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Parietal lobe epilepsy: the great imitator among focal epilepsies

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ABSTRACT – *Aim*. Comprising large areas of association cortex, the parietal lobe is part of an extensive synaptic network elaborately intertwined with other brain regions. We hypothesize that such widespread projections are responsible for producing inaccurate localisation readings on scalp EEG and clinical semiology in patients with parietal lobe epilepsies, as opposed to frontal or temporal lobe epilepsies. *Methods*. Our study included 50 patients with pharmacoresistant focal epilepsy, who were subsequently rendered seizure-free for ≥12 months (median: 23 months) following resections limited to the frontal (n=17), temporal (n=17), or parietal (n=16) lobes. Interictal and ictal EEG data with accompanying seizure video recordings were extracted from archived files of scalp video-EEG monitoring. Two blinded raters independently reviewed the EEG according to predetermined criteria. Videos of seizures were then observed, as raters formulated their final electroclinical impression (ECI), identifying patients' abnormal neuronal activities with parietal, temporal, and frontal lobe epilepsy, or unspecified localisation. *Results*. Groups did not differ significantly in demographics, age at epilepsy onset, or presence of MRI abnormalities. Interictal discharges in parietal lobe epilepsy showed the greatest magnitude of scatter outside the lobe of origin; the majority of patients with parietal lobe epilepsy had more than one spike population (p<0.045). Localised ictal EEG recognition was most frequent in temporal, followed by frontal and parietal lobe epilepsy cases (p=0.024). Whenever raters confidently limited their ECI to one lobar subtype, overall accuracy was excellent. Lobar classifications by ECI were highly accurate for temporal lobe epilepsy, vacillating in frontal lobe epilepsy, and least accurate in parietal lobe epilepsy subjects. *Conclusion*. Scalp EEG readings of parietal lobe epilepsy patients showed a more variable scatter of interictal discharges and a lower localisation value of ictal recordings compared to temporal and frontal lobe epilepsy subjects, suggesting an increased likelihood of misidentification and mislocalisation of parietal lobe epilepsy. Combining seizure semiology with scalp interictal and ictal EEG readings facilitates a more accurate lobar classification in patients with temporal and frontal, but not parietal, lobe epilepsy.

Key words: parietal lobe epilepsy, temporal lobe epilepsy, frontal lobe epilepsy, mislocalization, interictal EEG, ictal EEG, semiology

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Although the majority of epilepsy patients achieve seizure control through medical treatment, approximately one third of cases manifest a pharmacoresistant form of epilepsy (Kwan and Brodie, 2000; Kwan *et al*., 2010). Surgical resection may be a highly beneficial option for some patients with drug-resistant, focal epilepsy (Salanova *et al*., 1999; Jeha *et al*., 2006; Jeha *et al*., 2007; Kral *et al*., 2007; Jehi *et al*., 2009). Despite the significant progress in video-EEG recording technology, imaging techniques, and invasive mapping methods with depth and/or subdural grid electrodes, epilepsy surgery is successful in achieving complete and sustained seizure-freedom for more than 10 years in only nearly one half (49%) of patients (Salanova *et al*., 1999; Jeha *et al*., 2006; Jeha *et al*., 2007; Kral *et al*., 2007; Jehi *et al*., 2009; Téllez-Zenteno *et al*., 2007). The poorest outcomes resulted specifically from frontal resections (31% seizure-free at 10 years), as opposed to all other surgery types (57% seizure-free at 10 years after temporal lobe or posterior quadrant surgery) (Jeha *et al*., 2006; Jeha *et al*., 2007; Jehi *et al*., 2009). Within the frontal lobe resection group, the least favourable results were evident from those patients with non-lesional MRI (Jeha *et al*., 2006). The failure of resective frontal lobe surgery in controlling seizures may be attributed to an inability to entirely remove an epileptic, yet functional, cortex (Sarkis *et al*., 2010). Epilepsy surgery may, however, fail after the complete resection of the putative "epileptic focus," even when the epileptogenic zone is identified and delineated by direct cortical recordings (intraoperative or extraoperative) (Schwartz and Spencer, 2001; Sarkis *et al*., 2010). This ineffectiveness of epilepsy surgery in these cases may be due to some EEG patterns and electroclinical features that are erroneously interpreted as arising from the frontal lobe. However, these recorded and mapped epileptic patterns may represent "referred" EEG patterns from other areas of the brain, mainly the parietal and temporal lobes. The parietal lobe comprises large areas of association cortex extensively connected to other lobes. The tendency toward multiple spread patterns (in particular to the temporal and frontal lobes), although not an exclusive propensity, is characteristic of seizures originating in the parietal lobe (Williamson *et al*., 1992; Foldvary *et al*., 2001). This is reputedly not indicative of mesial temporal lobe seizures or most frontal neocortical seizures but was hypothesized to be due to spread from other areas of the brain (Williamson and Jobst, 2004).

Although the parietal cortex occupies the brain's second-largest cortical surface (after the frontal lobe) (Tramo *et al*., 1995), resections from the parietal lobe are rare in large, neocortical epilepsy surgical cases (Salanova *et al*., 1995; Binder *et al*., 2009). Out of the 1,212 patients who underwent epilepsy surgery at the Cleveland Clinic from 1996 to 2009, 758 cases required temporal lobe surgery, 323 had frontal resections, and 131 patients underwent posterior quadrant (parietal and/or occipital lobes) resections (Jehi *et al*., unpublished data). A salient ambiguity surrounds this variability among surgical case numbers, as these three anatomical areas (suspected surgical problem areas) occupy relatively similar volumes in the telencephalon (Tramo *et al*., 1995). We hypothesize that the mislocalisation of parietal lobe seizures, especially in patients with non-lesional focal epilepsies, to either the temporal or frontal lobes may be responsible for some of these statistical differences. Such misidentification leads to more focused, invasive evaluations of temporal and frontal lobe resections, thus contributing to ineffective surgery. This study investigates the reliability of electroclinical features in localising parietal lobe epilepsy (PLE), compared with the ability to recognise frontal (FLE) and temporal (TLE) lobe epilepsies.

Materials and methods

Patient population

From the surgical series of the Cleveland Clinic Epilepsy Center between January 2000 and December 2008, 16 consecutive patients with PLE were identified to have the following criteria: 1) habitual seizures recorded during video-EEG monitoring; 2) available pre-operative and postoperative MRI studies; 3) surgical resections limited to the parietal lobe; 4) Engel class 1 outcomes with a minimum follow-up duration of 12 months, and 5) no previous neurosurgery. In order to match the study samples, 17 consecutive patients with FLE and 17 cases of TLE (based on a standard, non-invasive diagnostic assessment and confirmed by a seizure-free period of at least one year) were also taken from the same series and included in the study. As part of the presurgical evaluation, all patients underwent surface video-EEG monitoring (median: 5 days; IQR: 4-6) and MRI scanning (including volume acquisition T1-weighted, T2-weighted, and fluid-attenuated inversion recovery [FLAIR] sequences).

Additionally, invasive recordings by means of subdural grid electrodes were collected from 19 patients: 59% (10/17) of the FLE cases and (9/16) 56% of the PLE patients. The presence, location, and magnitude of lesions, along with the extent of surgical resection, were analysed on pre-operative and postoperative MRI scans. This retrospective study was approved by the Cleveland Clinic Institutional Review Board.

Interictal and ictal EEG

Interictal and ictal EEG samples were extracted from digitally acquired and archived files. Samples

were printed on paper using the following three types of montage: longitudinal anterior-posterior (A-P) bipolar montage, referential montage (common average reference), and extended A-P bipolar montage incorporating additional anterior temporal scalp (FT9/10) or sphenoidal (Sp1/2) electrodes. Filter settings were set at a high frequency of 70 Hz and a low frequency of 1.6 Hz. When more than one interictal spike/sharp wave population appeared, each example of electrical burst was printed, studied, and mapped separately. A single representative ictal EEG sample was selected for each patient. Interictal and ictal recordings were independently interpreted by two raters (R1 and R2), experienced board-certified epileptologists/neurophysiologists, who were blinded to the epilepsy syndrome, historical data, and site of resection.

Each interictal sample was analysed and classified according to the following criteria: 1) morphology: sharp wave/spike; 2) localisation: (a) regional, (b) multiregional, (c) lateralised, or (d) bilateral (maximum bifrontal); 3) lateralisation: (a) left, (b) right, or (c) not applicable (NA, when generalised or non-localising); 4) site: (a) frontal, (b) temporal, (c) parietal, (d) occipital, (e) vertex, (f) fronto-temporal, or (g) NA (when generalised or non-localising); and 5) distribution of the electric field: spike/sharp wave maximum and distribution up to 90% of drop off (*i.e.* F4 maximum; 90% F8 and T8). Each ictal EEG seizure onset pattern was also analysed and mapped according to the following criteria: 1) localization: (a) regional, (b) multiregional, (c) lateralised, (d) bilateral (maximum bifrontal), or (e) non-localising; 2) lateralisation: (a) left, (b) right, or (c) not applicable (NA); and 3) site: (a) frontal, (b) temporal, (c) parietal, (d) occipital, (e) vertex, (f) frontotemporal, or (g) NA (bilateral or non-localising).

Electroclinical impression (ECI)

Raters independently reviewed clipped videos of representative seizures after analysis of each patient's available EEG samples. If the video did not show the type of aura, raters were provided access to the raw data. ECI characterisation reflected the rater's level of confidence in accurately assessing a lobar origin of the epilepsy. This recognition of the epileptogenic zone was based on the analysis of interictal and/or ictal EEG recordings, along with the evaluation of seizure semiology(ies) displayed on video(s). ECI was limited to one of the following: 1) FLE; 2) TLE; 3) PLE; or 4) unable to specify.

Statistical analysis

We performed empirical analyses on raters' classifications with Statistical Package for the Social Sciences Version 16.0 (SPSS, Chicago, IL, USA). Inter-rater agreement was estimated by intraclass correlation coefficient (ICC) and two-way mixed single measures (Absolute agreement) (ICC 3.1) (MacLennan, 1993). ICC is a more accurate measurement of consistency and reliability among observers, especially when subgroups (*i.e.* different lobar epilepsies) that vary in average measurements are included in the sample (Shrout and Fleiss, 1979). Inter-rater reliability was classified as follows: poor (ICC$<$0.40), moderate (ICC of 0.40 to 0.59), substantial (ICC of 0.60 to 0.79), and outstanding (ICC$\geq$0.80). The 95% CI for ICC was used to assess the reliability of the two independent reviewers' scores. Percentages of composite numbers (data from both reviewers) were calculated when the reliability of the analysed variable was moderate or better. A Student's t test, χ^2 test, Kruskal-Wallis test, and Mann-Whitney test were employed when appropriate. A value of $p<0.05$ was considered statistically significant.

Results

Encompassing a total of 50 patients with PLE, TLE, and FLE (19 paediatric patients; 7 with PLE, 6 with TLE, and 6 with FLE), the following variables were held constant for our study: age (PLE: 36.6±24.2; TLE: 23.4±10.6; FLE: 27.3±15.6 years); gender distribution (PLE: 10 males; TLE: 9 males; FLE: 7 males); absence of the interictal findings (5 FLE, 2 TLE, and 3 PLE); age at epilepsy onset (PLE: 10.8±12.1; TLE: 11.6±9.6; FLE: 7.3±5.2 years); median number of recorded seizures per monitoring (PLE: 4.5 [2-8]; TLE: 5 [1-9]; FLE: 5 [3-8]); presence of a specific aura (PLE: contralateral hand/arm somatosensory aura *n*=3, bilateral hand somatosensory aura *n*=1; TLE: olfactory aura *n*=2, abdominal aura *n*=5, psychic aura *n*=1; and FLE: psychic aura *n*=1, abdominal aura *n*=2, whole body somatosensory aura *n*=2, or chest somatosensory aura *n*=1); presence of non-lesional MRI scans (4 FLE, 1 PLE); and median follow-up in months (PLE: 30.5, IQR: 20.25-46; TLE: 22, IQR: 14-29; FLE: 21, IQR: 14.5-42.5).

Histopathological findings were different across the groups; PLE: cortical dysplasia *n*=1, cavernoma *n*=1, low grade glioma *n*=4, ganglioglioma *n*=1, DNET *n*=2, remote infarct *n*=2, pleomorphic xanthoastrocytoma *n*=1, non-specific gliosis *n*=3, not available *n*=1; TLE: cortical dysplasia *n*=1, hippocampal sclerosis *n*=10, cavernoma *n*=2, ganglioglioma *n*=4; and FLE: cortical dysplasia *n*=12, cavernoma *n*=3, remote infarct *n*=2.

The number of interictal spikes/sharp wave populations varied across the groups. In the PLE group, the majority of patients (10/13) had more than one spike population (three patients had one single spike population, six patients had two distinct spike distributions,

and four patients had three interictal spike populations). In the TLE group, the majority of patients (10/15) exhibited one single spike population (10 patients with one population, two patients with two spike populations, and three patients with three interictal spike populations). In the FLE group, a significant number of patients (9/12) exhibited one single spike population (nine patients had one, two patients had two, and one patient had three interictal populations). The number of interictal spike/sharp wave populations per patient was significantly higher in the PLE group than in the FLE group (p=0.045).

Inter-rater agreement was substantial or outstanding across different segments of the interictal EEG findings (*table 1*). R1 did not consider four samples as unequivocal, interictal epileptiform discharges (all in PLE group).

PLE interictal EEG readings displayed the greatest scatter outside the lobe of origin, in the: ipsilateral parietal region (P electrodes, 16%); ipsilateral antero-temporal region (electrodes Sp/FT, 28%); unilateral or bilateral frontal regions (Fp and F electrodes, 20%); central region (Cz electrodes, 8%); ipsilateral temporal region (T7/8 electrodes, 6%); bioccipital region (O1/O2 electrodes, 4%); and contralateral antero-temporal (12%), temporal (2%), or frontal regions (4%).

The FLE group did not exhibit contralateral interictal epileptic EEG abnormalities. Findings in this group were classified by the following: ipsilateral frontal (38.5%), bifrontal, or generalised/maximum bifrontal regions (38.7%); central region (16.3%); and ipsilateral temporal region (6.5%). TLE interictal EEG recordings showed dominant findings in the temporal lobe, in the: ipsilateral antero-temporal region (43.7%); ipsilateral temporal region (12.5%); ipsilateral or bilateral frontal (20.8%) area; and ipsilateral parietal (12.5%), contralateral antero-temporal (6.3%), or contralateral temporal (4.2%) regions (*figure 1*).

Inter-rater agreement was moderate or substantial across several intervals of the ictal EEG analysis (*table 1*).

The observations of localised or lateralised ictal EEG patterns differed among the three epilepsy groups; both raters were able to localise ictal EEG recordings to the respective epileptogenic region in all TLE cases. Correctly classified ictal EEG patterns were, however, less frequent in FLE (76.5%) and PLE (53.1%) patients (p<0.001). Similarly, localised ictal EEG readings were most frequent in the TLE group (70.6%), followed by the FLE (50%) and PLE groups (37.5%) (p=0.024). In the PLE group, ictal EEG signals were mostly localised within the parietal region (66.7%), and rarely within

Table 1. Inter-rater agreement across different aspects of the interictal and ictal EEG findings.

	Morphology	**Localisation**	**Lateralisation**	**Site**
Interictal EEG Population #1 (n=40)	ICC: 0.63 95%CI: 0.40-0.78 p<0.0001	ICC: 0.83 95%CI: 0.70-0.90 p<0.0001	ICC: 0.96 95%CI: 0.92-0.98 p<0.0001	ICC: 0.74 95%CI: 0.55-0.85 p<0.0001
Interictal EEG Population #2 (n=19)	ICC: 0.62 95%CI: 0.23-0.84 p=0.002	ICC: 1 p<0.0001	ICC: 0.82 95%CI: 0.57-0.93 p<0.0001	ICC: 0.75 95%CI: 0.44-0.90 p<0.0001
Interictal EEG Population #3 (n=8)	ICC: 0.72 95%CI: 0.10-0.94 p=0.02	ICC: 1 p<0.0001	ICC: 1 p<0.0001	ICC: 0.73 95%CI: 0.11-0.94 p<0.0001
Ictal EEG overall (n=50)		ICC: 0.58 95%CI: 0.33-0.74 p<0.0001	ICC: 0.76 95%CI: 0.61-0.85 p<0.0001	ICC: 0.58 95%CI: 0.37-0.74 p<0.0001
Ictal EEG PLE (n=16)		ICC: 0.62 95%CI: 0.18-0.85 p=0.001	ICC: 0.67 95%CI: 0.29-0.87 p=0.001	ICC: 0.71 95%CI: 0.35-0.89 p<0.0001
Ictal EEG TLE (n=17)		ICC: 0.44 95%CI: 0.04-0.76 p=0.036	ICC: 1 p<0.0001	ICC: 0.59 95%CI: 0.16-0.83 p=0.007
Ictal EEG FLE (n=17)		ICC: 0.44 95%CI: 0.00-0.74 p=0.014	ICC: 0.61 95%CI: 0.21-0.83 p=0.02	ICC: 0.48 95%CI: 0.05-0.76 p=0.014

Populations were ordered according to their abundance during the scalp video-EEG monitoring.

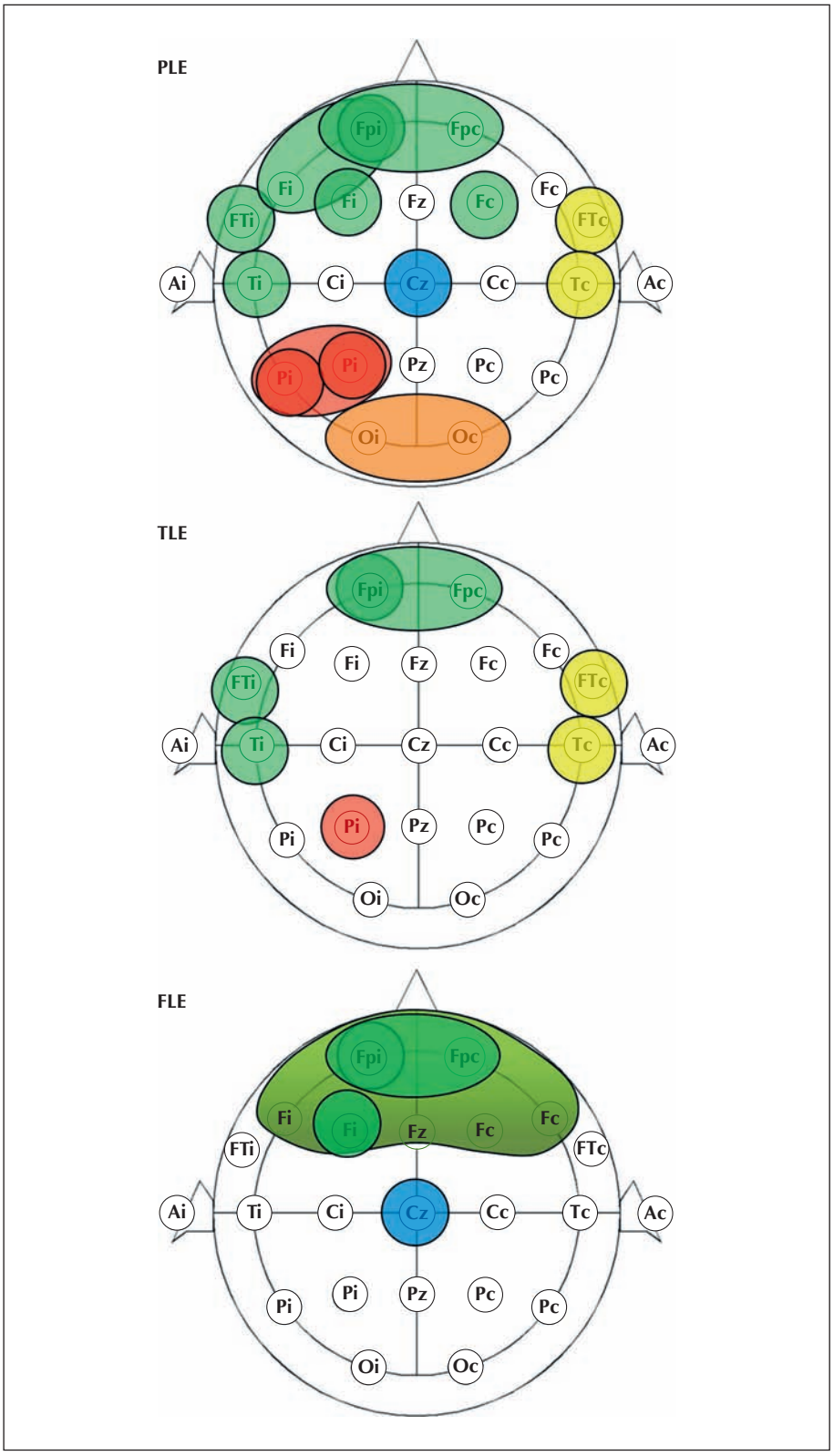

Figure 1. Maximum electric field interictal EEG distribution among the groups.
Green: frontal or ipsilateral temporal lobe; green gradient: generalized discharges; yellow: contralateral temporal; red: parietal ipsilateral; orange: occipital; blue: vertex.

the temporal (16.7%), fronto-temporal (8.4%), or vertex regions (8.4%). In the FLE group, ictal EEG recordings were limited to the frontal (70.6%) and vertex regions (17.6%), but rarely within temporal (5.4%) and fronto-temporal regions (5.4%). In the TLE group, ictal EEG waves were localised to temporal (83.3%), parietal (4.2%), and fronto-temporal regions (12.5%).

ECI agreement for all epilepsy types was poor (ICC: 0.39; 95%CI: 0.14-0.60; p=0.02). Raters specified the correct lobe in the majority of TLE (R1: 82%, R2: 76%) and FLE (R1 47%, R2 82%) cases, but least frequently in PLE cases (R1: 50%, R2: 62%). Whenever raters confidently categorised their ECI as one lobar subtype, overall accuracy was excellent (R1 80%, R2 89%) (ICC: 0.53; 95%CI: 0.20-0.75; p=0.001). However, PLE was significantly misidentified by R1 and R2 in 50% and 40% of cases, respectively; corrected/specified cases for R1 were: FLE 8/8, TLE 12/14, PLE 4/8 (p=0.034), and R2: FLE 14/14, TLE 13/13, PLE 6/10 (p=0.002). Similarly, TLE (correctly specified/rest; R1: 14/17; R2 13/17) was more often accurately specified compared to PLE (R1: 4/16; R2: 6/16), (p=0.032; p=0.013).

According to the ECIs of both raters, two patients in the PLE group (Patient 32 with cavernous angioma and resection in the right precuneus and Patient 40 with remote ischaemic lesion and resection in the left angular gyrus) were incorrectly classified. On the contrary, four patients were correctly identified as PLE cases by both raters (Patient 9 with DNET and resection in the right superior parietal lobule, Patient 13 with remote infarct and resection in right precuneus, Patient 17 with ganglioglioma and resection in the left superior parietal lobule, and Patient 30 with pleomorhic xanthoastrocytoma and resection in the right superior parietal lobule) (*figure 2*).

Discussion

In this study, we independently compared the EEG recording trends of three groups of patients, all of whom had well-defined focal epilepsies as evidenced by seizure freedom following focal lobar or sublobar resections. Our results show significant differences in the electrical scalp EEG features of PLE cases, compared to the two most frequent focal epilepsy subtypes encountered in surgical epilepsy centres.

Both interictal and ictal EEG findings are more variable in their anatomical distribution and/or less localising in PLE patients as compared to FLE and TLE cases. These findings underscore the possibility of mislocalisation of the epileptic focus to other lobes (*e.g.* temporal or frontal) in patients with PLE. This misidentification can be particularly problematic in patients with non-lesional, parietal lobe epilepsy, where seizure onset may be erroneously localised to other lobes (mainly frontal or temporal). The rather elaborate connectivity of the parietal lobe to various distant regions of the brain presents the most probable reason for extraparietal localisation of the ictal and interictal EEG patterns in PLE patients. The spread and distribution of EEG activity have been shown to follow certain cortical-subcortical pathways. For instance, deep sources of electrical activity in the temporal lobe tend to propagate to the ipsilateral neocortex (Alarcon *et al*., 1994). This same mechanism is most likely characteristic of PLE.

In accordance with a previous study reported from our centre (Foldvary *et al*., 2001), localising ictal EEG patterns were observed in approximately 40% of all PLE patients. This percentage differs from other published reports (11%) (Williamson *et al*., 1992; Cascino *et al*., 1993). This discrepancy is likely to be due to the possibility that the patients with assumed PLE in the latter series may not have had true PLE, in contrast to the confirmed localisation of PLE in our patients, since we included only patients who achieved complete seizure control following focal parietal resection. Nevertheless, all published series show that the frequency of non-localising EEG recordings is greatest in PLE patients (compared to FLE and TLE cases), likely reflecting the richly entangled connectivity of the parietal lobe.

Our study shows that interictal EEG findings are most widely distributed in PLE cases, regardless of patient age. Recent functional imaging studies suggest a central role of the parietal lobe as an anatomical intermediary, integrating visuo-spatial imagery, episodic memory retrieval, and self-processing operations (Cavanna and Trimble, 2006). Integrative functions imply the elaborate connectivity of mesial, dorsolateral, and ventrolateral areas of the parietal lobe to other brain regions (Rushworth *et al*., 2006). Furthermore, several intricate, bilateral interconnections exist between the two parietal lobes, providing the physiological basis for functional coupling (He *et al*., 2007). These anatomical connections may account for the diffuse, interictal EEG distribution observed in PLE cases. Similarly to our results, exclusively temporal spikes or broad hemispheric spikes with temporal preponderance were described in non-lesional intractable epilepsy patients with parietal lobe symptomatology (Aghakhani *et al*., 2004).

Synchronous or at least temporal overlapping activation of 10-20 cm^2 of gyral cortex is sufficient to produce scalp EEG spikes in the temporal lobe (Tao *et al*., 2005). We have observed a relative absence of genuine parietal spiking in our data, in addition to the complete non-existence of contralateral parietal scalp EEG spiking. These findings can be attributed to the insufficiency of parietal cortex synchronicity in generating scalp detectable spikes prior to the

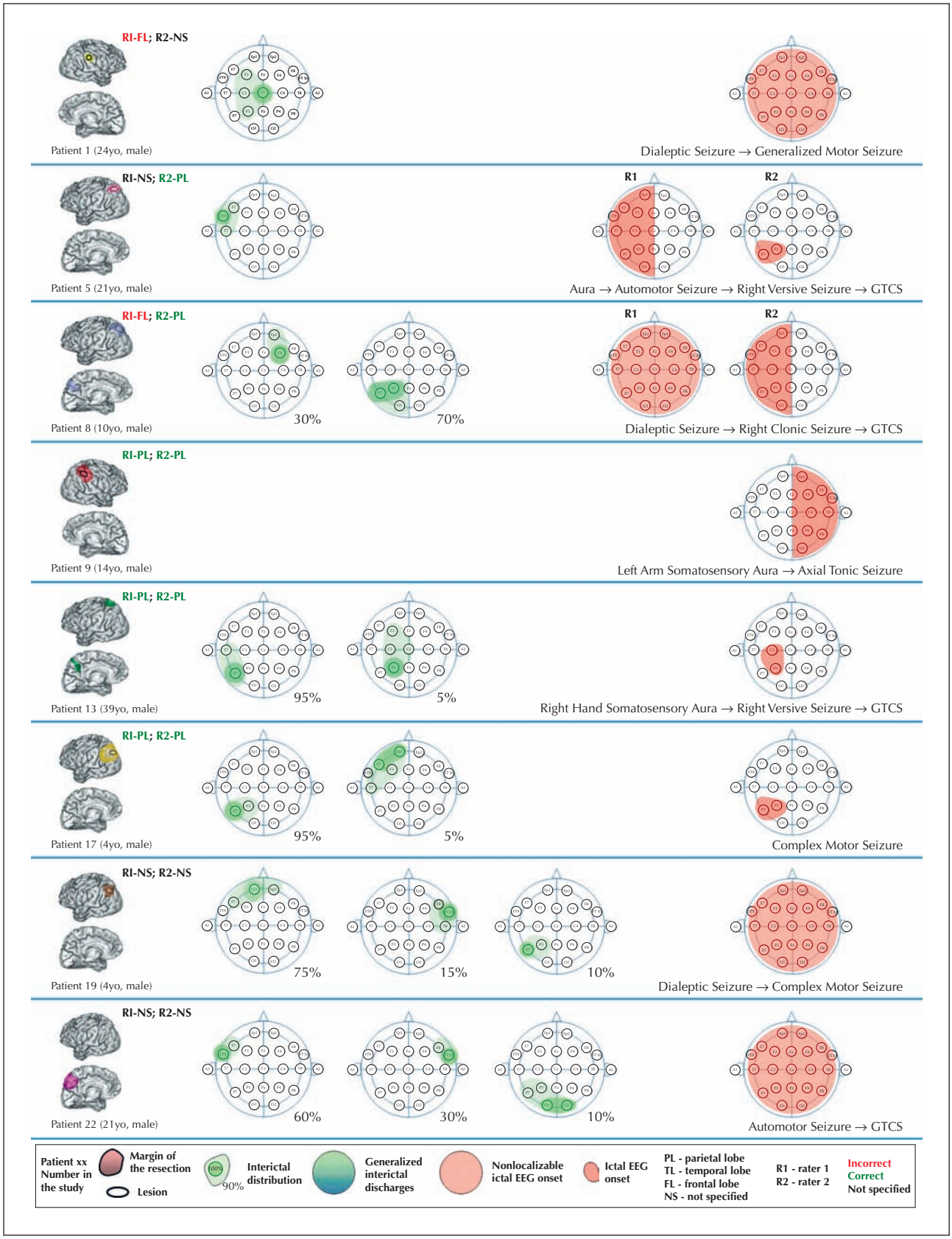

Figure 2. Interictal and ictal EEG findings, semiology, and ECI of PLE patients.

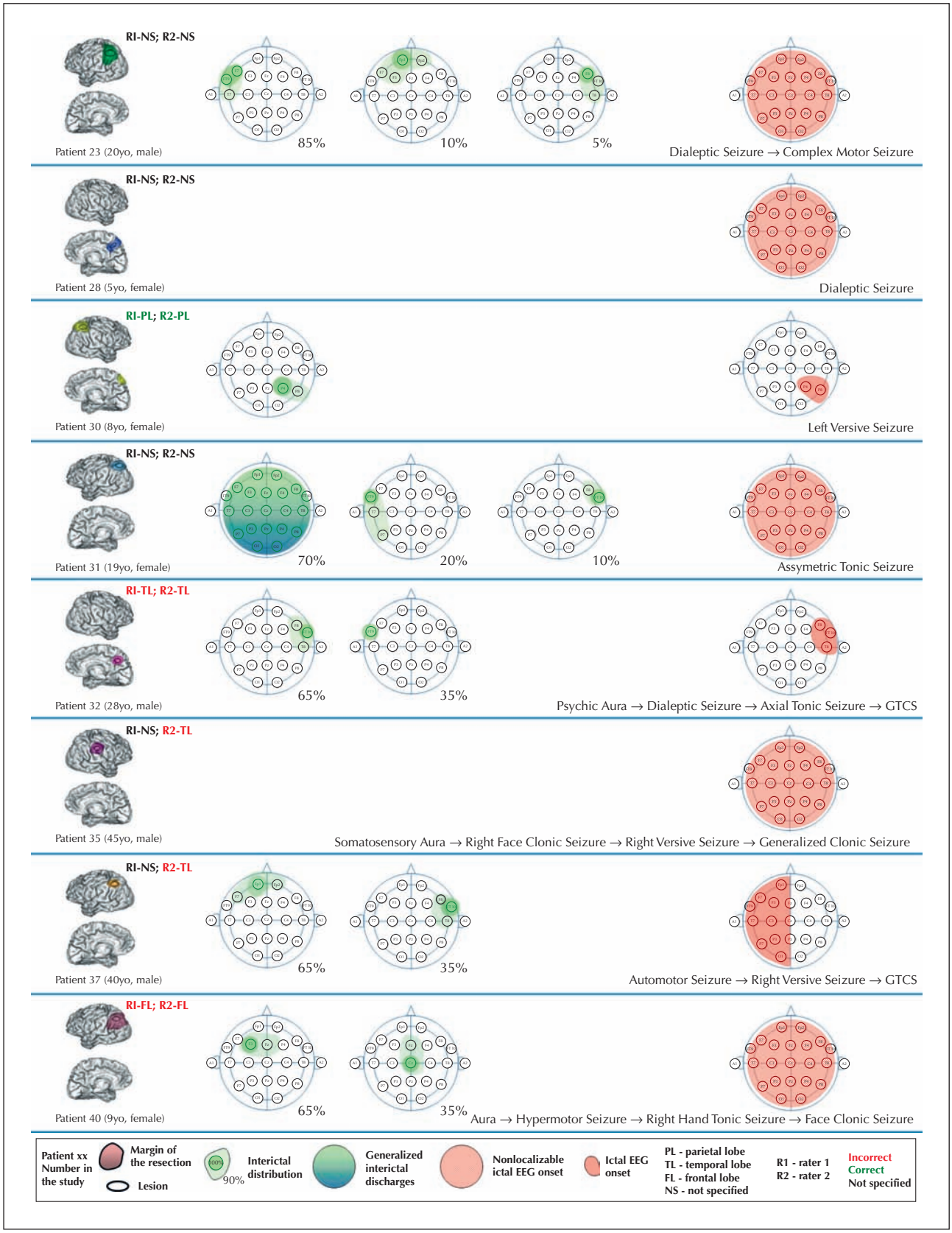

Figure 2. (*Continued.*)

propagation of epileptic activity into anatomically bounded regions. The absence of biparietal spiking in scalp EEG recordings, when functionally-proven biparietal connections (Grefkes and Fink, 2005; Santens *et al*., 2010) are particularly germane, is surprising. In our experience, contralateral spiking from homologous parietal regions is commonly observed in intracranial EEG investigations, specifically when sampling is taken from both parietal lobes in which anatomical connectivity is structurally intact. Significant selection bias in our study (decision making with regards to surgical treatment eligibility) may have contributed to some of our findings. For instance, the presurgical observation of biparietal spiking on scalp EEG readings may be perceived as an admonition against surgical treatment consideration. Therefore, the analysis of network connectivity using intracranial (preferably stereo-EEG due to a precise anatomical placement) depth electrodes would be a more precise method in addressing these issues. The retrospective nature of this study and the unavoidable selection/referral bias are both critical limitations of our investigation. Although our findings cannot be generalised to all patients with PLE, the conditions of our study do closely simulate the actual routine clinical practices of the epilepsy monitoring unit and the presurgical non-invasive evaluation process.

Both raters consistently misidentified PLE in a significant number of cases. This may be due to the relatively small number of PLE patients reported by already preoccupied epilepsy centres (Comair *et al*., 2008). In fact, very few reports of non-lesional PLE cases are found in the literature. As reported in previous studies, somatosensory or vestibulary sensations are the most prevalent subjective complaints in PLE patients (Bartolomei *et al*, 2011; Boesebeck *et al*., 2002). Concerning seizure onset localisation, these symptoms are clinically most valuable when preceding motor features that are more frequently associated with extraparietal cortices (especially premotor regions and temporal lobe). Concordantly, all PLE cases that were misidentified by both raters exhibited no electrical or clinical indications of parietal lobe involvement. We hypothesize that the expression of electroencephalographic and semiological features of temporal or frontal lobe involvement in patients with PLE is most likely responsible for the misidentification of many PLE patients. Conversely, the presence of interictal EEG findings, along with ictal EEG patterns localised to the parietal lobe or typical parietal lobe auras, alone, may facilitate the identification of patients with PLE (especially if guided by the presence of parietal lobe lesions on MRI).

Although seizure semiology may substantially differ between different age groups (Fogarasi *et al*., 2007), equal distribution of the lobar epilepsy subtypes among the paediatric population makes the bias in our study unlikely.

The parietal lobe is at the centre of multisensory integration. The dorsal fronto-parietal network (superior parietal lobule to frontal eye field) links incoming sensory information to corresponding motor responses, while the ventral fronto-parietal network (temporo-parietal junction to ventral frontal cortex) plays a significant role in detecting novel/unexpected stimuli. Damage to the latter neuronal network has been highly suspected in patients showing unilateral spatial neglect (Corbetta and Shulman, 2002; He *et al*., 2007). The highly interconnected nature of the parietal region (Borra *et al*., 2008) likely accounts for the mislocalisation of "referred" electroclinical patterns. Indeed, a preferential spread of ictal activity from the superior parietal lobule to the frontal lobe (including the supplementary sensorimotor area and the premotor cortex) and an electrical propagation from the inferior parietal lobule to temporo-limbic areas have been observed (Salanova *et al*., 1995), thus emphasizing differences in underlying functional connectivity.

In conclusion, PLE scalp EEG readings show an increased scatter of interictal discharges and non-localising ictal EEG patterns. These characteristics may account for mislocalisation of non-lesional PLE to other lobes in some patients. These results suggest the requirement of a more comprehensive invasive EEG evaluation (that includes sampling of the ipsilateral parietal lobe) for some patients with EEG and/or clinical features of frontal (and to a lesser extent temporal) lobe epilepsy, particularly in the context of normal ("non-lesional"), high resolution MRI studies. □

Disclosures.

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We confirm that we have read the Journal's position on issues concerning ethical publications and affirm that our report is consistent with these guidelines.

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