Session 4B: Parietal lobe epilepsy

Non-Invasive and Invasive Investigations of Parietal Lobe Epilepsy.

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To Professor Philippe Kahane and Doctor Lorella Minotti for accepting to share their experience from University Grenoble-Alpes.
Introduction

Semiology of PLE is heterogeneous and mimic seizures originating from extra-parietal cortex.

Parietal lobe is subdivided in distinct ”epileptic regions”:
- A case of precuneal epilepsy
- A case of parietal cingulate gyrus epilepsy
- A case of inferior parietal lobule epilepsy.

Scalp EEG is also misleading

Diagnostic accuracy of non-invasive modalities in presurgical evaluation in PLE.

Conclusion and comments.
objectives

Emphasize the wide difference in clinical and scalp EEG manifestations in PLE.

Review the diagnostic accuracy and sensitivity of non-invasive localisation modalities in PLE.

Review the value of invasive EEG, cases-based discussion.
**Representation of regions of the parietal lobe generating seizures** (adapted from Bartolomei 2011).

- **Group 1 (n=7)**. Brodmann Area 7 Superior parietal lobule and precuneus.
- **Group 2 (n=2)**. BD area 5 Superior parietal lobule.
- **Group 3 (n=4)**. BD area 39,40 Inferior parietal lobule (supramarginal and angular gyri).
- **Group 4 (n=4)**. BD area 40,43 Parietal operculum.

**PCC**
Posterior cingulate cortex.
The parietal cortex is at the centre of multisensory integration. It is also highly interconnected to other cortical regions. This explains the heterogeneity of PL seizures semiology.

- **Visual**
  1. elementary visual hallucinations / amaurosis
  2. visual illusions/distortion
  3. complex visual hallucinations

- **Somatosensory**
  4. elementary somatosensory
  5. pain
  6. sensations of warmth/cold
  7. somatognostic illusions

- **Vestibular**

The parietal lobe and associative cortex is highly connected to other cortical regions:

**Dorsal fronto-parietal network**
- SPL → frontal eye field

**Ventral fronto-parietal network**
- TP junction → ventral frontal cortex
Introduction
Semiology of PLE is heterogeneous and mimic seizures originating from extra-parietal cortex.

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Scalp EEG is also misleading

Diagnostic accuracy of non-invasive modalities in presurgical evaluation in PLE.

Conclusion and comments.
Scenarios of intracerebral SEEG schemes in PLE.
Adapted from Bartolomei et al., Epilepsy Res 2011.

gr 1. SPL. BA 7.
gr 3. IPL. BA 39,40.
gr 2. SPL. BA 5.
gr 4. parietal operculum. BA 40.
Case 1. Precuneal seizures.

A case-based discussion

21 yo R-handed woman with seizures since age 12:


Seizures started at 12. Typically, daytime, monthly, seizures with prominent motor features and occasional GTC szs.

Low average/borderline intelligence.

Normal examination and phenotype.

Refractory to several AEDs.
Case 1 presentation con’t

Seizure semiology

Aura is present and with vestibular flavor i.e. sudden impression of unsteadiness or of a movement, not further described, but often attempted to prevent a fall → L UL increased tone and LOC → clonic jerks L arm and inconsistent bilateral eyes blinking. May fall and occ. 2ary generalization.

Slow recuperation and fatigue. Amnestic or partly amnestic, but no other apparent deficits.

No triggers. No somatosensory, visual or temporal-like features.

**Key point**: vestibular aura and early lateralized motor features
21 yo R-handed woman

Anatomical MRI : normal
Interictal FDG-PET : normal
MEG : no
Ictal SPECT : no
EEG-fMRI : no

Summary of neuropsychological evaluation :
R-handed and left hemispheric speech dominance with a low average IQ.

Neuropsychology profile consistent with bilateral posterior quadrant dysfunction. Mild impairment of attention, memory and executive function deficits and mild visuo-spatial impairments.
Case

History

Seizures

EEG

Cognition

Imaging

Video-EEG

Hypothesis

SEEG


Conclusion

21 yo R-handed woman

Interictal scalp EEG findings (10-20 and 10-10):
- bilateral occipital sharp activity (O1, O2)
- R centro parietal (C4, P4)
- R T (F8, T4, T6)

Ictal scalp EEG findings:
- R CP (C4, P4)

Key point: EEG not localizing but pointing toward posterior quadrant generator, probably right.
Case 1 presentation cont

1st admission

typical seizures were recorded with vestibular aura and ictal motor manifestations involving predominantly the L UL.

interictal and ictal scalp discharges suggestive of a generator in to the right posterior quadrant.

neuropsychology profile consistent with bilateral posterior quadrant dysfunction.

MRI and PET revealed no anatomical lesion or hypometabolism.

What’s next?
Lesion resected
FCD type 2B
F/U > 2 yrs
sz-free
Precuneal epilepsy

clinical-EEG correlations although often inaccurate, point to the posterior quadrant in most cases (n=6). Structural imaging has a better yield compared to functional imaging (MRI (4/6), SPECT (1/4), PET (1/3) and MEG (2/3)).

Harroud et al., Epilepsy & Behavior 2017

<table>
<thead>
<tr>
<th>Pt</th>
<th>AO</th>
<th>MRI</th>
<th>Ss</th>
<th>Vest.</th>
<th>visual</th>
<th>E+H</th>
<th>motor</th>
<th>others</th>
<th>II EEG</th>
<th>I EEG</th>
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<tr>
<td>f, 17</td>
<td>10</td>
<td>L post. PreCu</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>lat, floc</td>
<td>diffuse</td>
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<td>F, 21</td>
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<td>n</td>
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<td>+</td>
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<td>+</td>
<td>-</td>
<td>loc, bil</td>
<td>bil</td>
</tr>
<tr>
<td>M, 50</td>
<td>16</td>
<td>n</td>
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<td>+</td>
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<td>loc, floc</td>
<td>loc</td>
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<tr>
<td>M, 23</td>
<td>13</td>
<td>R post PreCu</td>
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<td>-</td>
<td>+</td>
<td>+</td>
<td>lat, floc</td>
<td>lat</td>
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<tr>
<td>M, 59</td>
<td>12</td>
<td>L ant PreCu</td>
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<td>+</td>
<td>+</td>
<td>none</td>
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<td>F, 21</td>
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<td>R ant PreCu</td>
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<td>+</td>
<td>-</td>
<td>loc, bil, floc</td>
<td>loc</td>
</tr>
</tbody>
</table>

4/6 0 5 3 2 5 4
Case 2. Posterior cingulate seizures.

A case-based discussion

22 yo L-handed woman with seizures since age 14:

Uneventful obstetrical history. No antecedents.

Seizures started at 14. Typically, daytime monthly seizures with prominent temporal features.

Low average/borderline intelligence.

Normal examination except discreet R facial paresis.

Refractory to several AEDs.
Case 2 presentation con’t

Seizure semiology

Aura is present, epigastric and +/- gustatory) → automotor behavior with loss of contact and confusion.

Slow recuperation, post-ictal confusion and fatigue. Amnestic or partly amnestic, but no other apparent deficits.

No triggers. No somatosensory or visual features.

**Key point**: temporal-like seizures.
22 yo L-handed woman

**Anatomical MRI** : L thalamus hypersignal.

**Interictal FDG-PET** : L hemispheric hypometabolism, max temporo-parietal.

**MEG** : no

**Ictal SPECT** : no

**EEG-fMRI** : no

**Summary of neuropsychological evaluation** :
L-handed and left hemispheric speech dominance with verbal IQ = 99 and non verbal IQ = 76.
22 yo L-handed woman

- F7-Fb1
- Fb1-Tp1
- Tp1-T5
- FP1-F7
- F7-T3
- T3-T5
- T5-O1
- FP1-F3
- F3-C3
- C3-P3
- P3-O1
- Fz-Cz
- Cz-Pz
- F8-Fb2
- Fb2-Tp2
- Tp2-T4
- FP2-F8
- F8-T4
- T4-T6
- T6-O2
- FP2-F4
- F4-C4
- C4-P4
- P4-O2

T3-T5, F7-T3
**Key point**: ictal > interictal discharges not typical for mesial TLE.
Symptomatogenic zone

EZ

differential for other possible EZ.
Semiology of PCE varies depending upon the seizure spread patterns.

7 patients (Enatsu et al., JNNP 2014)

3/7 with motor manifestations (spread to frontal - premotor area, OF, SMA, ACC - and to parietal lobe - precuneus, PCC, IPL, SS).

4/7 with dialeptic seizures or automotor seizures (spread to medial temporal or IPL areas).
Case 3. Inferior parietal lobule seizures.

A case-based discussion

13 yo man with seizures since age 8:

Focal motor seizure involving R side at 2 days of age.

Seizures started at 8. Typically, daytime with R side somatosensory and prominent motor features.

Refractory to several AEDs.
Case 3 presentation con’t

Seizure semiology

Aura is present, cephalic and somatosensory manifestations with paresthesia involving R arm and head. → R arm dystonia or tonic posture, unresponsive although appears alert. Seizures often triggered during meals. Slow recuperation and fatigue, but no post-ictal motor deficits.

No triggers except maybe eating. No pain, visual, gustatory or temporal-like features.

Key point: lateralized somatosensory aura followed by prominent focal motor manifestations.
13 yo male

**Anatomical MRI**: L posterior perisylvian and post-central mild atrophy.

**Interictal FDG-PET**: no

**MEG**: no

**Ictal SPECT**: no

**EEG-fMRI**: no

**Scalp EEG**: lateralized to the left hemisphere but did not localized.

**Hypothesis and SEEG planification:**

Generator in involving SS, opercular region and possibly IPL.
Somatosensory cortex

Inferior parietal lobule

Motor and premotor cortex

FCP operculum
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A case of inferior parietal lobule epilepsy.

Scalp EEG is also misleading

Diagnostic accuracy of non-invasive modalities in presurgical evaluation in PLE.

Conclusion and comments.
**EEG correctly localizes the epileptic generator in a small proportion of patients with PLE.**

<table>
<thead>
<tr>
<th>Occipital epilepsy (n=25)*</th>
<th>Parietal epilepsy (n=11)**</th>
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</thead>
<tbody>
<tr>
<td><strong>Interictal EEG</strong></td>
<td></td>
</tr>
<tr>
<td>occipital only</td>
<td>2</td>
</tr>
<tr>
<td>occipital ‘plus’</td>
<td>4</td>
</tr>
<tr>
<td>temporal only</td>
<td>14</td>
</tr>
<tr>
<td>frontal bil. synch.</td>
<td>5</td>
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<tr>
<td><strong>Interictal EEG</strong></td>
<td></td>
</tr>
<tr>
<td>parietal only</td>
<td>1</td>
</tr>
<tr>
<td>parietal ‘plus’</td>
<td>3</td>
</tr>
<tr>
<td>temporal only</td>
<td>3</td>
</tr>
<tr>
<td>frontal bil. synch.</td>
<td>2</td>
</tr>
<tr>
<td>no spikes</td>
<td>2</td>
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</table>

*, ** Williamson et al., *Ann Neurol* 1992

EEG can mimic temporal patterns in patients with PLE.

<table>
<thead>
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<th>Frontal Pattern</th>
<th>Occipital epilepsy (n=25)*</th>
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<tr>
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<td>occipital only 2</td>
<td>parietal only 1</td>
</tr>
<tr>
<td></td>
<td>occipital ‘plus’ 4</td>
<td>parietal ‘plus’ 3</td>
</tr>
<tr>
<td></td>
<td>temporal only 14</td>
<td>temporal only 3</td>
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<td></td>
<td>frontal bil. synch. 5</td>
<td>frontal bil. synch. 2</td>
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<tr>
<td></td>
<td>Ictal clinical</td>
<td>No spikes 2</td>
</tr>
<tr>
<td></td>
<td>TL type 11</td>
<td>temporal lobe type 4</td>
</tr>
<tr>
<td></td>
<td>FL type 3</td>
<td>frontal lobe type 4</td>
</tr>
<tr>
<td></td>
<td>≥ 2 seizure types 11</td>
<td>≥ 2 seizure types 3</td>
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**EEG can mimic frontal patterns in patients with PLE.**

### Frontal Pattern

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<tr>
<td><strong>Ictal clinical</strong></td>
</tr>
<tr>
<td>TL type</td>
</tr>
<tr>
<td>FL type</td>
</tr>
<tr>
<td>≥ 2 seizure types</td>
</tr>
</tbody>
</table>

### Parietal epilepsy (n=11)**

<table>
<thead>
<tr>
<th><strong>Interictal EEG</strong></th>
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<tr>
<td>parietal only</td>
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<tr>
<td>parietal ‘plus’</td>
</tr>
<tr>
<td>temporal only</td>
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<tr>
<td>frontal bil. synch.</td>
</tr>
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<td>no spikes</td>
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</tbody>
</table>

<table>
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<tr>
<th><strong>Ictal clinical</strong></th>
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<tbody>
<tr>
<td>temporal lobe type</td>
</tr>
<tr>
<td>frontal lobe type</td>
</tr>
<tr>
<td>≥ 2 seizure types</td>
</tr>
</tbody>
</table>

**Interictal more than ictal activity mis-localizes or mis-lateralizes epileptic discharges in post Q epilepsy.**

<table>
<thead>
<tr>
<th>62 operated children (mean age 7.9 yrs)</th>
<th>Pediatric epilepsy surgery in the posterior cortex.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Interictal EEG</strong></td>
<td><strong>62</strong></td>
</tr>
<tr>
<td>localising</td>
<td>22 (35%)</td>
</tr>
<tr>
<td>regional</td>
<td>25 (40%)</td>
</tr>
<tr>
<td>falsely localising</td>
<td>6 (2 F, 3 FT, 1 T) (9.7%)</td>
</tr>
<tr>
<td>lateralising only</td>
<td>3 (4.8%)</td>
</tr>
<tr>
<td>falsely lateralising</td>
<td>6 (9.7%)</td>
</tr>
<tr>
<td><strong>Ictal EEG</strong></td>
<td><strong>56</strong></td>
</tr>
<tr>
<td>localising</td>
<td>21 (37%)</td>
</tr>
<tr>
<td>regional</td>
<td>18 (32%)</td>
</tr>
<tr>
<td>falsely localising</td>
<td>7 (F) (12.5%)</td>
</tr>
<tr>
<td>lateralising only</td>
<td>1 (1.8%)</td>
</tr>
<tr>
<td>falsely lateralising</td>
<td>3 (5.35%)</td>
</tr>
<tr>
<td>bil hemispheric</td>
<td>6 (10.7%)</td>
</tr>
</tbody>
</table>

Liava et al., *Epileptic Dlsord* 2014
Maximum electrical field interictal EEG distribution in PLE vs TLE and FLE

Ristic et al., Epileptic Disord 2012

PLE patients

• show a more variable scatter of interictal discharges

• the majority have more than one spike population

• they also show a lower localisation value of ictal recordings.

➢ lobar classification by electroclinical impression is least accurate in PLE patients.
Diagnostic accuracy of pre-surgical evaluation.  
(n = 26 patients operated and > 1 yr f/u)

<table>
<thead>
<tr>
<th>Diagnostic modality</th>
<th>No.</th>
<th>Seizure-free</th>
<th>Persistent seizure</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Focal lesion on MRI</td>
<td>26</td>
<td>64%</td>
<td>25%</td>
<td>0.06</td>
</tr>
<tr>
<td>Hypometabolism on PET</td>
<td>26</td>
<td>50%</td>
<td>17%</td>
<td>0.11</td>
</tr>
<tr>
<td>Focal hyperperfusion on SPECT</td>
<td>21</td>
<td>45%</td>
<td>50%</td>
<td>1.00</td>
</tr>
<tr>
<td>Localized ictal EEG</td>
<td>26</td>
<td>36%</td>
<td>42%</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Kim et al., *Epilepsia* 2004

Only structural imaging appears to have a predictive value in terms of seizure outcome. A good understanding and interpretation of the semiological features and concordance of different diagnostic modalities is also associated with higher seizure-free rate.
Diagnostic sensitivity of individual modalities in 22 (85%) operated patients with a favorable (14 seizure-free) surgical outcome.

<table>
<thead>
<tr>
<th>Diagnostic modality</th>
<th>No.</th>
<th>localizing</th>
<th>lateralizing</th>
<th>nonlateral.</th>
<th>false loc.</th>
<th>false lat.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interictal EEG</td>
<td>22</td>
<td>3 (14)</td>
<td>6 (27)</td>
<td>8 (36)</td>
<td>5 (23)</td>
<td>0</td>
</tr>
<tr>
<td>Ictal EEG</td>
<td>22</td>
<td>8 (36)</td>
<td>5 (23)</td>
<td>1 (4,5)</td>
<td>8 (36)</td>
<td>0</td>
</tr>
<tr>
<td>PET</td>
<td>22</td>
<td>8 (36)</td>
<td>5 (23)</td>
<td>4 (18)</td>
<td>4 (18)</td>
<td>1 (4,5)</td>
</tr>
<tr>
<td>SPECT</td>
<td>18</td>
<td>9 (50)</td>
<td>0</td>
<td>2 (11)</td>
<td>7 (39)</td>
<td>0</td>
</tr>
</tbody>
</table>

No. in parenthesis are %.
Conclusions

The characterization of adequate diagnostic criteria is difficult in parietal lobe epilepsy:

• Parietal seizures are rare compared to those observed in temporal or frontal lobe epilepsy.
• The anatomical boundaries of the parietal lobe are not well defined and not clearly related to function.
• The cortex, mostly associative, is concerned with higher sensory, perceptual and cognitive processes or functions. It is highly interconnected with other brain regions and multiple systems.
• Seizures are highly variable with inconsistent semiologic features, often mimicking extra-parietal seizures.
• The yield of the different diagnostic modalities is low, particularly in MR-negative cases. Scalp EEG for instance is often poorly localizing and even often mis-localizing.
Comments:
How can we improve diagnostic yield of parietal seizures and epilepsy?

Subjective phenomena and elementary aura are frequent, often complex and difficult to characterize, and must be well documented.

Parietal lobe seizures propagate typically to frontal lobe and motor regions, through a dorsal frontoparietal stream. They may also spread through ventral pathways toward the ventral frontal regions or to the temporal lobe. The parietal lobe has also intimate interconnectivity in the posterior insula.

Studies of the surface epileptic activity with non-invasive modalities such as EEG-fMRI are scarce. Understanding the hemodynamic responses of interictal epileptic activity may increase the localisation value of scalp EEG.
Schematic cortical representation of the main subjective manifestations (aura) in parietal lobe seizures.

They are frequent and must be well documented.

Adapted from Bartolomei et al., *Epilepsy Res* 2011.
Objective signs (motor) in parietal lobe seizures reflect the involvement of systems participating in oculomotor controls and in the involvement of distant cortices.

Adapted from Bartolomei et al., Epilepsy Res 2011.
Functional organization of the dominant and non-dominant parietal cortices using neurostimulation.

172 patients using high- (50 Hz) and low- (1 Hz) electrical stimulations

<table>
<thead>
<tr>
<th></th>
<th>Postcentral gyrus, posterior part of paracentral lobule and parietal operculum, significant association with:</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>• Somatosensory sensations</td>
</tr>
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<td></td>
<td>• Motor symptoms</td>
</tr>
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<td>• Dysarthria.</td>
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</table>

2. Superior and inferior parietal lobules associated multiple types of responses including:  
   • Somatosensory  
   • Visual illusions and body scheme alterations  
   • Motor symptoms  
   • Eye movements /sensations  
   • Multimodal.

3. Precuneus associated with visual illusions or hallucinations, and with vertigo.

4. Intraparietal sulcus associated with visual illusions and hallucinations and eye movements/sensations.

5. Posterior cingulate gyrus associated with somatosensory and motor symptoms, with vertigo and with neurovegetative manifestations.

Balestrini et al., *Brain* 2015
Patients with focal epilepsy and negative BOLD often have PQE with diffuse bilateral spike-and-wave \cite{Pittau2013}.