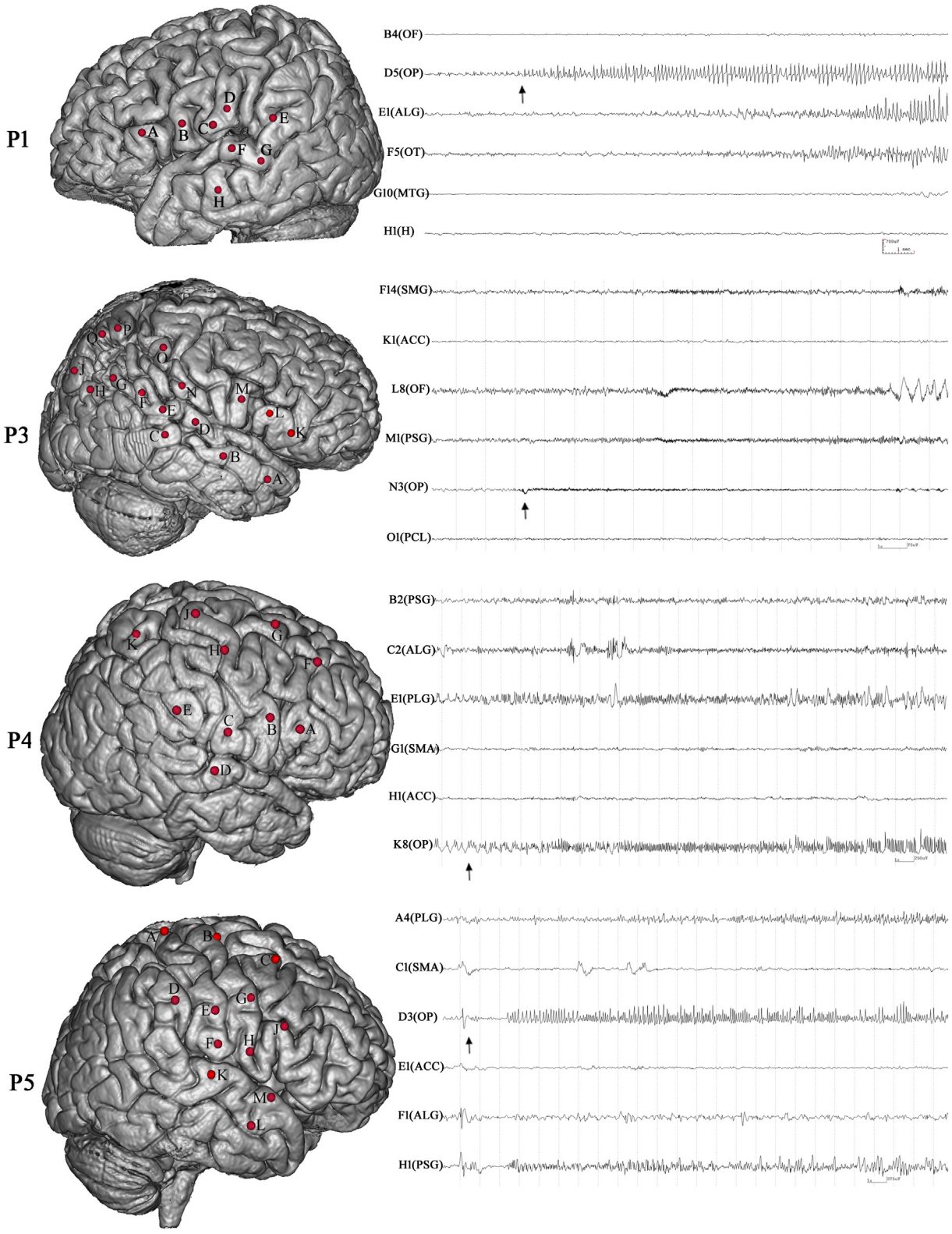

Fig. 2. SEEG recordings from group 2 demonstrate that the propagation network involved perisylvian structures (the left is SEEG implantation scheme with electrode labels, and the right is SEEG recording from the contact of each electrode corresponding to a brain region; arrows indicate seizure onset). OP, parietal operculum; OF, frontal operculum; OT, temporal operculum; SMG, supramarginal gyrus; PCL, paracentral lobule; PSG, posterior short insular gyrus; ALG, anterior long insular gyrus; PLG, posterior long insular gyrus; SMA, supplementary motor area; ACC, anterior cingulate gyrus; MTG, middle temporal gyrus; H, hippocampus.

stimulation. The data indicated that patients' seizures originated from the OP. Somatosensory responses elicited by the OP (SII) and posterior insular cortex differed from those elicited by the postcentral gyrus (SI): pain and temperature responses were only obtained in the former. Moreover, skin territories involved in somatosensory responses obtained after OP and insula stimulation may be larger than those associated with SI stimulation; the latter revealed a marching phenomenon. This finding was consistent with the previous study that suggested

Table 3
Main electrical cortical stimulation results for each patient.

Patient	OP	PSG	ALG	PLG	PoCG	SMA
1	Right arm and facial anesthesia; abdominal pain and nausea[a]	Right arm cold sensation	Right leg anesthesia	—	Right facial anesthesia	—
2	Left leg or left body electric paresthesia[a]	—	Left foot anesthesia	—	Left leg anesthesia marching to left arm	Left arm electric shock and clonus
3	Left hand anesthesia and palpitation[a]	—	—	—	Left foot anesthesia marching to left crus; left hand anesthesia	—
4	Left hand pain and tonic seizure[a]	—	Left hand pain[a]	Left auditory hallucination	—	—
5	Left arm anesthesia and throbbing[a]	Head throbbing	Lower jaw throbbing	—	Left hand or tongue anesthesia	Perioral paresthesia
6	Right facial anesthesia	—	—	—	Right thumb anesthesia	—
7	Right half body anesthesia and burning sensation[a]	—	—	Right limbs anesthesia	—	Right arm anesthesia

OP, parietal operculum; PSG, posterior short gyrus; ALG, anterior long insular gyrus; PLG, posterior long insular gyrus; PoCG, postcentral gyrus; SMA, supplementary motor area; MCC, middle cingulate gyrus; —, stimulations were negative or no electrode covered the regions.
[a] Habitual aura or seizures.

ictal pain was reproduced by stimulation of the insula or SII region but not by stimulating the SI area. Additionally, pain did not spread from one somatotopic territory to adjacent territories, and the spatial extent of the pain was larger [25]. However, it might be difficult to differentiate the responses from SII and the dorsal posterior insular cortex. The cytoarchitectures of these two regions were similar and there was no clear demarcation between them. Hence, the localization value of this initial specific symptom could be considered in the presurgical evaluation.

With regard to the objective manifestation, hypermotor seizure was a prominent and frequent sign (3/7), consistent with prior reports.

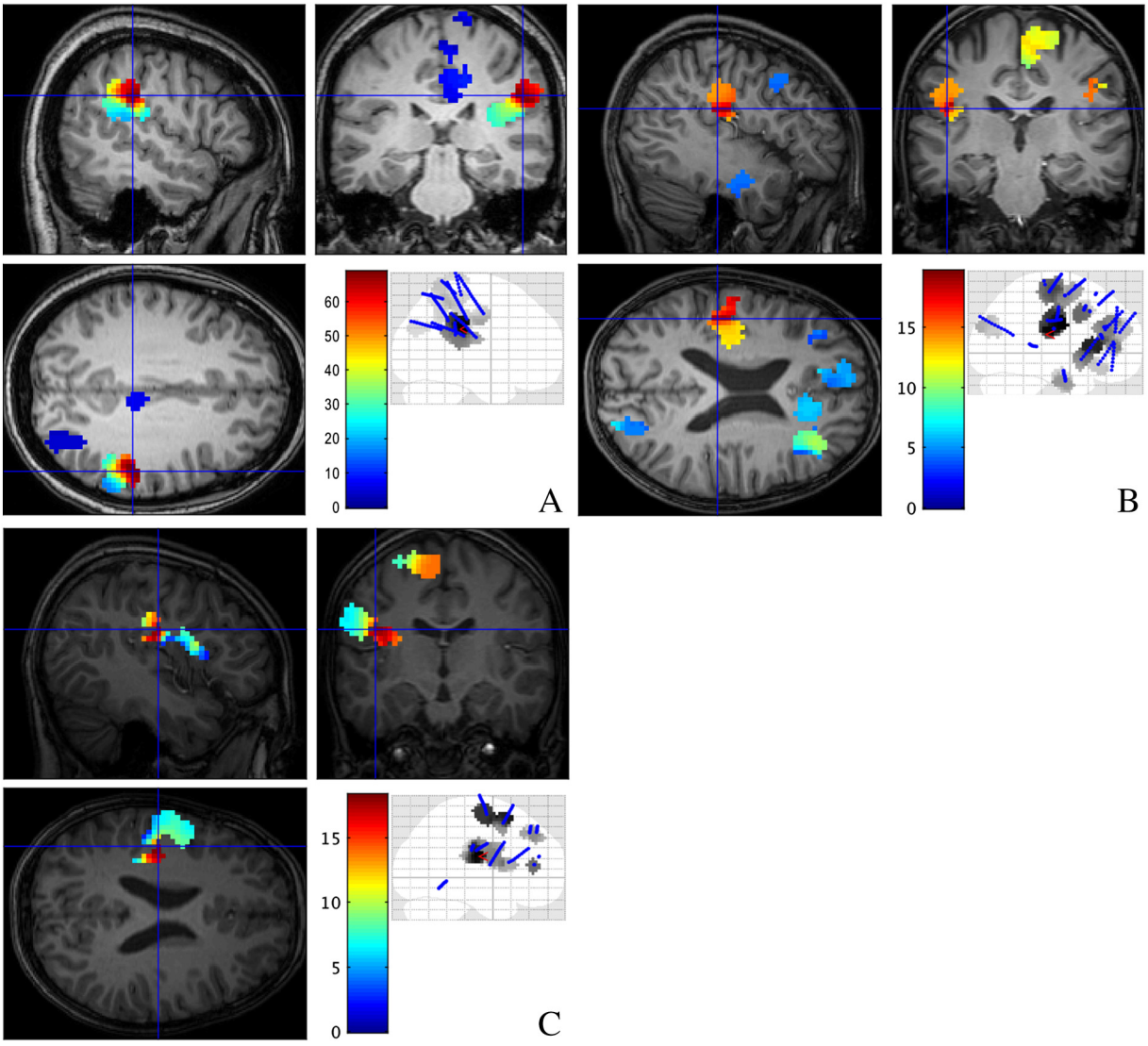

Fig. 3. Epileptogenicity mapping of the three patients from group 1. (A–C) Map of significant ($p < 0.05$, family-wise error corrected) ictal HFOs obtained at the onset of all seizures recorded for patients 2, 6, and 7, respectively. Colors indicate the *t*-statistics. Electrodes and maps were drawn over the MNI152 atlas. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

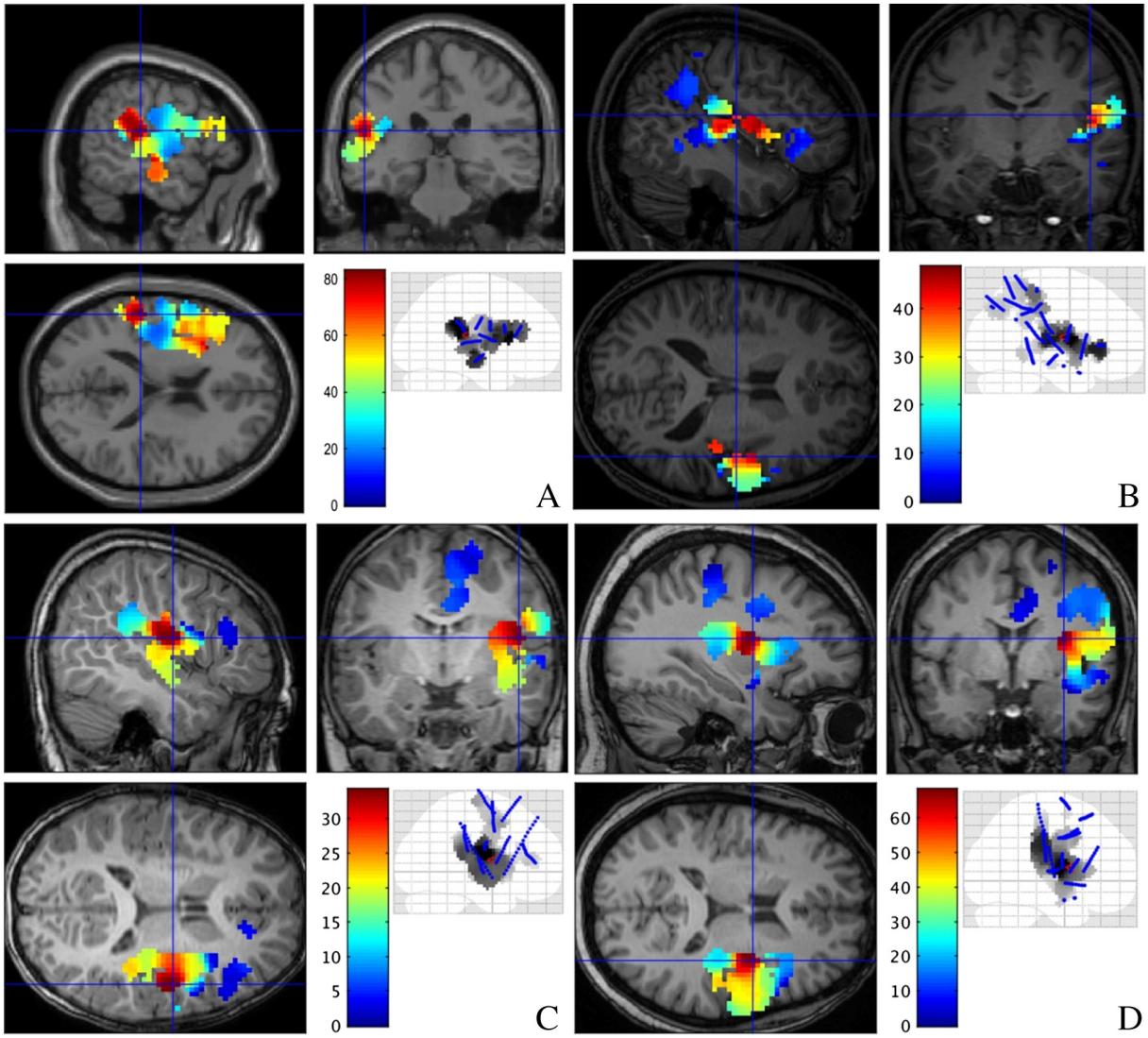

Fig. 4. Epileptogenicity mapping of the four patients from group 2. (A–D) Map of significant ($p < 0.05$, family-wise error corrected) ictal HFOs obtained at the onset of all seizures recorded for patients 1, 3, 4, and 5, respectively. Colors indicate the *t*-statistics. Electrodes and maps were drawn over the MNI152 atlas. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Hypermotor seizure is a classical manifestation of frontal lobe seizures but has also been reported in patients with seizures in other regions, including the operculoinsular cortex [6,26] and parietal lobe [5,27]. The presence and type of aura might help to distinguish the various types of epilepsy that give rise to hypermotor seizures [27]. Therefore, hypermotor seizures preceded by somatosensory sensation might be of OP origin. Other objective signs included contralateral focal motor seizures mainly involving the face and hands.

Regarding the scalp EEG recordings, interictal and ictal scalp EEG provided lateralizing information in the majority of patients, but these discharges were widely distributed over perisylvian or "rolandic-like" regions and the vertex. This characteristic discharge distribution may be ascribed to the anatomic localization of the OP, which is deeply buried in the sylvian fissure. As a result, spike dipoles could be recorded in both up and down directions.

4.2. Subgroups of epileptogenic networks organization in POS

We found two subgroups of epileptogenic network organization in POS by SEEG, visually and quantitatively, by using EM analysis: group 1 (mesial frontal/cingulate networks) and group 2 (perisylvian networks). It was notable that the two distinct networks corresponded to two respective main objective manifestations. For the mesial frontal/cingulate networks, hypermotor seizures were primarily reported in mesial frontal or orbitofrontal epilepsies [11]. Accordingly, the occurrence of hypermotor movements in extrafrontal epilepsies was suggested to reflect seizure spread to the frontal lobe [6,26]. Furthermore, Hagiwara found significant functional coupling between the insula and mesial frontal/cingulate regions by using nonlinear regression analysis [27]. Frontal semiology was expressed by strong functional couplings between the insula and mesial frontal regions. Our results demonstrated that the OP gave rise to the hypermotor seizures by the organization of mesial frontal regions, including the SMA and cingulate gyrus. For the perisylvian networks, contralateral focal motor seizures mainly involving the face and hands were the predominant manifestation. These motor signs were attributed to the propagation to the precentral gyrus, frontal operculum, and sensorimotor area of the insular cortex.

When we compared the quantitative EM with visual SEEG analysis, the results were concordant in most patients. However, the regional propagation network involved was more broadly distributed in patients 5 and 6. The ictal discharges might spread faster than the late involved regions, as calculated by EM. The subgroups of epileptogenic networks were also compared with scalp EEG recordings. We found that ictal

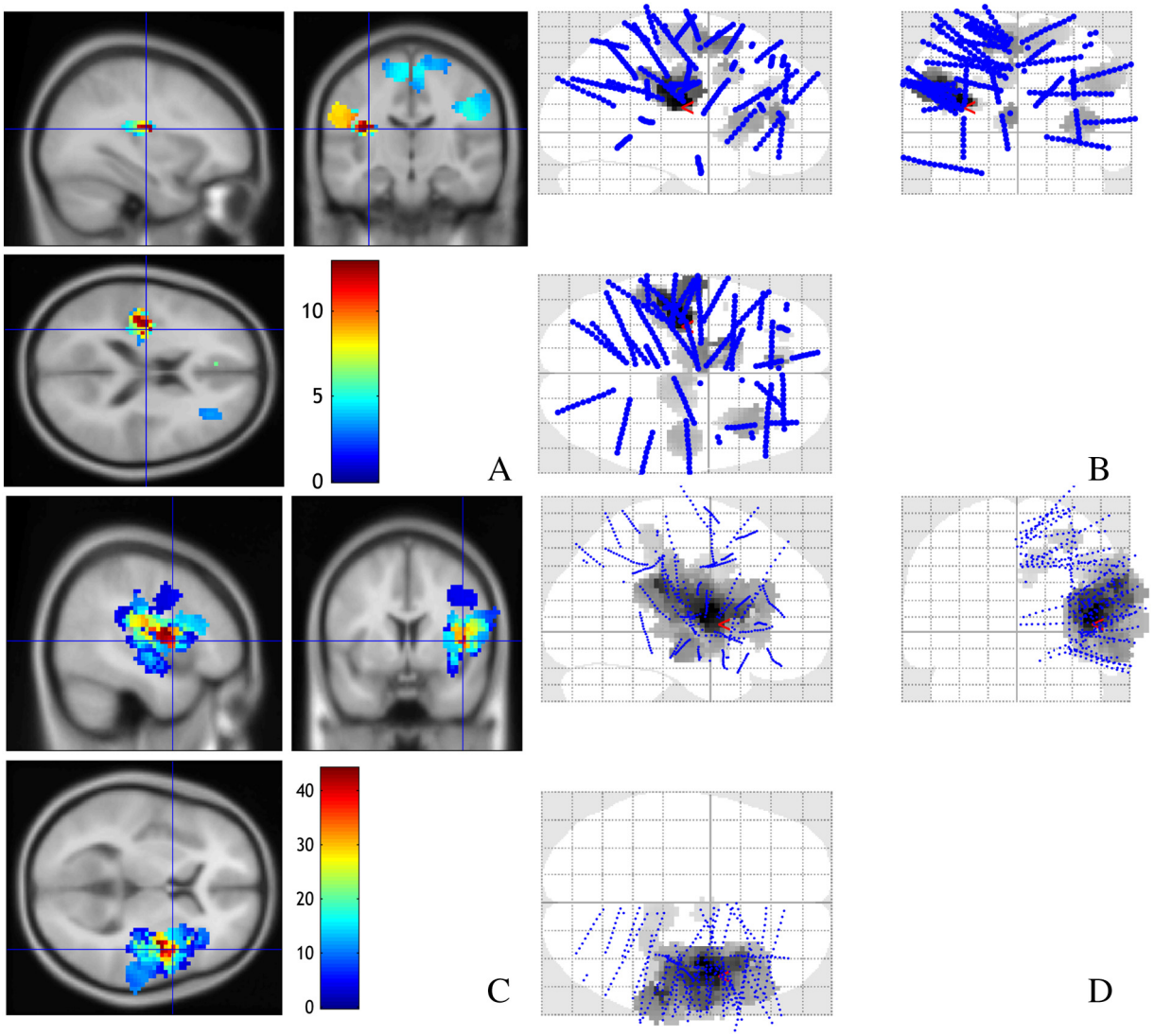

Fig. 5. Epileptogenicity mapping averaged among patients at the group level. (A–B: group 1; C-D: group 2). Electrodes and maps were drawn over the MNI152 atlas. Epileptogenicity mapping of one patient from each group (patient 2 in group 1 and patient 1 in group 2) was left–right reversed for visual illustration.

discharges of scalp EEG in all patients from group 1 involved midline regions: patient 1 involved C4, P4, T4, T6, Cz, and Pz; patient 6 involved F3, C3, F8, T4, and Fz; and patient 7 involved Fz and Cz. Therefore, the epileptologist might consider a spread from the OP when midline discharges are encountered in clinical practice.

When considering patient differences in age, histopathology, and hemispheric localization, with respect to the network groups, we could not perform statistical tests of age and hemispheric localization between the two network groups because this was a small retrospective study, although such variables could show potential effects on the reported network groups. Regarding histopathology results, 6/7 patients showed developmental disorders (focal cortical dysplasia (FCD) and tuberous sclerosis complex (TSC)); the exception was patient 3 (encephalomalacia).

5. Conclusions

This study found that POS were characterized by initial specific somatosensory sensations, followed by frequently nocturnal hypermotor seizures or contralateral focal motor seizures. The distinctive seizure semiology depended on two primary epileptogenic networks.

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Conflicts of interest

None of the authors have any conflict of interest to disclose.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.yebeh.2018.08.031.

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