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SEEG: Interpreting the Noninvasive Data

1. Is the patient a surgical candidate?
2. Does the patient need an invasive evaluation?
3. What type of invasive evaluation?
4. What is the hypothesis?

Indication for Epilepsy Surgery

• Confirmed diagnosis of epilepsy
• Medical intractability
• Disabling seizures
• Resectable focus (except callosotomy candidates, vagus nerve stimulation, and deep brain stimulation).
• Motivated patient
• No progressive underlying cause (except Rasmussen’s encephalitis).
• High probability that better seizure control will improve quality of life.

Concept of Epilepsy Surgery

<table>
<thead>
<tr>
<th>Description of cortical zones</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epileptogenic Zone</td>
</tr>
<tr>
<td>Invasive Zone</td>
</tr>
<tr>
<td>Epileptogenic Zone</td>
</tr>
<tr>
<td>Epileptogenic Zone</td>
</tr>
<tr>
<td>Functional Deficit Zone</td>
</tr>
<tr>
<td>Symptomatogenic Zone</td>
</tr>
<tr>
<td>Eloquent Cortex</td>
</tr>
</tbody>
</table>

Evaluation for Epilepsy Surgery

<table>
<thead>
<tr>
<th>Lesion</th>
<th>MRI</th>
<th>FDG PET</th>
<th>NPS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Invasive Zone</td>
<td>EEG</td>
<td>MEG</td>
<td>Stereo-EEG</td>
</tr>
<tr>
<td>Epileptogenic Zone</td>
<td>Scalp Video EEG</td>
<td>Ictal SPECT</td>
<td>Extraoperative ECoG</td>
</tr>
</tbody>
</table>
Epilepsy Surgery Evaluation Phase I-III

<table>
<thead>
<tr>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>History, general medical and neurological examination</td>
<td>Intracranial electrode placement</td>
<td>Cortical resection (with and without ECOG)</td>
</tr>
<tr>
<td>EMU monitoring</td>
<td>EMU monitoring</td>
<td>Functional Mapping</td>
</tr>
<tr>
<td>Neuropsychological assessment</td>
<td>Functional Mapping</td>
<td>Functional Mapping</td>
</tr>
<tr>
<td>MRI imaging</td>
<td>MRI imaging</td>
<td>MRI imaging</td>
</tr>
<tr>
<td>Functional Imaging (PET, SPECT)</td>
<td>Electrical and Magnetic Source Imaging (fMRI, TMS)</td>
<td>Electrical and Magnetic Source Imaging (fMRI, TMS)</td>
</tr>
<tr>
<td>Functional Imaging (fMRI, Wada testing)</td>
<td>Functional Mapping (Functional MRI, Wada testing)</td>
<td>Functional Mapping (Functional MRI, Wada testing)</td>
</tr>
</tbody>
</table>

Indication for Invasive EEG Monitoring

A. MR – Lesional (70-80% of invasive cases)
- Discordant findings
- Lesions with poorly defined borders (e.g. FCD)
- Proximity to eloquent cortex
- Dual pathology; multiple lesions
- Surgical failure

70% of epilepsy surgeries do not require invasive EEG

Lesional Case

31 yo old right handed male
Onset: age 25 yrs

EPILEPSY CLASSIFICATION: Left temporal lobe epilepsy
SEIZURE CLASSIFICATION: Psychic aura > Automotor Seizure (daily)

EEG CLASSIFICATION:
- Interictal: Continuous slow, regional, left temporal
- Sharp Waves, regional, left anterior temporal
- Spike, regional, left posterior temporal
- Ictal: Clinical: Psychic Aura > Automotor Seizure

EEG: EEG seizure, regional, left temporal

ETIOLOGY: Left temporal cystic lesion/ presumed glioneuronal tumor

ASSOCIATED CONDITIONS: MRI – unable to lateralize language due to braces
Wada: Right hemisphere dominant for language; bilateral memory representation;
NPS – high functioning without memory deficits.

Left anterior temporal cystic tumor

Intraoperative ECOG
**Repetitive spikes and polyspikes**

**Postresection ECOG**

**Indication for Invasive EEG Monitoring**

**When good is good enough**

1/22/2021

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Indication for Invasive EEG Monitoring

B. MR – Non lesional

1. Temporal
   a. Non-dominant
   b. Dominant

2. Extratemporal

Do I really need invasive monitoring?

MTLE patients do really well
ECOG may suffice

Invasive Monitoring may be indicated:
- No corroborating imaging (PET)
- Atypical clinical features
- Atypical/insufficient EEG data
- Suspected neocortical focus
- LITT procedure

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39 yo RH woman with epigastric rising followed by automotor (ictal kissing) seizure
EEG: Right temporal interictal and ictal epileptic activity
MRI: no abnormality
FDG-PET: hypometabolism in the right mesial temporal lobe (R MTL)
MEG: right basal, mid-temporal lobe
Ictal SPECT: congruent ictal hyperperfusion over the R MTL

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Indication for Invasive EEG Monitoring

B. MR – Non lesional

1. Temporal
   a. Non-dominant
   b. Dominant

2. Extratemporal

Am I really considering to remove the dominant hippocampus?

Reasons to go forward with invasive Monitoring:
- Pre-existing memory deficit
- PET = Wada asymmetry
- 100% sure it is temporal
- Selective neocortical resection
- Consideration for Laserablation
- Backup Neurostimulation

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Indication for Invasive EEG Monitoring

B. MR – Non lesional

1. Temporal
   a. Non-dominant
   b. Dominant

2. Extratemporal

What is the chance for a good outcome?

Good outcome associated with:
- Distinct aura/clinical onset
- Localized IED
- FDG-PET hypometabolism
- Complete resection of EZ
- Short epilepsy duration
- FCD Type II

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**Invasive Evaluation**

**SEEG: Noninvasive Data and Hypothesis**

1. Is the patient a surgical candidate?
2. Does the patient need an invasive evaluation?
3. What type of invasive evaluation?
4. What is the hypothesis?

**Comparison SEEG vs. SDG**

<table>
<thead>
<tr>
<th>Outcome Stereo EEG vs. Subdural Grids</th>
<th>SEEG</th>
<th>SDGs</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td># patients</td>
<td>200</td>
<td>102</td>
<td></td>
</tr>
<tr>
<td>EZ located</td>
<td>154 (77%)</td>
<td>99 (97%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Resection</td>
<td>90 (45%)</td>
<td>84 (82%)</td>
<td></td>
</tr>
<tr>
<td>Seizure-free</td>
<td>61 (68%)</td>
<td>59 (70%)</td>
<td>0.85</td>
</tr>
</tbody>
</table>

SEEG = Stereo-encephalography; SDGs = subdural grids; EZ = epileptogenic zone

**Table 6.1 Stereoe EEG Versus Subdural Grid Exploration**

<table>
<thead>
<tr>
<th>Case Features</th>
<th>SEEG</th>
<th>SDGs</th>
<th>P</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depth electrodes</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Nonlesional cases</td>
<td>34</td>
<td>56</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deep cortical structures</td>
<td>x</td>
<td>x</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lesions with poorly defined borders (e.g. FCD, complex lesions)</td>
<td>x</td>
<td>x</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multiple lesions</td>
<td>x</td>
<td>x</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior nonlesional/surgical failure</td>
<td>x</td>
<td>x</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>&gt;2 years old</td>
<td>No specific age limit</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Timing of resection</td>
<td>Monitoring is followed by resection 6-8 weeks later</td>
<td>Monitoring and resection within single hospitalization</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

SEEG: Noninvasive Data and Hypothesis

Invasive Evaluation

**In favor of Stereo EEG:**
- MR nonlesional
- Lesions with poorly defined borders (e.g. FCD, complex lesions)
- Dual pathology; multiple lesions
- Surgical failure

**In favor of Subdural grids:**
- Proximity to eloquent cortex

**In selected patients:**
- Combination of stereotactic depths and grids
- Serial invasive evaluation: Stereo EEG followed by subdural grid

**Phase I: Creating a hypothesis**

<table>
<thead>
<tr>
<th>Phase of Epilepsy Surgery Evaluation</th>
<th>SEEG emphasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>History, general, medical and neurological examination</td>
<td>Anatomy-clinico-electrical correlation, anatomy-pathological-clinico-electrical</td>
</tr>
<tr>
<td>EMG monitoring/Genealogy</td>
<td>Morphometric analysis, e.g. BOS CFD</td>
</tr>
<tr>
<td>Neurosurgical assessment</td>
<td>MRI-negative case</td>
</tr>
<tr>
<td>MR imaging</td>
<td>Electrical and Magnetic Source Imaging (ESI; MEG; EEG-MRI)</td>
</tr>
<tr>
<td>Functional Imaging (PET, fMRI)</td>
<td>Functional Mapping (Functional MRI, Wada testing)</td>
</tr>
<tr>
<td>Connectivity — deep lesions, functional pathways</td>
<td>Targeting the source</td>
</tr>
</tbody>
</table>
### Lobe: Frontal

<table>
<thead>
<tr>
<th>Aura</th>
<th>Motor seizure</th>
<th>Non-motor seizure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Olfactory</td>
<td>Simple motor: Tonic, clonic, myoclonic, asymmetric tonic, versive</td>
<td>Dyscognitive: Aphasia, dysphasia</td>
</tr>
<tr>
<td>Autonomic</td>
<td>Hypermotor</td>
<td>Dialeptic</td>
</tr>
<tr>
<td></td>
<td>Gelotic</td>
<td></td>
</tr>
</tbody>
</table>

### Lobe: Temporal

<table>
<thead>
<tr>
<th>Aura</th>
<th>Motor seizure</th>
<th>Non-motor seizure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Olfactory</td>
<td>Simple motor: Tonic, clonic, myoclonic, asymmetric tonic, versive</td>
<td>Dyscognitive: Aphasia, dysphasia</td>
</tr>
<tr>
<td>Auditory</td>
<td>Auditory aura (abrupt ringing, buzzing, chirping, voices, etc.)</td>
<td>Dialeptic</td>
</tr>
<tr>
<td>Gustatory</td>
<td>Gustatory aura (often metallic taste)</td>
<td></td>
</tr>
<tr>
<td>Olfactory</td>
<td>Olfactory aura (often noxious smell)</td>
<td></td>
</tr>
<tr>
<td>Visual</td>
<td>Visual illusions (e.g., macropsia, micropsia, blurred images)</td>
<td></td>
</tr>
</tbody>
</table>

### Lobe: Insular

<table>
<thead>
<tr>
<th>Aura</th>
<th>Motor seizure</th>
<th>Non-motor seizure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatosensory (paresthesias: tingling, warmth, tension, electrical, pain)</td>
<td>Speech aphasias</td>
<td>Dyscognitive/Dialeptic</td>
</tr>
<tr>
<td>Large areas, bilateral, no Jacksonian spread</td>
<td>Automatisms</td>
<td></td>
</tr>
<tr>
<td>Viscerosensitive (constriction, suffocation, choking), visceromotor</td>
<td>Multimodal early olfactory, gustatory, viscerosensory, auditory</td>
<td></td>
</tr>
<tr>
<td>Psychic aura: anxiety, panic, fear</td>
<td>Autonomic: dysgeusia, breathlessness</td>
<td></td>
</tr>
</tbody>
</table>

### Lobe: Parietal

<table>
<thead>
<tr>
<th>Aura</th>
<th>Motor seizure</th>
<th>Non-motor seizure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatosensory auras (e.g., tingling, numbness, pain, thermal sensation)</td>
<td>Uncontrollable eye movements: eye pulling, nystagmus</td>
<td></td>
</tr>
<tr>
<td>Disturbances of body image</td>
<td>Complex visual illusions</td>
<td></td>
</tr>
<tr>
<td>Sensation of movement of a part of the body</td>
<td>Tonic motor, focal motor</td>
<td></td>
</tr>
</tbody>
</table>

### Lobe: Occipital

<table>
<thead>
<tr>
<th>Aura</th>
<th>Motor seizure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual</td>
<td>Uncontrollable eye movements: eye pulling, nystagmus</td>
</tr>
<tr>
<td>Elementary visual hallucinations (e.g., colored shapes)</td>
<td>Head deviation</td>
</tr>
<tr>
<td>Flashing colors</td>
<td>PostictalTodd</td>
</tr>
<tr>
<td>Total blindness</td>
<td>Postictal dysphasia</td>
</tr>
</tbody>
</table>

### Lobe: Parietal

<table>
<thead>
<tr>
<th>Aura</th>
<th>Motor seizure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychomotor aura (e.g., tingling, numbness, pain, thermal sensation)</td>
<td>Uncontrollable eye movements: eye pulling, nystagmus</td>
</tr>
<tr>
<td>Disturbances of body image</td>
<td>Head deviation</td>
</tr>
<tr>
<td>Sensation of movement of a part of the body</td>
<td>PostictalTodd</td>
</tr>
<tr>
<td>Complex visual illusions</td>
<td>Postictal dysphasia</td>
</tr>
</tbody>
</table>

### SEEG: Noninvasive Data and Hypothesis

1. Is the patient a surgical candidate?
2. Does the patient need an invasive evaluation?
3. What type of invasive evaluation?
4. What is the hypothesis?
Pertinent History

31 year old RH man
- Seizure onset 2010 (age 21) after an assault. He was punched from behind resulting in a broken jaw
- Three days later after the assault he started having episodic déjà vu and lightheadedness followed by whole body tingling sensation. This can then progress to witnessed unresponsiveness and convolution
- He has since been refractory to medications and continues to have multiple focal seizures per week

Medications
- Oxcarbazepine 450-450
- Vimpat 200-200
- Divalprox ER 1000-1000
- Onfi 10 mg BID
- Clonazepam 1 mg PRN

Semiology

Seizure Type A: Psychic aura → Automotor Sz → Right head/body service seizure → GTCS

- Description: Patient describes a Déjà vu sensation followed by a feeling of dizziness and then whole body tingling bilaterally. Partner reports that his speech slows down and he then has automatisms (lip smacking, repetitive wrist movements). This lasts about 15-25 seconds. Events can end here or progress to head deviation to right and then full body shaking.
- Frequency: auto-motor several times per month, one convulsion per month

Video EEG

- Interictal: Left temporal sharp wave

Imaging

NPS

Summary

Action Items

Invasive Plan

Epilog
Imaging Findings

The F-18 FDG PET images of the brain suggest minimal decreased metabolic activity in the anteromedial left temporal lobe as compared to the right. This is a subtle abnormality, but this focal site of decreased metabolic activity has been described in seizure foci. Subtle asymmetric prominence of the mesial left temporal lobe with faint T2 signal abnormality in this region. These findings are nonspecific but may represent cortical dysplasia. The combined PET/MR studies show decreased activity and subtle abnormality in the left mesial temporal lobe, which may represent an epileptogenic focus.

Language & Visuospatial Functions

Temporal-Memory Functions

What is the Hypothesis
Hypothesis

Hypothesis:
- Primary: Left mesial temporal lobe epilepsy
- Secondary: Other temporal subtype

Indication:
- “100%” sure patient has temporal lobe epilepsy
- Understand temporal lobe subtype for targeted treatment approach
- Potential candidate for Laserablation vs. RNS

What are the risks of a SEEG evaluation

Epilepsy related

- Unable to localize EZ
- Unable to operate

Surgical complications

- Hemorrhage:
  - 1-2%: ICH > SDH > EDH; intervention in 1/3
  - 2-2% symptomatic, 0.4% permanent deficit
  - post-explantation CT: 50% some signs of hemorrhage
  - 3.5% delayed hemorrhage
- Infection
  - 0.8%: abscess, meningitis, superficial infections
- Hardware problems
  - 0.4%: accidental removal, fracture, recording issues; anchor bolt fracture
- Other
  - CSF leak; cerebral edema, pressure ulcers, vascular injury

Implantation Scheme

Population 1: SPK over B3

Seizure 1PG/2PG

- Apneic seizure
- Right Vertex seizure
- Repetitive episodes over B3/B2 for 1.5 seconds
- DC shift over B2/B3, D2/B2, B3/D3
- High amplitude spiking in epileptogenic zone over mesial contacts
- LVFA over basal temporal and superior temporal
- Spread to lateral temporal contacts

Seizure stimulation mapping: low frequency
### Decision: Laserablation vs. RNS implantation

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Yes/No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Resection in the dominant hemisphere</td>
<td>No</td>
</tr>
<tr>
<td>2. MRI findings other than exclusively unilateral MTS</td>
<td>Yes</td>
</tr>
<tr>
<td>3. Intact preoperative immediate verbal memory function</td>
<td>No</td>
</tr>
<tr>
<td>4. Intact preoperative delayed verbal memory function</td>
<td>No</td>
</tr>
<tr>
<td>5. Intact intracarotid amobarbital procedure (IAP) memory performance</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Stroud E, Neurology 2003

### Summary

- 20-30% of surgical epilepsy patients undergo an invasive evaluation prior to resection.
- The majority of invasive studies are performed on patients with lesional epilepsy.
- Advanced imaging and improvements in noninvasive understanding of the EZ has shifted the indications for invasive monitoring to more complex cases.
- Particularly for patients with multiple or deeper lesions and non-lesional epilepsies, SEEG has become the method of choice.
- SEEG relies heavily on hypothesis creation for electrode placement and emphasizes three dimensional information (semiologic onset and propagation, source imaging, functional imaging and functional connectivity).
- Particularly “nonlesional” cases benefit from advanced MRI analysis.
- Hypothesis is crucial and should take potential surgical procedure into account.