2023 GW Epilepsy Board Review & Best Practices

Invasive EEG recordings

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Epilepsy Board Review Course
George Washington University

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Disclosure

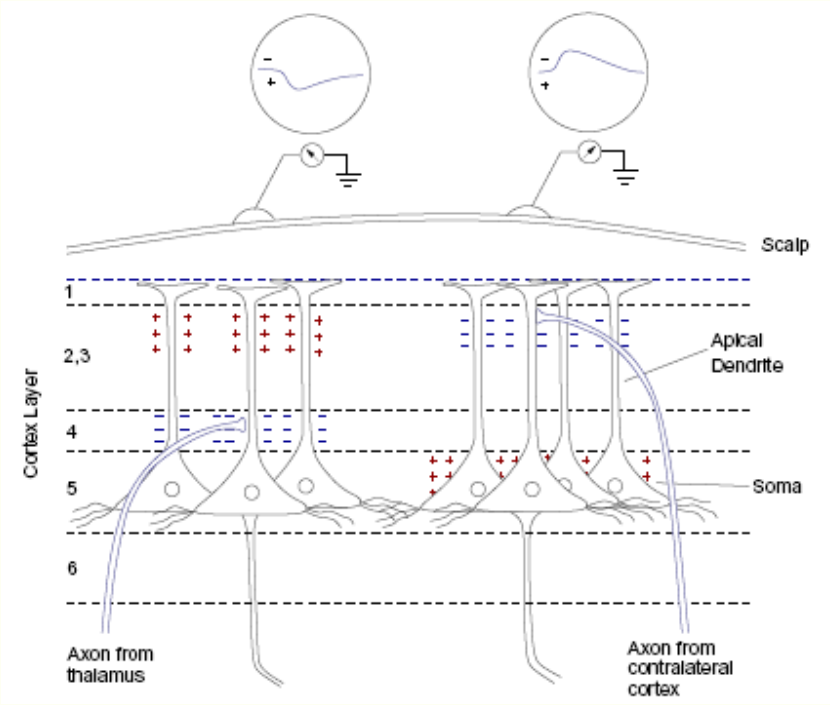
No relevant conflict of interest

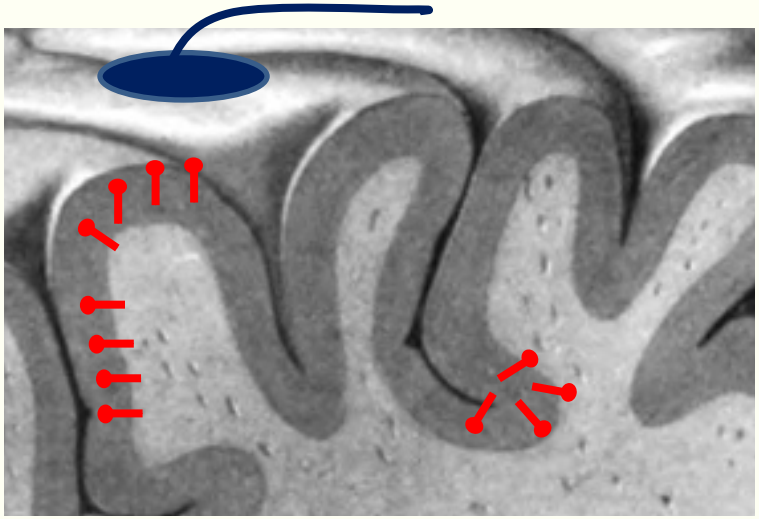
Primary Objective

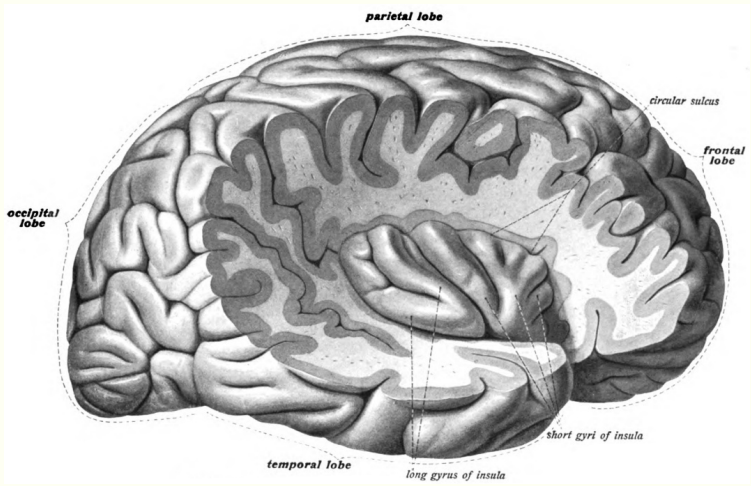
Invasive EEG recordings:

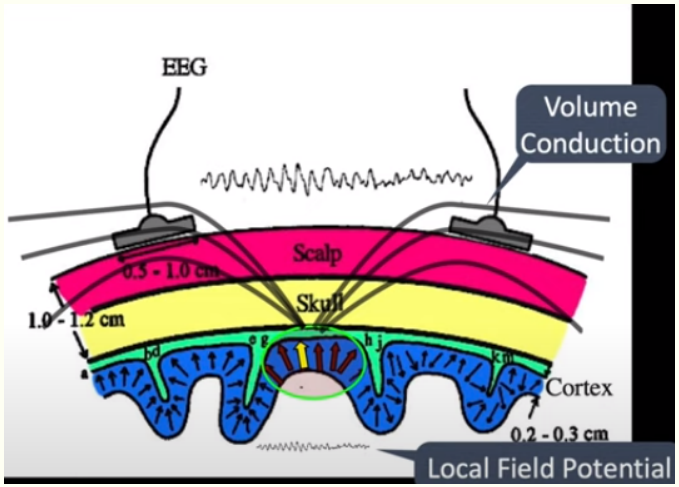
1. Subdural grid electrodes
2. Stereo EEG and other depth electrodes
3. Functional mapping

EEG Generators









Speckmann et al, 20001

Why Intracranial Electrodes?

- Spike visibility on scalp depends on: *synchrony, location, & cortical extent*
  - Linear relationship between *amplitude* and the *above factors*

- Power of scalp EEG: inversely proportional to a power of 2.5 of the frequency *($P=1/F^{2.5}$)*

  - Decreases at higher frequency bands. Fast oscillations (80–200 Hz) are rarely detected on scalp EEG because of low amplitude (>10 times smaller than IEDs, c/w with cortical generators of ~ 1 cm^2)

  von Ellenrieder et al, 2014

Why Intracranial Electrodes?

- 6 cm^2 of synchronized cortical activity is necessary to record scalp electrodes Cooper et al, 1965

- Only a few of cortical spikes are associated with scalp potentials:
  - 90% of cortical spikes with source area of >10 cm^2 produced scalp EEG spikes
  - only 10% of cortical spikes of <10 cm^2 source area produced scalp potentials
  - Intracranial spikes with <6 cm^2 of area are never associated with scalp EEG spikes Tao et al, 2005, 2007

- Recent studies indicate that a significantly smaller generator may be visible on the scalp von Ellenrieder et al, 2014

Invasive Monitoring – Historical Perspective

- First invasive serial EEG recording using epidural electrodes (1939 at MNI):
  - Potential for delineation of the seizure focus

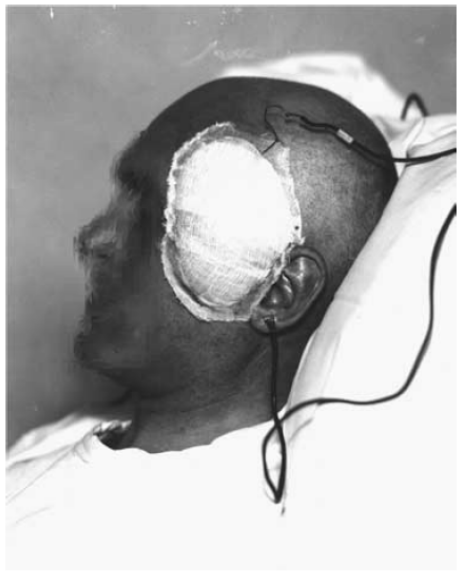


FIG. 2. Patient with the epidural lead and reference electrode on ear lobe positioned for recording. He was monitored for 3 days until enough data were gathered for surgery.

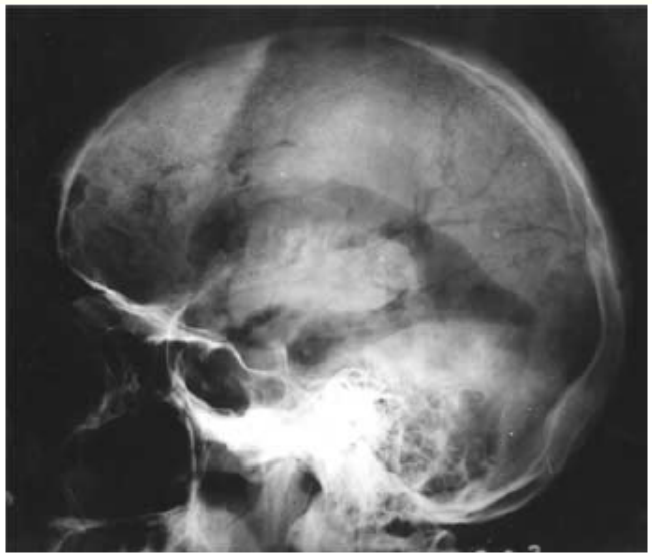


FIG. 1. Patient's pneumoencephalogram done in 1938, 1 year after his car accident, showing bilaterally enlarged ventricles with selective dilatation of the left temporal horn.

Epidural single-contact electrodes placed through burr holes over both temporal lobes. Lt temporal lesionectomy; seizures did not improve

Almeida et al., Epilepsia, 2005. Reif et al, Seizure, 2016

Invasive Monitoring – Historical Perspective

- ***Subcortical/deep brain lesions*** e.g., thalamus, basal ganglia, identified as sources of slow and epileptiform activity (*potential seizures generators)*

  Lennox & Brody, 1946

- First report on stereotactically implanted EEG electrodes in humans with epilepsy:

  - Independent seizure activity in cortical and subcortical structures (hence the notion of *simultaneous recording* of *cortical and deep brain* tissues)

  Hayne et al, 1949

- Jean Talairach's first experience with stereotactic procedures

  Talairach et al., 1949

- Stimulation studies in animals and their impact on generalized epilepsies showed the Influence of *thalamocortical circuits*

  Jasper & Marsan, et al, 1951

Invasive Monitoring – Historical Perspective

- In temporal lobe epilepsy (TLE) the importance of the resection of mesial TL structures was recognized Penfield, 1950; Jasper et al., 1951

- Interhemispheric connectivity (observation of a fast shift of EEG patterns in TLE) highlighted the role of subcortical regions/ tracts in seizure generation and *propagation* Jasper et al., 1951

- Improved implantation technique (using pneumencephalogram to adapt implantation coordinates with respect to ventricles):

  - Defined a system of reference lines and structures that allowed an individualized and optimized approach for investigation of deep brain structures and their anatomical localization

- First atlas of stereotactically defined brain structures Talairach, 1957

Invasive Monitoring – Historical Perspective

- First *combined subdural, epidural and depth* electrodes to investigate temporal lobe epilepsy Marsan & van Buren, 1958

- Association between a cerebral lesion, the *irritative zone*, and the *epileptogenic* focus conceptualized Talairach & Bancaud, 1966

- Spikes likely did not localize the epileptogenic focus (in contrast to Jasper). The ‘‘irritative zone’’ was considered solely an interictal phenomenon; seizures could be recorded elsewhere

- Depiction of *epileptogenic focus* based on spatio-temporal characteristics of seizures:

  - An “anatomo-electro-clinical” correlate for the definition of epileptogenic Zone (‘‘epileptogenic network’’)

Invasive Monitoring – Subdural Grids

- Safety of subdural strips in epilepsy patients established, since 1952 (London Hospital; strips over frontal lobe and amygdala through burr-holes) Fischer-Williams, 1963
- Since the 1980s, subdural grids became more popular, esp. subtemporal access to mesial temporal lobe Wyler, Ojemann et al, 1984
- Functional analysis and cortical evoked potentials through subdural electrode stimulation Luders, Lesser, et al, 1983

Invasive Monitoring - Indications

- Precise delineation of the region of seizure onset when:

  - Noninvasive data are inconclusive

  - To resolve divergence of noninvasive data pointing to 2 or more regions

  - To map eloquent cortex

  - Sources are too deep or too weak to be seen on scalp

  - Scalp recording is thought to be a propagation pattern

Jobst et al, 2020

Invasive Monitoring - Indications

- Common clinical scenarios requiring iEEG:

  (a) Nonlesional, likely extratemporal epilepsy

  (b) There is a lesion, but it is either large or deep/small, or there are multiple lesions so one needs to be confirmed as the epileptogenic zone

  (c) Close proximity of the putative EZ to eloquent cortex

  (d) Previous failed surgery

- Presence of a lesion increases the chances of seizure freedom after a resection by a factor of 2.5

Tellez-Zenteno et al., 2010; Kovac et al, 2017

Intracranial Electrodes - Safety

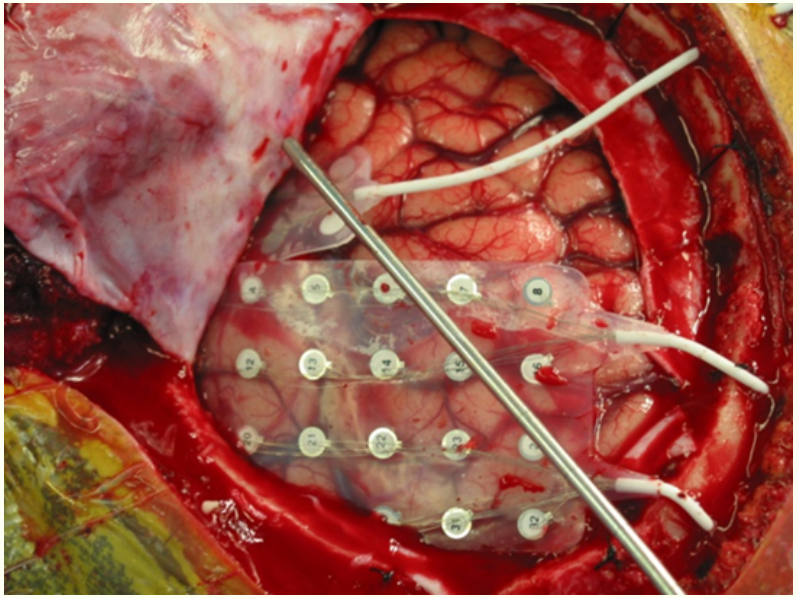
- Data from 452 implantations in 420 patients (1999 to 2019):
  - n = 160 subdural electrodes, n = 156 depth electrodes, n = 136 combination of both approaches

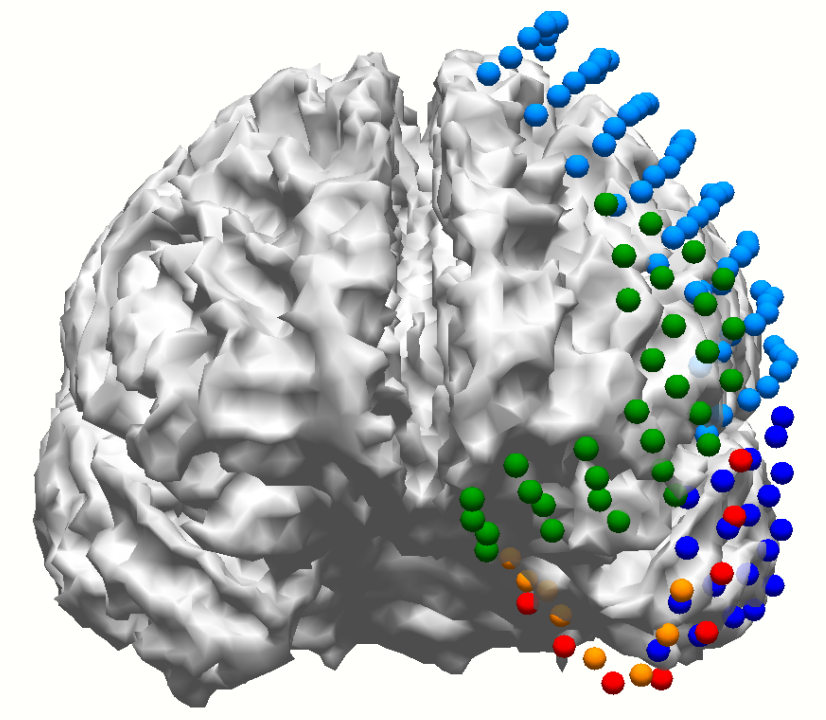
- Most frequent complications in both implantation groups:
  - Hemorrhage
    - Subdural electrode: more symptomatic
  - Hemorrhage risk higher for 64-contact grids (than for smaller grids)
  - Infection: 0.2%
  - Transient neurological deficit: 8.8% (more with grids)

Julia Männlin et al, 2023

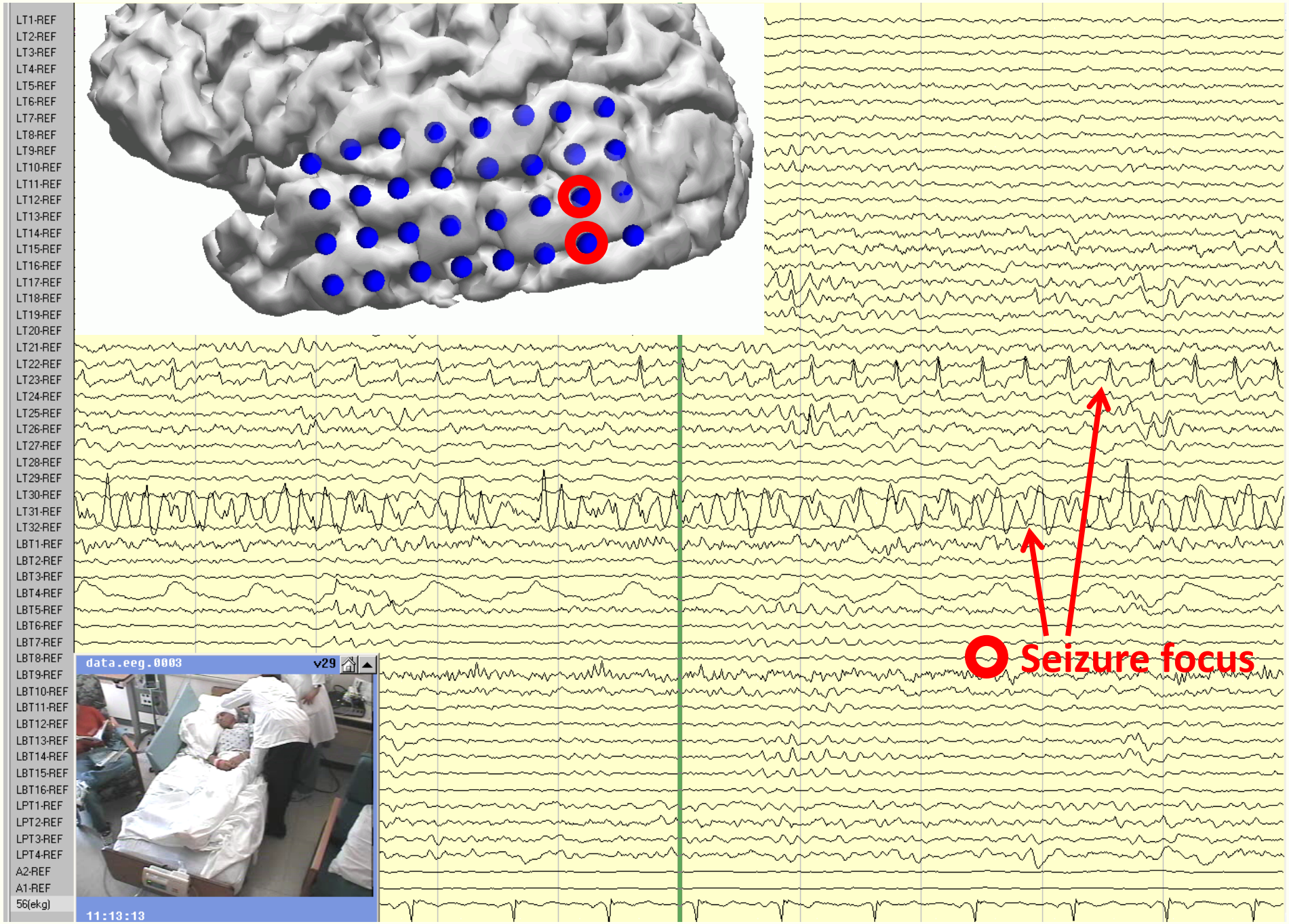
Invasive Monitoring – Subdural Grids

- 4 × 8 subdural grid over Lt lateral temporal lobe
- 2 × 8 strip inserted forward to wrap around the anterior tip of the temporal lobe and cover basal and medial regions
- 2– 3 mm opening in the silastic cover on the opposite side provides a direct contact with brain





3D reconstruction. Left lateral temporal, pre/post-rolandic frontoparietal, frontopolar, fronto-orbital, and two 1 × 8 strips inserted towards the medial temporal regions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Seizure focus

Cortical Stimulation Mapping

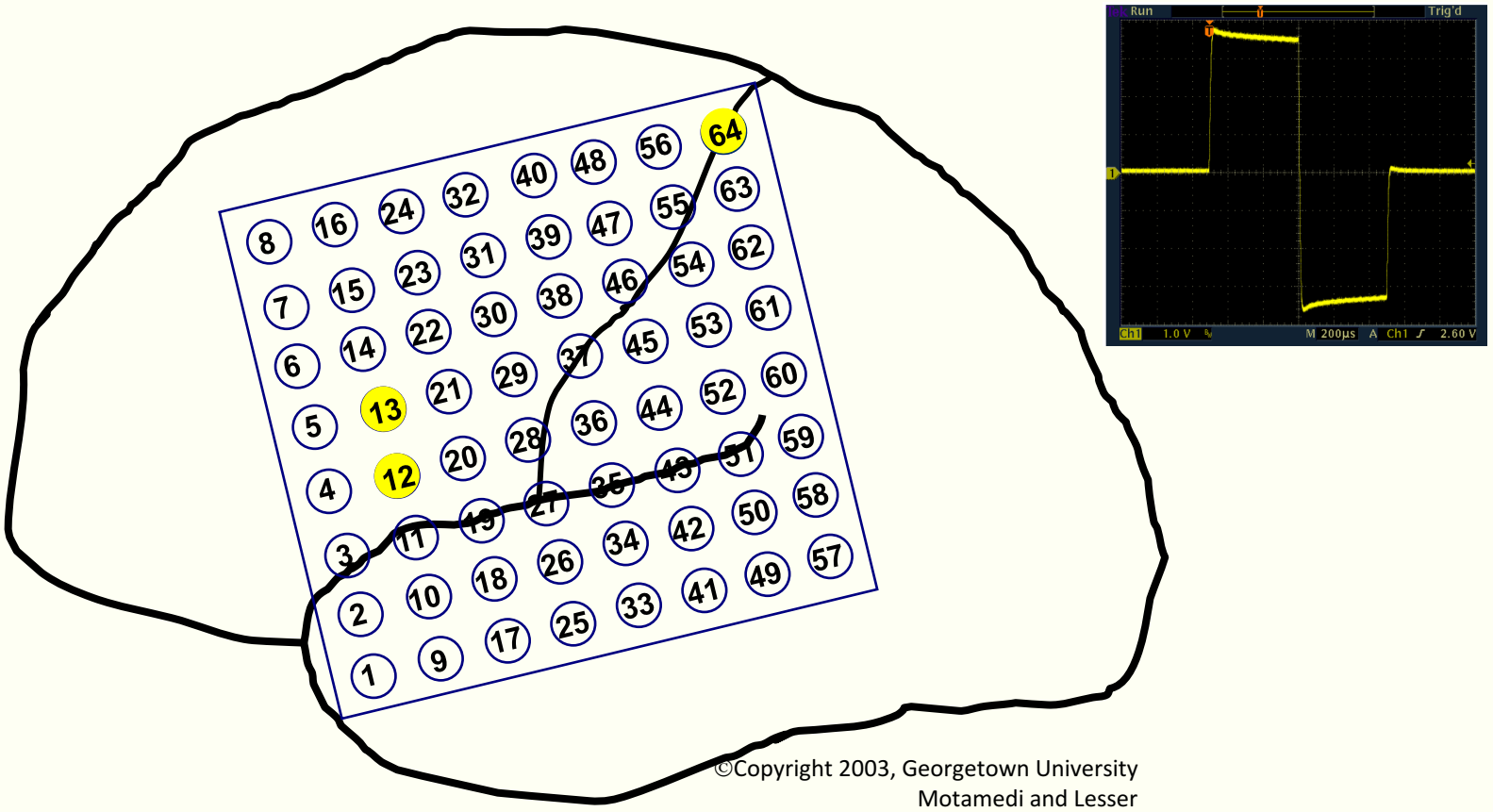
- 2.3-mm-diameter platinum-iridium electrodes embedded in a plastic/silastic sheet; 1cm center-to-center distances

  (Ad-Tech, Racine, Wisconsin)

- Alternating polarity square wave pulse pair stimulation (0.3 msec, 50 or 100 Hz), 3-5 seconds

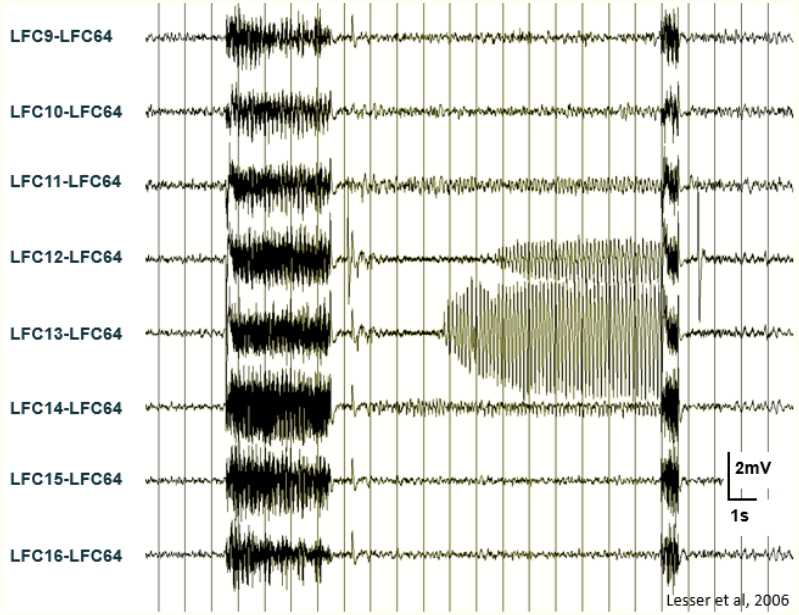
- One electrode pair stimulated at a time starting at a low intensity (1 to 2 mA) titrated up by 0.5 to 1.0 mA at a time till a functional change occurs, or max current of 15-17.5 mA is reached, or ADs develop

Cortical Stimulation Mapping



Left hemispheric subdural grid electrode; stimulated pair of electrodes (12 & 13) and reference (64)

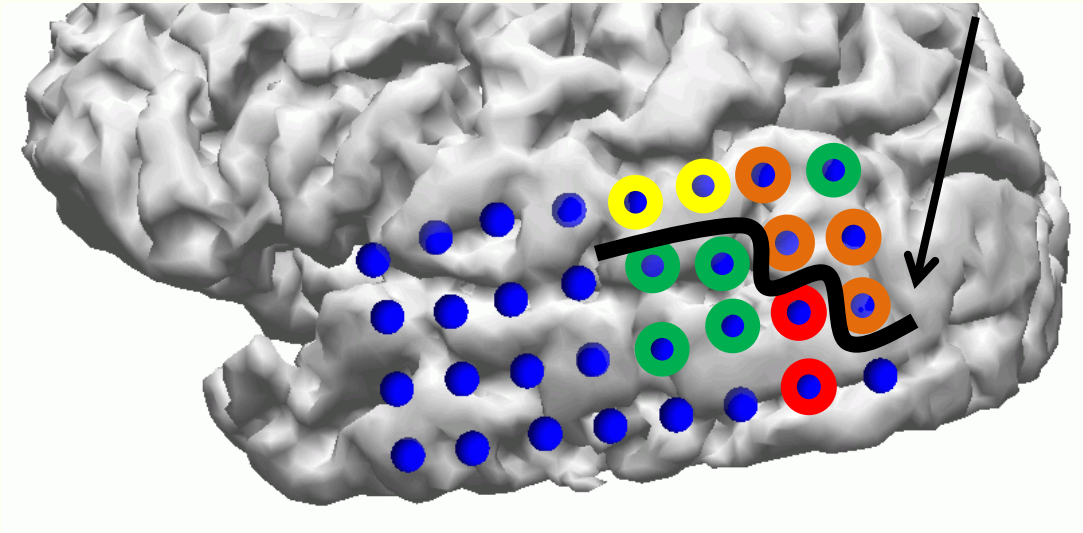
Cortical Stimulation Mapping



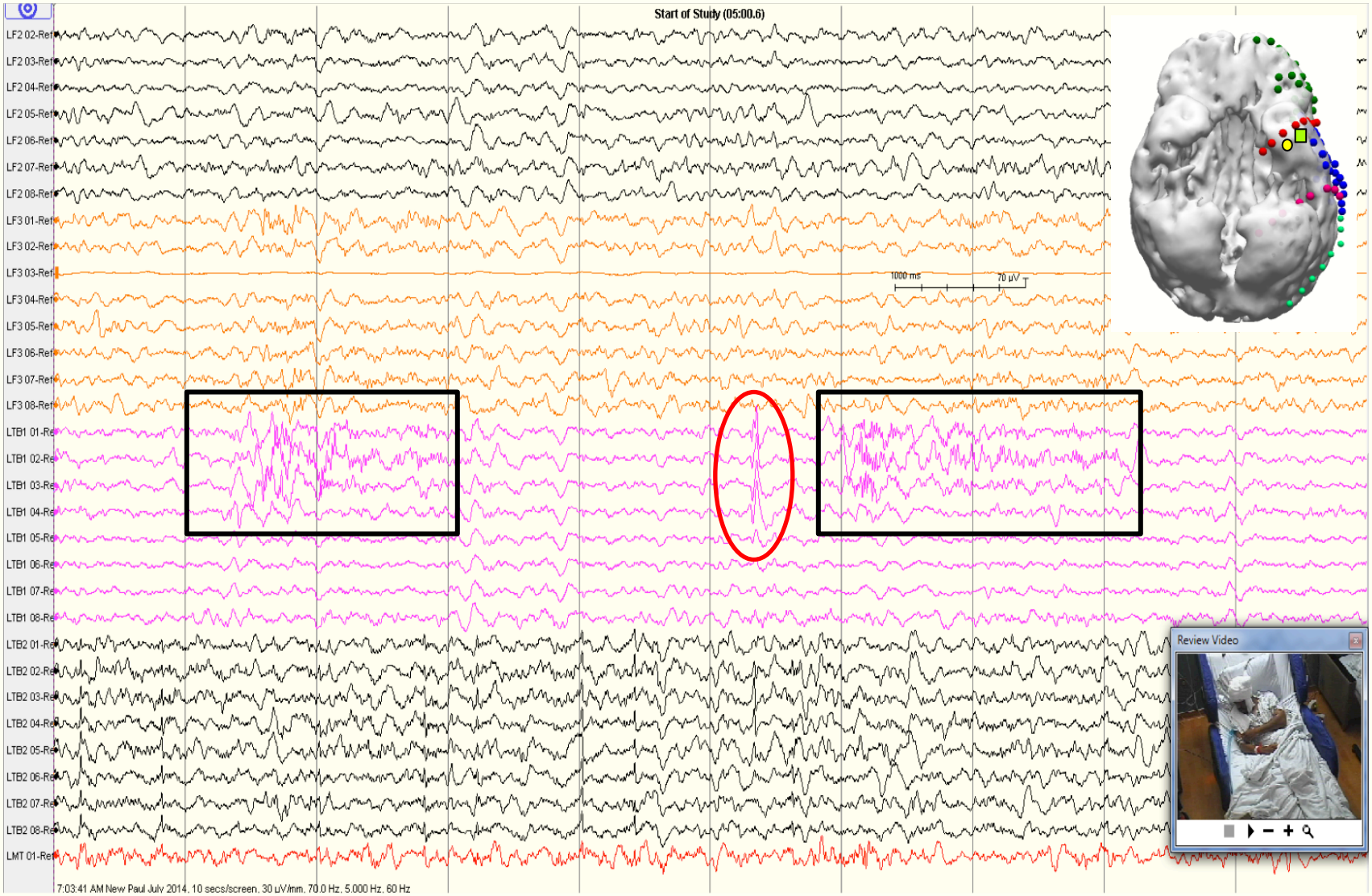
LS BPS

- Seizure focus
- Language
- Sensory
- "Clear"

Resection line

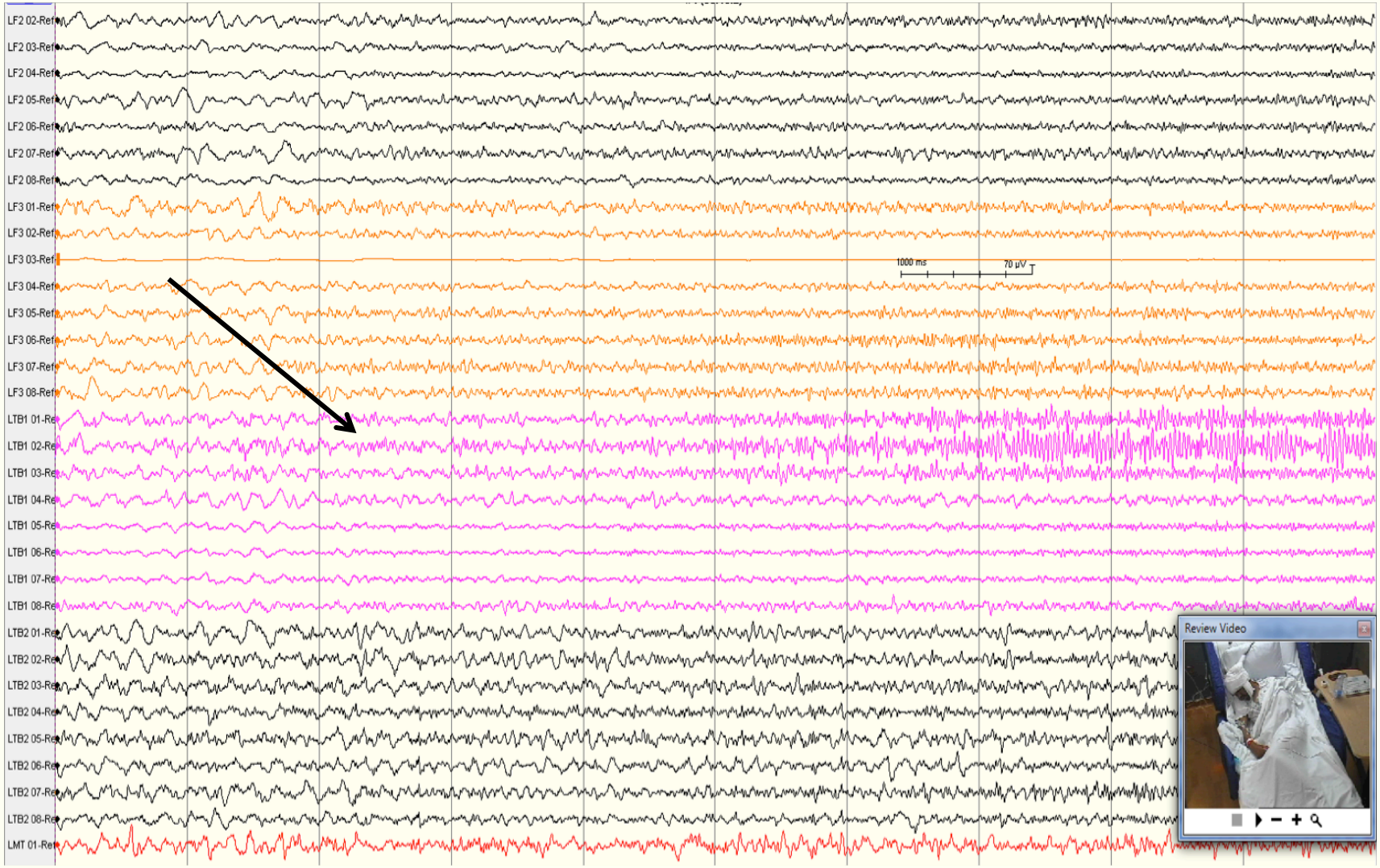


High Frequency Oscillations (HFOs)



Interictal HFOs and spike. Fast oscillations (80–200 Hz) are hard to record on scalp EEG (amplitude >10 times smaller than IEDs), i.e., cortical generators of ~ 1 cm^2

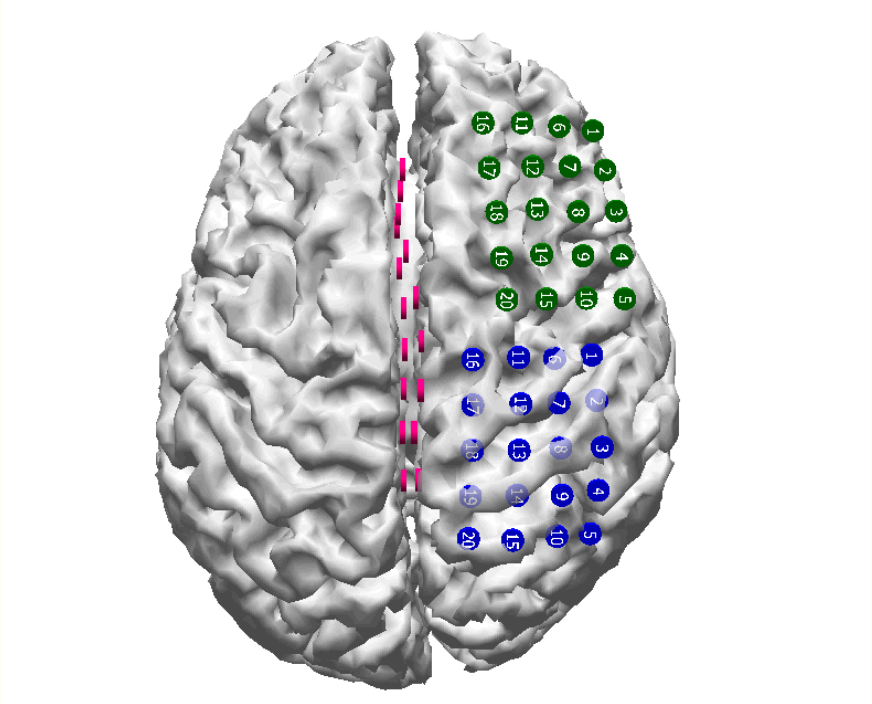
High Frequency Oscillations (HFOs)



HFOs at ictal event

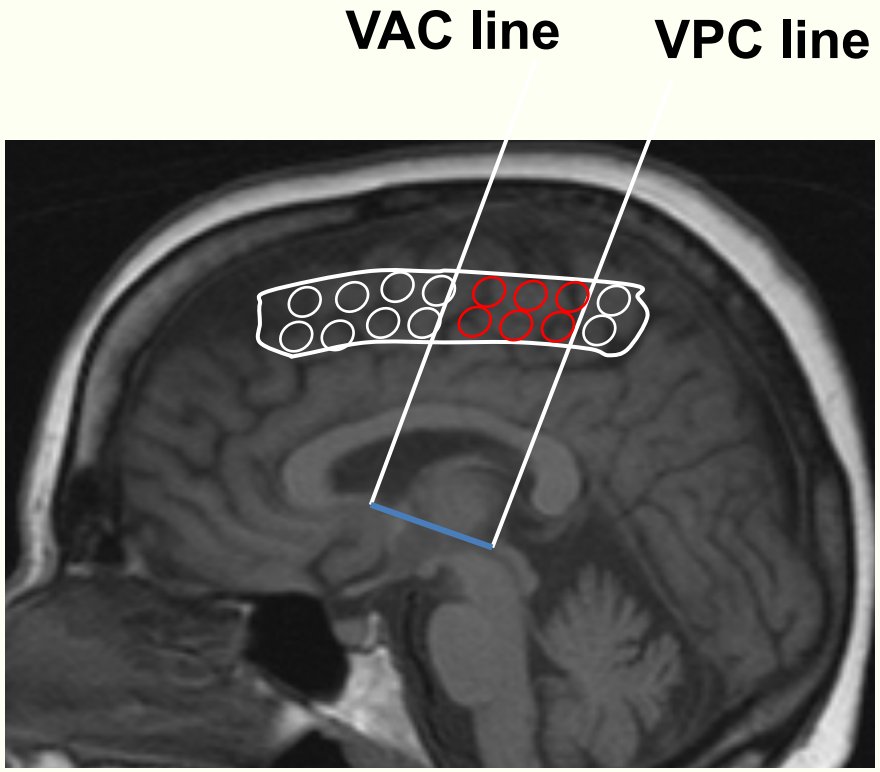
Subdural Evaluation

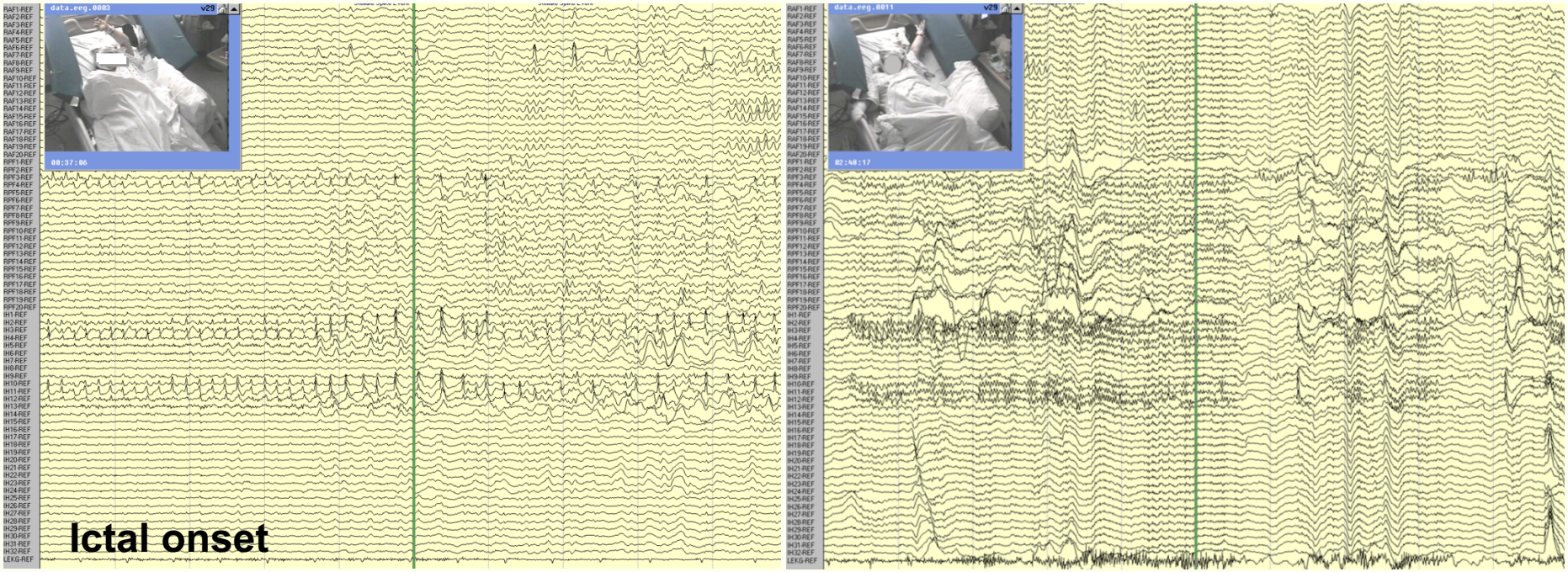
Frontal (Supplementary Motor Area/SMA) Epilepsy



Subdural Evaluation

Frontal (Supplementary Motor Area/SMA) Epilepsy

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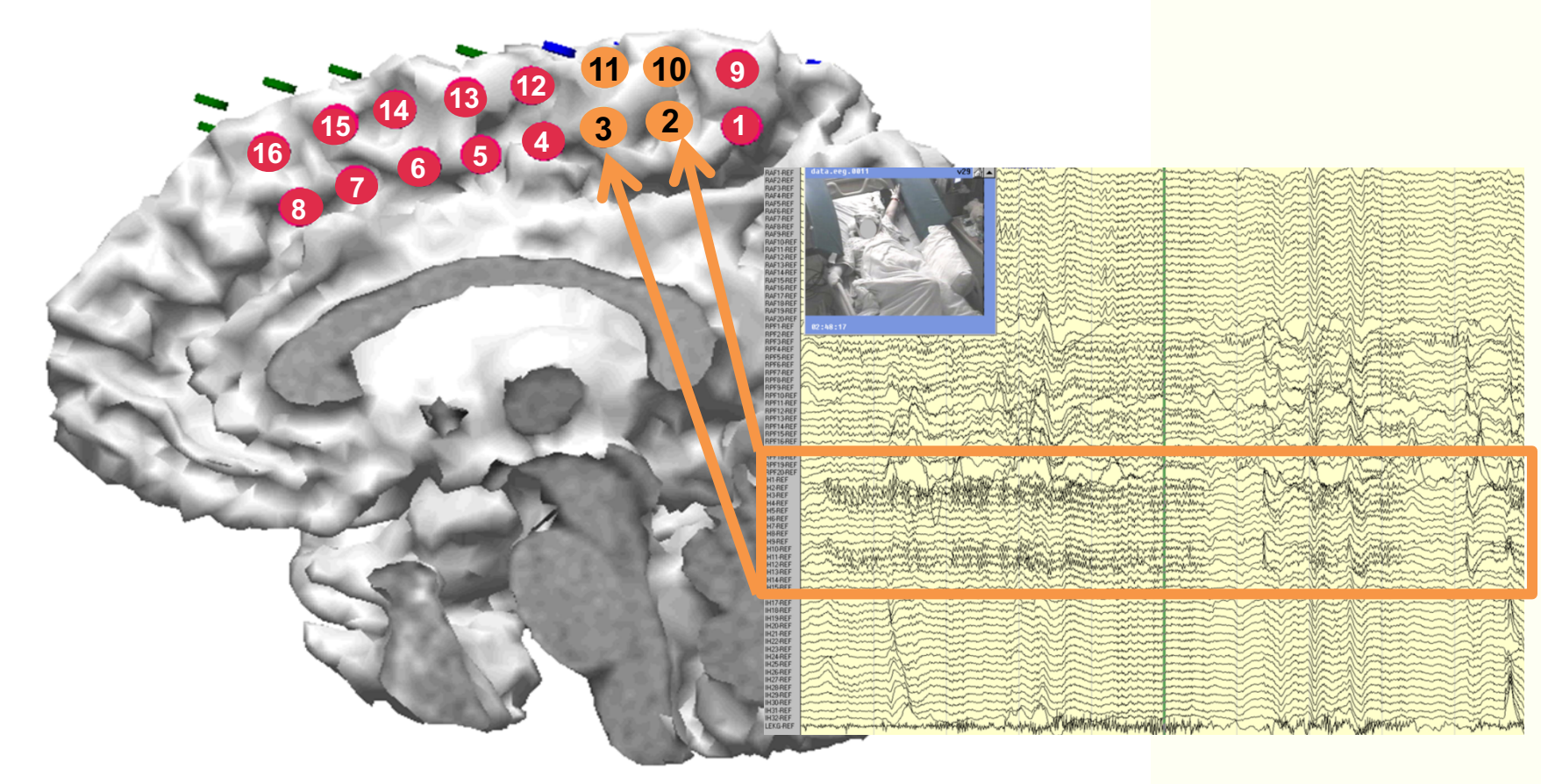
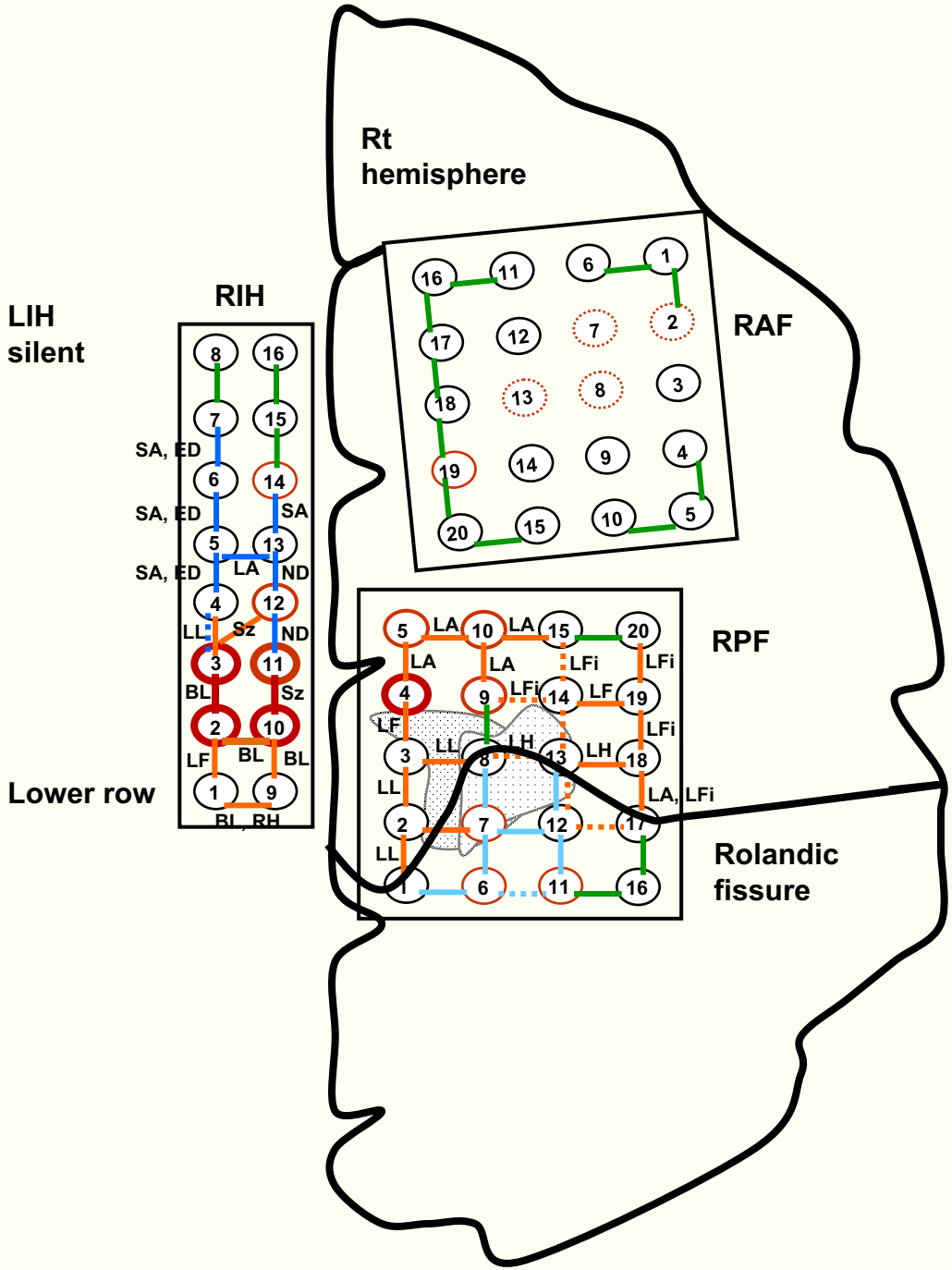
Ictal onset

Subdural Evaluation

Frontal (Supplementary Motor Area/SMA) Epilepsy



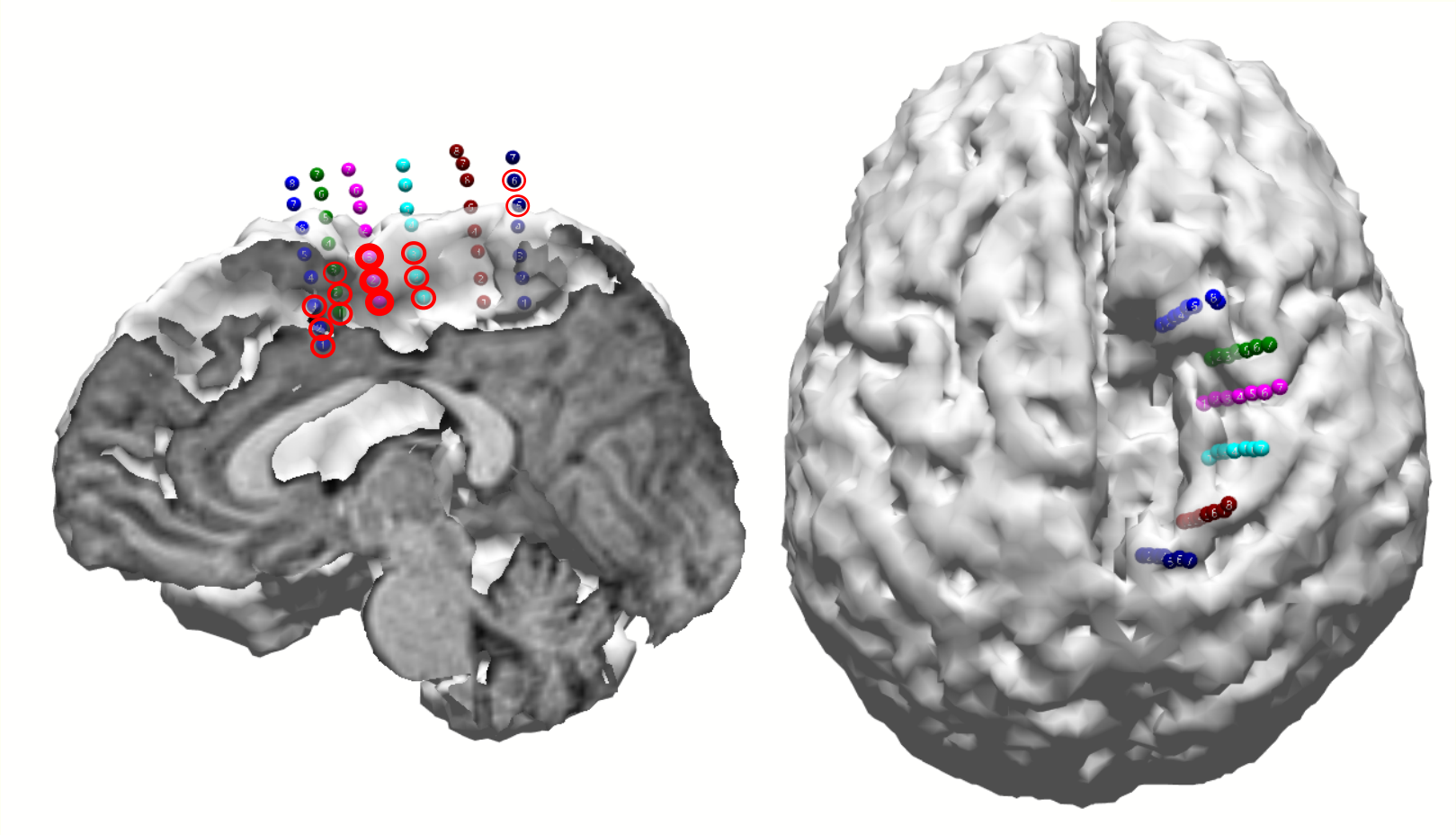


Ictal
Interictal
Motor
Sensory
SMA
Clear
Dysplasia

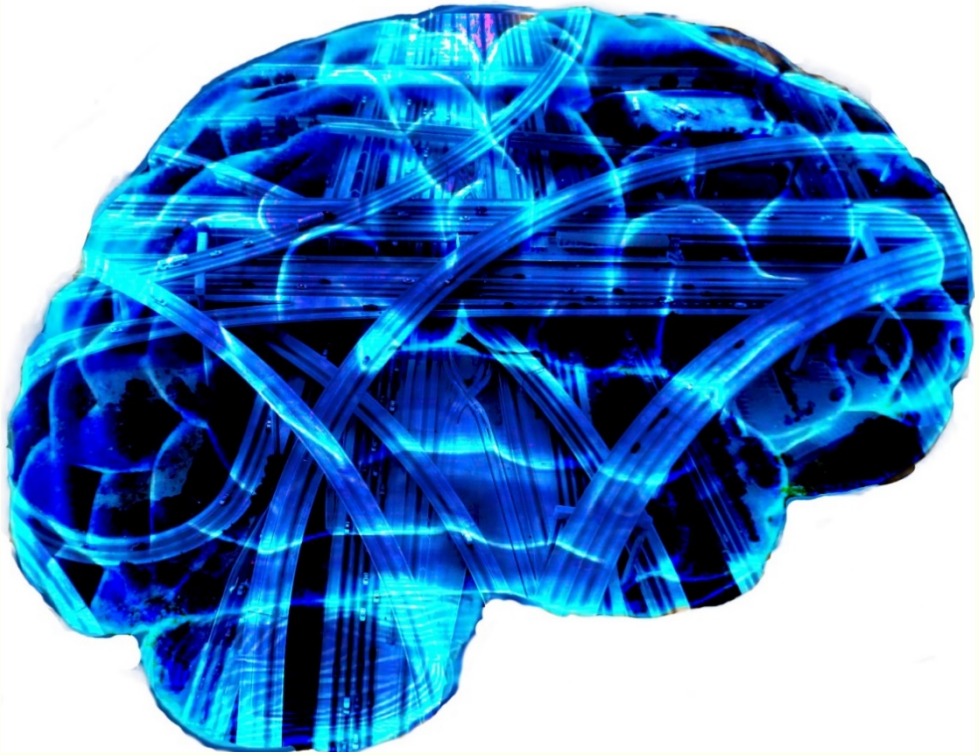
LH, LA, LFi: left hand, left arm, left finger
LL, LF, LT: left leg, left foot, left toe
BL: both legs (motor)
LS, TS, AS: Leg, trunk, arm sensory
SA: speech arrest
ED: eye deviation to Lt
ND: neck drop
Sz: induced seizure

Ictal and interictal

Interictal



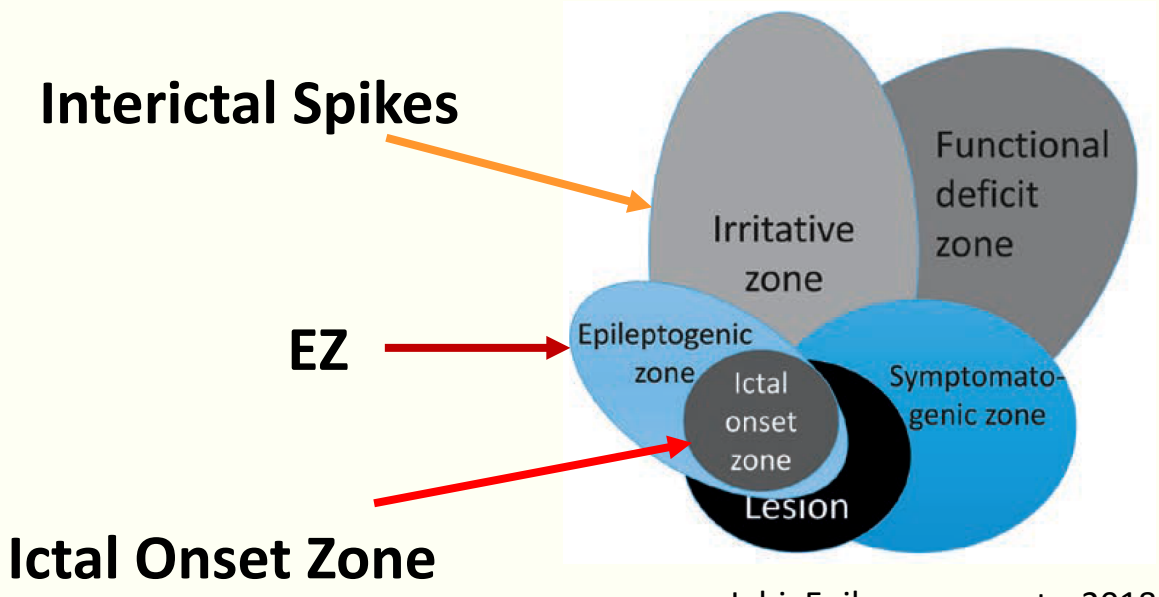
A Network Disorder



Epileptogenic Zone

- Area of cortex necessary and sufficient for initiating seizures whose removal is necessary for complete abolishment of seizures

Luders et al, 1993



Jehi. Epilepsy currents, 2018

Epilepsy as a Network Disorder

- Insufficient resection of the mesial temporal structures: higher risk for seizure recurrence Hennessy et al., 2000
- Surgical failure when only one focus is removed
  - Repetitive identical behavioral seizures suggest one focus, but epileptogenesis might be distributed
    - Connected by functional/structural networks
- Post-surgical prognosis may be related to the extension of the epileptogenic network to subcortical structures (e.g., thalamic)
- Better ATL outcome when low values of thalamocortical synchrony are found, and worse in case of high index of synchrony Guye et al, 2006

Epilepsy as a Network Disorder

- High prevalence of comorbid neuropsychiatric disorders
  - Similar networks postulated in neuropsychiatric disorders, (depression/anxiety)
  - Overlapping with epilepsy networks (hence the high prevalence of comorbid neuropsychiatric disorders in epilepsy)

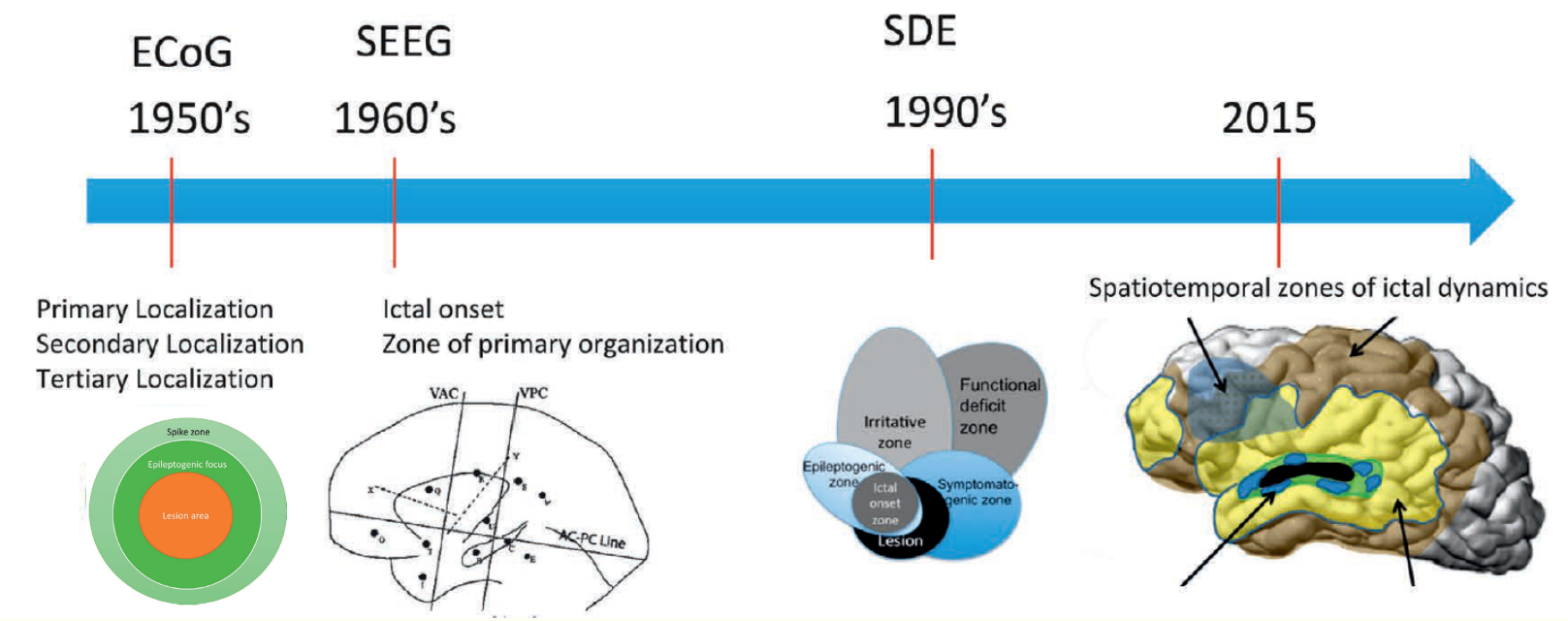
- Functional, anatomical, and bilateral connection of the cortical and subcortical brain regions

- Multiple foci in a circuit can act as independent seizure generators in an abnormal network

The epileptogenic zone *is* the brain

Spencer et al, 2002, 2018; Phi and Cho, 2019

Network vs Focus



(Jehi. Epilepsy currents, 2018)

Broadly applied treatment (directed at any region of the network) should be as effective as treatments directed at a specific seizure 'focus'

Simultaneous recording **of SEEG signals from** ***deep regions*** **and buried cortex &** ***superficial structures*** **allows delineation of a 3-D,** ***spatial and temporal organization*** **of seizure** Minotti et al, 2018

French Guidelines on SEEG - Indications

- **Insufficient anatomo-electroclinical concordance** (a surgical hypothesis exists)
- **Most appropriate method for simultaneous recording:**
  - Superficial & Deeply located cortical structures (e.g., insulo-opercular system, the limbic system)
  - Sulcal cortical zones (e.g., focal cortical dysplasia)
  - Deeply located or periventricular lesions (e.g., periventricular heterotopia, hypothalamic hamartoma)
- **SEEG is preferred to subdural explorations:**
  - Lower morbidity
  - In bilateral foci
  - Prior craniotomy
  - MRI-negative cases

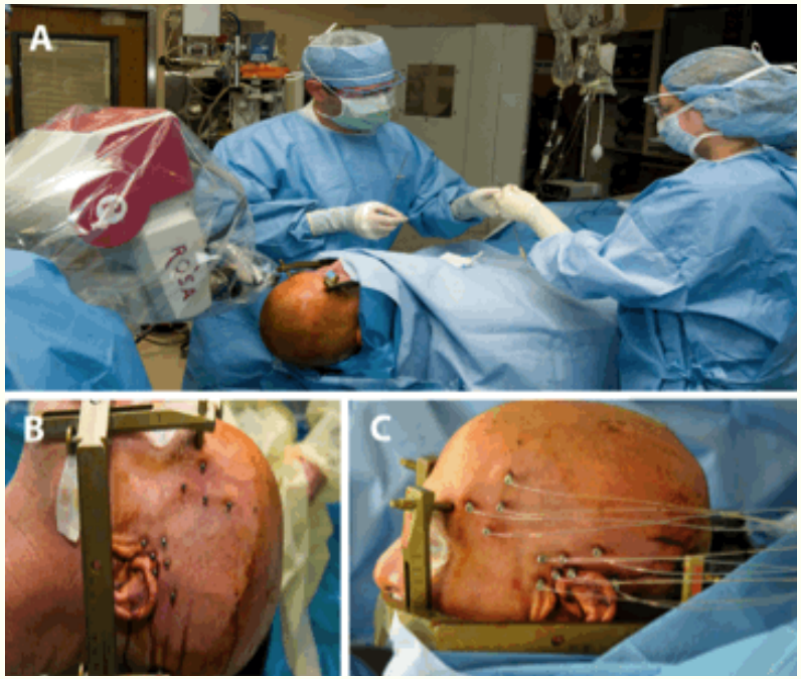
Limitations

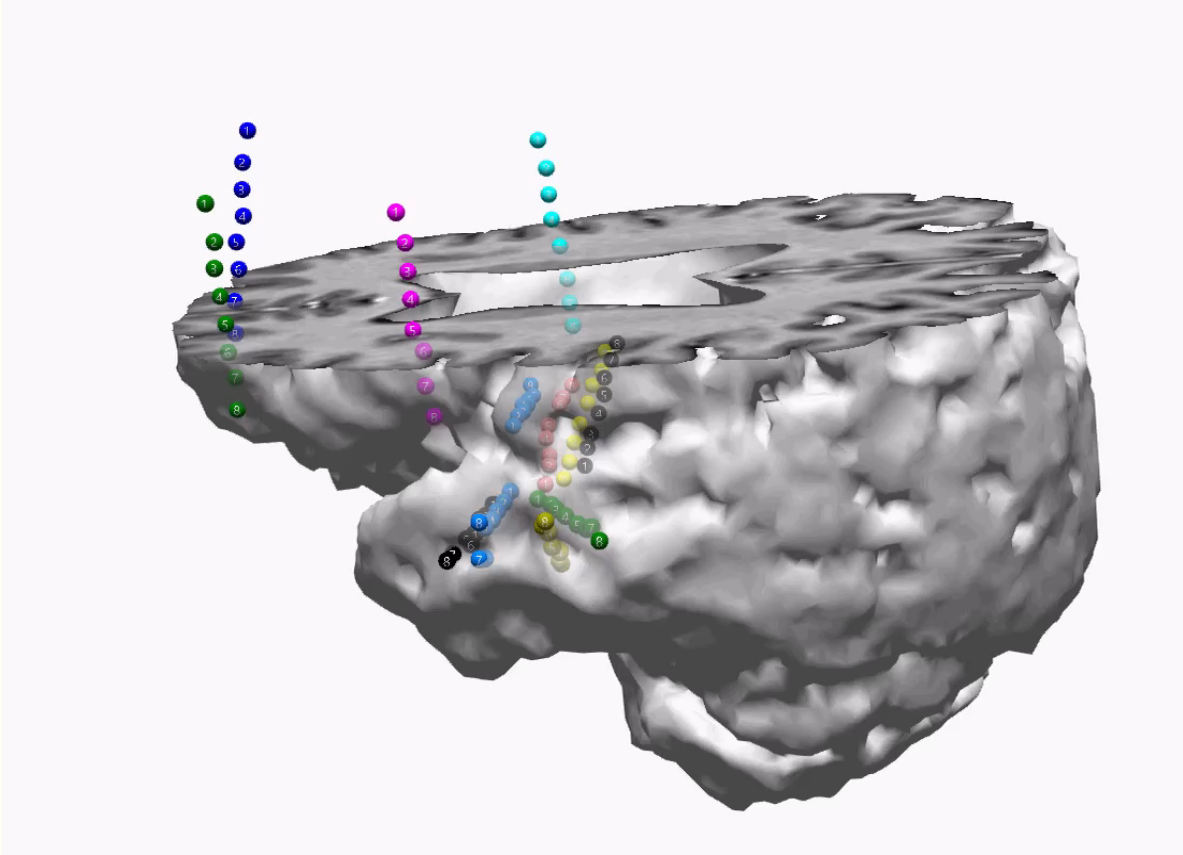
- Sampling bias
- Less spatial resolution than grids for cortical mapping

Isnard et al., 2018

Robotic Stereotactic Assistance

- Frameless and robot-assisted SEEG implantation provide easier, safer, and accurate insertion of sEEG
- No stereotactic frame
- Co-registering images, 3D image added to software which pinpoints exact location and trajectory
- Choosing entry point and a target
- Multiple pinhole sized incisions

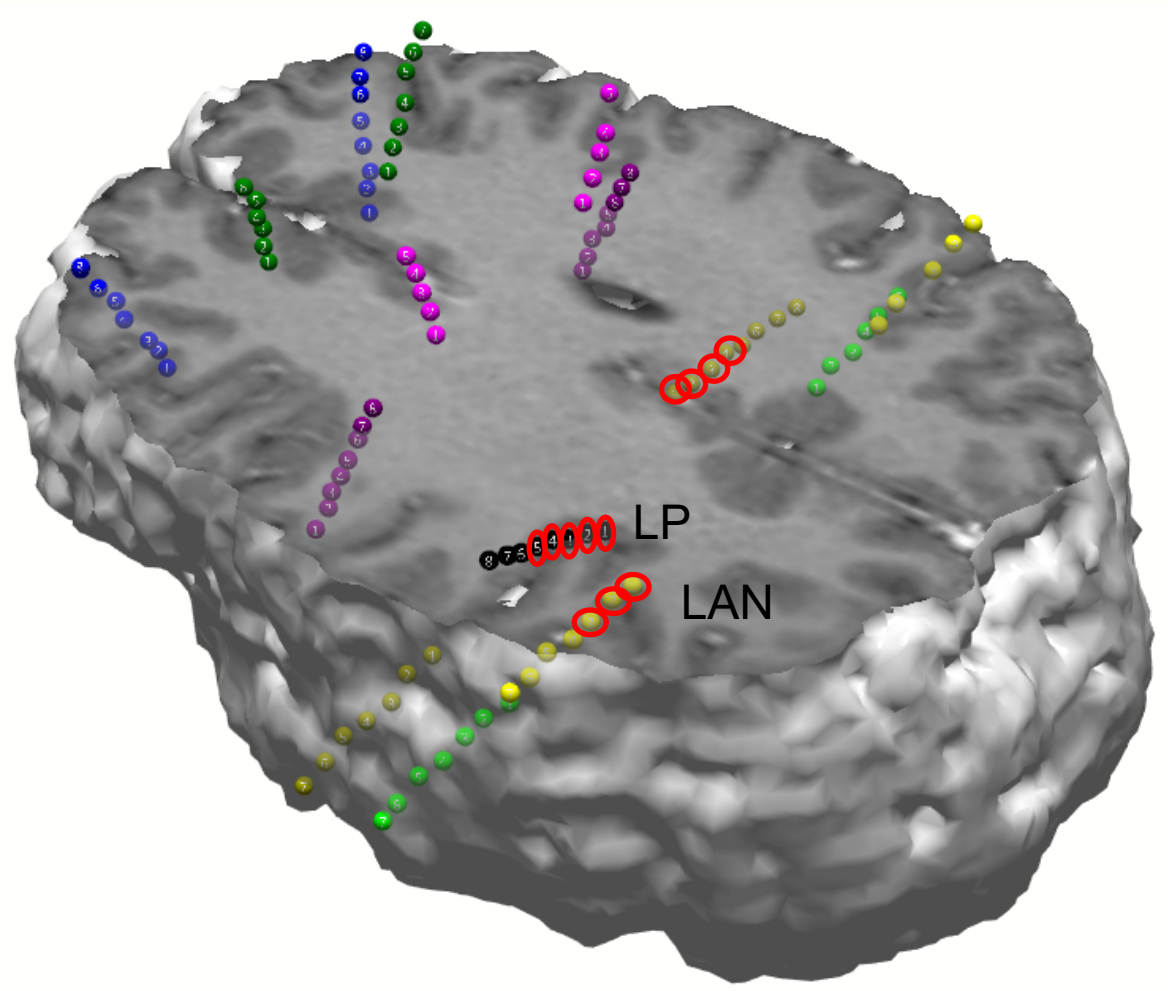




Surgical Hypothesis: A suspected Fronto-temporo-parietal epileptogenic network

Bilateral depth electrodes implanted through Robotic Stereotactic Assistance (ROSA)

- Interictal and ictal
  LP=Lt Parietal
  LAN=Lt Angular



Thalamus

- Relay center: connecting cortical & subcortical networks

- Association nuclei: anterior (ANT), mediodorsal (MD), pulvinar (PUL)

  - Limbic system (alertness, emotion, memory)

- Animal studies: potential role for thalamus in secondary generalization:

  - Pharmacological manipulation in the midline thalamic nuclei sig. limits 2^{ndary} generalization

    (Turski et al.,1984)

  - Thalamic inactivation by lidocaine shortens Hippocampal discharges in kindled rats (Bertram et al., 2001)

Thalamus

doi:10.1093/brain/awl151 *Brain* (2006), 129, 1917–1928

The role of corticothalamic coupling in human temporal lobe epilepsy

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The EEG activity of the thalamus and temporal lobe structures (hippocampus, entorhinal cortex and neocortex) was obtained using intracerebral recordings (stereoelectroencephalography, SEEG) performed in patients with TLE seizures undergoing pre-surgical evaluation. Synchrony was studied using a statistical measure of SEEG signal interdependencies (non-linear correlation). The results demonstrated an overall increase of synchrony between the thalamus and temporal lobe structures during seizures. Moreover, although there was great inter-individual variability, we found that values from seizure onset period were significantly higher than values from the background period ($P = 0.001$). Values at the end of seizure were significantly higher than values from the seizure onset ($P < 0.0001$). Several indices were also defined in order to correlate some clinical features to the degree of coupling between cortical structures and the thalamus. In patients with mesial TLE seizures, a correlation was found between the degree of thalamocortical synchrony and the presence of an early loss of consciousness but not with other clinical parameters. In addition, surgical prognosis seemed better in patients with low values of thalamocortical couplings at the seizure onset. This report demonstrates that the thalamus and remote cortical structures synchronize their activity during TLE seizures and suggest that the extension of the epileptogenic network to the thalamus is a potential important factor determining surgical prognosis.

Thalamus

Subcortical (thalamic) automated seizure detection: A new option for contingent therapy delivery

*Ivan Osorio, †Mark G. Frei, ‡Andres M. Lozano, and ‡Richard Wennberg

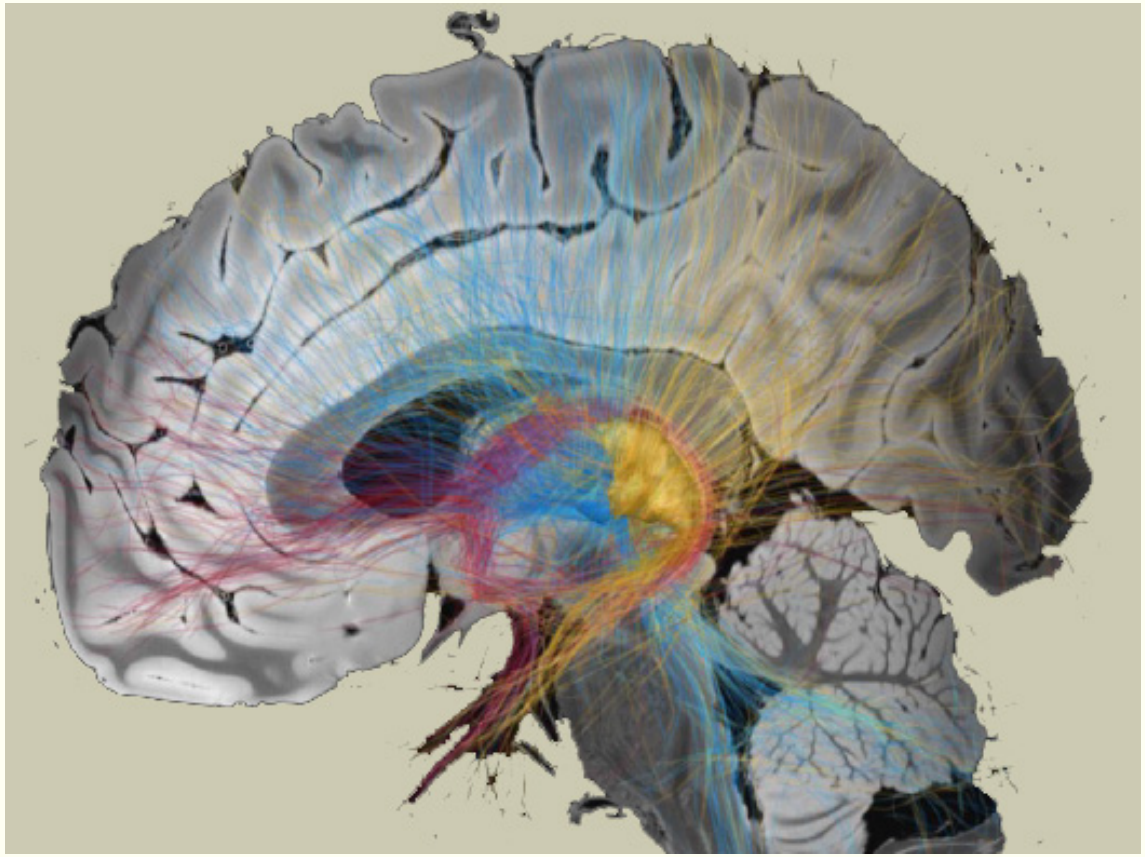
Epilepsia, 56(10):e156–e160, 2015
doi: 10.1111/epi.13124

SUMMARY

The feasibility of automated detection of cortical-onset epileptic seizures from subcortical structures such as the thalamus was investigated via simultaneous recording of electroencephalography (EEG) and anterior and centromedian thalamic nuclei electrical signals (electrothalamography) in nine subjects with pharmacoresistant seizures admitted to an epilepsy monitoring unit after deep brain stimulating electrode implantation. Thalamic electrical signals were analyzed using a validated seizure detection algorithm, and times of seizure onset and termination were compared to those determined through visual analysis of video-EEG. Ictal activity was recorded from the scalp and thalamic nuclei in three subjects who had seizures during the 3–4-day recording period. In the majority of seizures, ictal activity in the thalamic nuclei preceded electrographic onset as determined from the EEG or clinical onset as determined from behavioral observations. Interictal epileptiform discharges were also recorded from the thalamus and in certain instances had no scalp representation. Subcortical/thalamic detection of cortical-onset seizures is feasible. This approach would enable contingent therapy delivery and may be particularly valuable for subjects with multiple or difficult-to-localize epileptogenic regions.

KEY WORDS: Electrothalamography, EEG, Subcortical, Computerized seizure detection, Computerized electrical current delivery.

Thalamus, Recording & stimulating



Where to record and stimulate in the thalamus?

Thalamus, Recording & stimulating

Epilepsy & Behavior 112 (2020) 107354

Brain-responsive corticothalamic stimulation in the centromedian nucleus for the treatment of regional neocortical epilepsy

David E. Burdette [a,*], M. Ayman Haykal [a], Beata Jarosiewicz [b], Rachel R. Fabris [a], Gabe Heredia [a], Kost Elisevich [a], Sanjay E. Patra [a]

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ABSTRACT

Objective: The aim of the study was to determine if corticothalamic responsive stimulation targeting the centromedian nucleus of the thalamus (CMT) is a potential treatment for neocortical epilepsies with regional onsets.

Methods: We assessed efficacy and safety of CMT and neocortical responsive stimulation, detection, and stimulation programming, methods for implantation, and location and patterns of electrographic seizure onset and spread in 7 patients with medically intractable focal seizures with a regional neocortical onset.

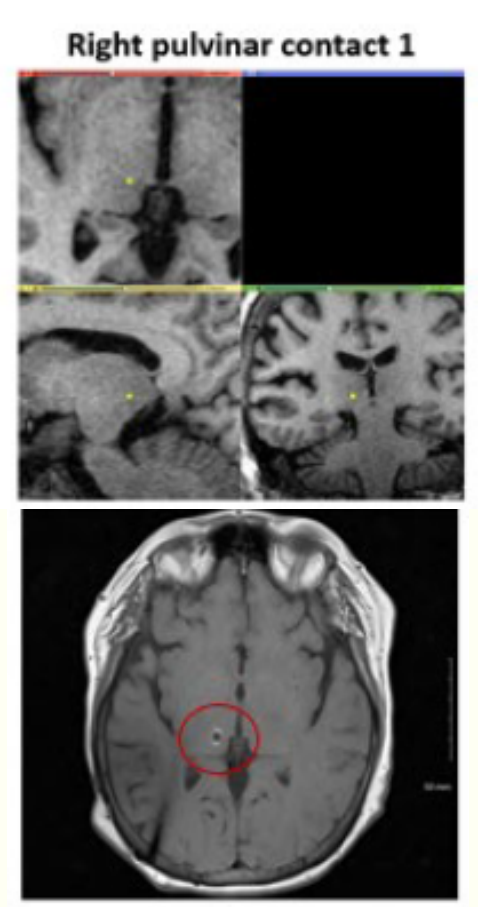
Results: The median follow-up duration was 17 months (average: 17 months, range: 8–28 months). The median % reduction in disabling seizures (excludes auras) in the 7 patients was 88% (mean: 80%, range: 55–100%). The median % reduction in all seizure types (disabling + auras) was 73% (mean: 67%, range: 15–94%). There were no adverse events related to implantation of the responsive neurostimulator and leads or related to the delivery of responsive stimulation. Stimulation-related contralateral paresthesias were addressed by adjusting stimulation parameters in the clinic during stimulation testing.

Electrographic seizures were detected in the CMT and neocortex in all seven patients. Four patients had simultaneous or near simultaneous seizure onsets in the neocortex and CMT and three had onsets in the neocortex with spread to the CMT.

Conclusion: In this small series of patients with medically intractable focal seizures and regional neocortical onset, responsive neurostimulation to the neocortex and CMT improved seizure control and was well tolerated.

Significance: Responsive corticothalamic neurostimulation of the CMT and neocortex is a potential treatment for patients with regional neocortical epilepsies.

Thalamus - Pulvinar



Brain-responsive corticothalamic stimulation in the pulvinar nucleus for the treatment of regional neocortical epilepsy: A case series

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Abstract

Drug-resistant focal epilepsy with regional neocortical seizure onsets originating from the posterior quadrant can be particularly difficult to treat with resective surgery due to the overlap with eloquent cortex. Published reports indicate that corticothalamic treatment targeting the anterior or centromedian nucleus of the thalamus with direct brain-responsive stimulation may be an effective approach to treat regional neocortical epilepsy. The pulvinar has remained largely unstudied as a neurostimulation target to treat refractory epilepsy. Because the pulvinar has connections with the posterior quadrant, neurostimulation may be effective if applied to seizures originating in this area. We performed a retrospective chart review of patients with regional neocortical seizure onsets in the posterior quadrant treated with the RNS System. Demographics, epilepsy history, clinical seizure frequencies, and neuropsychological testing results were obtained from the chart. Electrocorticogram (ECoG) records stored by the RNS System were reviewed to evaluate electrographic seizure onset patterns. Our patients were followed for 10, 12.5, and 15 months. All patients were responders (≥50% seizure reduction), and two of the three patients experienced a ≥90% reduction in seizures at the last follow-up. Pre- and postsurgical neuropsychological evaluations were compared for two of the patients, and there was no evidence of cognitive decline found in either patient. Interestingly, mild cognitive improvements were reported. The third patient had only postimplant neuropsychological testing data available. Findings for this patient suggested executive dysfunction that was present prior to the RNS System which did not worsen with surgery. A visual inspection of ECoGs revealed near-simultaneous seizure onsets in neocortical and pulvinar leads in two patients. Seizure onsets in the third patient were more variable. This is the first published report of brain-responsive neurostimulation targeting the pulvinar to treat refractory regional onset epilepsy of posterior quadrant origin.

Thalamus, Connectivity

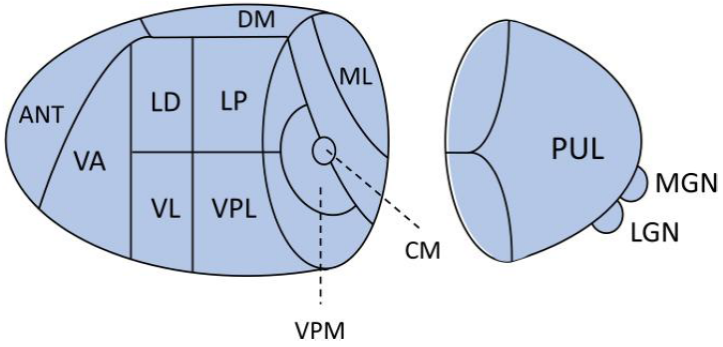

Figure 1 Thalamic structure, connectivity, and neuromodulation. Diagram of major thalamic nuclei. ANT = anterior nucleus of thalamus; CM = centromedian nucleus; DM = dorsomedial nucleus; LD = lateral dorsal; LGN = lateral geniculate nucleus; LP = lateral posterior; MGN = medial geniculate nucleus; ML = midline nuclei; PUL = pulvinar; VA = ventral anterior; VL = ventrolateral; VPL = ventral posteriolateral; VPM = ventral posteriomedial.

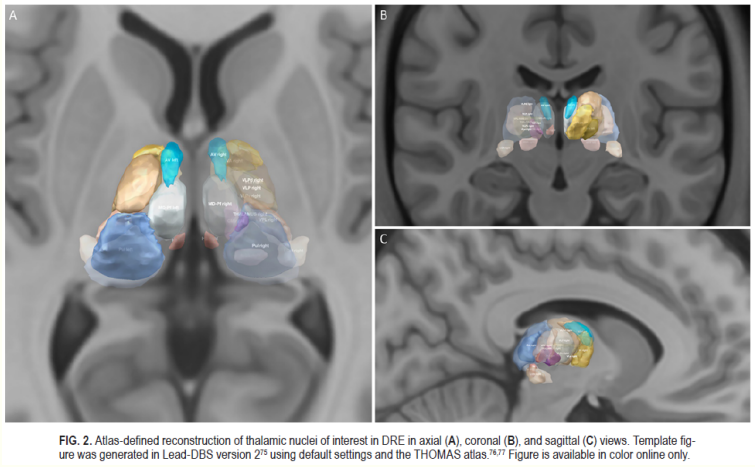

FIG. 2. Atlas-defined reconstruction of thalamic nuclei of interest in DRE in axial (**A**), coronal (**B**), and sagittal (**C**) views. Template figure was generated in Lead-DBS version 2[73] using default settings and the THOMAS atlas.[76,77] Figure is available in color online only.

Gadot et al., 2022

Table 1 Connectivity of selected thalamic nuclei and potential uses of neurostimulation

Nucleus	Connectivity	Seizures treated
ANT	Frontal lobe, temporal lobe, hippocampus	Focal onset seizures with or without generalization
CM	Striatum, sensorimotor cortex, premotor cortex, cingulate	1° generalized seizures or multifocal seizures
DM	Globus pallidus, amygdala, prefrontal cortex	None yet
PUL	Visual system, amygdala, hippocampus, temporal lobe, cingulate, orbitofrontal cortex	Posterior quadrant focal epilepsy

ANT = anterior nucleus of thalamus; CM = centromedian nucleus; DM = dorsomedial nucleus; PUL = pulvinar.

Bernabei et el., Brain, 2023

Thalamus, Recording & stimulating

Thalamic stereoelectroencephalography in epilepsy surgery: a scoping literature review

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OBJECTIVE Stereoelectroencephalography (sEEG) is a well-established surgical method for defining the epileptogenic network. Traditionally reserved for identifying discrete cortical regions for resection or ablation, sEEG in current practice is also used for identifying more broadly involved subcortical epileptic network components, driven by the availability of brain-based neuromodulation strategies. In particular, sEEG investigations including thalamic nuclei are becoming more frequent in parallel with the increase in therapeutic strategies involving thalamic targets such as deep brain stimulation (DBS) and responsive neurostimulation (RNS). The objective to this study was to evaluate existing evidence and trends regarding the purpose, techniques, and relevant electrographic findings of thalamic sEEG.

METHODS MEDLINE and Embase databases were systematically queried for eligible peer-reviewed studies involving sEEG electrode implantation into thalamic nuclei of patients with epilepsy. Available data were abstracted concerning preoperative workup and purpose for implanting the thalamus, thalamic targets and trajectories, and electrophysiological methodology and findings.

RESULTS sEEG investigations have included thalamic targets for both basic and clinical research purposes. Medial pulvinar, dorsomedial, anterior, and centromedian nuclei have been the most frequently studied. Few studies have reported any complications with thalamic sEEG implantation, and no studies have reported long-term complications. Various methods have been utilized to characterize thalamic activity in epileptic disorders including evoked potentials, power spectrograms, synchronization indices, and the epileptogenicity index. Thalamic intracranial recordings are beginning to be used to guide neuromodulation strategies including RNS and DBS, as well as to understand complex, network-dependent seizure disorders.

CONCLUSIONS Inclusion of thalamic coverage during sEEG evaluation in drug-resistant epilepsy is a growing practice and is amenable to various methods of electrographic data analysis. Further study is required to establish well-defined criteria for thalamic implantation during invasive investigations as well as safety and ethical considerations.

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KEYWORDS thalamus; stereoelectroencephalography; sEEG; epilepsy; invasive EEG; drug resistant

Thalamus, Recording & stimulating

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Multisite thalamic recordings to characterize seizure propagation in the human brain

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Neuromodulation of the anterior nuclei of the thalamus (ANT) has shown to be efficacious in a subset of patients with refractory focal epilepsy. One important uncertainty is to what extent thalamic subregions other than the ANT could be recruited more prominently in the propagation of focal onset seizures. We designed the current study to simultaneously monitor the engagement of the ANT, mediodorsal (MD) and pulvinar (PUL) nuclei during seizures in patients who could be candidates for thalamic neuromodulation.

We studied 11 patients with clinical manifestations of presumed temporal lobe epilepsy (TLE) undergoing invasive stereo-encephalography (sEEG) monitoring to confirm the source of their seizures. We extended cortical electrodes to reach the ANT, MD and PUL nuclei of the thalamus. More than one thalamic subdivision was simultaneously interrogated in nine patients. We recorded seizures with implanted electrodes across various regions of the brain and documented seizure onset zones (SOZ) in each recorded seizure. We visually identified the first thalamic subregion to be involved in seizure propagation. Additionally, in eight patients, we applied repeated single pulse electrical stimulation in each SOZ and recorded the time and prominence of evoked responses across the implanted thalamic regions.

Our approach for multisite thalamic sampling was safe and caused no adverse events. Intracranial EEG recordings confirmed SOZ in medial temporal lobe, insula, orbitofrontal and temporal neocortical sites, highlighting the importance of invasive monitoring for accurate localization of SOZs. In all patients, seizures with the same propagation network and originating from the same SOZ involved the same thalamic subregion, with a stereotyped thalamic EEG signature. Qualitative visual reviews of ictal EEGs were largely consistent with the quantitative analysis of the corticothalamic evoked potentials, and both documented that thalamic nuclei other than ANT could have the earliest participation in seizure propagation. Specifically, pulvinar nuclei were involved earlier and more prominently than ANT in more than half of the patients. However, which specific thalamic subregion first demonstrated ictal activity could not be reliably predicted based on clinical semiology or lobar localization of SOZs.

Our findings document the feasibility and safety of bilateral multisite sampling from the human thalamus. This may allow more personalized thalamic targets to be identified for neuromodulation. Future studies are needed to determine if a personalized thalamic neuromodulation leads to greater improvements in clinical outcome.

Thalamus, Indications: Recording/Stimulating

- **Enough evidence to routinely implanting the thalamus?**
- Thalamus itself not a seizure onset zone in focal epilepsy
  - It has not been part of standard SEEG implants
- No class 1 evidence to support Routine diagnostic thalamic implantation
  - Strong evidence for electrical stimulation of ANT to reduce seizures
- Growing evidence of seizure spread to thalamic nuclei but no evidence of therapeutic stimulation of these nuclei
- Hypothesis driven implant

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Thalamus, Indications: Recording/Stimulating

- **Non-localizable / poorly localized seizures:**
  - Scalp EEG, MRI, or PET findings non-localizing, or suggestive of multifocal seizures
- **Specific seizure semiologies:**
  - Gelastic seizures (hypothalamic hamartomas)
  - Focal cortical dysplasia (frontal, temporal, thalamus)
- **Lennox-Gastaut Syndrome/other diffuse encephalopathies:**
  - Multiple seizure types
  - Diffuse EEG abnormalities
- **Limbic system involvement:**
  - Thalamus is a critical relay in the limbic circuit