

EEG E STEREO-EEG

LAURA TASSI – CENTRO CHIRURGIA EPILESSIA «CLAUDIO MUNARI»

ASST GOM NIGUARDA - MILANO

Lunedì 25 ottobre

Moderatori:

Adriana Magaudda (Messina) – Mario Zappia (Catania)

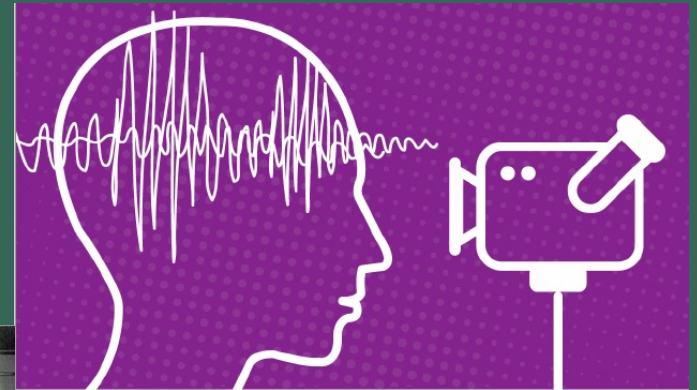
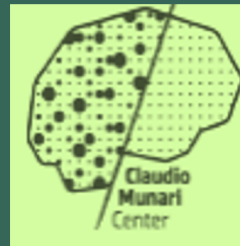
09:00 – 10:00 Manifestazioni ed EEG nel bambino
Nicola Specchio (Roma)

10:00 – 11:00 Manifestazioni ed EEG nell'adulto
Stefano Meletti (Modena)

11:00 – 11:20 *Pausa*

11:20 – 12:20 Video session
Giuseppe d'Orsi (Andria)

12:20 – 13:20 EEG e Stereo-EEG
Laura Tassi (Milano)



**CORSO VIDEO EEG LICE
3° EDIZIONE
CATANIA, 24-27 OTTOBRE 2021**



Stereo-EEG

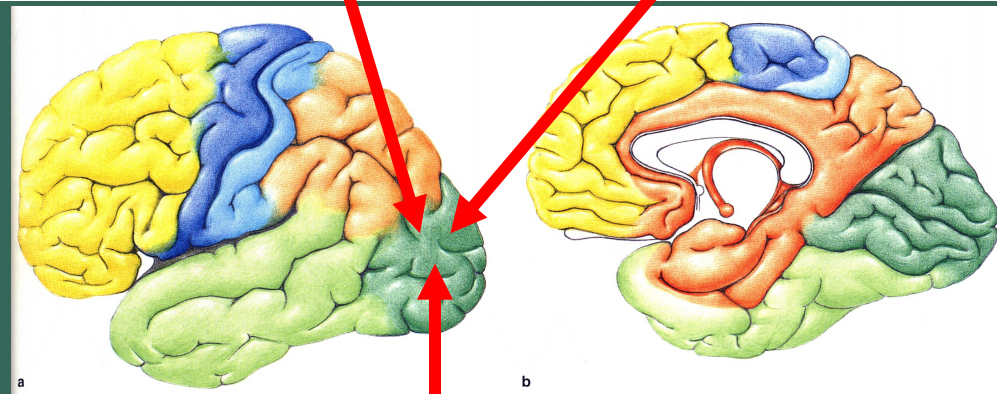
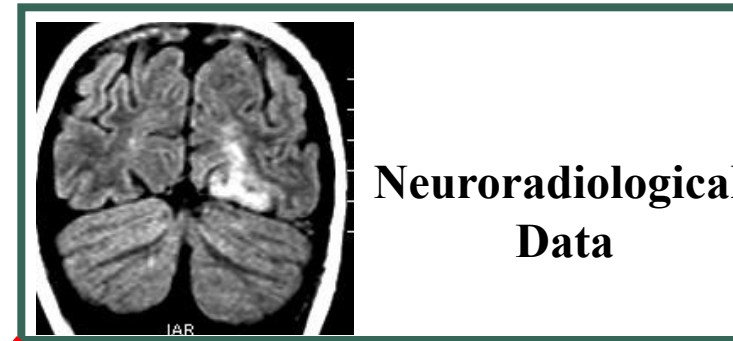
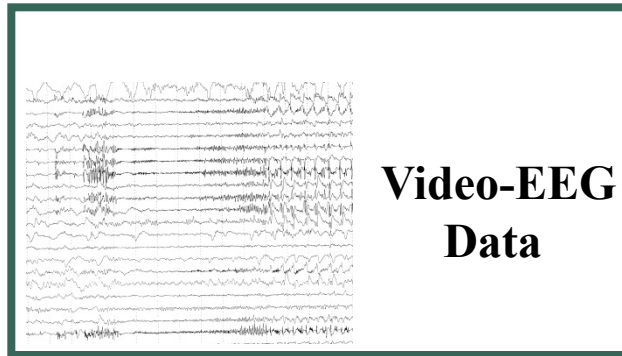
EPILEPTOGENIC ZONE

**Defined on the basis
of anatomo-electro-
clinical correlations**

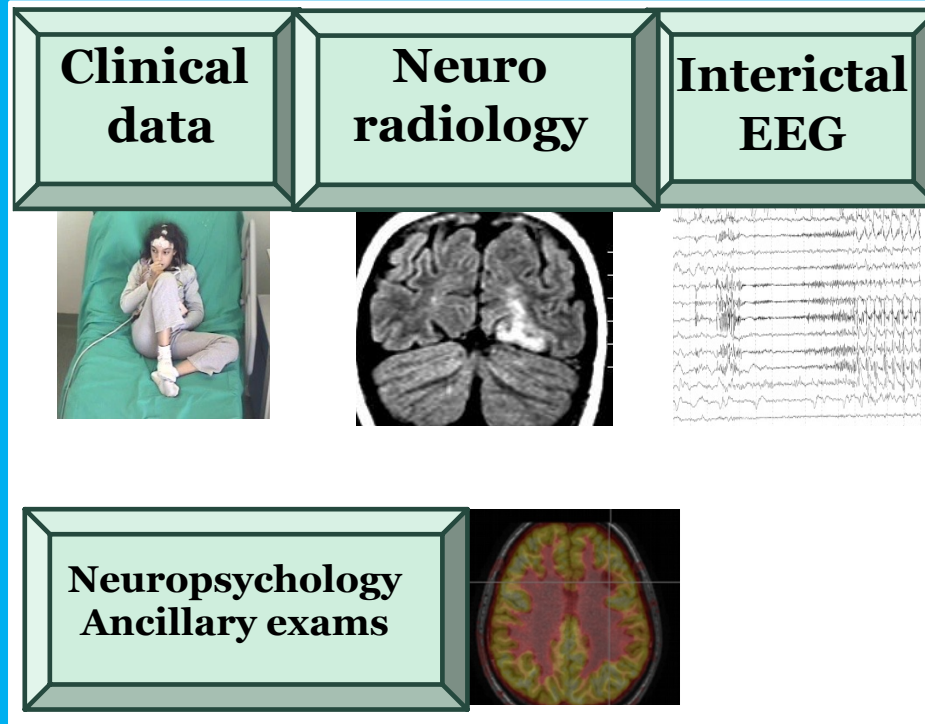
Cortical area/areas:

- in which ictal discharges originate?
- whose removal cure the patient?
- anatomical lesion?
- symptomatogenic zone?

Anatomo-electro-clinical Correlations



The Patient



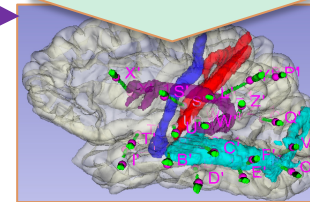
VEEG

45%

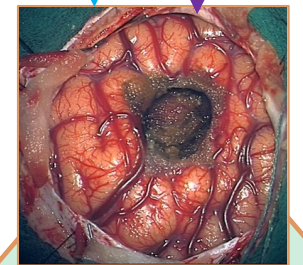


iEEG

35%



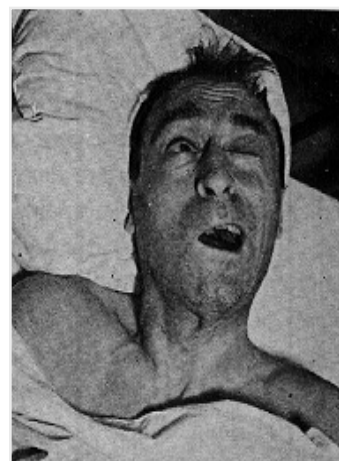
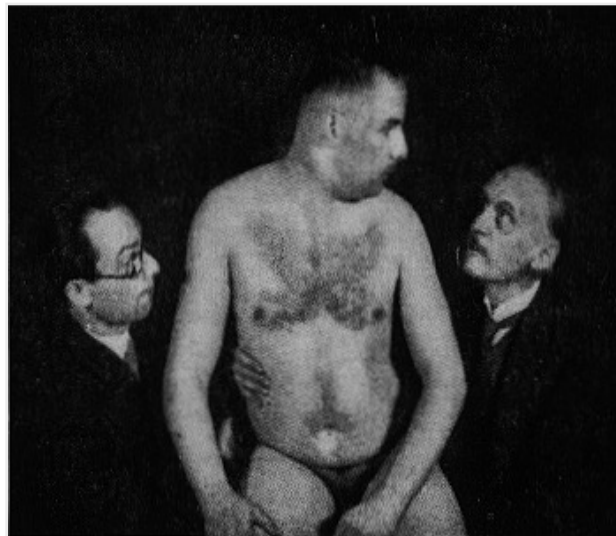
20%



Surgery

**Epilepsy Surgery:
presurgical workflow**

Once upon a time...





**Lesional Area: slow waves,
background activity absent**



**Irritative Area: spikes, spikes and
waves**



**Functional Area: electrical
stimulations**

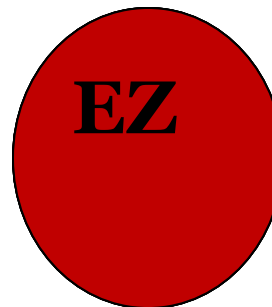
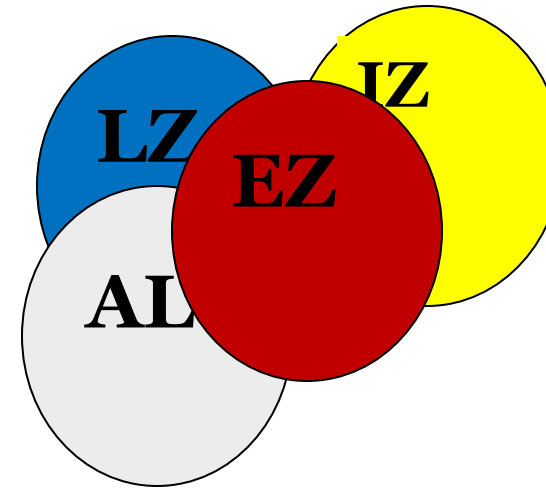
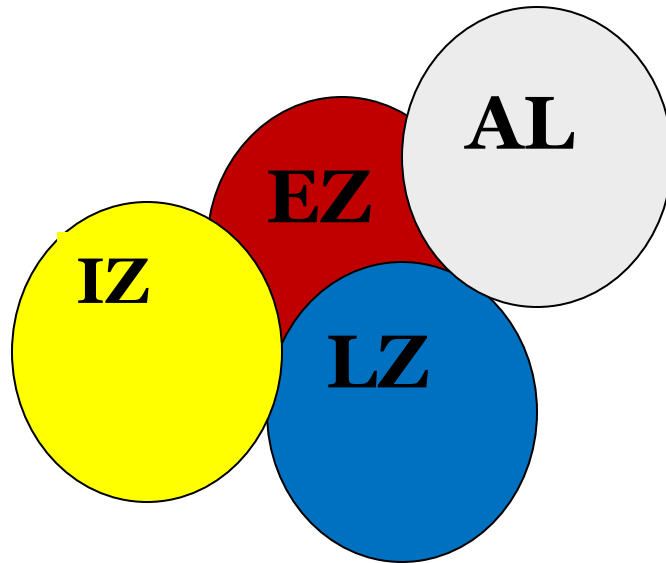


**Epileptogenic
Area**

Interictal paroxysms
Spontaneous seizures
Electrical stimulations
.....

Stereo-EEG Analysis and interpretation

EZ and invasive recordings: how many regions?



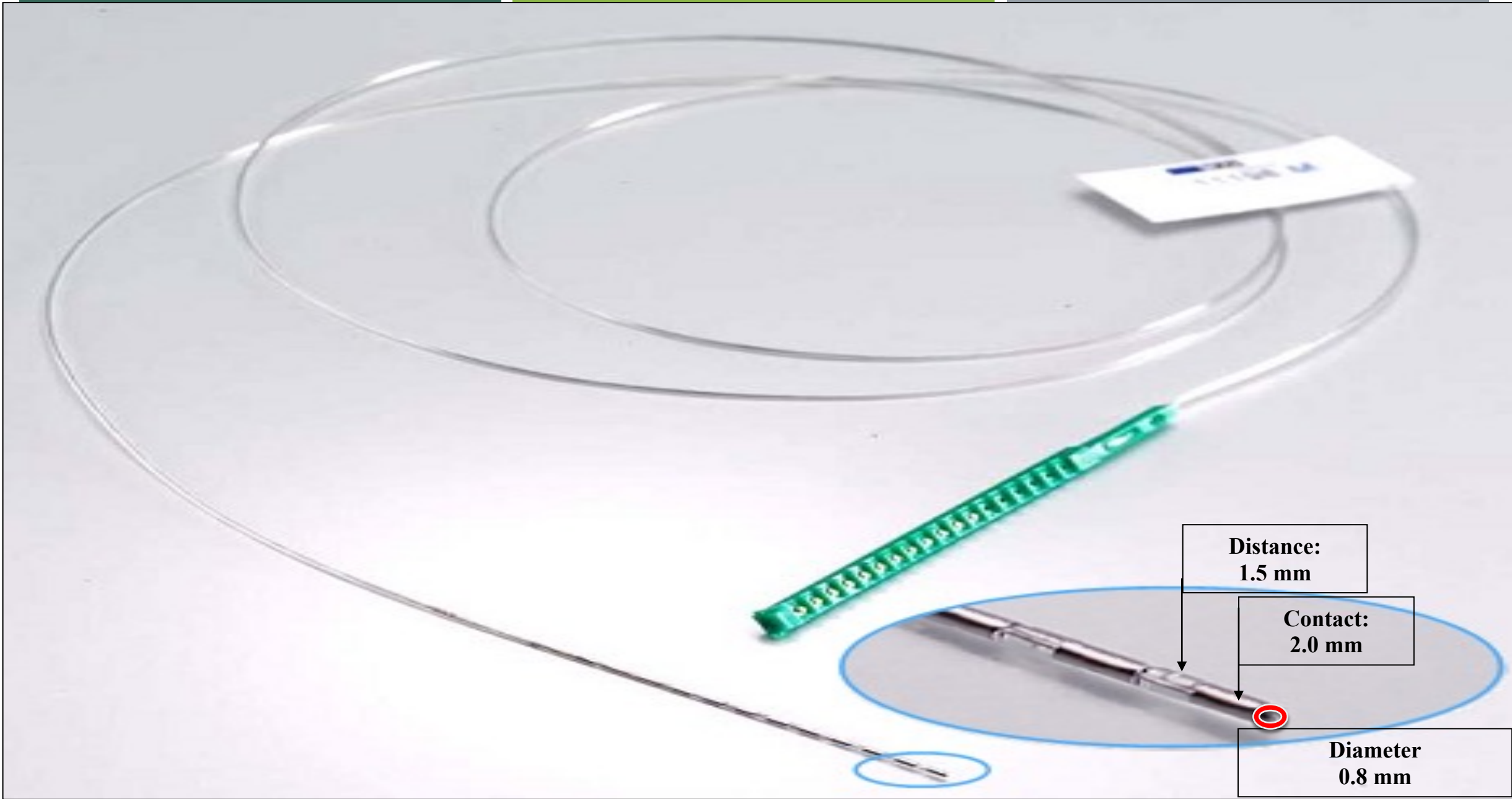
AL: anatomical lesion
LZ: lesional zone
IZ: irritative zone
EZ: epileptogenic zone

Indications and utility of Stereo-EEG

- discrepancies between:
 - anatomical and clinical data;
 - anatomical and electrical data;
 - electrical and clinical data;
- supposed bi- or multi-lobar ictal discharges;
- early ictal involvement of functional cortical areas (suggested by the clinical semiology);



1. Individually tailored surgical removal
2. THC: Thermocoagulation



**Distance:
1.5 mm**

**Contact:
2.0 mm**

**Diameter
0.8 mm**

Concerning the observation of an electrical potential at a distance from an intracranial electrode contact

Hitten P. Zaveri, Robert B. Duckrow, Susan S. Spencer

Clinical Neurophysiology 120 (2009) 1873–1875

The source???

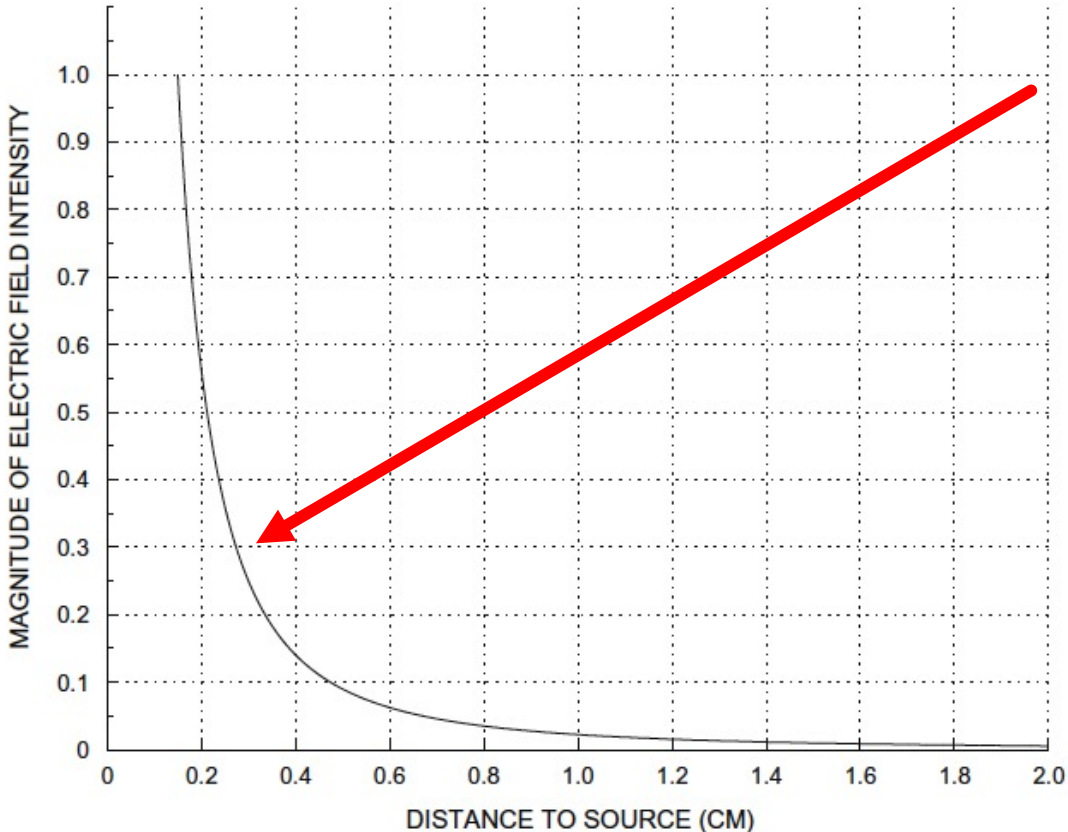
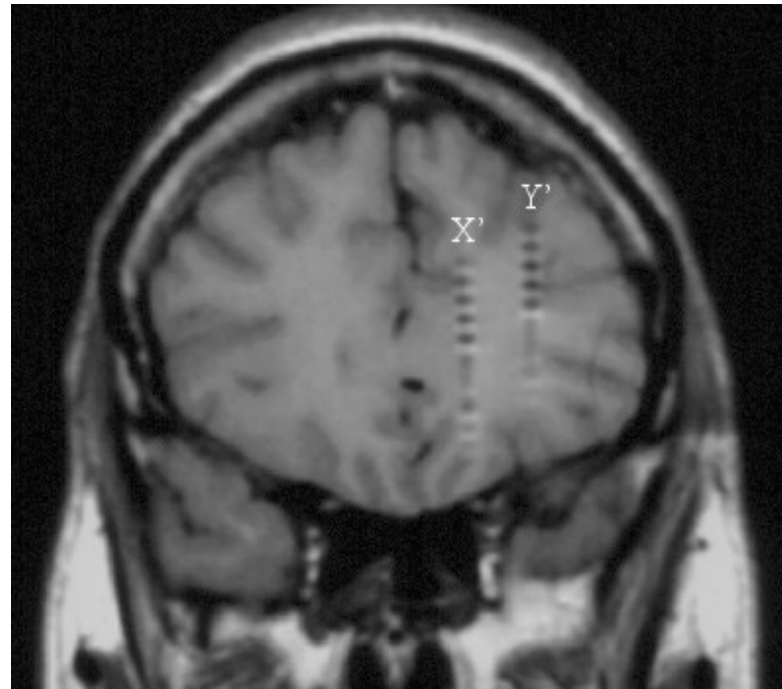
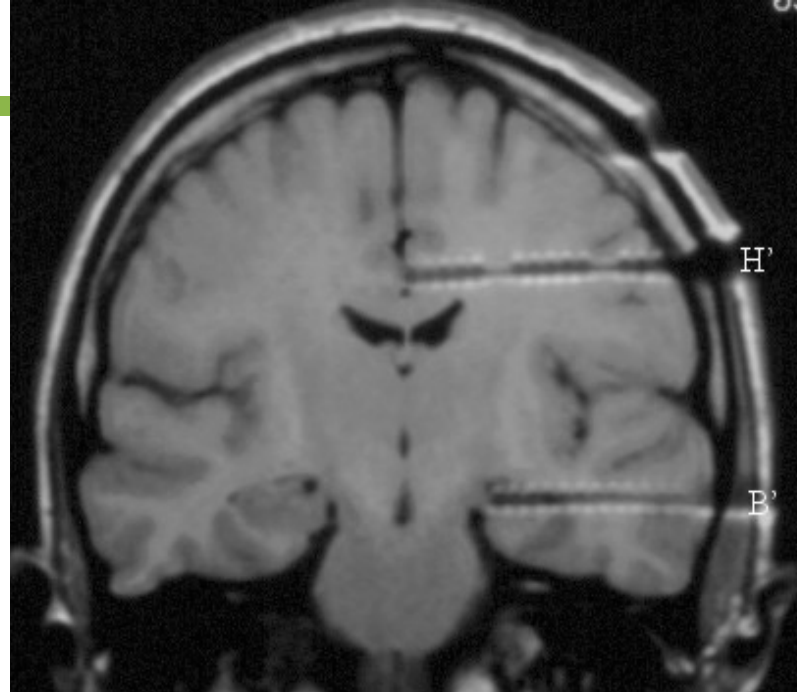
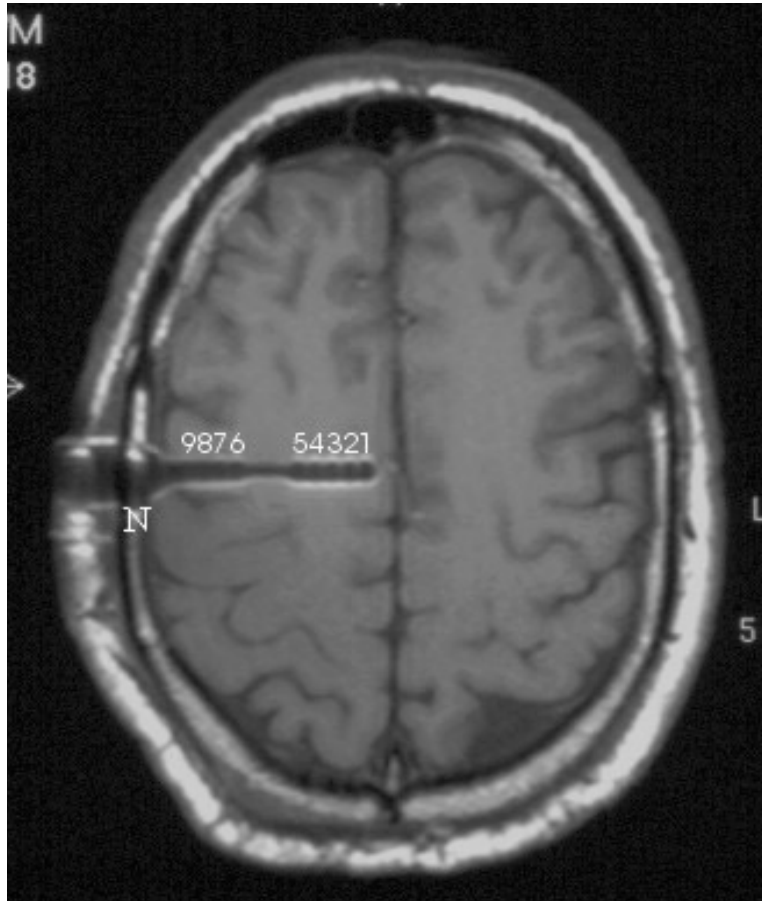
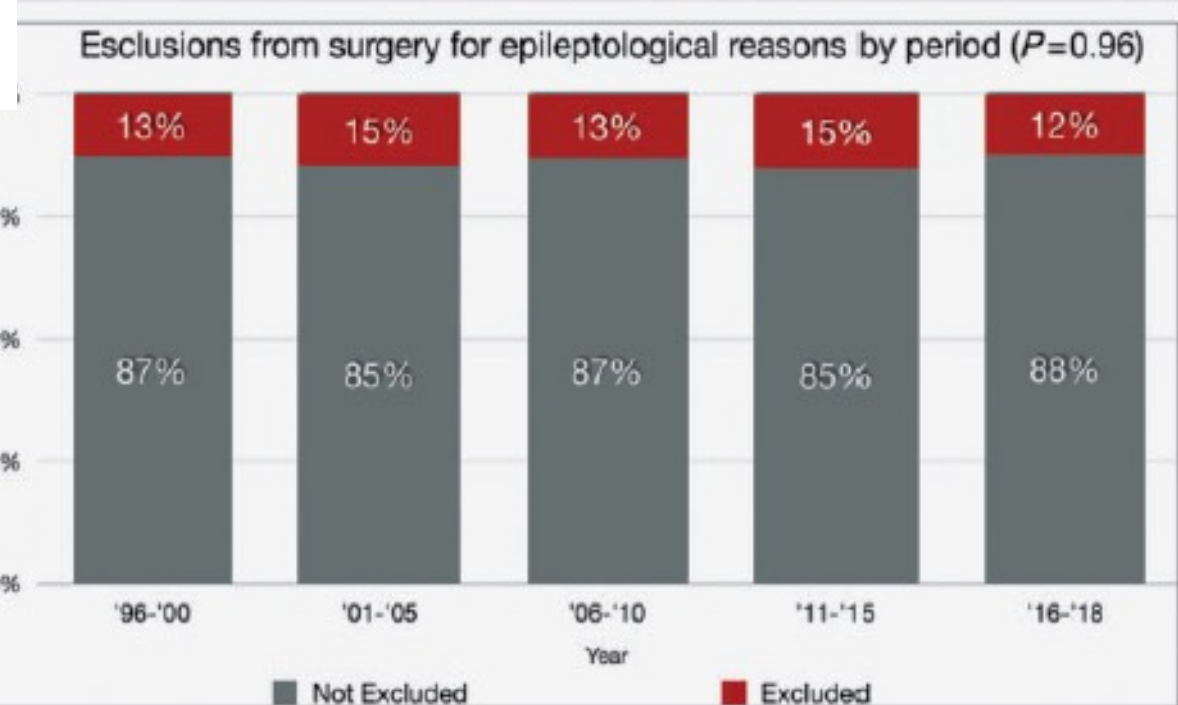
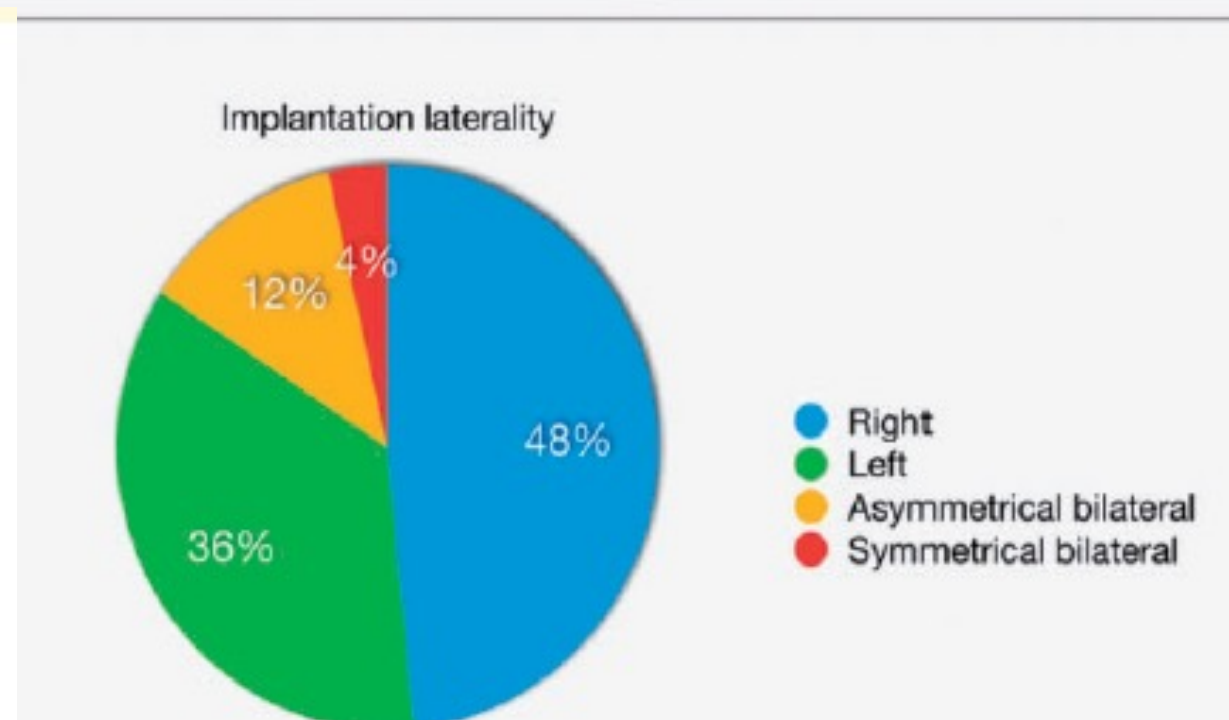
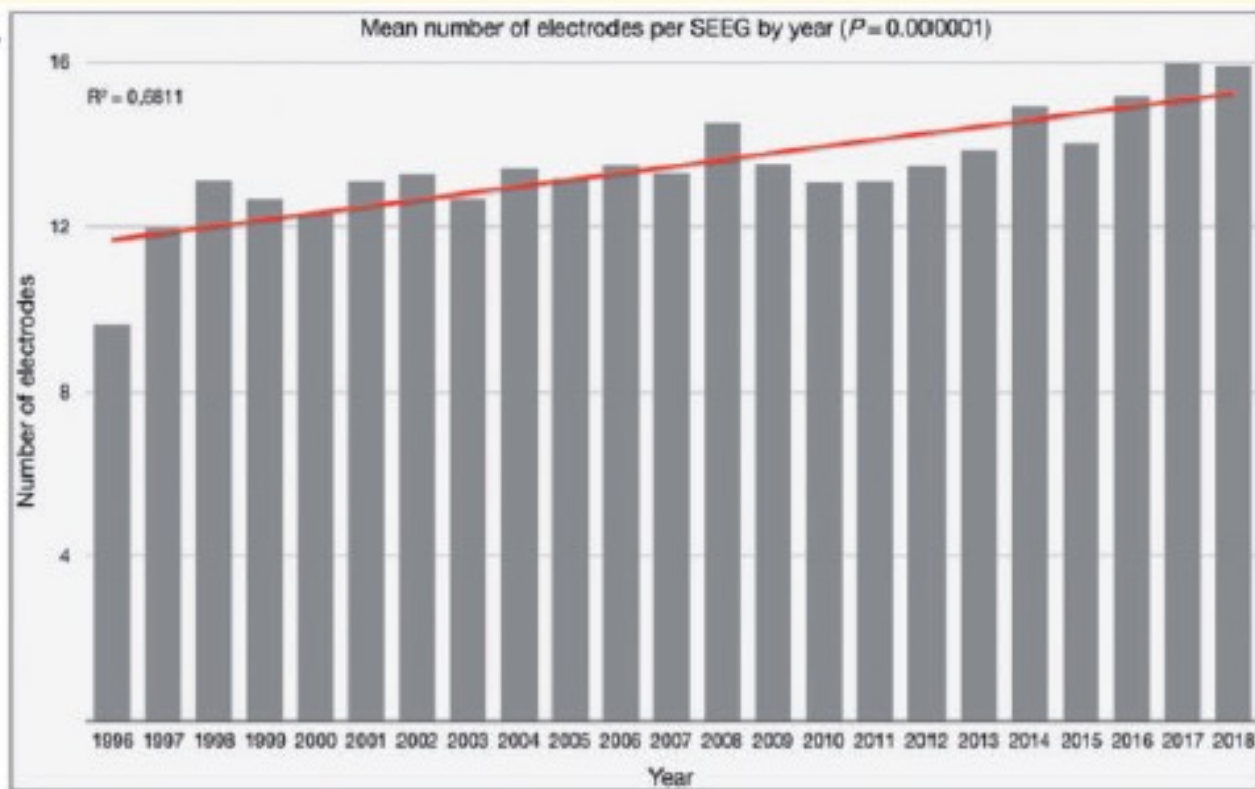


Fig. 1. The magnitude of the electric field intensity as a function of distance from a point charge. In this evaluation a charge is assumed to result in an electric field intensity of magnitude 1 at a distance of 0.15 cm. The magnitude of the electric field intensity decreases dramatically as a function of distance; the decrease in the magnitude of the electric field intensity is specified by the inverse-square law.



A



Brain 2019, Cardinale et al.

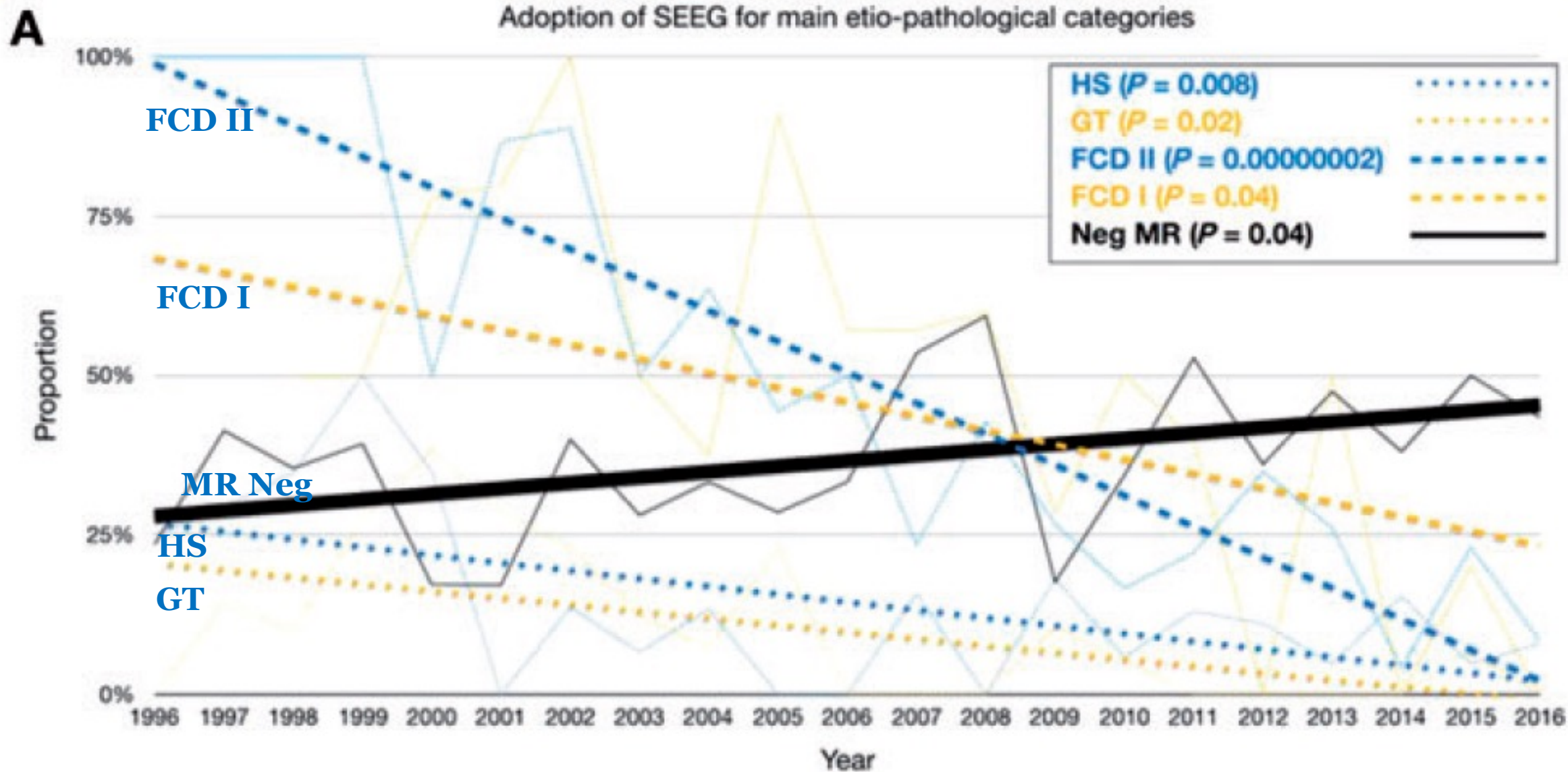
HS: hippocampal sclerosis

GT: glioneuronal tumors

FCD II: Focal Cortical Dysplasia Type II

FCD I: Focal Cortical Dysplasia Type I

Brain 2019, Cardinale et al.



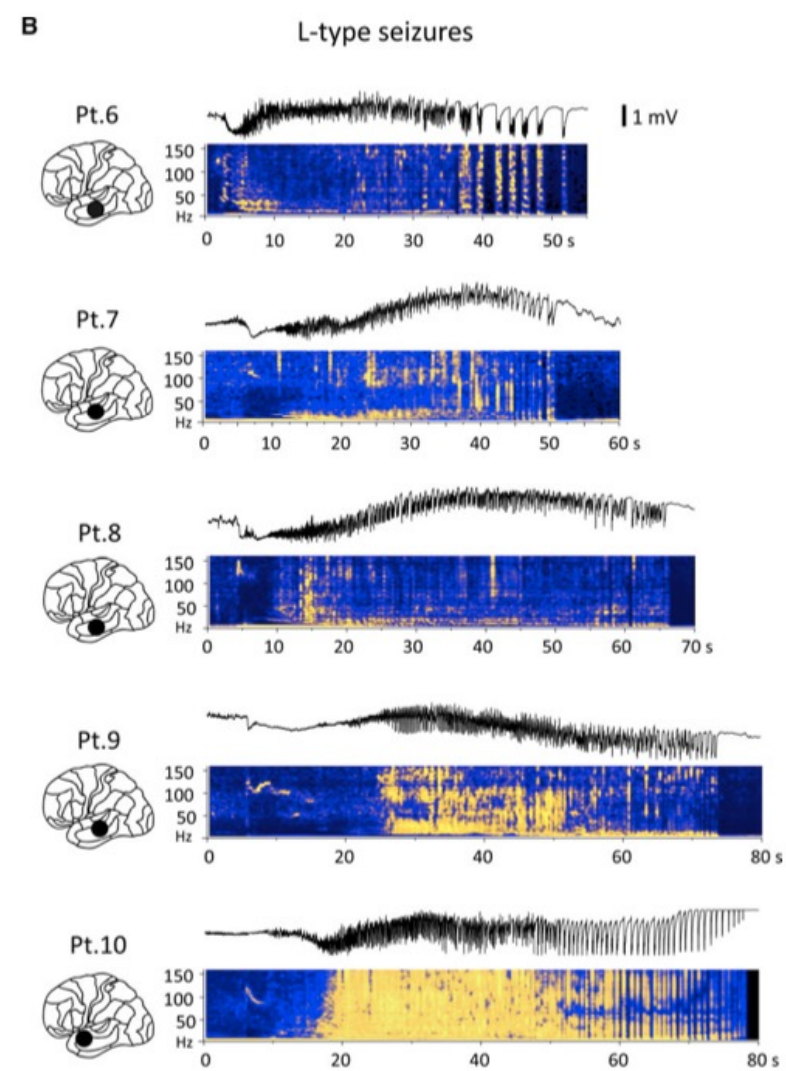
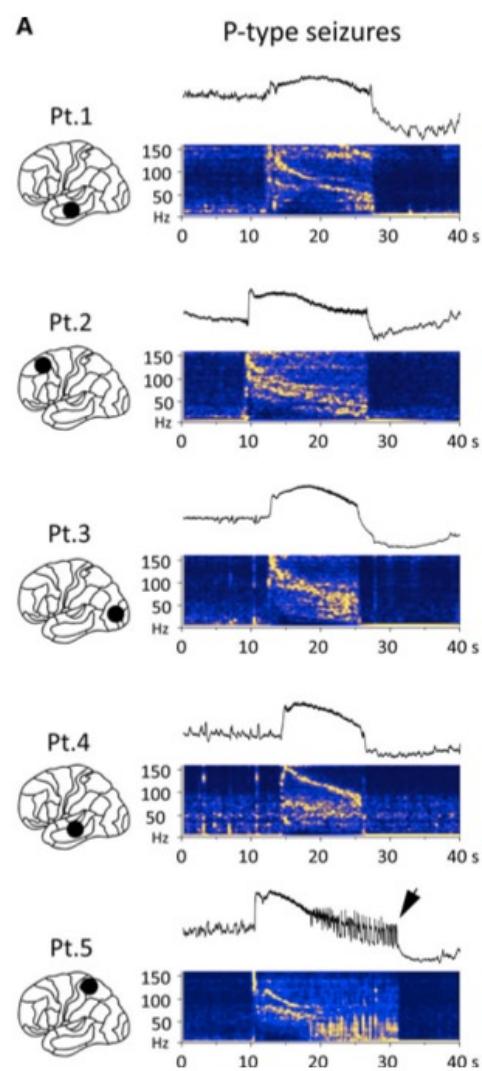
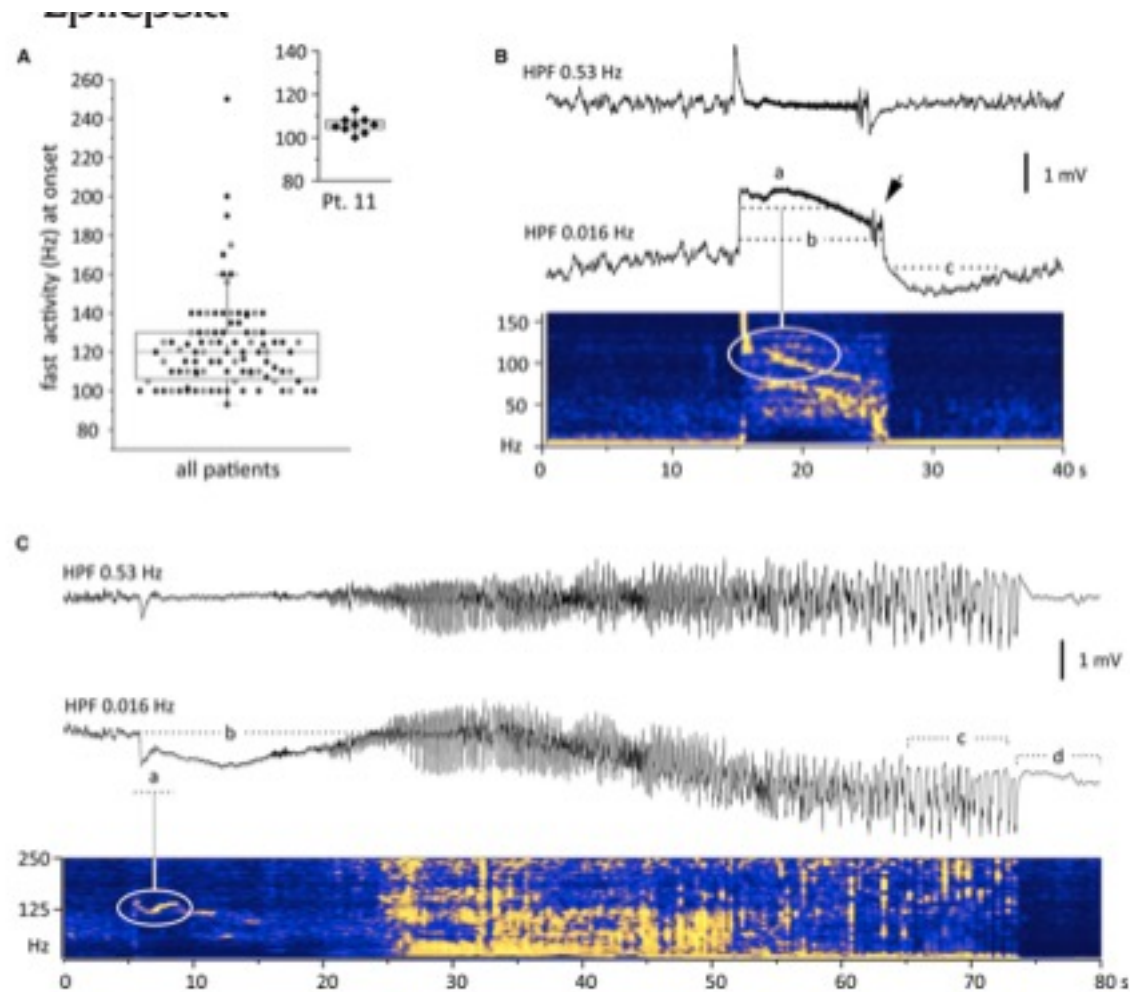
What did we learn over
the past 30 years?

HINTS

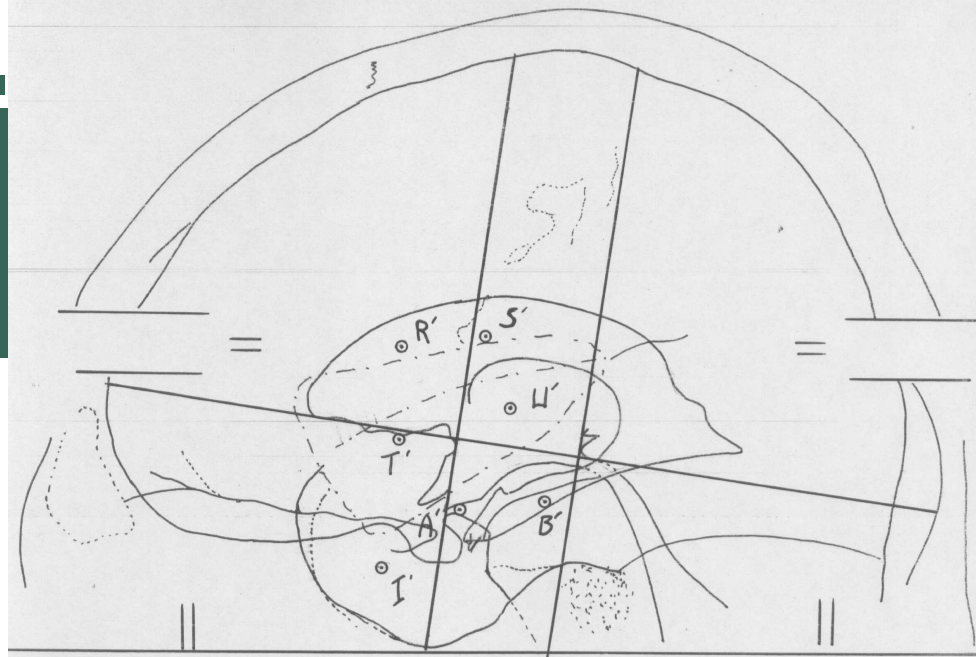
1. **Not all patients** need invasive recordings (HS, FCD II, tumors)
2. The **temporal lobe** is different from all other lobes
3. Always look for the **low voltage fast activity**
4. **Archicortex** not able to produce LVFA (Hippocampus, Amygdala, Cingulum)
5. Better to think in terms of **networks** than cortical structures
6. Need to know the **actual position** of the electrodes
7. **Intracerebral stimulations** are essential for the mapping and definition of EZ
8. Some etiologies, however, **require invasive recordings** (PMG, PNH, scars)
9. **Thermocoagulation** is useful for healing but also for prognosis

Two main focal seizure patterns revealed by intracerebral electroencephalographic biomarker analysis

Vadym Gnatkovsky¹ | Veronica Pelliccia² | Marco de Curtis¹ | Laura Tassi²



The past

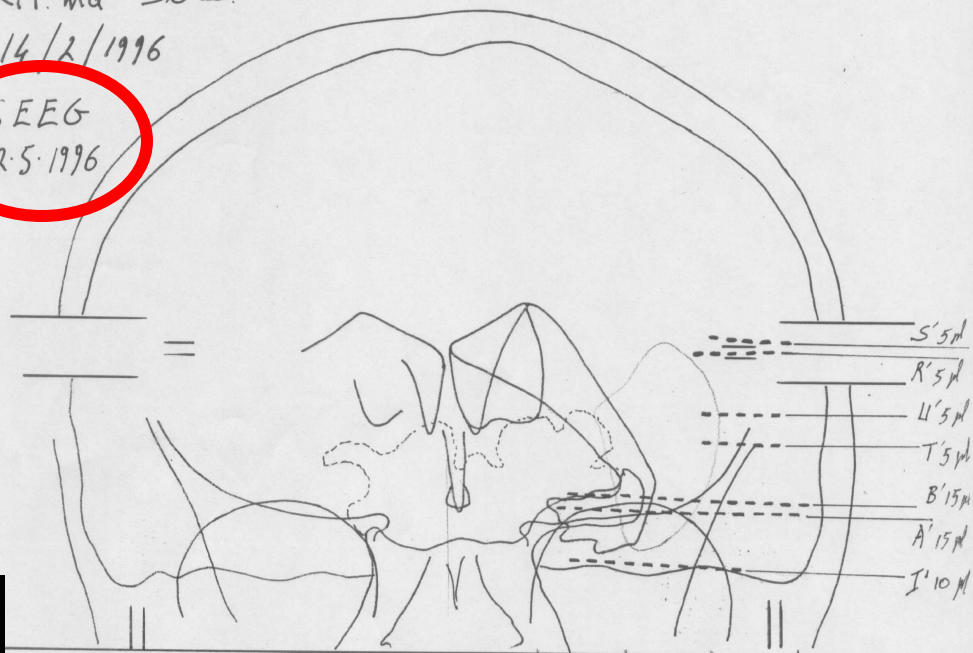


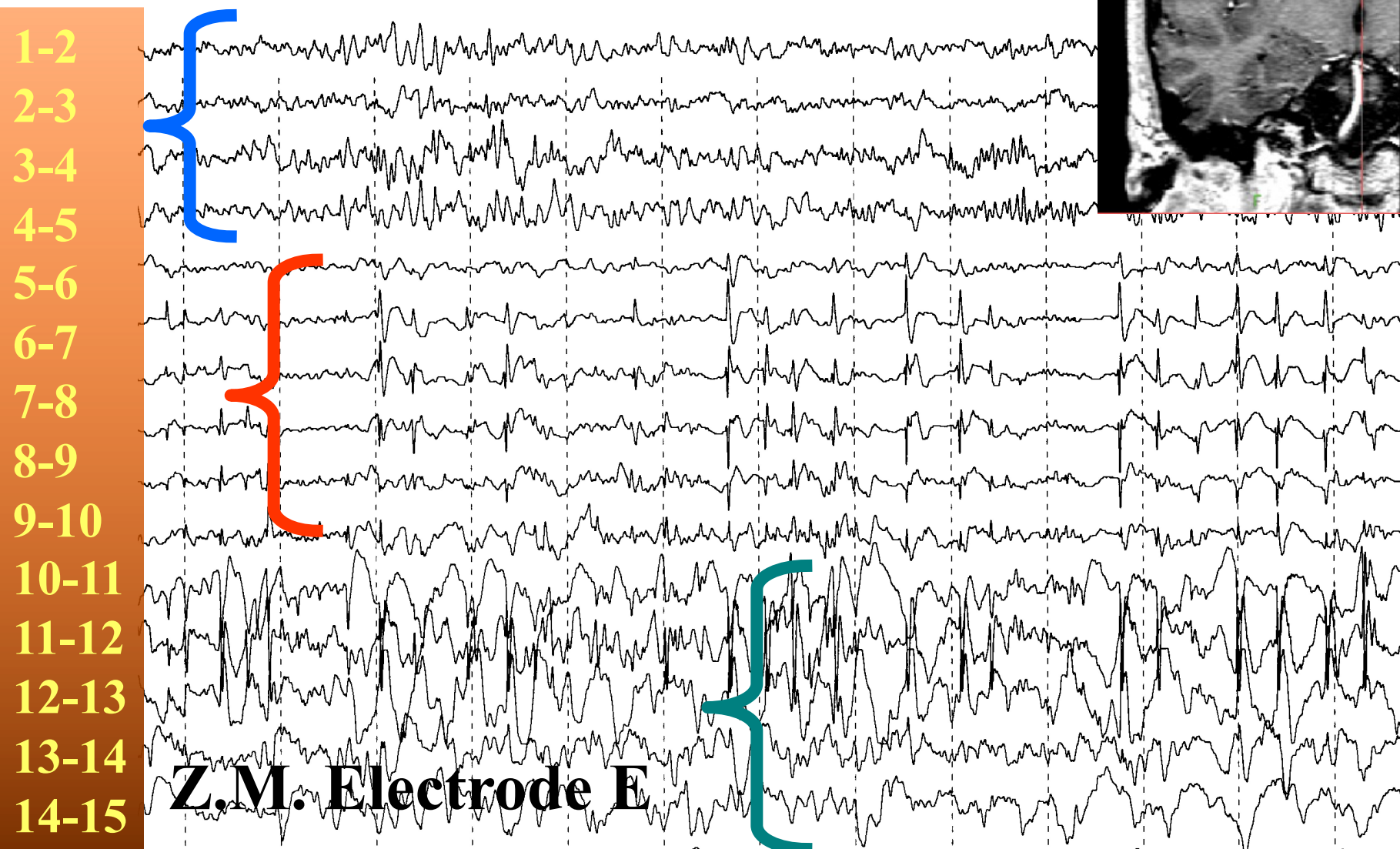
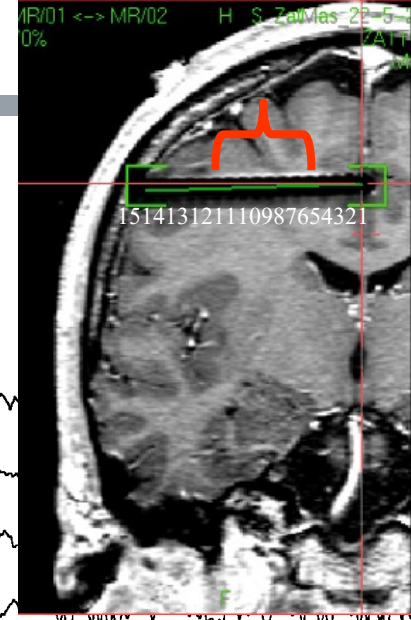
ZA. ma 36^{oo}.

14/2/1996

SEEG

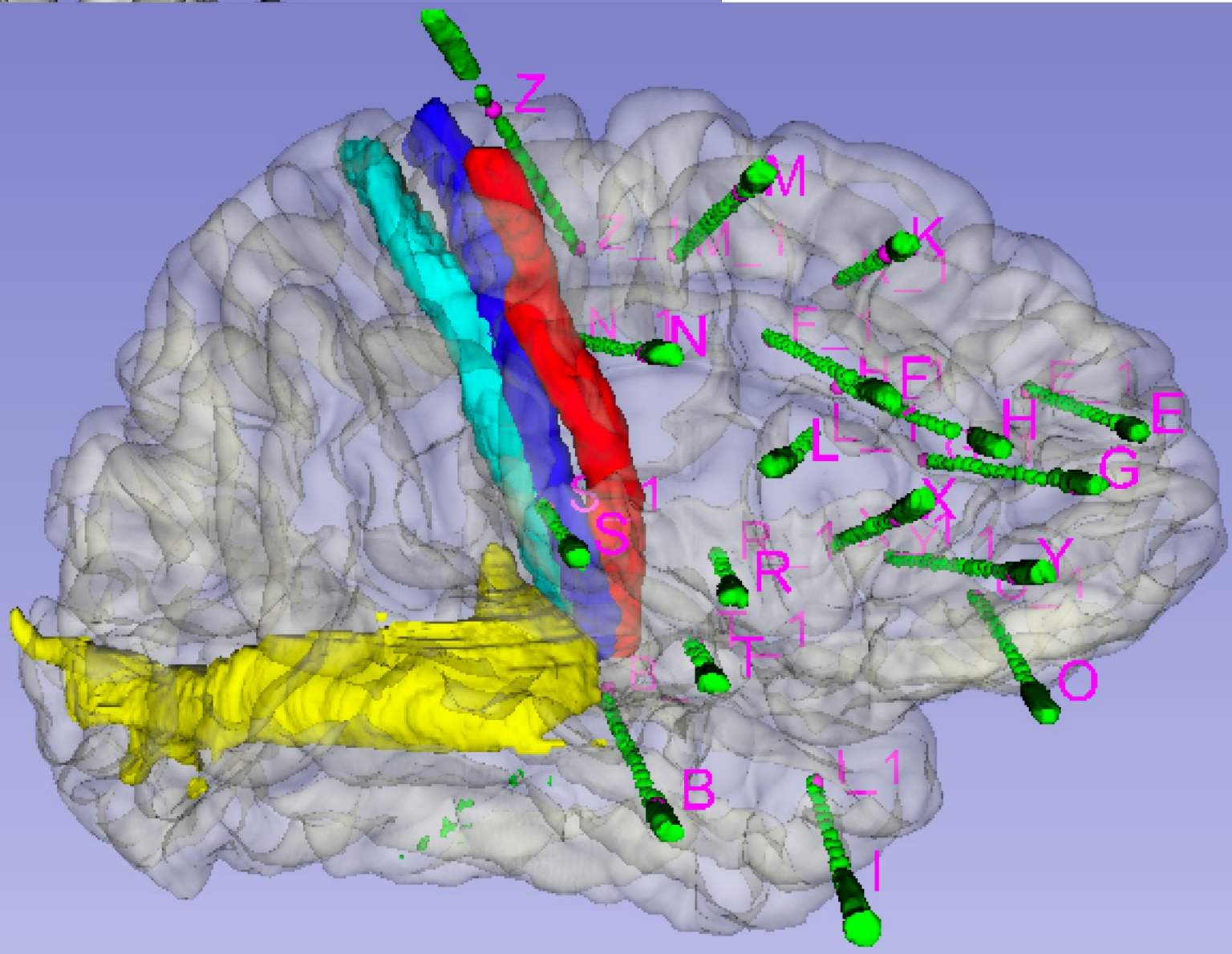
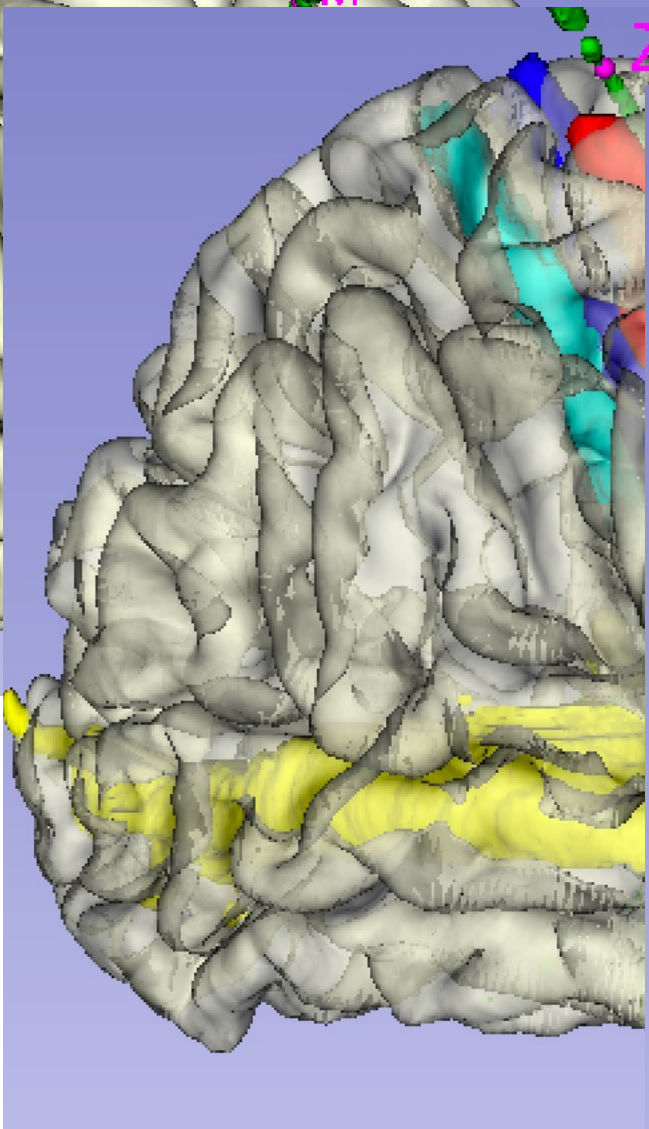
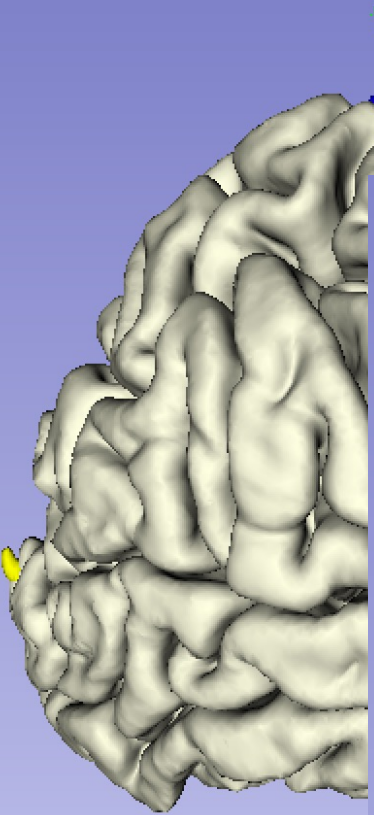
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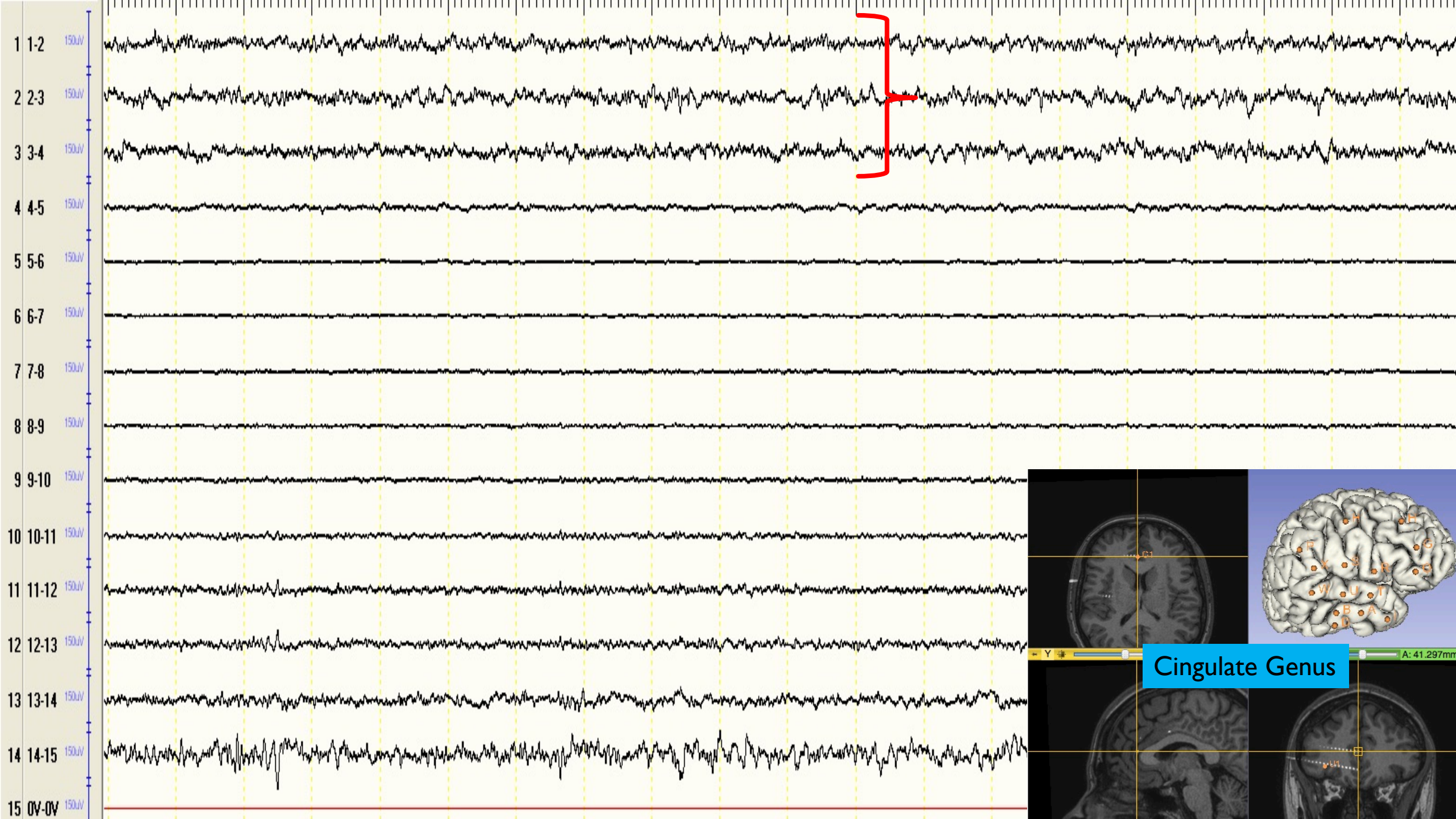
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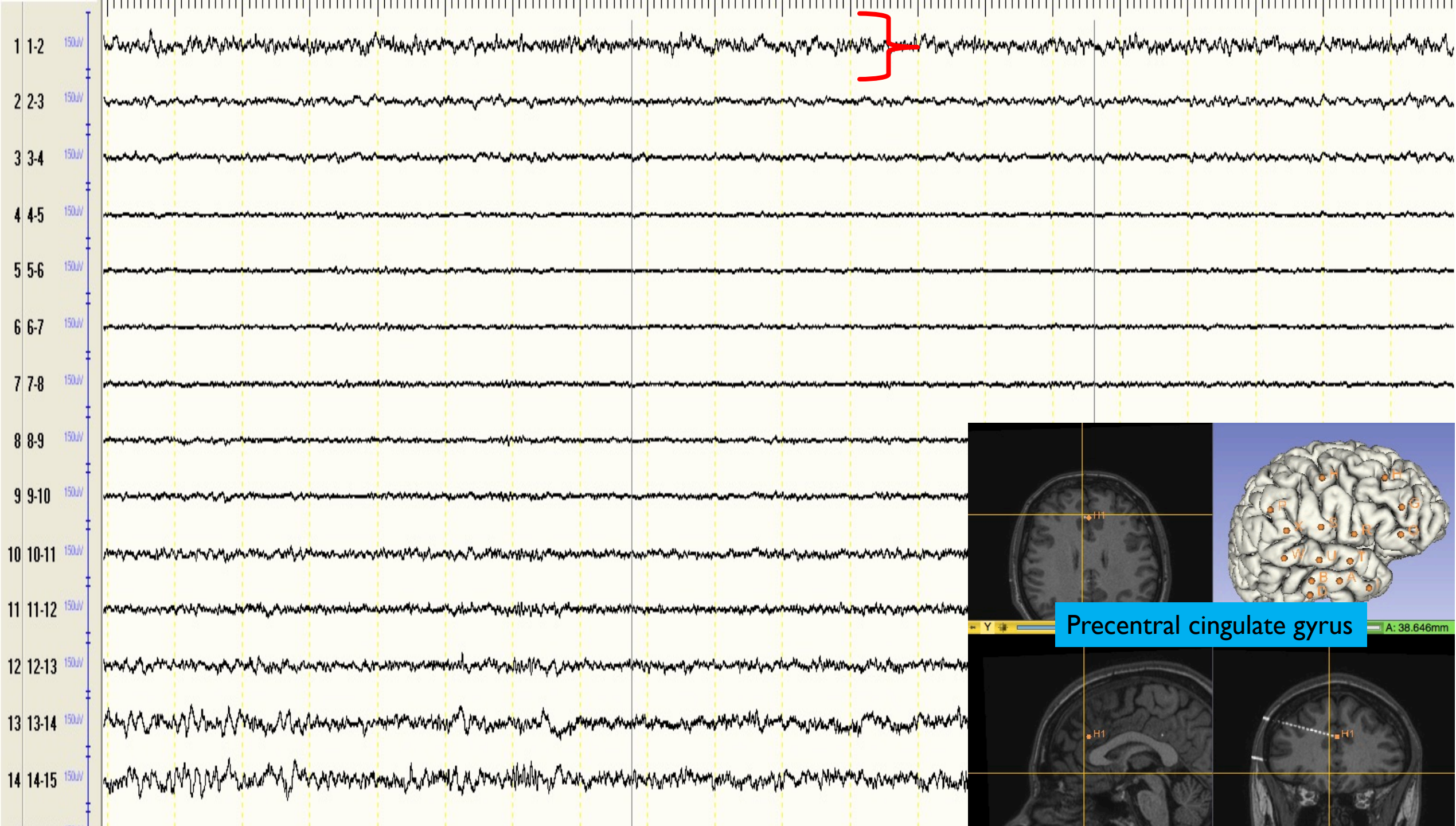
Z.M. Electrode E

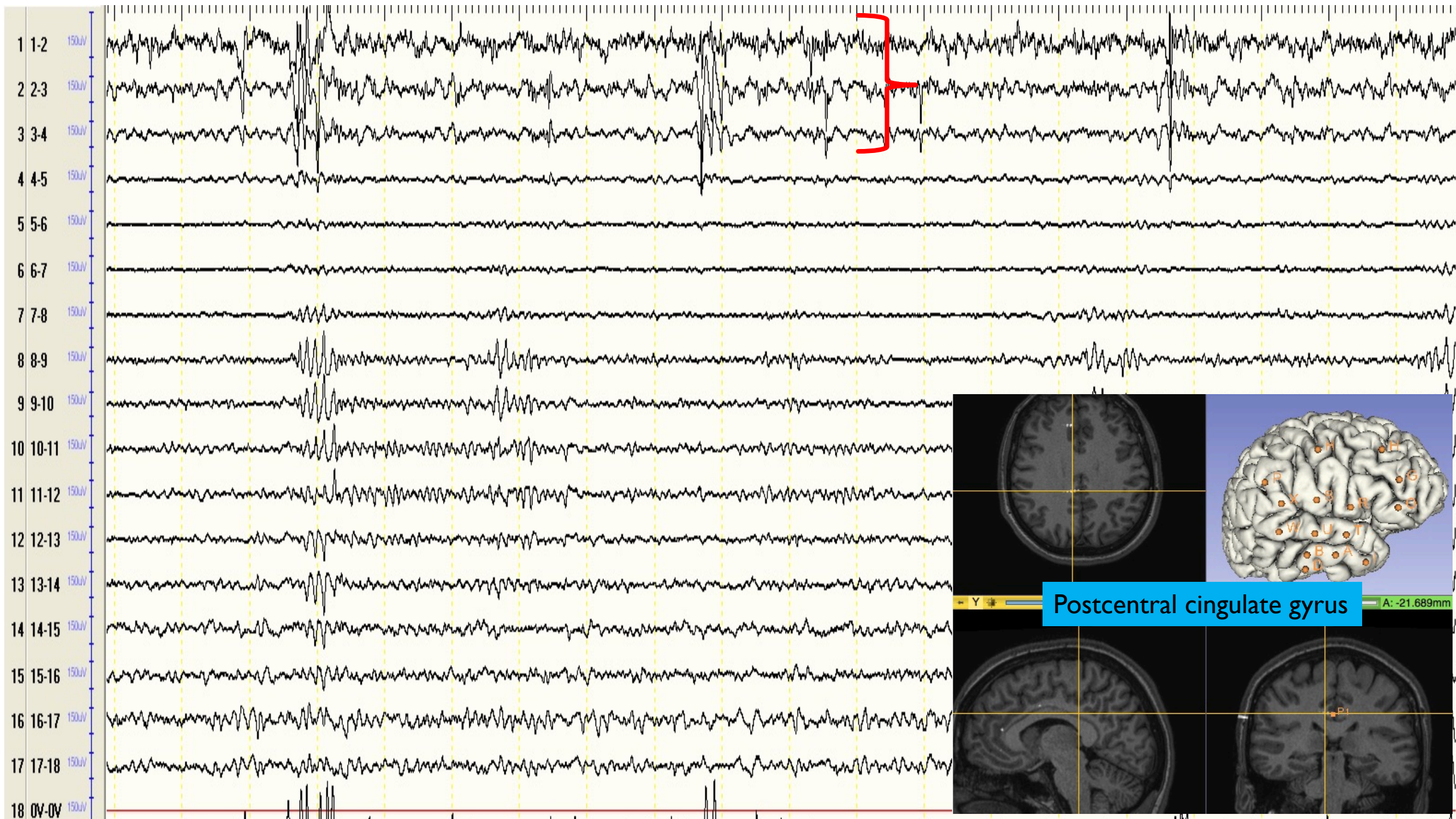


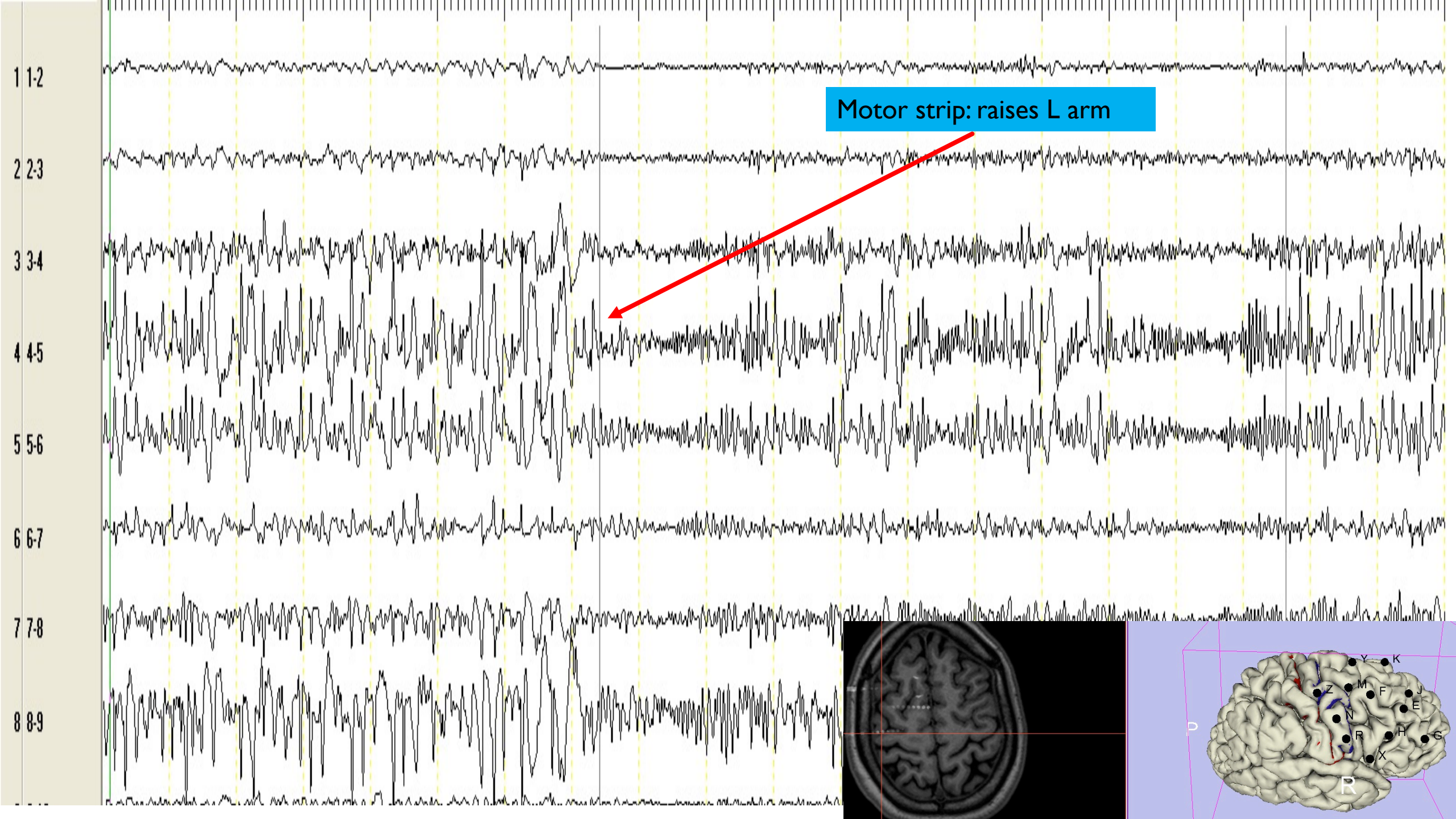
PHYSIOLOGY

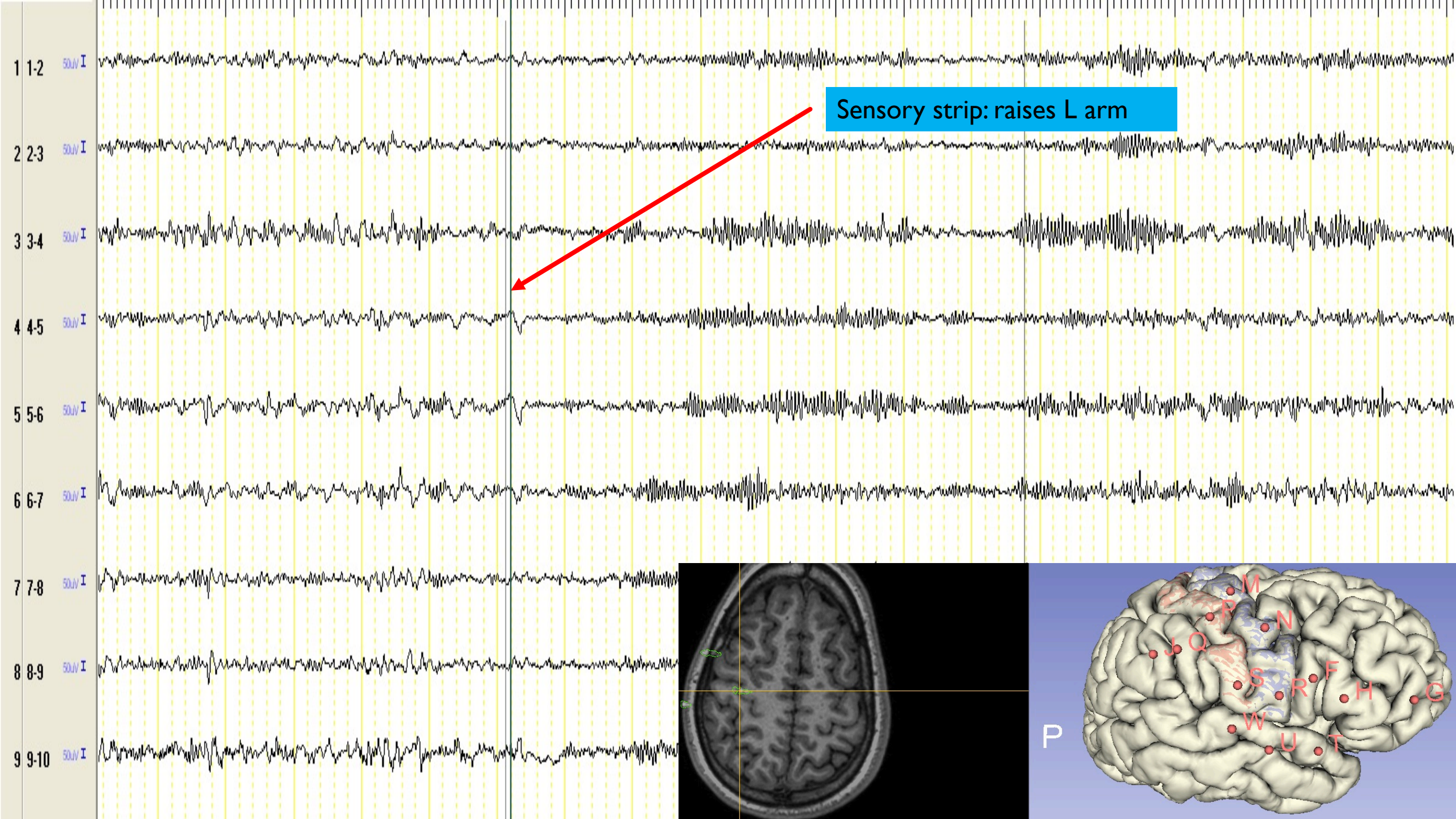


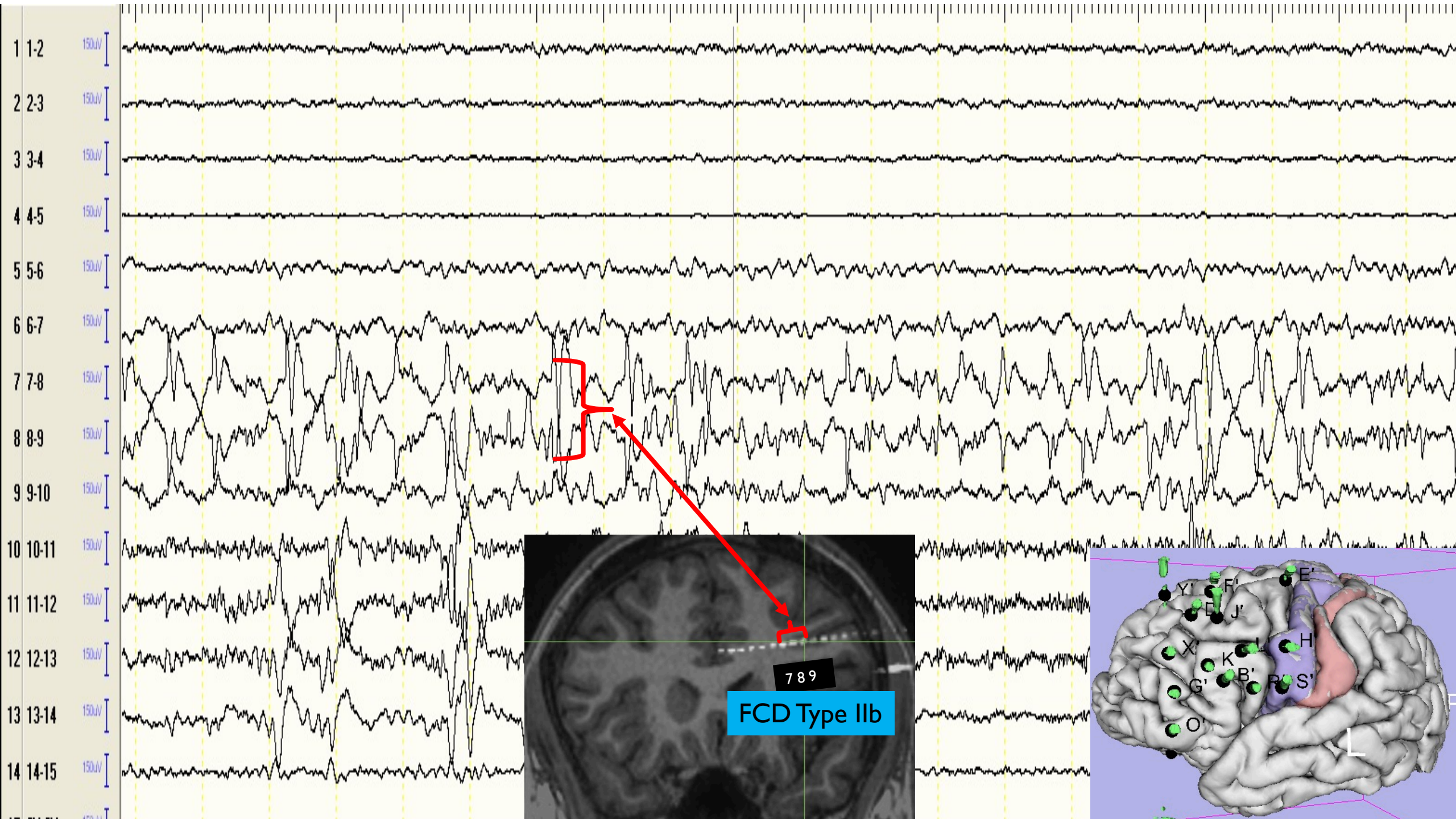






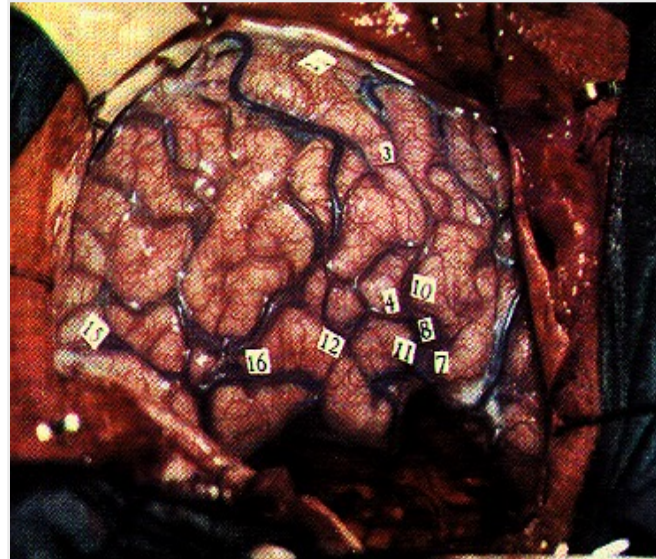
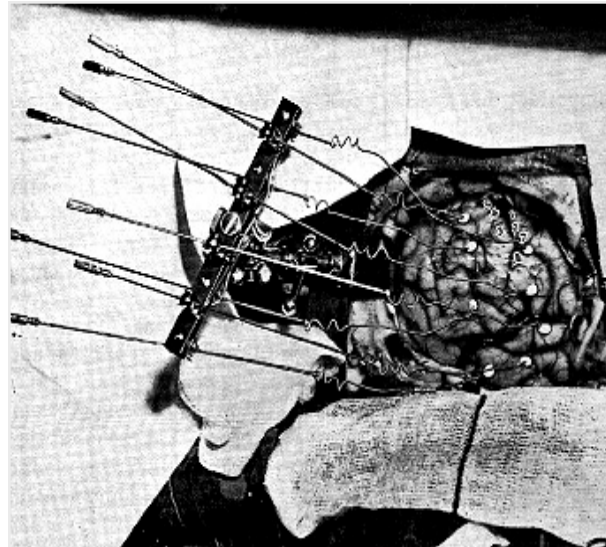
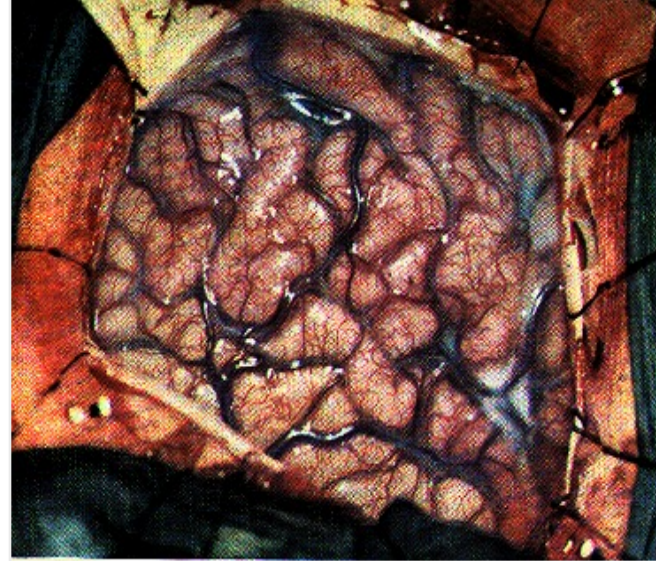
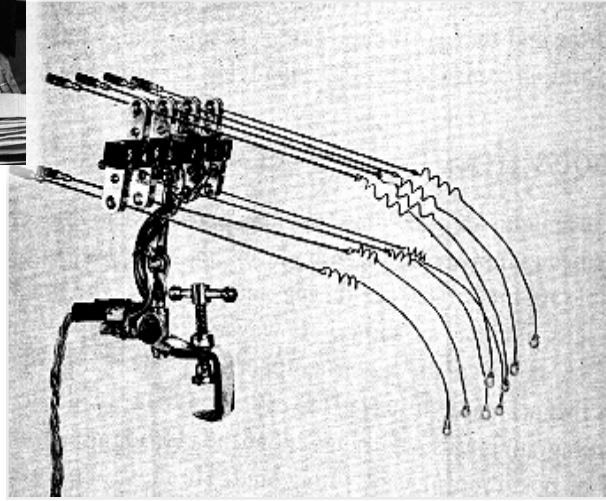






Intracerebral Stimulations

Epilepsy and the functional anatomy of the brain. (Penfield and Jasper, 1954).



Stereo-EEG in acuto



STIMULATIONS

Low Frequency

- ✓ Frequency: 1-18 Hz
- ✓ Pulse duration: 3 msec
- ✓ Intensity: 0.1 - 10 mA
- ✓ Mean duration 30-40 sec.



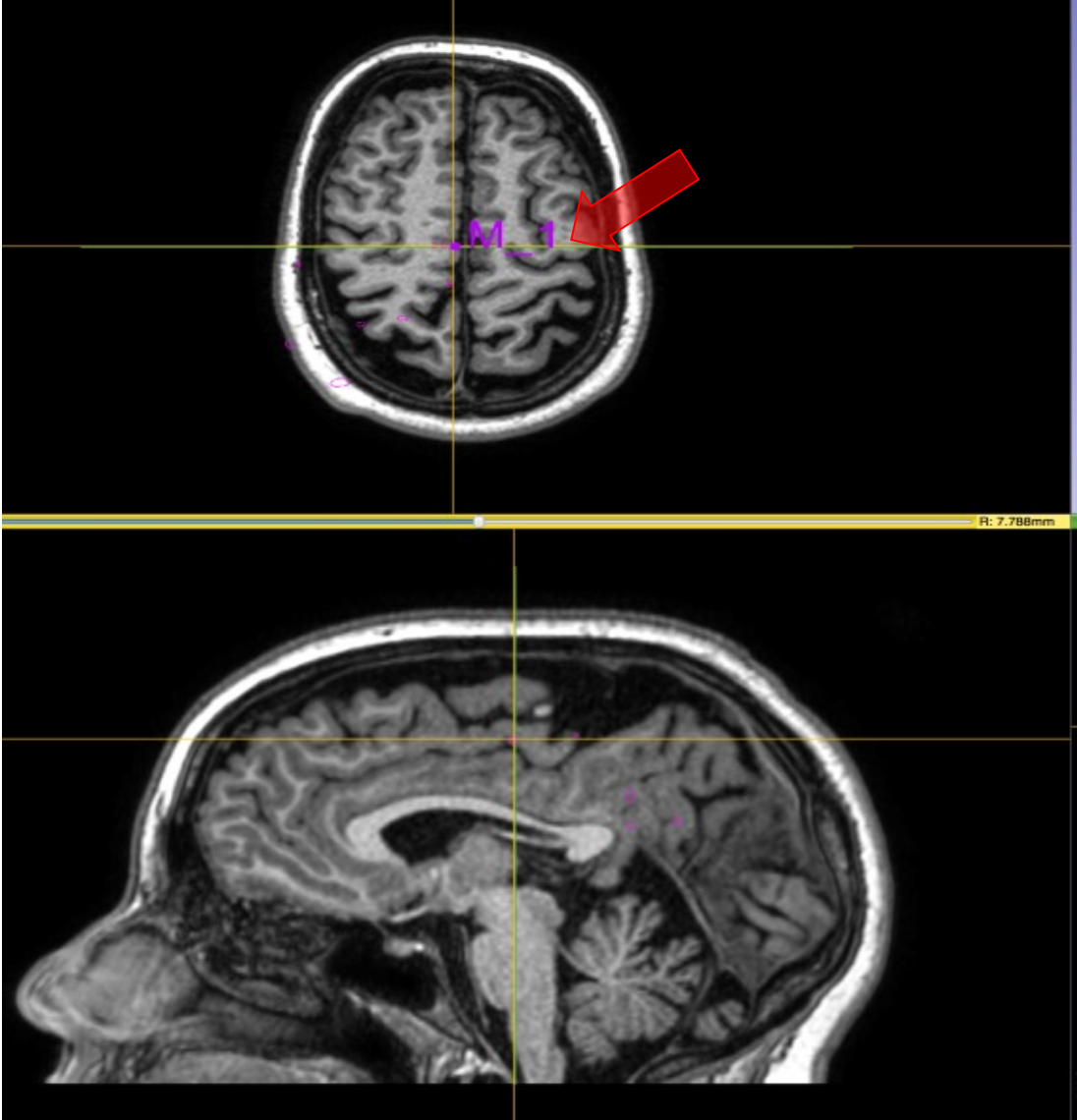
Neurophysiological mapping
Seizures

High Frequency

- ✓ Frequency: 50 Hz
- ✓ Pulse Duration: 1 msec
- ✓ Intensity : 0.5 - 3 mA
- ✓ Mean duration: 5 sec.



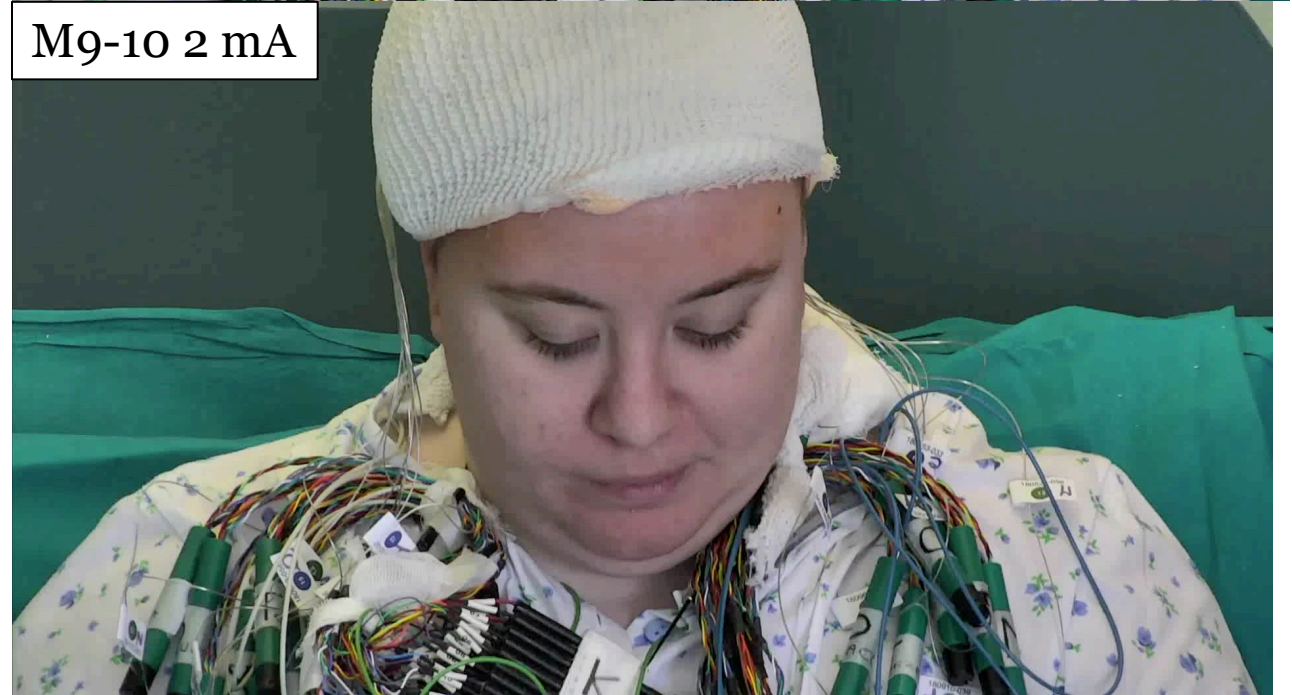
Neurophysiological mapping
Seizures

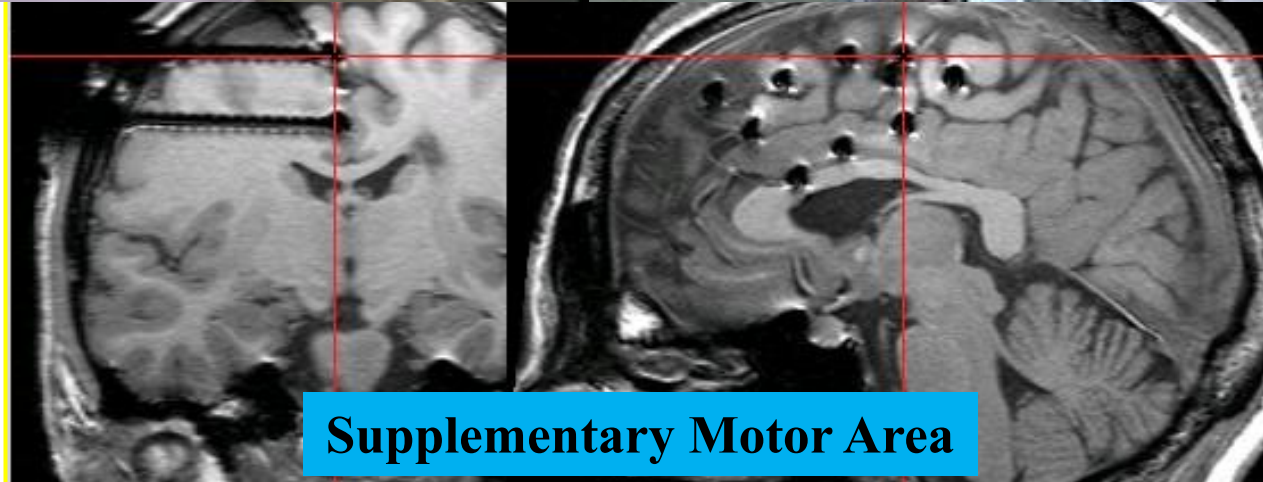


M9-10 1 mA

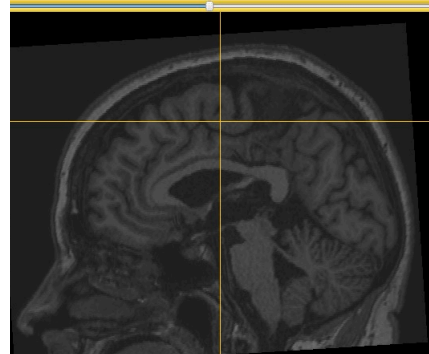
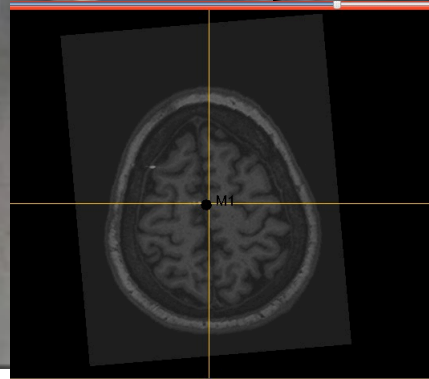


M9-10 2 mA



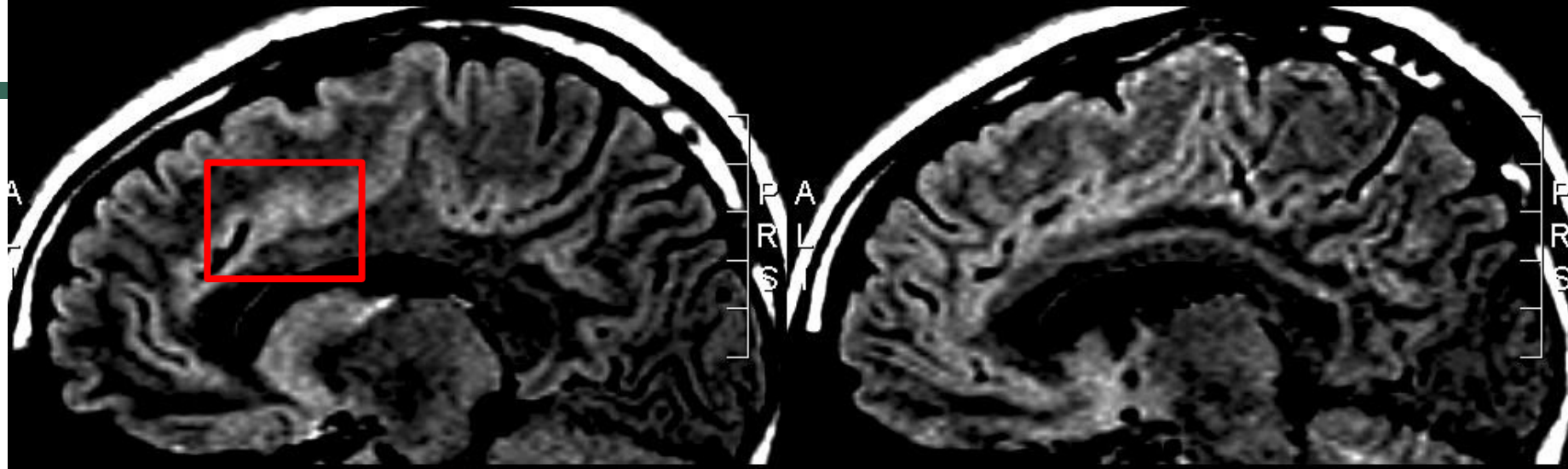


Supplementary Motor Area

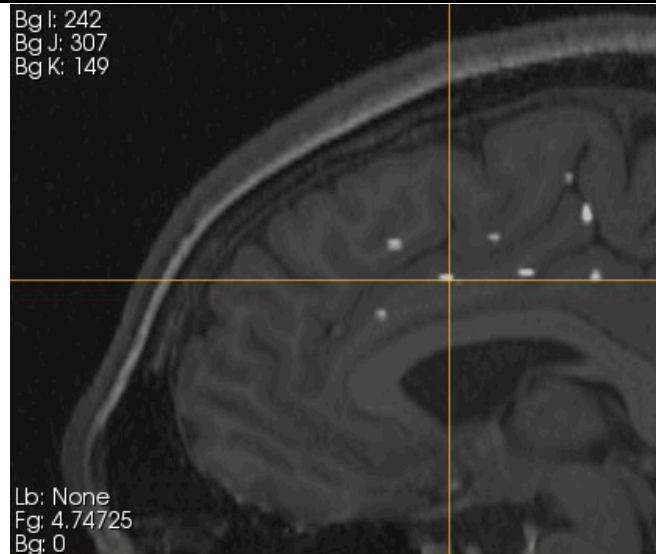


Supplementary Motor Area





Bg I: 242
Bg J: 307
Bg K: 149

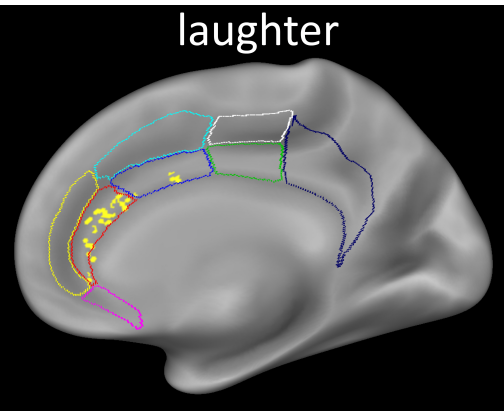


Lb: None
Fg: 4.74725
Bg: 0

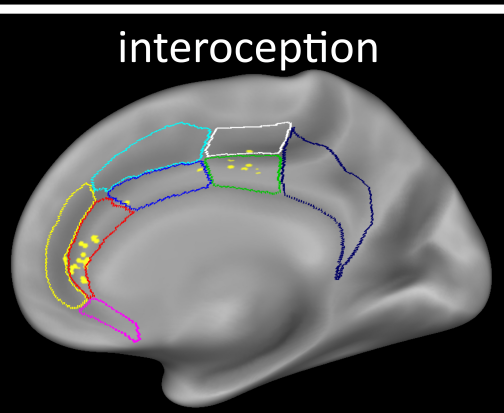


Anterior cingulate

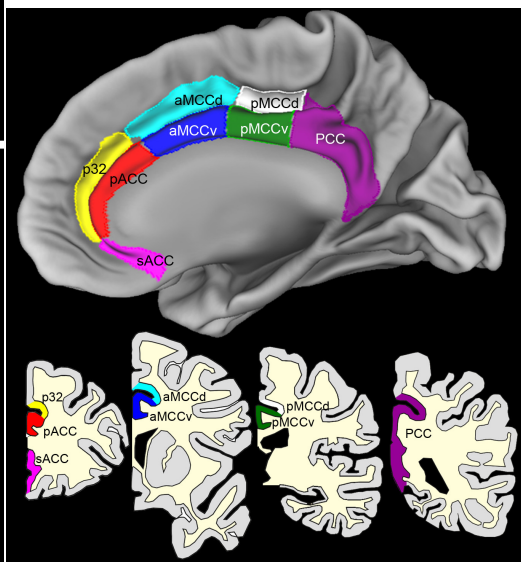
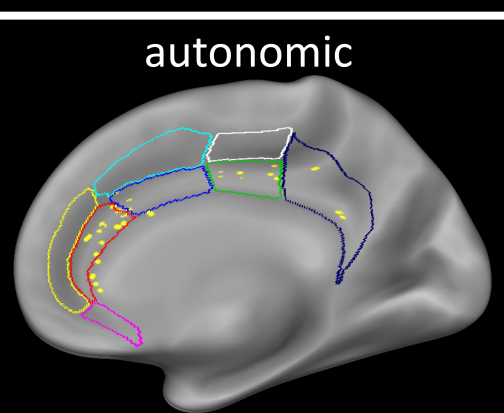
laughter



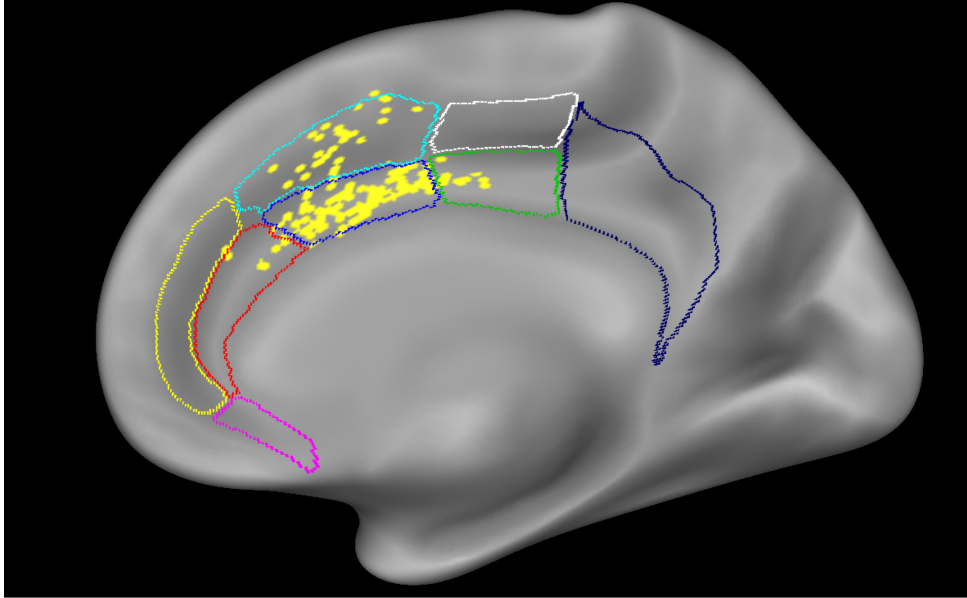
interoception



autonomic

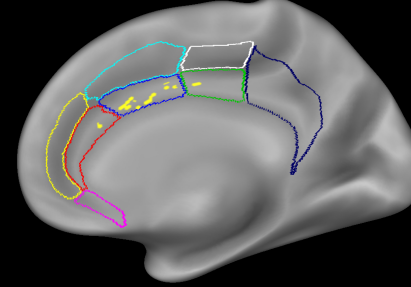


goal-oriented behavior



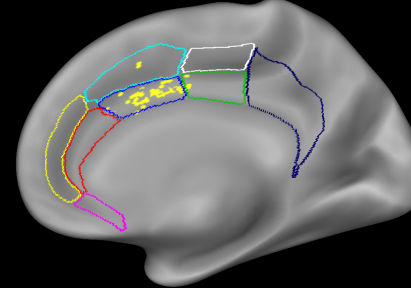
1

getting up impulse



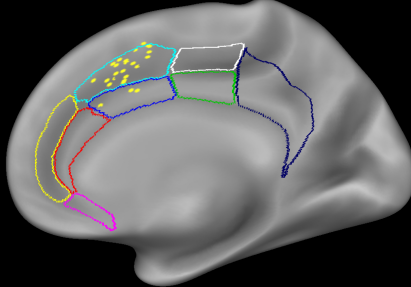
2

reaching and grasping



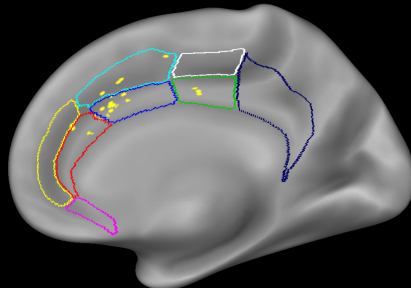
3

body-directed actions



4

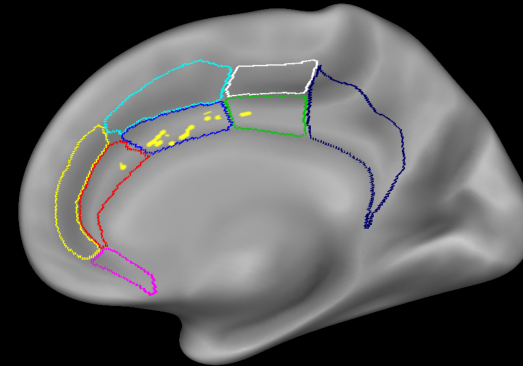
exploratory gazing



MID CINGULATE



getting up impulse

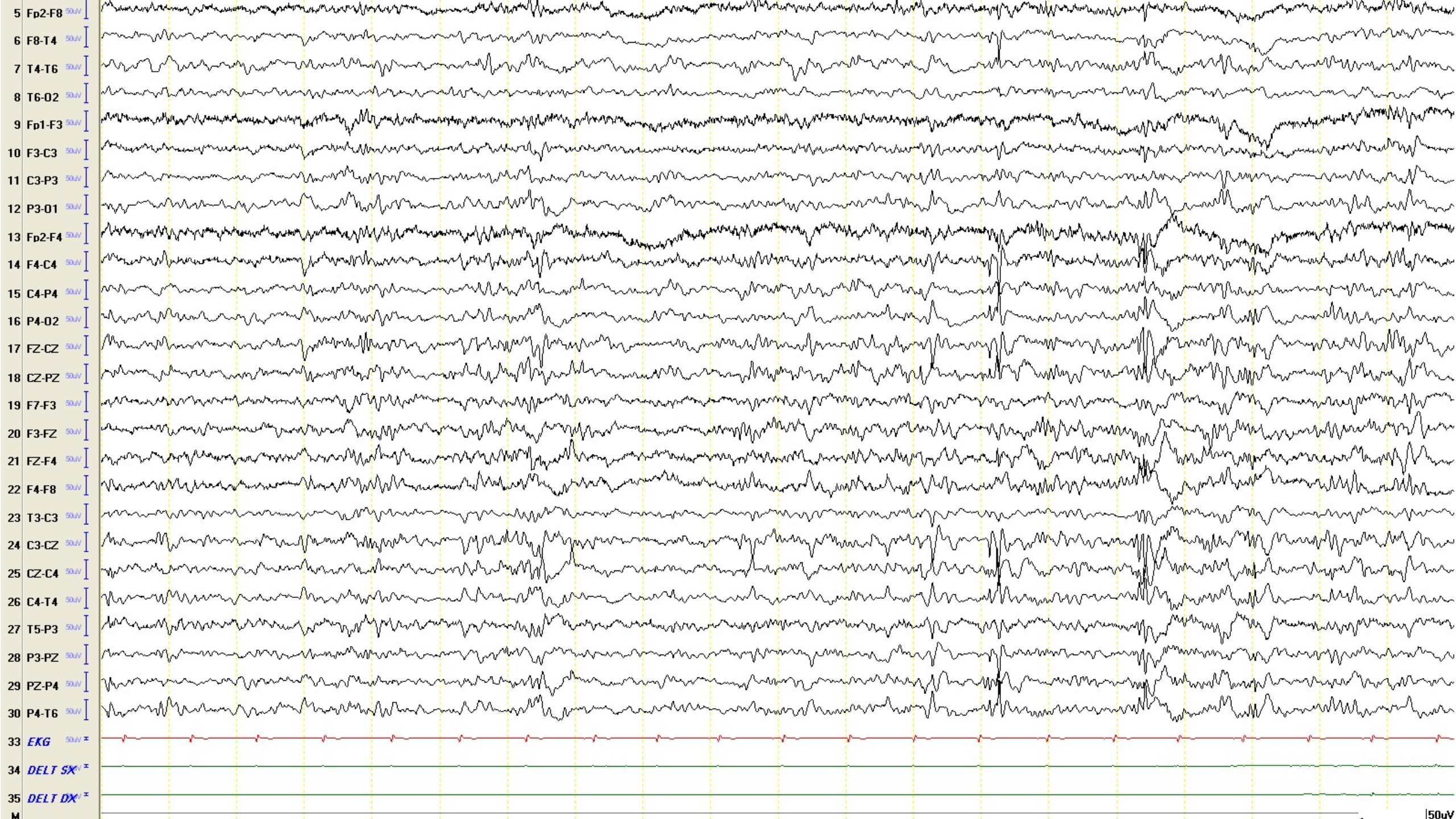


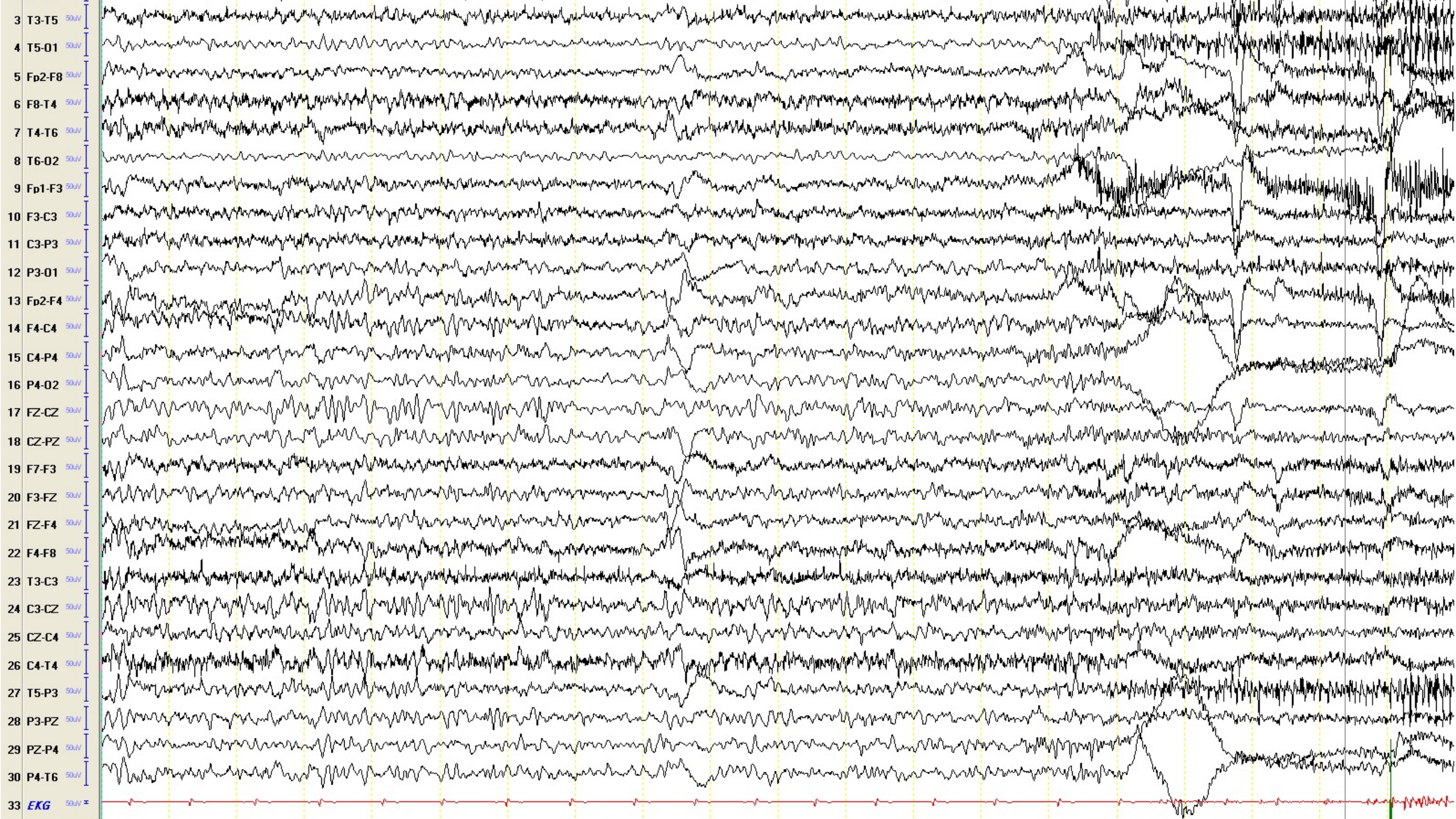
MID CINGULATE

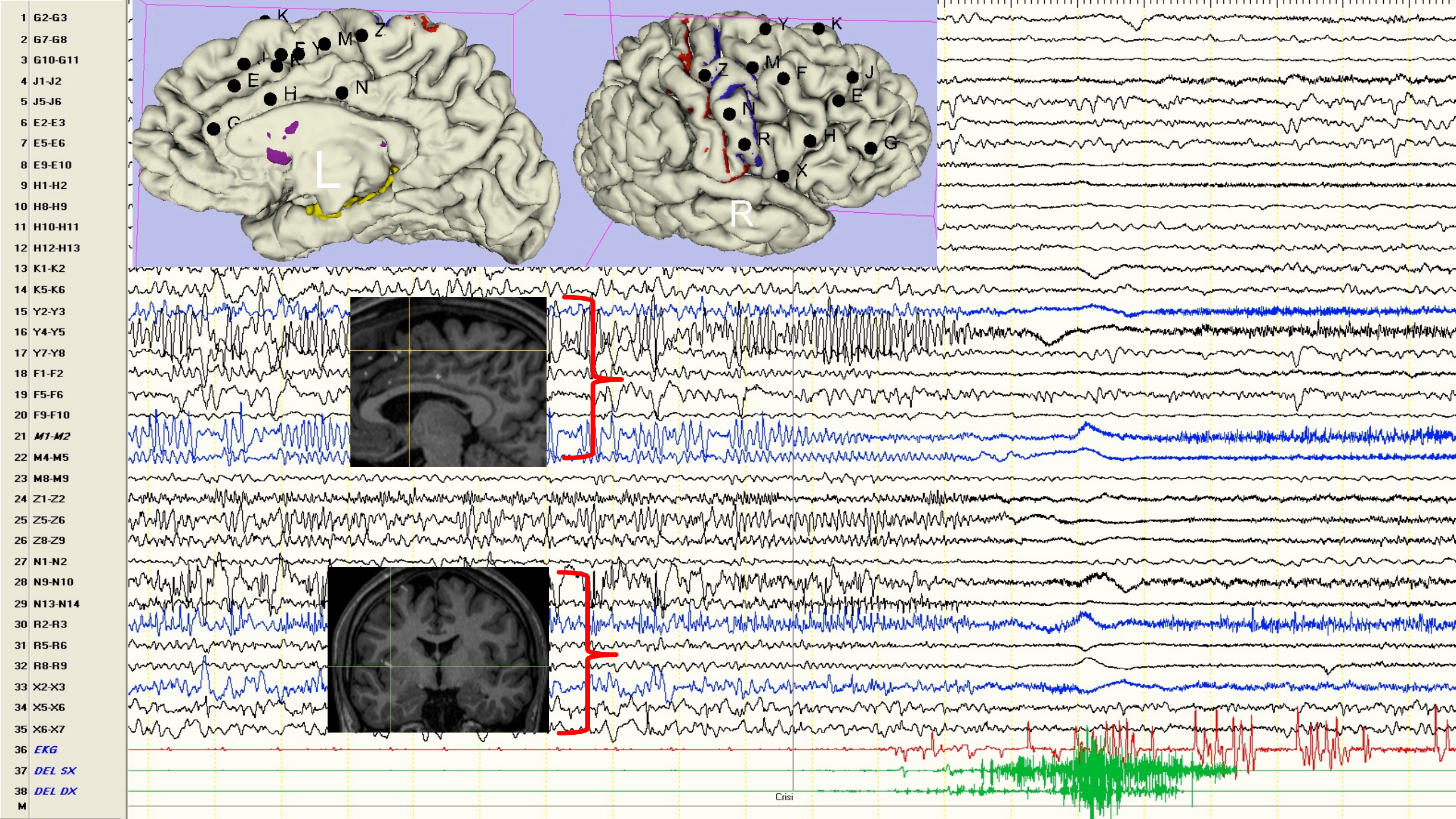


Ictal semeiology



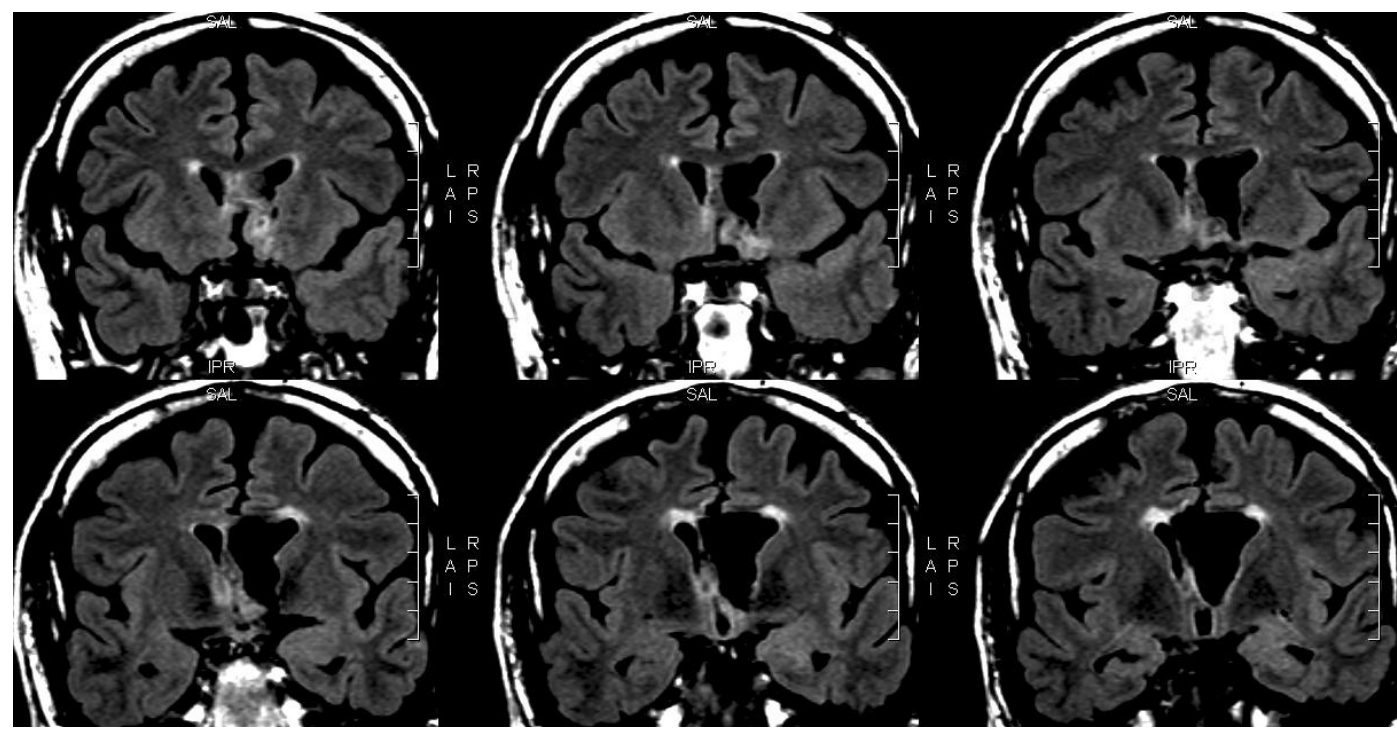
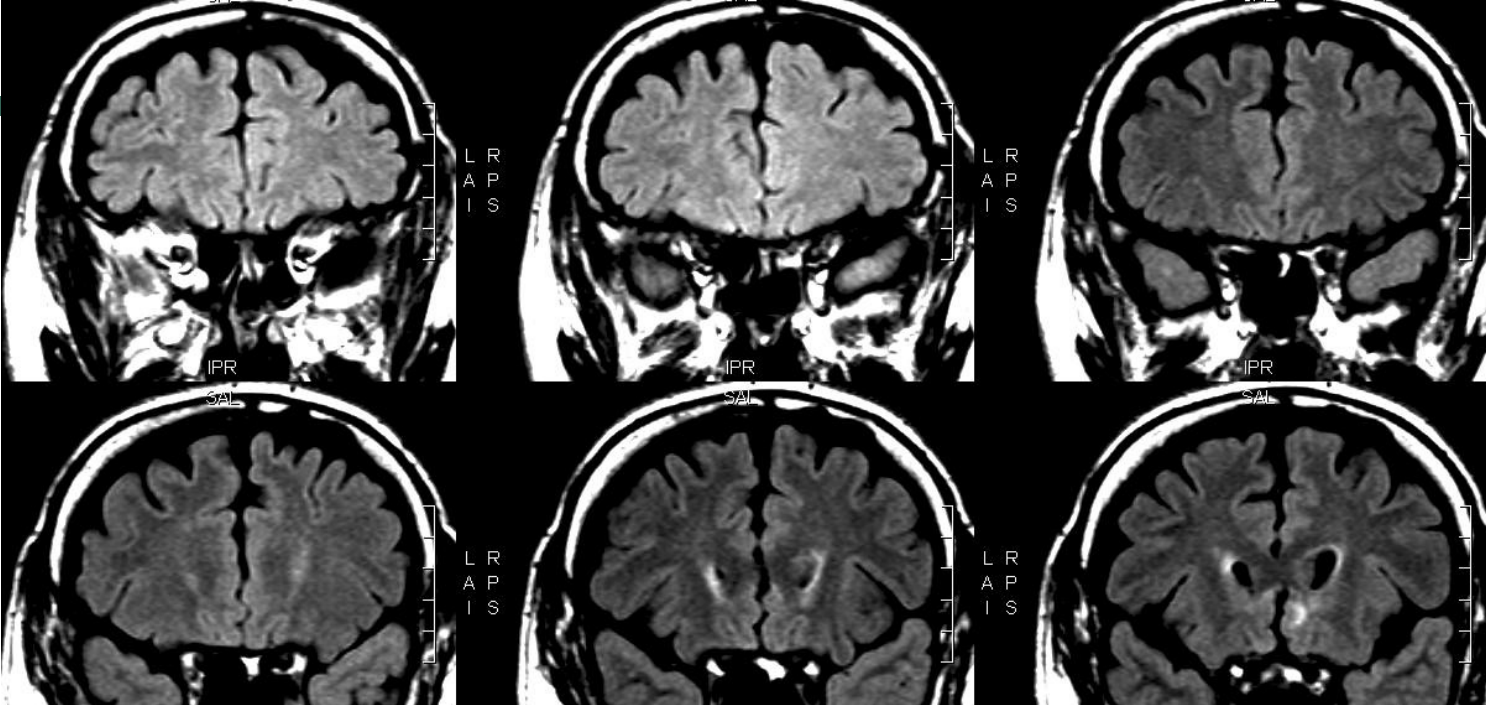


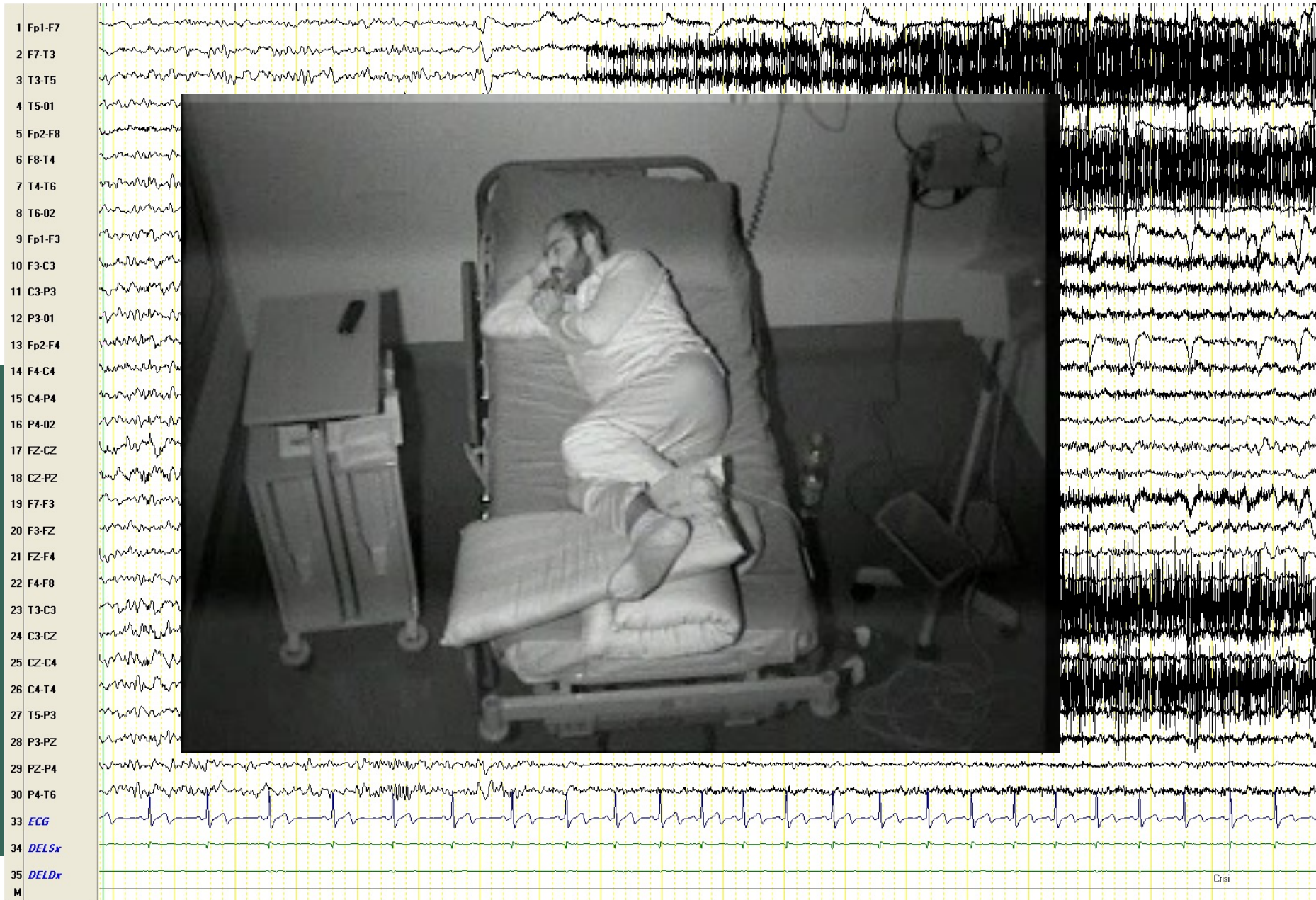




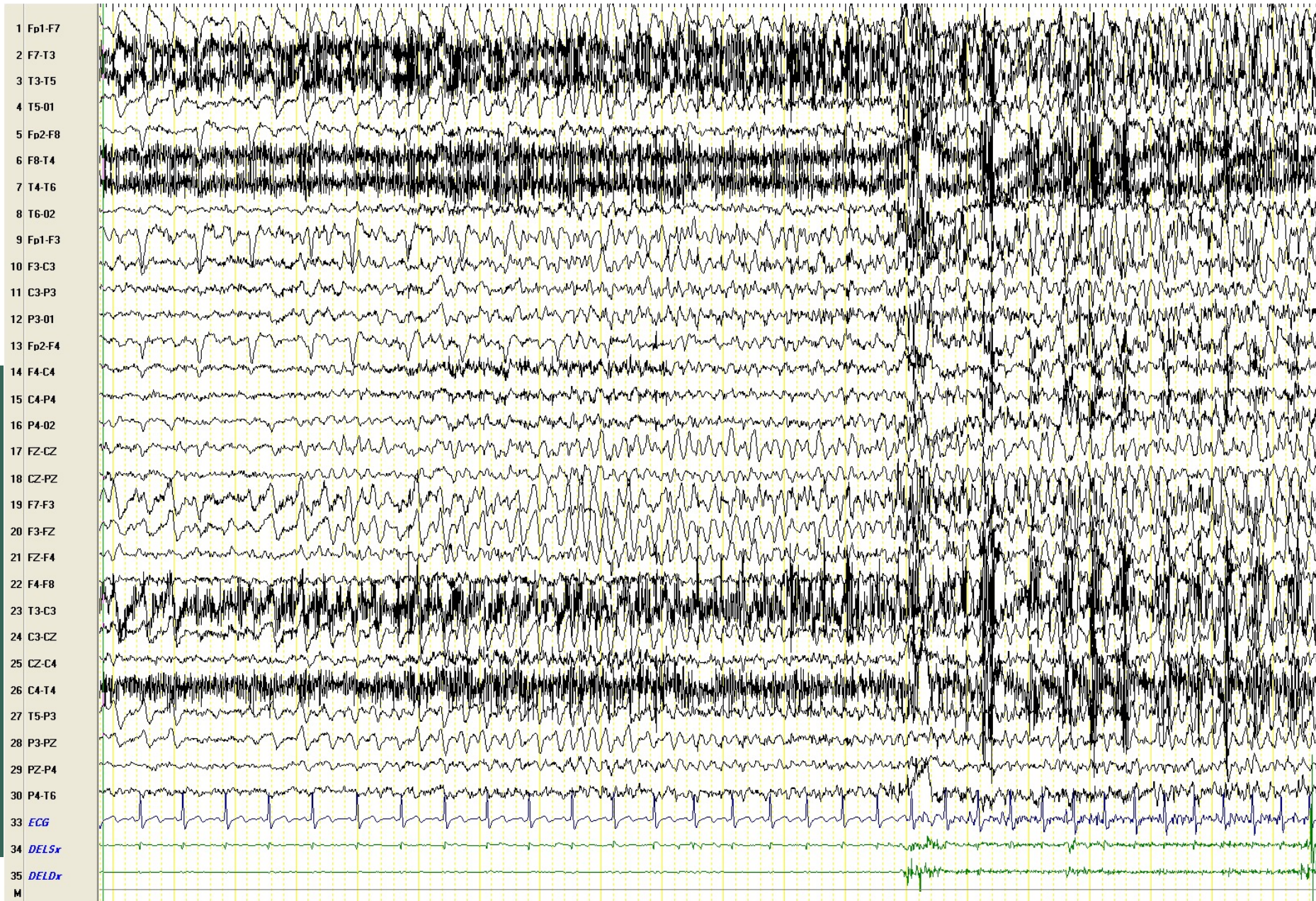
Treno M3-4 a 2 mA Treno M3-4 a 2 mA

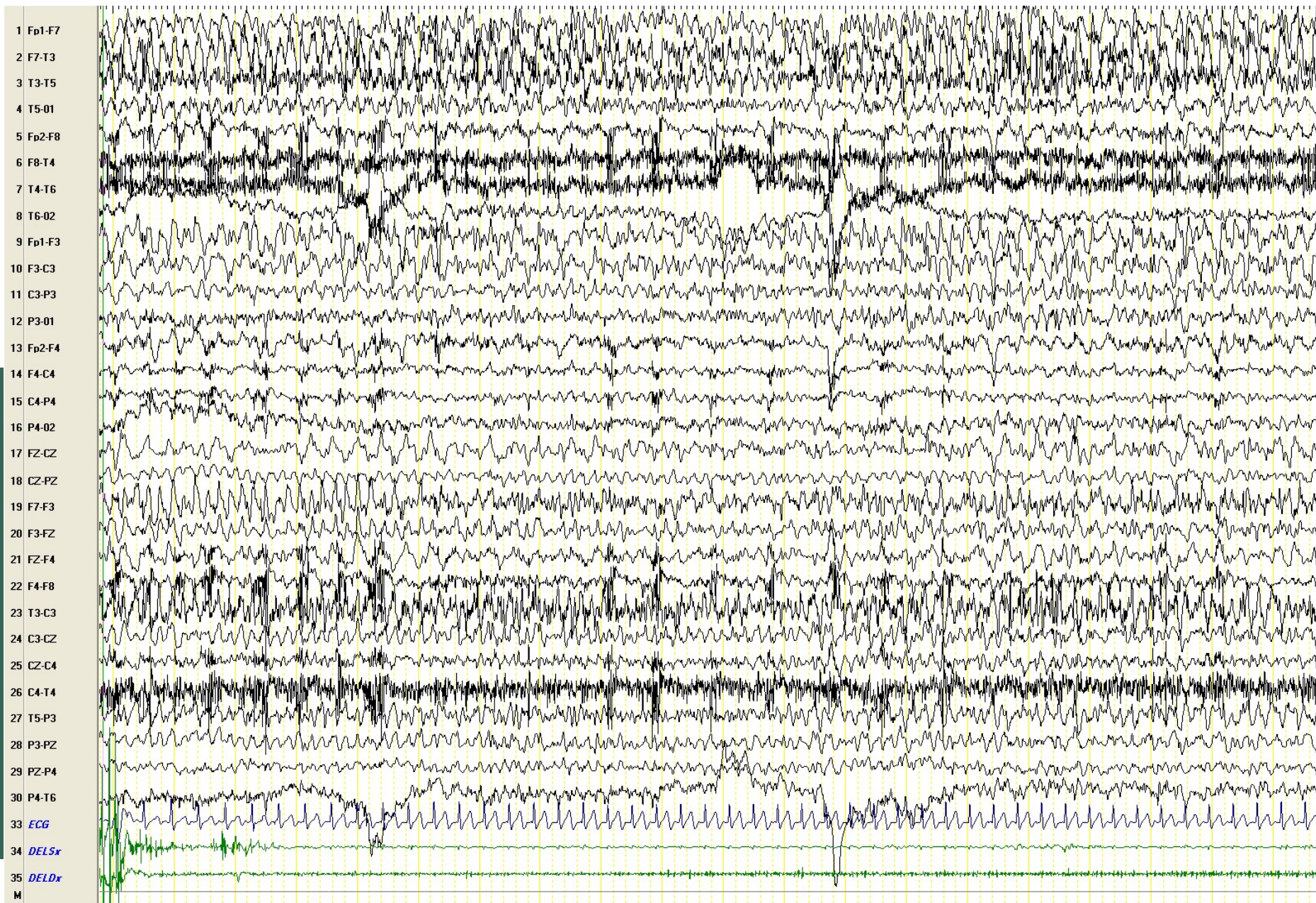




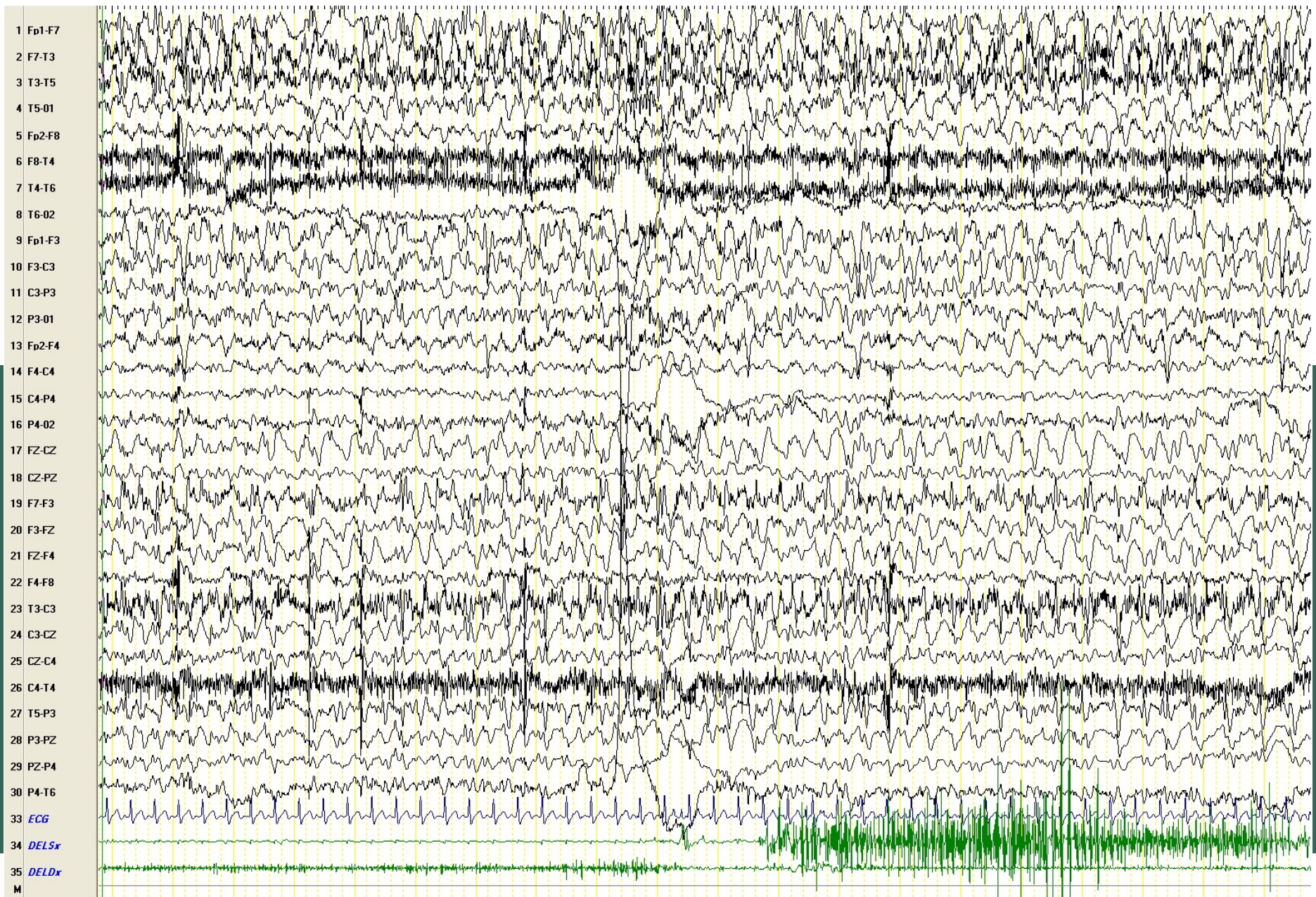


B



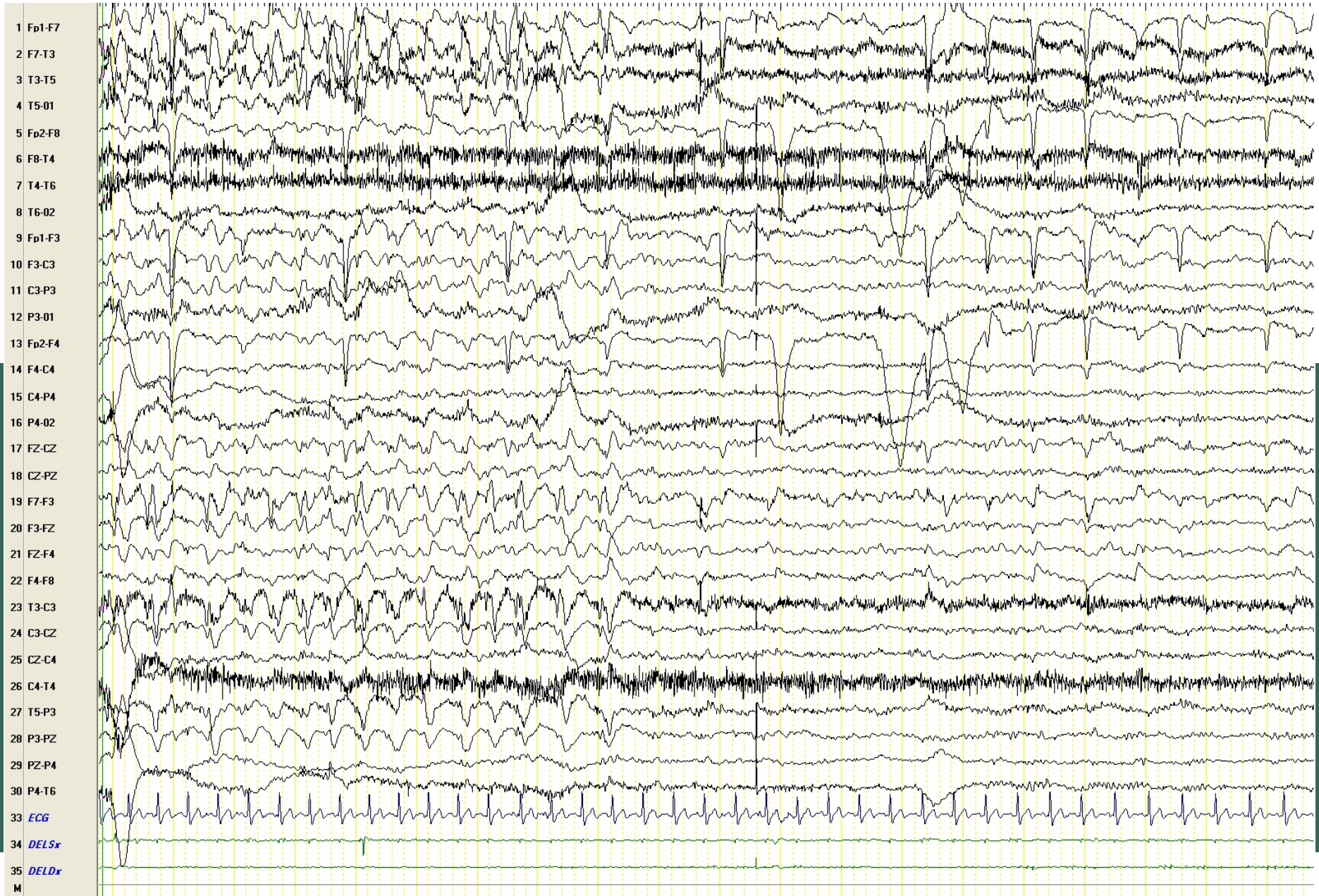


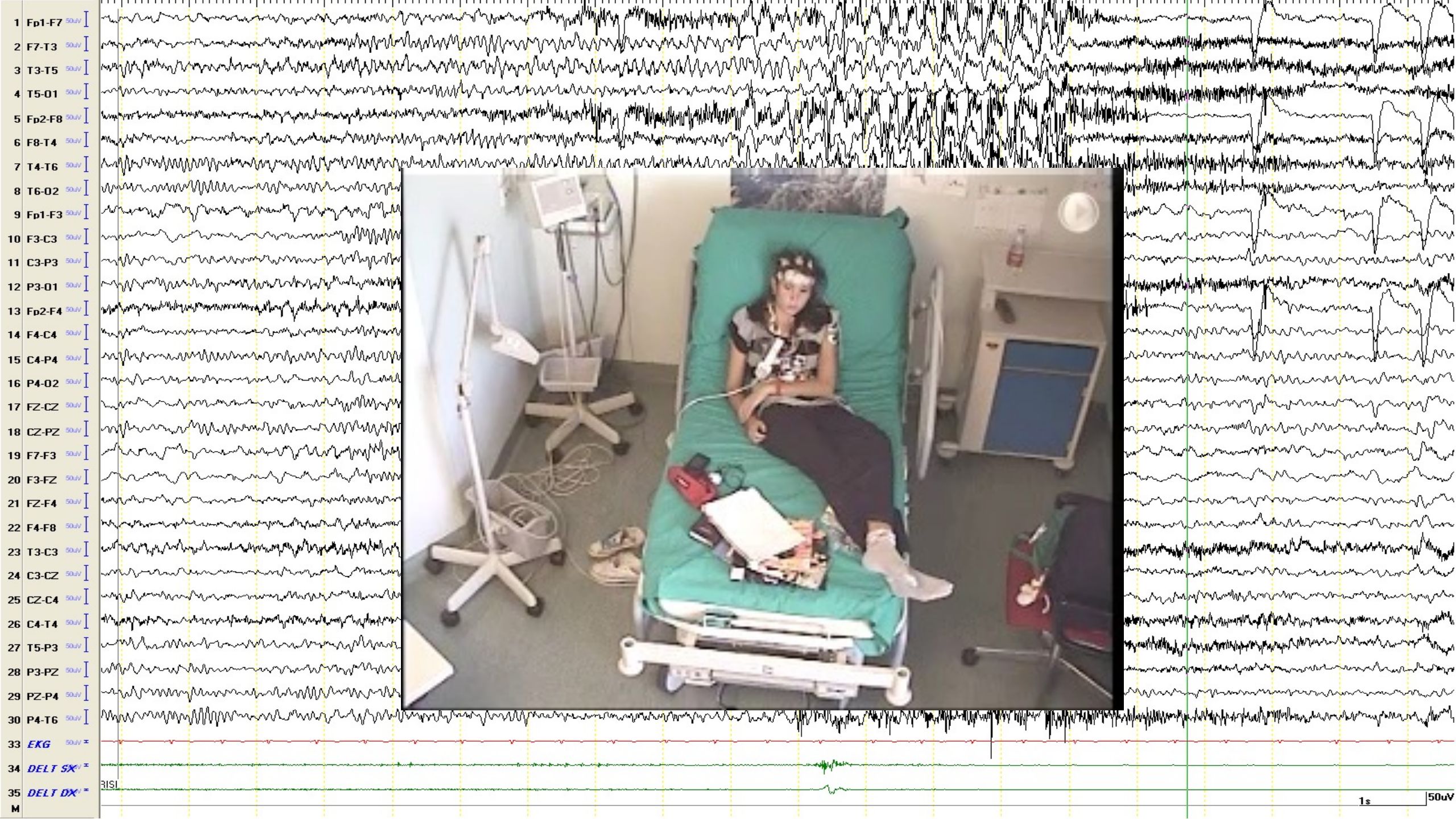
D

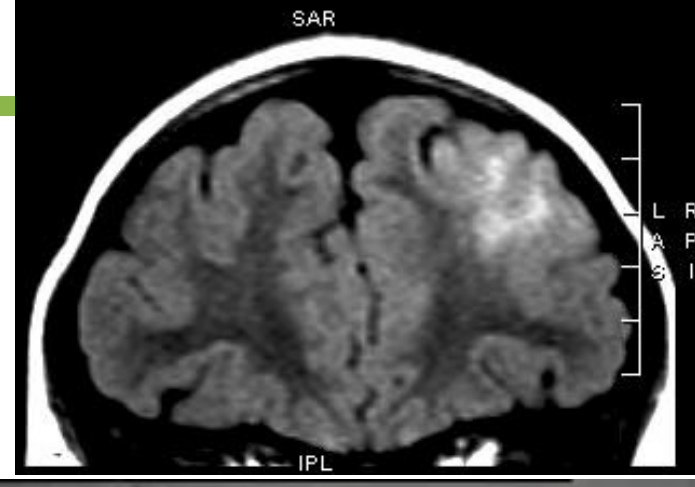
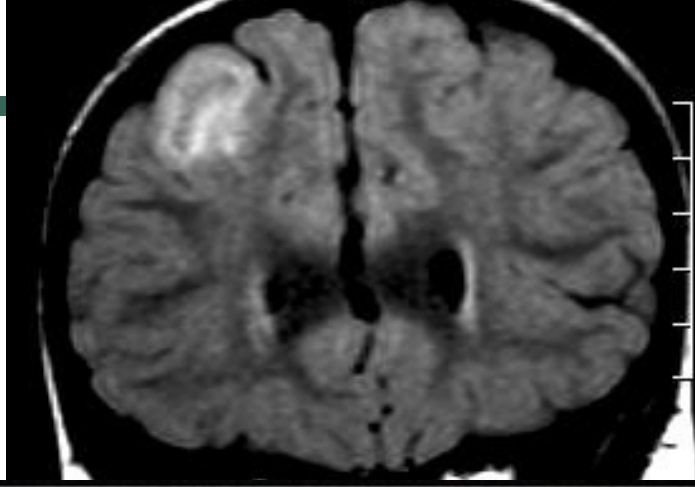


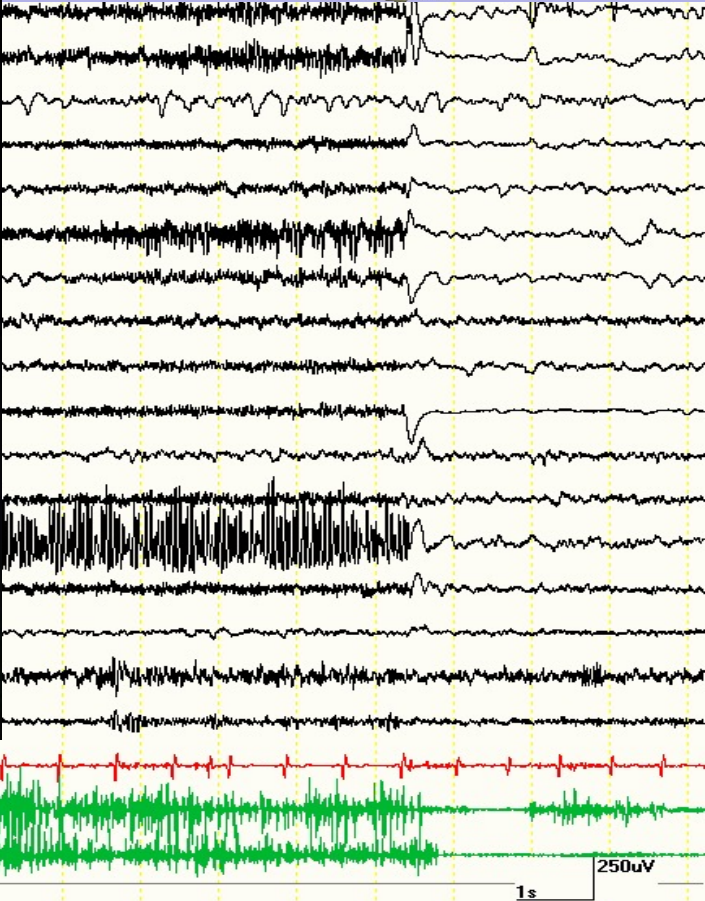
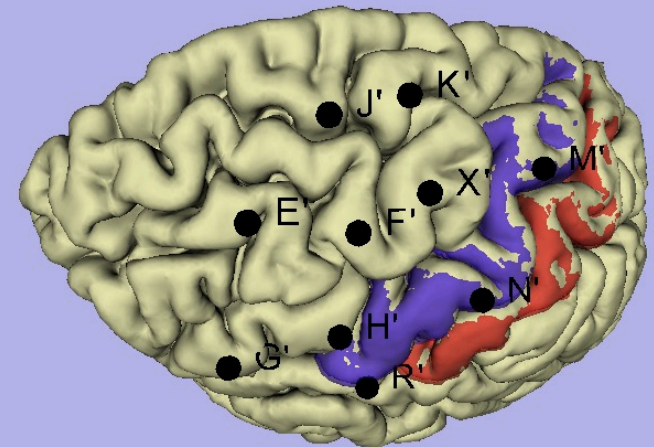
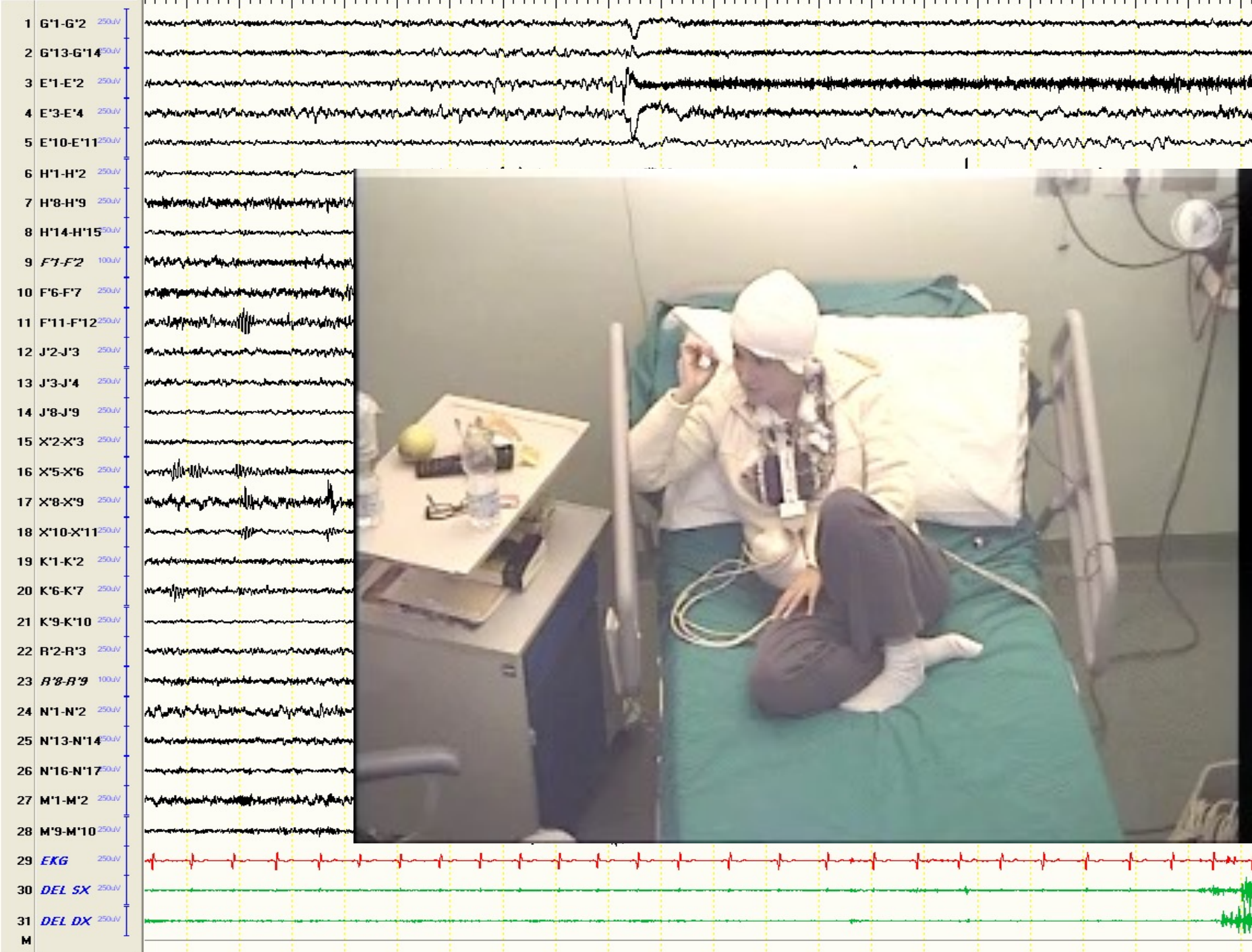


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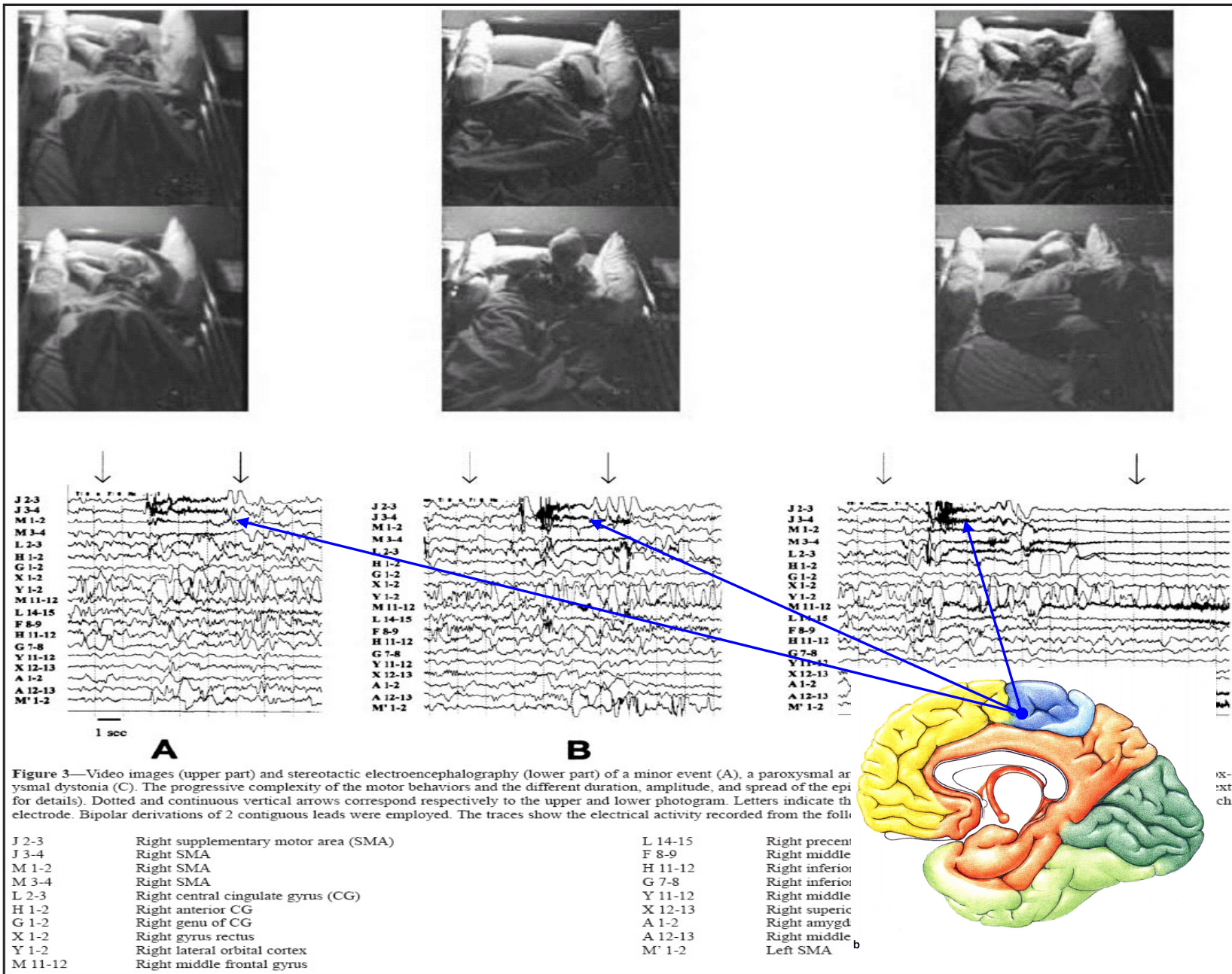












Frontal lobe seizures: From clinical semiology to localization

*†¹Francesca Bonini, *†¹Aileen McGonigal, *†²Agnès Trébuchon, *†³Martine Gavaret, *†⁴Fabrice Bartolomei, *†⁵Bernard Giusiano, and *†⁶Patrick Chauvel

Epilepsia, 55(2):264–277, 2014
doi: 10.1111/epi.12490

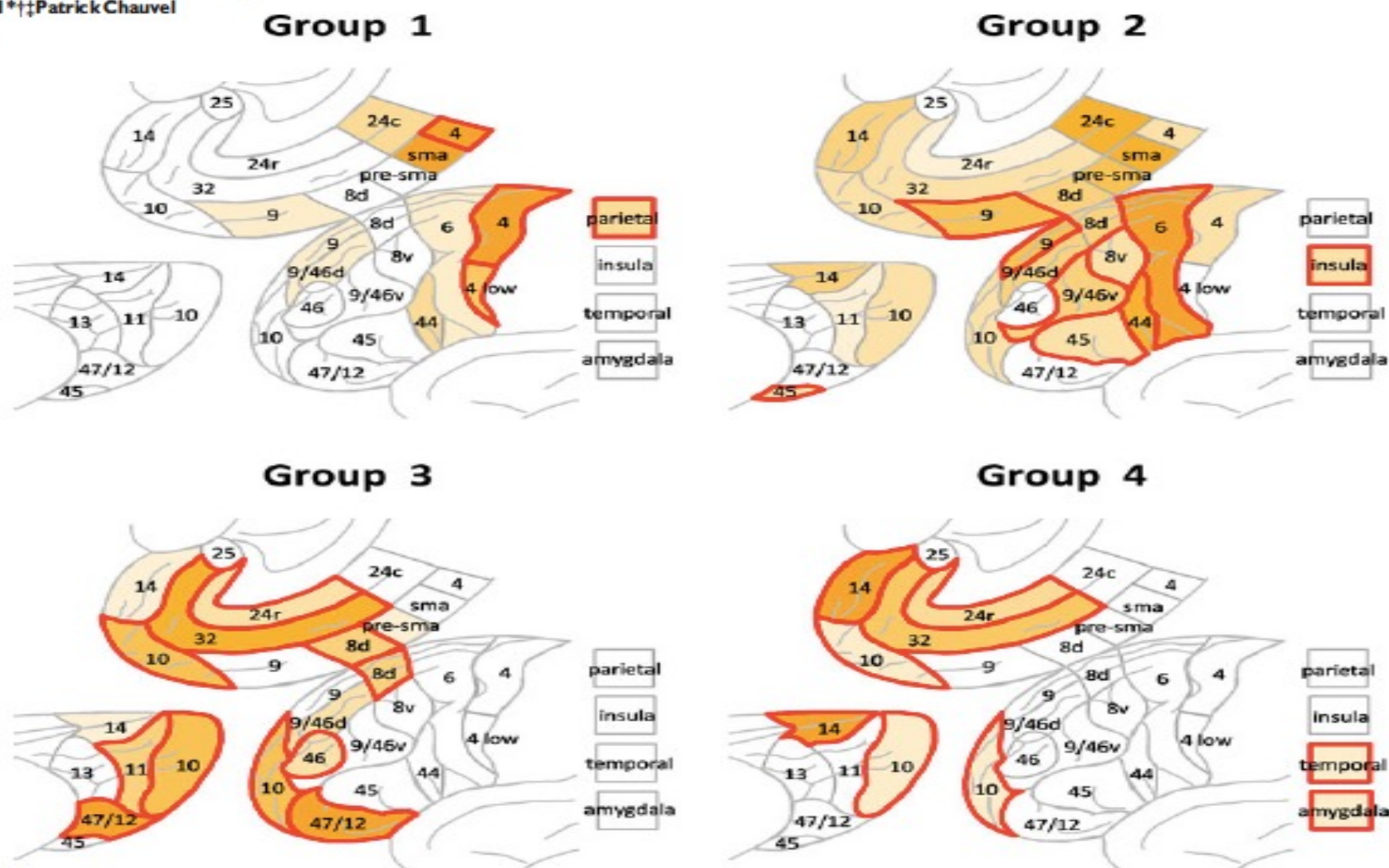
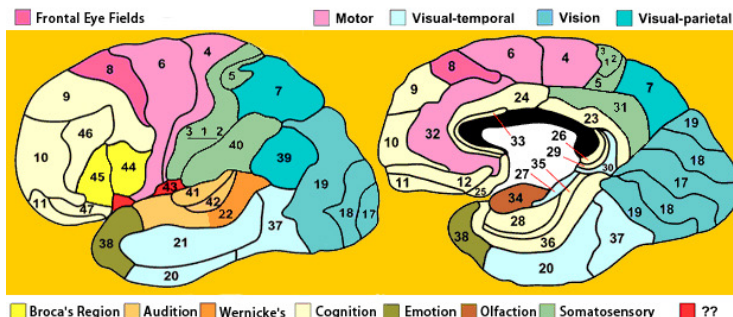
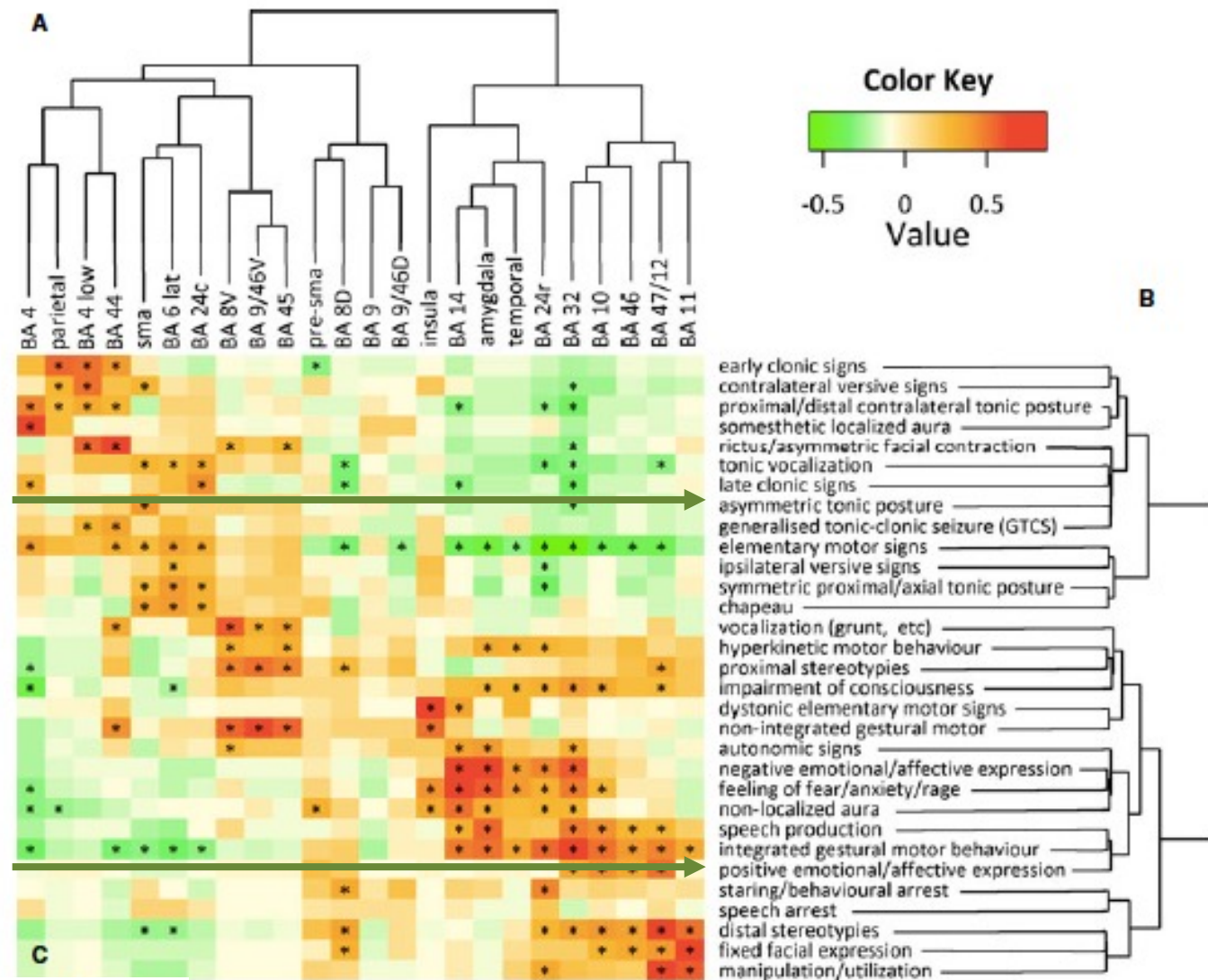


Figure 2. Cortical regions that characterize the four groups of patients. Brain areas forming part of the early spread network (scored as 2 basing on ictal SEEG) are colored with darker shading, based on the proportion of patients in the group with implication of that area. Brain areas significantly more often involved in one group than the others (value-test > 2) are red bordered. Architectonic subdivision of lateral and medial prefrontal Brodmann areas according to (Petrides & Pandya, 1994).

Frontal lobe seizures: From clinical semiology to localization

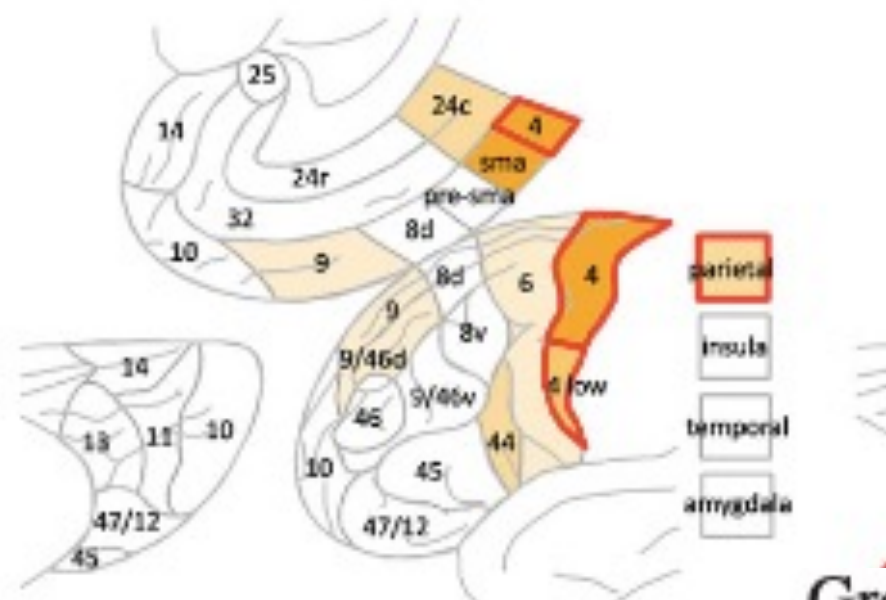
*††Francesca Bonini, *††Aileen McGonigal, *††Agnès Trébuchon, *††Martine Gavaret, *††Fabrice Bartolomei, *§§Bernard Giustiano, and *††Patrick Chauvel

Epilepsia, 55(2):264-277, 2014
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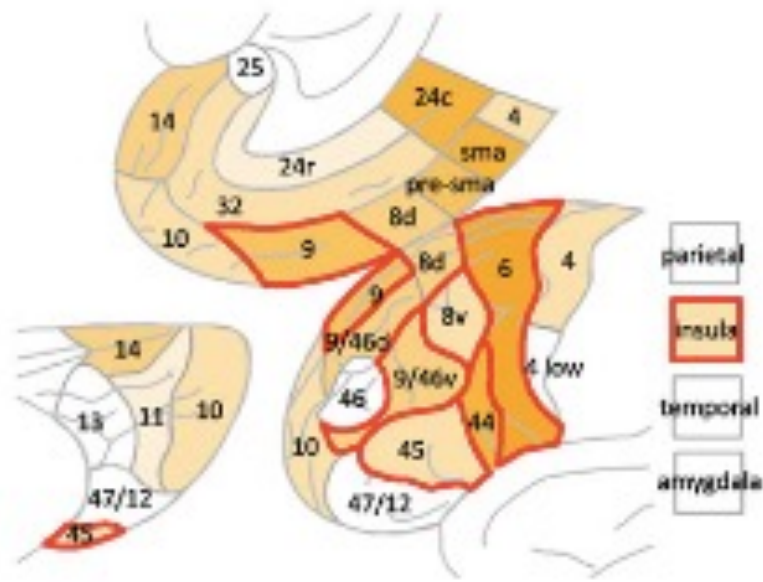
between cortical areas and clinical features, as ordered by hierarchical clustering basing on their reciprocal distance. The main regions is shown on the horizontal axis. (B) Clustering of clinical signs is shown on the vertical axis. In these two neighboring regions as well as neighboring signs occur more frequently together than distant ones. (C) Correlation signs as a function of the color (red, positive correlation; green, negative correlation; starred squares, $p < 0.05$) follow an axis of association running from more posterior (top left corner) to more anterior regions (bottom right corner). This axis served for significant correlation ($p < 0.05$ starred squares), indicates the emergence of clinical spectrum developing from primary motor cortex to the frontal pole (BA, Brodmann area; SMA, supplementary motor area; pre-SMA, pre-motor area). Architectonic subdivision of lateral and medial prefrontal Brodmann areas according to (Petrides & Pand-

Group 1



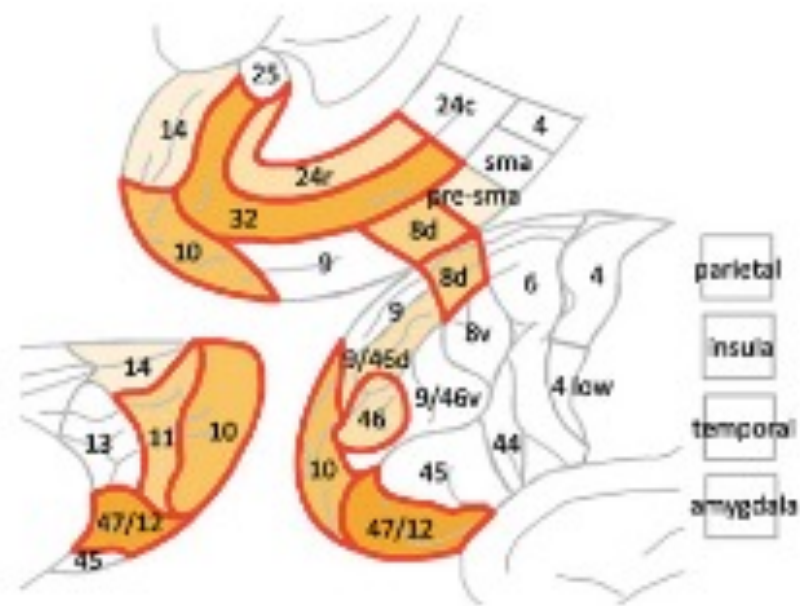
Group 1, composed of 16 patients, is characterized by the presence of one or more of the following elementary motor signs: clonic signs, contralateral tonic posture, contralateral versive signs, asymmetric tonic posture, secondary generalization, or asymmetric facial contraction. Somesthetic localized aura and tonic vocalization could also typically occur in these patients. Moreover, this group is characterized by the absence of gestural motor behavior and of emotional features (Table 1, group 1). Significant involvement

Group 2



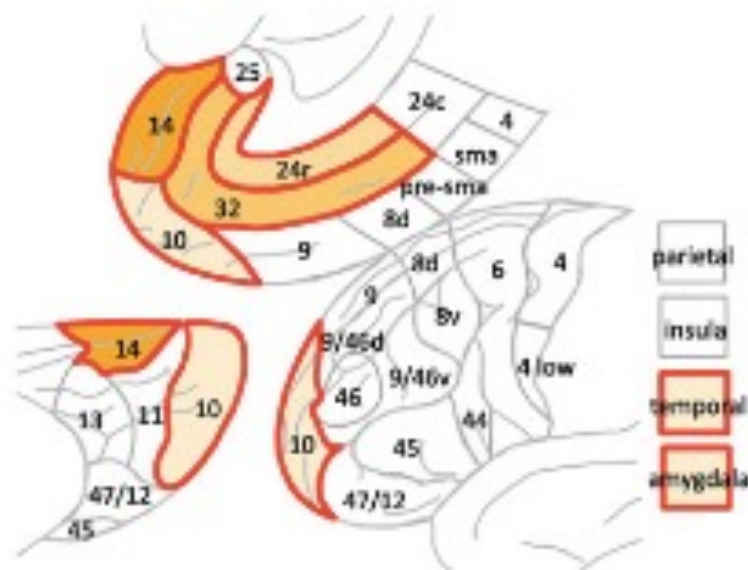
Group 2 (23 patients) was characterized by the co-occurrence of elementary motor signs (typically symmetric axial tonic posture and facial contraction such as “chapeau de gendarme”) and nonintegrated gestural motor behavior. In this more heterogeneous group, nonlocalized aura and more complex nonverbal vocalization were also frequently present, whereas integrated gestural motor behavior, distal stereotypies, early clonic signs, and fixed facial expression never occurred (Table 1). Nonintegrated gestural motor behavior could include proximal stereotypies and could have a hyperkinetic character or not (example illustrated in

Group 3



Typical semiologic features of group 3 (10 patients) were the following: integrated gestural motor behavior with distal stereotypies, fixed facial expression or, alternatively, positive emotional expression, proximal stereotypies, and speech production. On the other hand, the absence of any elementary motor sign was a significant characteristic of these patients (Table 1). Early spread network underlying

Group 4



Group 4 was composed of five patients presenting with integrated gestural behavior of fear, sometimes hyperkinetic, with attempt to fight or to escape, frightened facial expression, sometimes screaming or swearing, and autonomic signs. Elementary motor signs never occurred and underlying involved regions corresponded to the orbital and medial-prefrontal network (BA 14, BA 32 and 24r, BA 10) with propagation to amygdala and anterior temporal regions (Fig. 2), but not propagation to lateral frontal cortex.

Epilepsia. 2019 Mar 13

Clinical features of sleep-related hypermotor epilepsy in relation to the seizure-onset zone: A review of 135 surgically treated cases.

Gibbs SA^{1,2}, Proserpio P¹, Francione S¹, Mai R¹, Cardinale F¹, Sartori I¹, Castana L¹, Plazzi G^{3,4}, Tinuper P^{3,4}, Cossu M¹, Russo GL¹, Tassi L¹, Nobili L^{1,5}.

Sleep-related hypermotor epilepsy (SHE) arises more frequently in the frontal lobe than in the extrafrontal regions. We classified seizure semiology according to four semiology patterns (SPs)

1. elementary motor signs (SP1)
2. unnatural hypermotor movements (SP2)
3. integrated hypermotor movements (SP3)
4. gestural behaviors with high emotional content (SP4).

RESULTS:

Within the frontal lobe, SPs were organized in a posteroanterior gradient (SP1-4) with respect to the SOZ. In temporal SHE, SP1 was rare and SP3-4 frequent, whereas in operculoinsular and posterior SHE, SP4 was absent. Nonmotor manifestations were frequent (70%) and some could provide valuable localizing information.

Epilepsia. 2018 Sep;59(9):e130-e134. doi: 10.1111/epi.14517. Epub 2018 Jul 15.

Seizure duration and latency of hypermotor manifestations distinguish frontal from extrafrontal onset in sleep-related hypermotor epilepsy.

Gibbs SA^{1,2}, Proserpio P¹, Francione S¹, Mai R¹, Cossu M¹, Tassi L¹, Nobili L^{1,3}.

Sleep-related hypermotor epilepsy (SHE) is an epilepsy syndrome that is characterized by the occurrence of sleep-related hypermotor seizures of variable complexity and duration. Seizures usually arise in the frontal lobe, but extrafrontal seizure onset zones are well described.

We found that the mean duration of electrographic seizures and clinically observable ictal manifestations were significantly shorter in frontal SHE compared to extrafrontal SHE.

The mean latency between electrographic seizure onset and the onset of hypermotor manifestations was also shorter in frontal SHE. Accordingly, a latency > 5 seconds between the first video-detectable movement (eg, eye opening or a minor motor event) and the onset of hypermotor manifestations yielded a sensitivity of 75% and a specificity of 90% for an extrafrontal onset, thereby indicating that specific ictal features in SHE can provide clinically useful clues to increase diagnostic accuracy in this syndrome.

1. Duration of EEG changes and symptoms shorter in FLE
2. Mean latency EEG → Clinic shorter in FLE
3. Latency EEG → Clinic >5 sec: extraFL origin

Clinical Manifestations of Insular Lobe Seizures: A Stereo-electroencephalographic Study

*Jean Isnard, †Marc Guénot, †Marc Sindou, and *François Mauguière

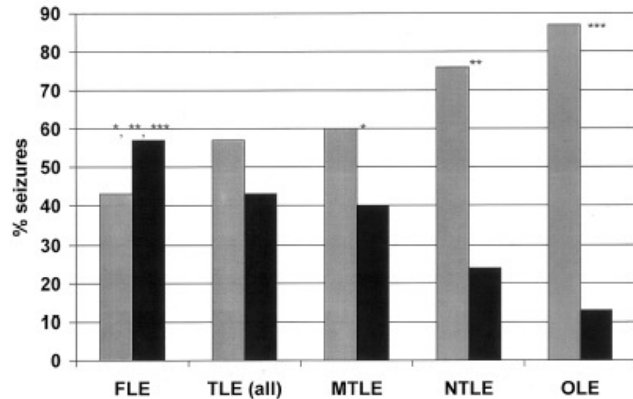


Figure 2. Percentage of partial seizures arising during sleep from various seizure onset zones. Frontal lobe seizures were more likely to occur during sleep than seizures arising from other foci ($p < 0.0001$). FLE = frontal lobe epilepsy ($n = 112$), TLE = temporal lobe epilepsy ($n = 382$), MTLE = mesial temporal lobe epilepsy ($n = 164$), NTLE = neocortical temporal lobe epilepsy ($n = 54$), OLE = occipital or parietal lobe epilepsy ($n = 45$). Gray bars, awake; black bars, asleep. * $p < 0.05$; *** $p < 0.001$; **** $p < 0.001$.

Herman, et al

NEUROLOGY 2001;56:1453–1459

Results: This ictal sequence occurred in full consciousness, beginning with a sensation of laryngeal constriction and paresthesiae, often unpleasant, affecting large cutaneous territories, most often at the onset of a complex partial seizure (five of the six patients). It was eventually followed by dysarthric speech and focal motor convulsive symptoms. The insular origin of these

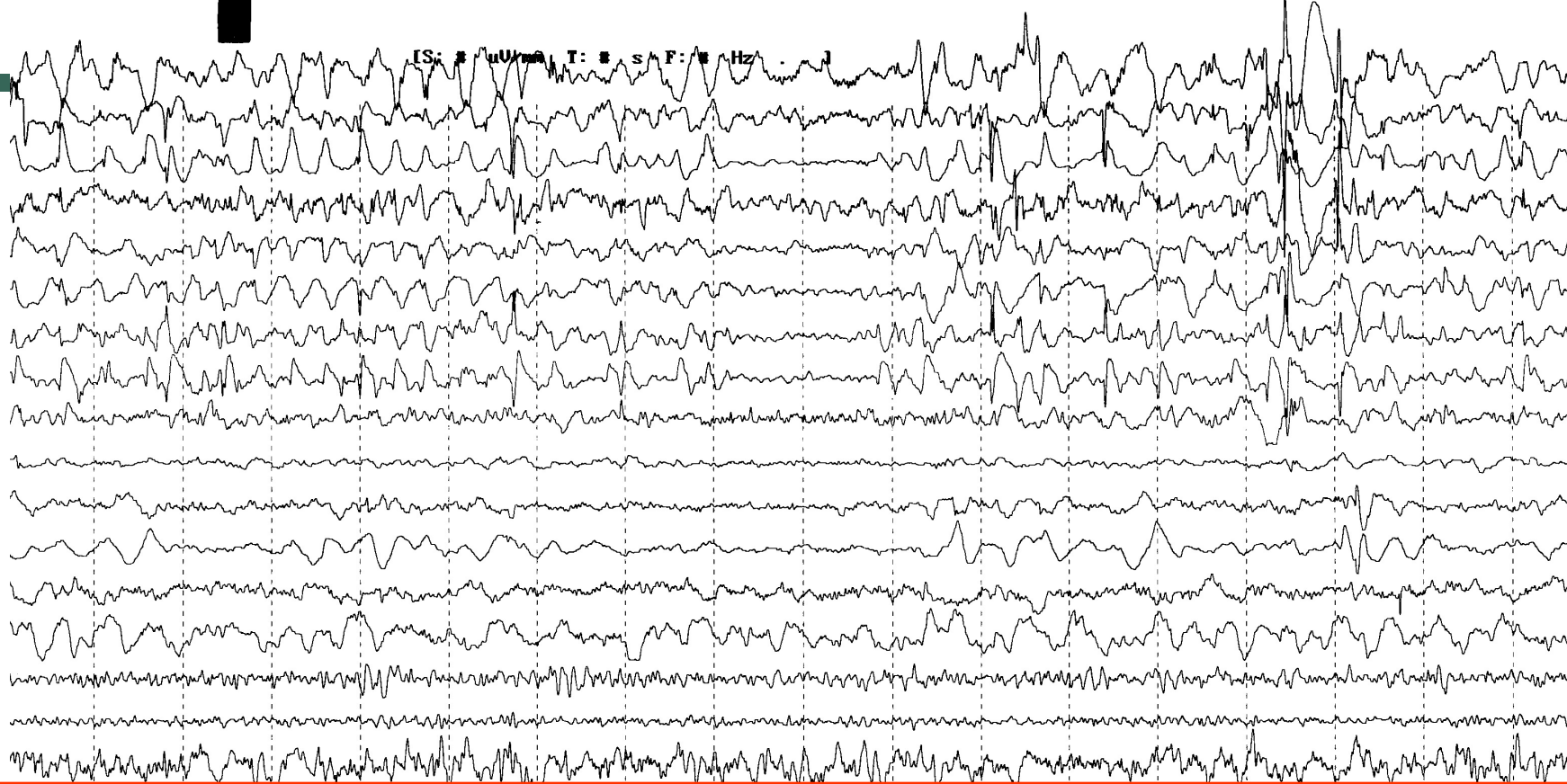
Nocturnal Hypermotor Seizures, Suggesting Frontal Lobe Epilepsy, Can Originate in the Insula

*Philippe Ryvlin, †Lorella Minotti, *Geneviève Demarquay, §Edouard Hirsch, ¶Alexis Arzimanoglou, †Dominique Hoffman, †Marc Guénot, ¶Fabienne Picard, *Sylvain Rheims, and †Philippe Kahane

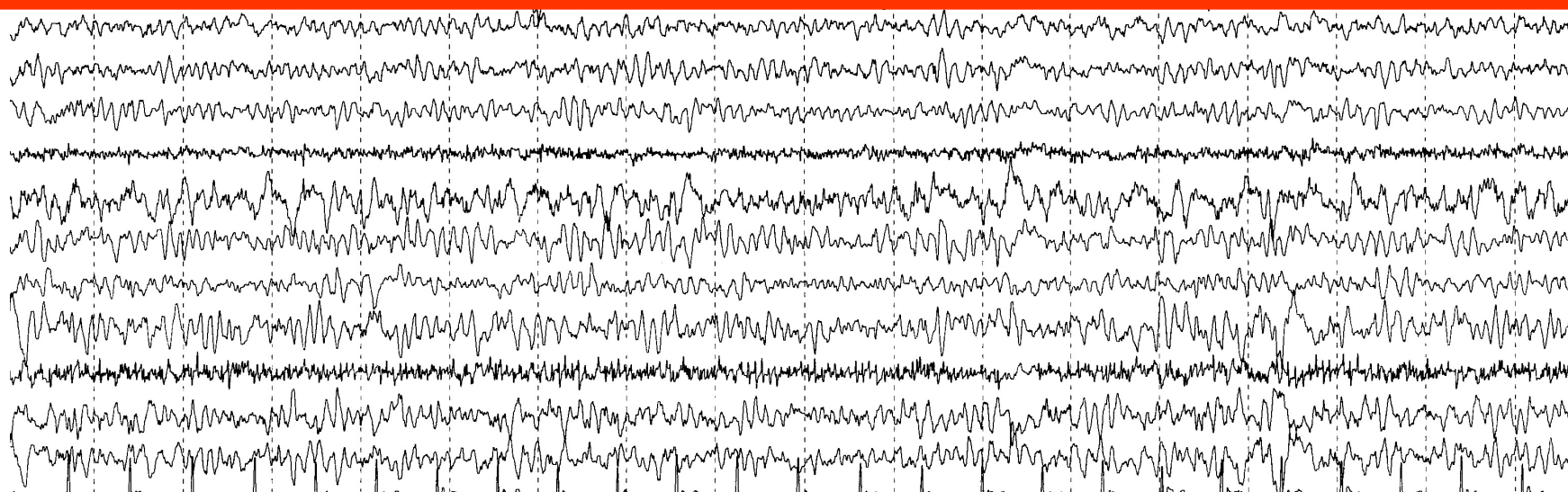
- 3 pts Ryvlin et al., 2006
- 1 pt Kaido et al., 2006
- 1 pt Dobesberger et al., 2008
- 1 pt Nguyen et al., 2009

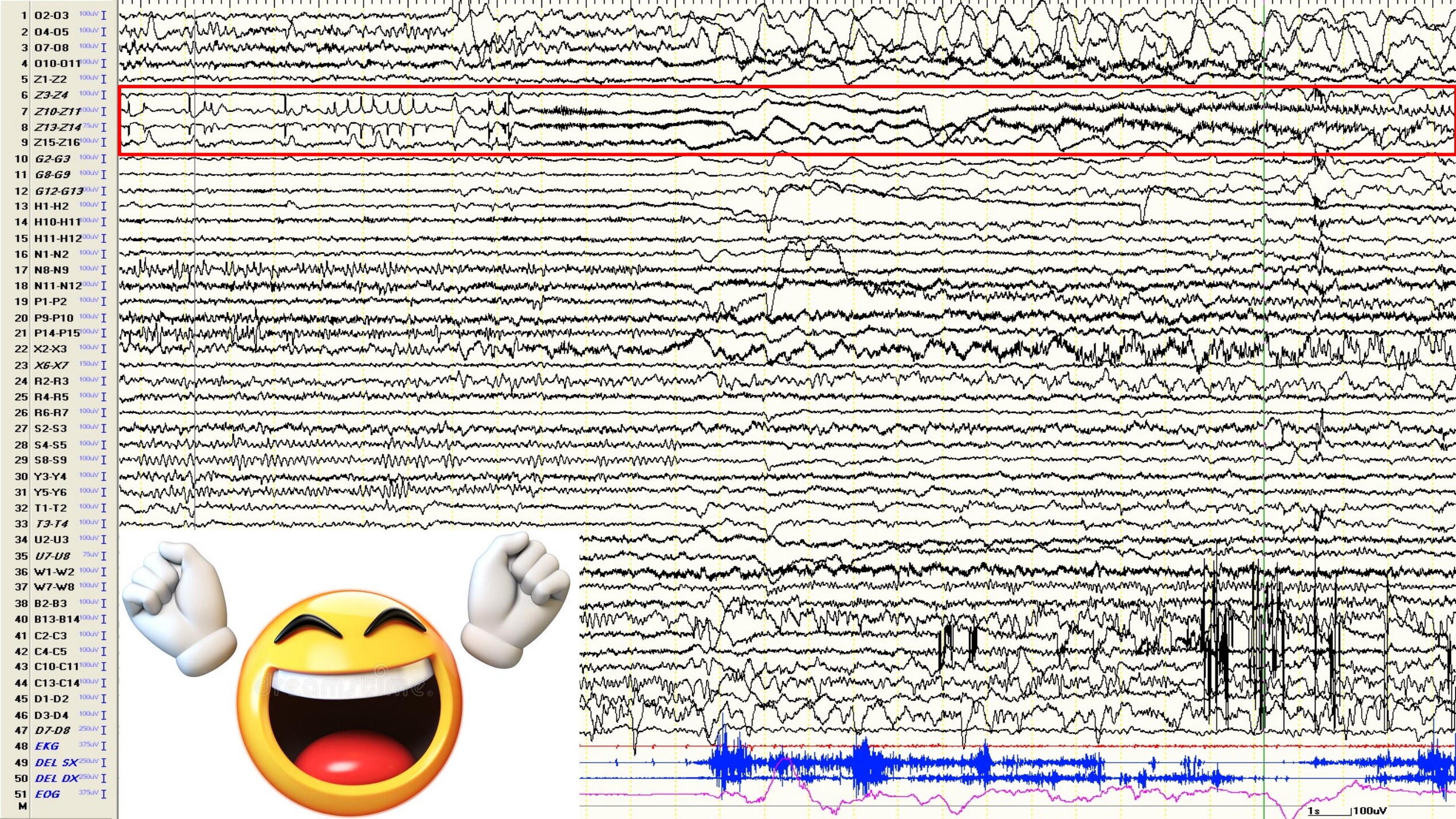
The ictal EEG

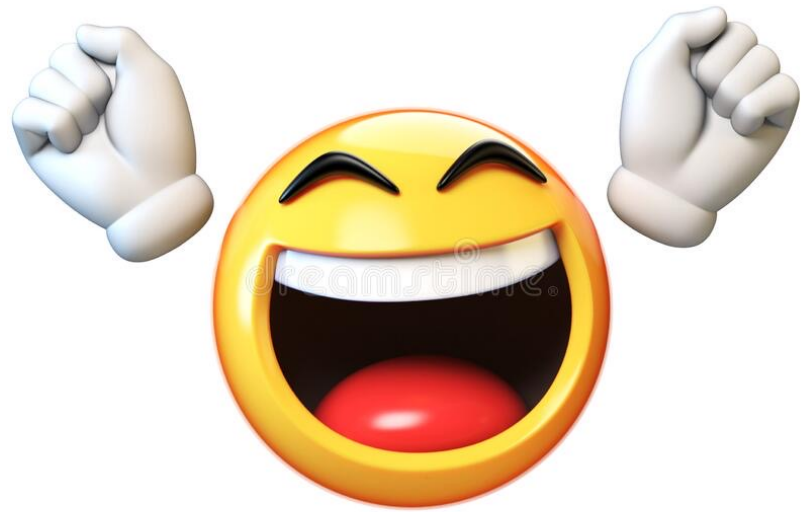
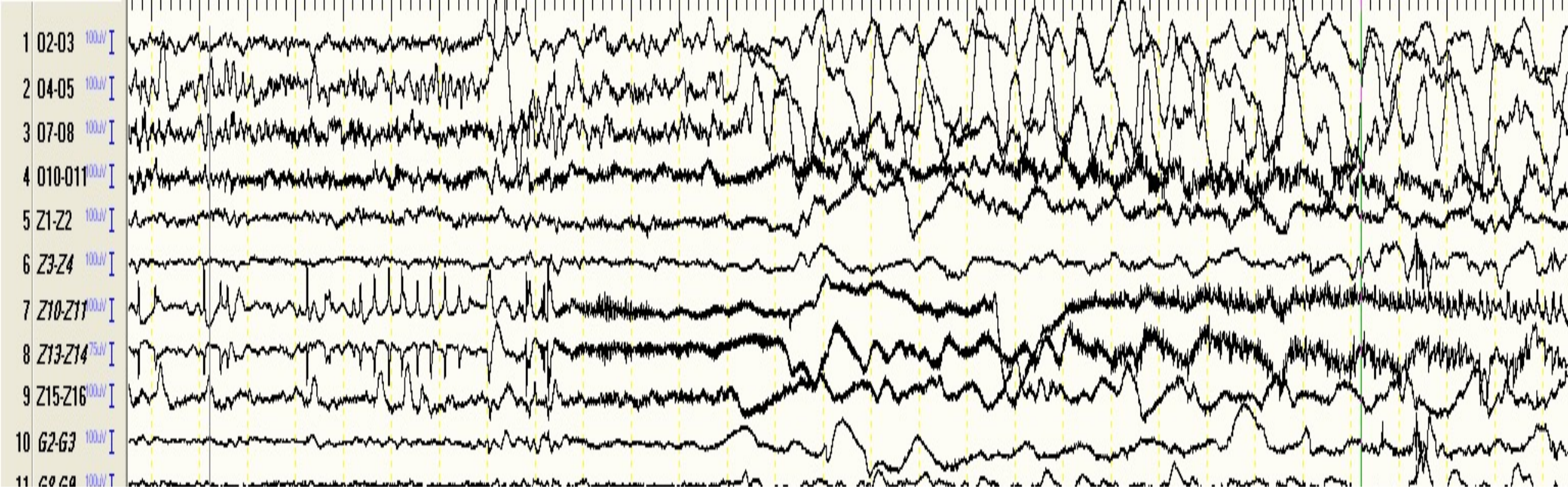
SEEG

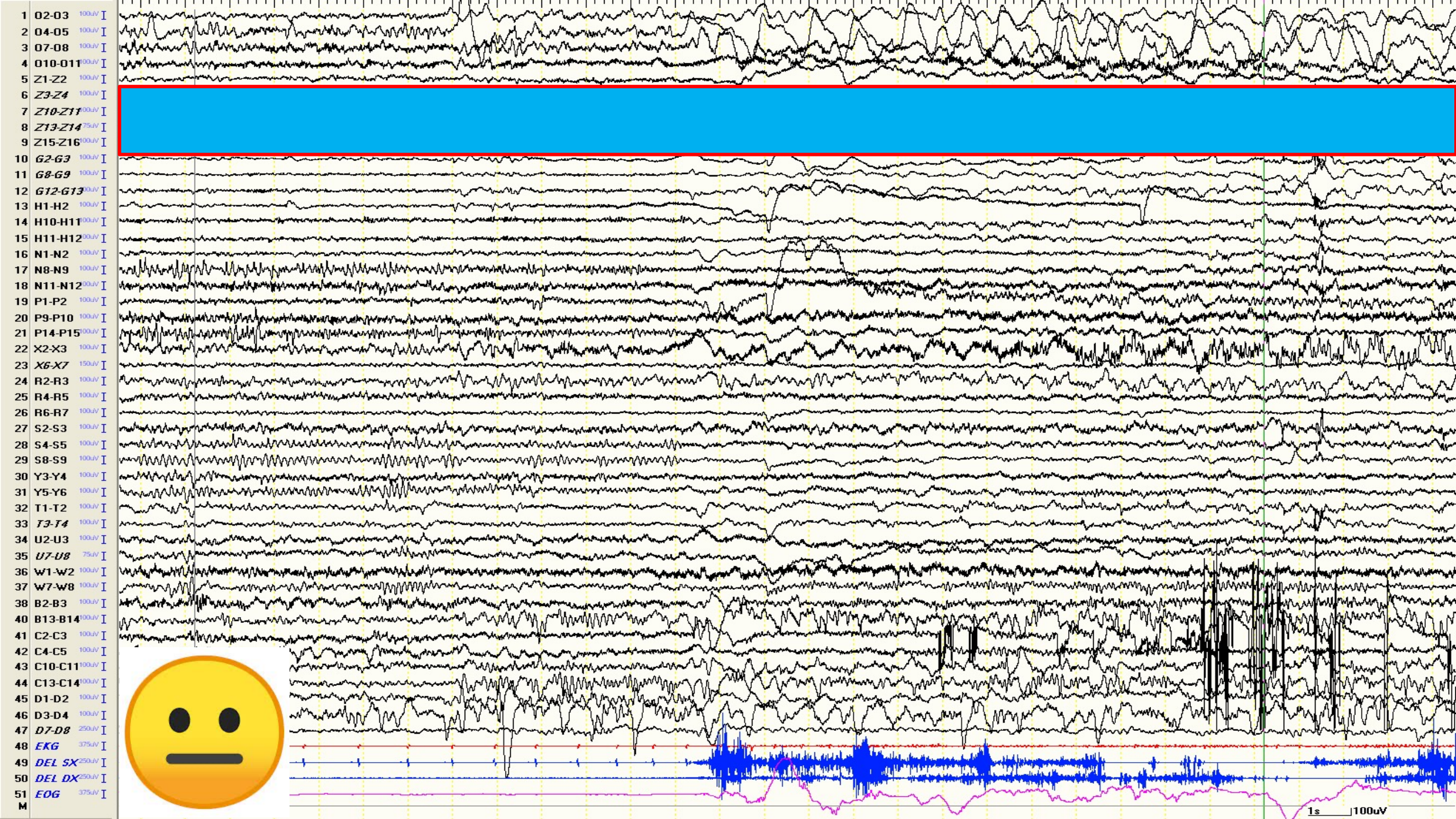


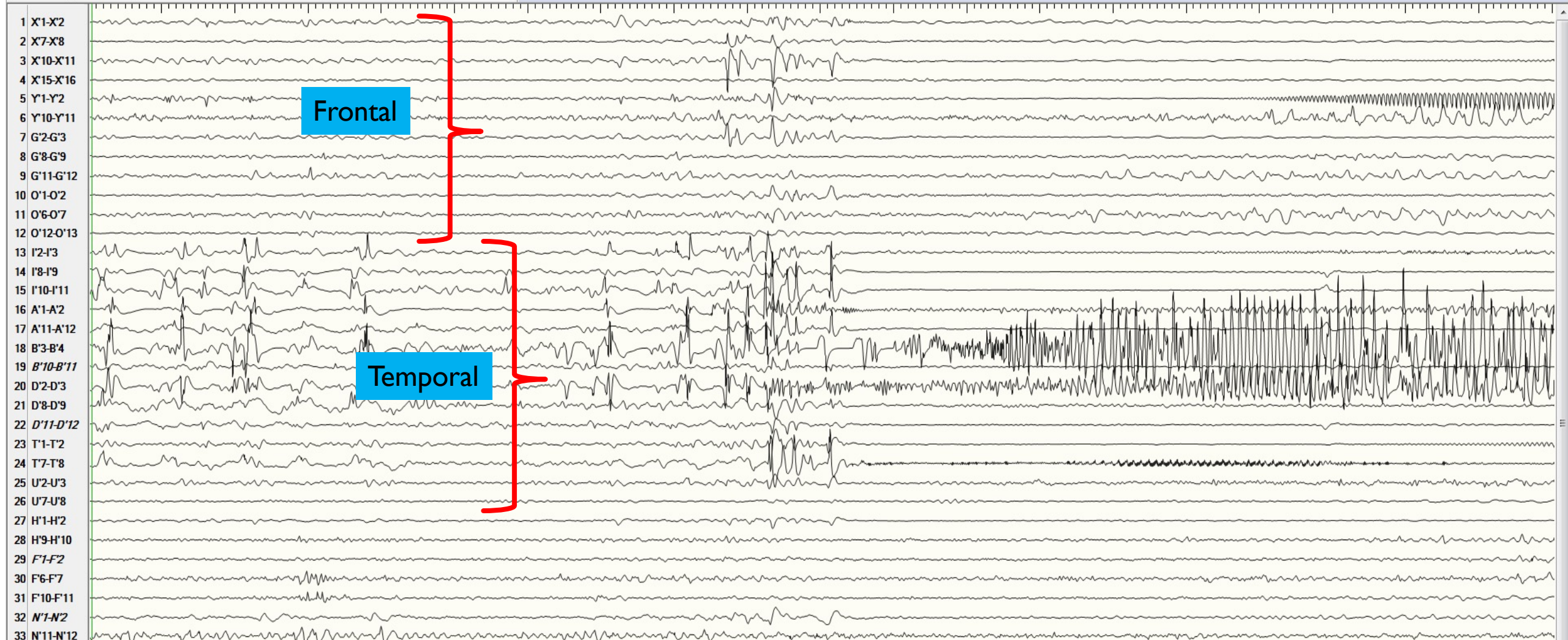
VEEG





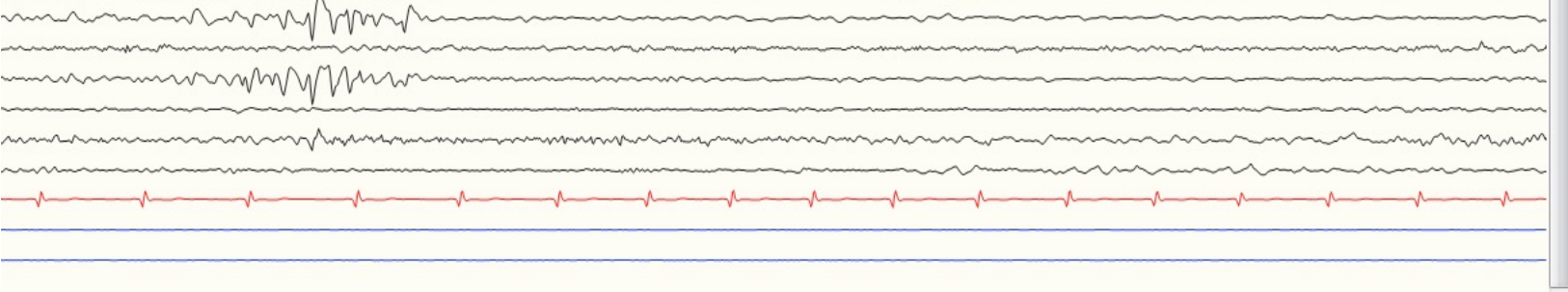
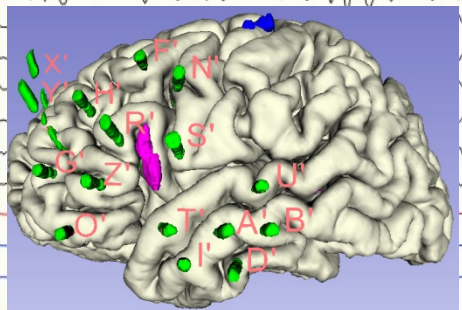


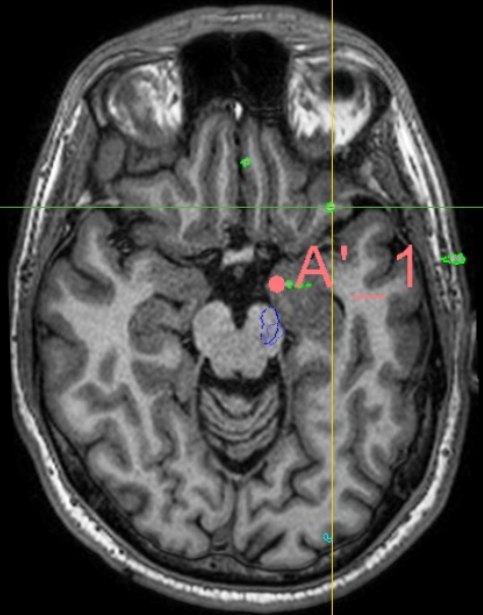




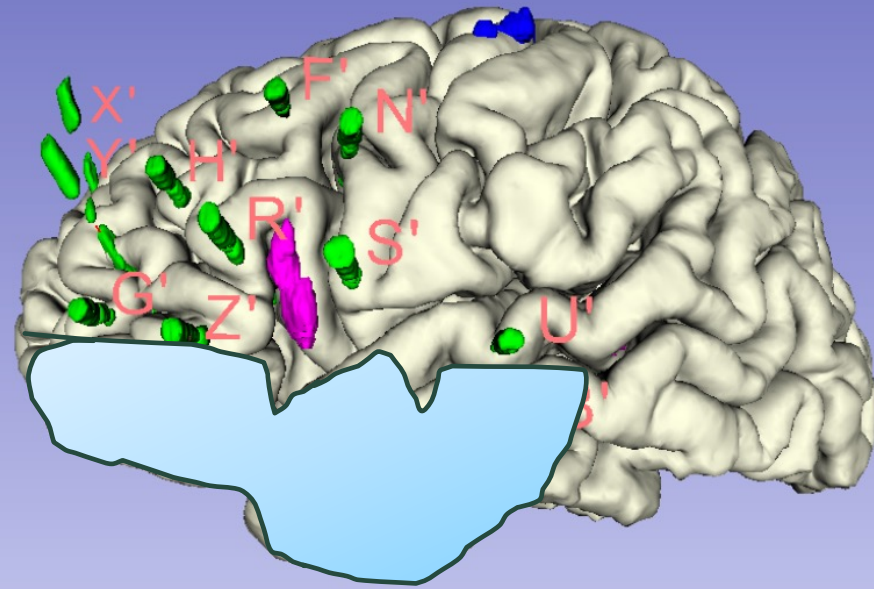
Frontal

Temporal





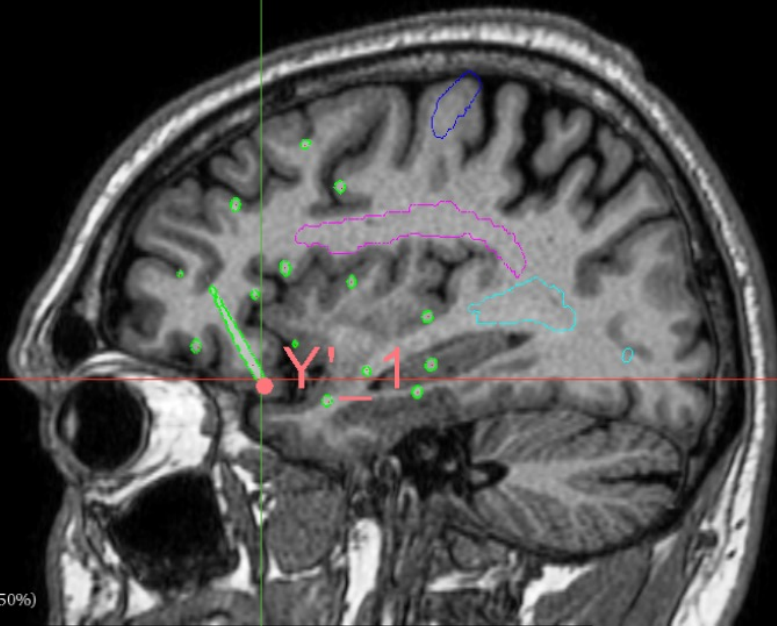
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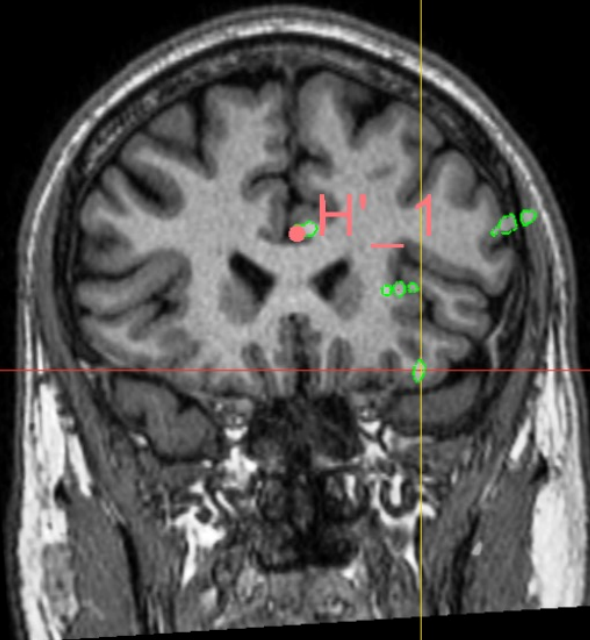
R: -33.508mm

G

A: 42.055mm

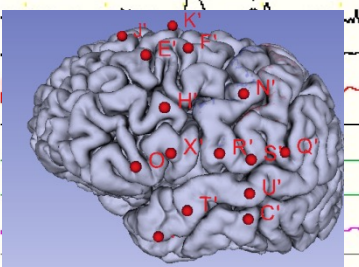
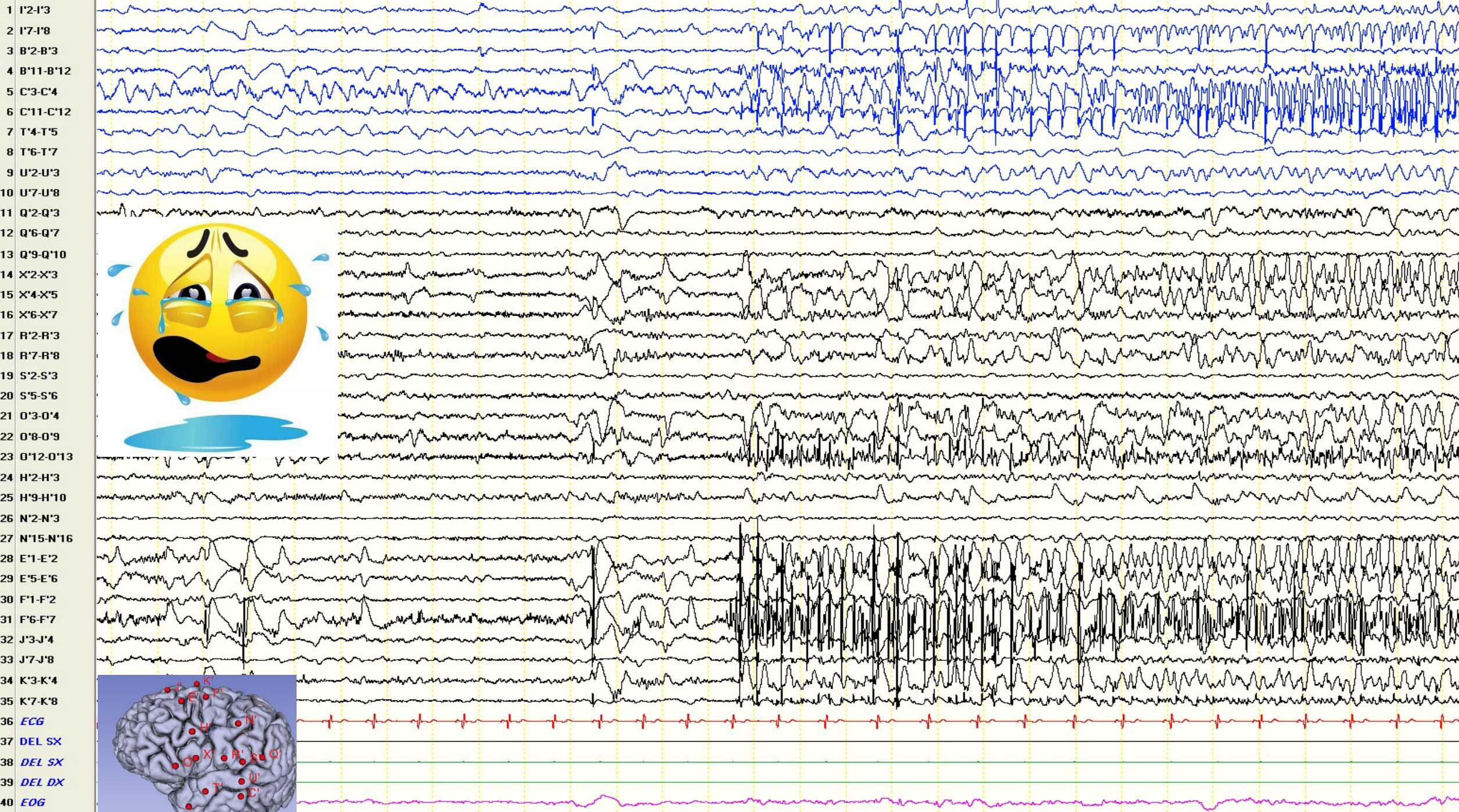


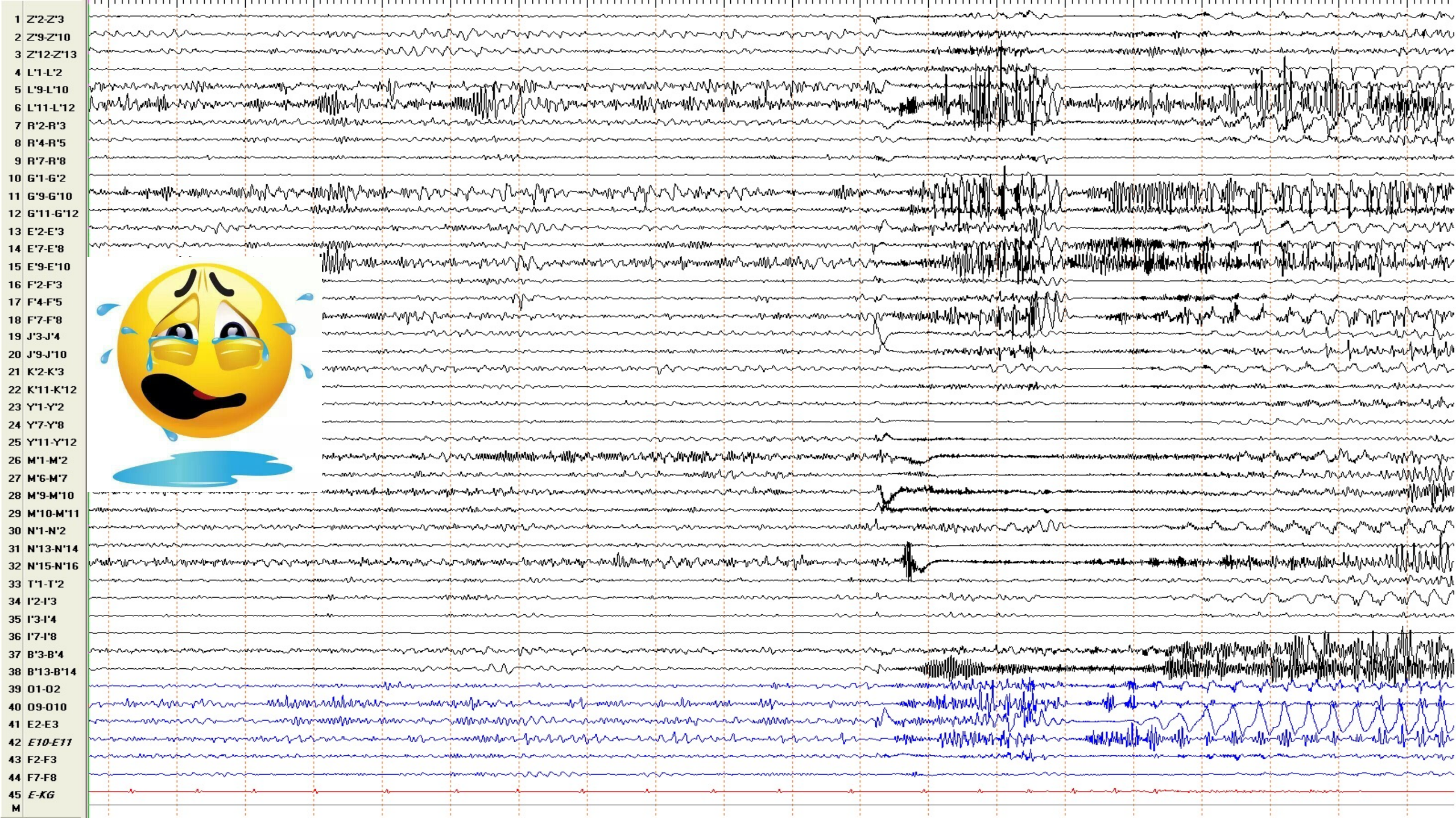
_seeg (50%)

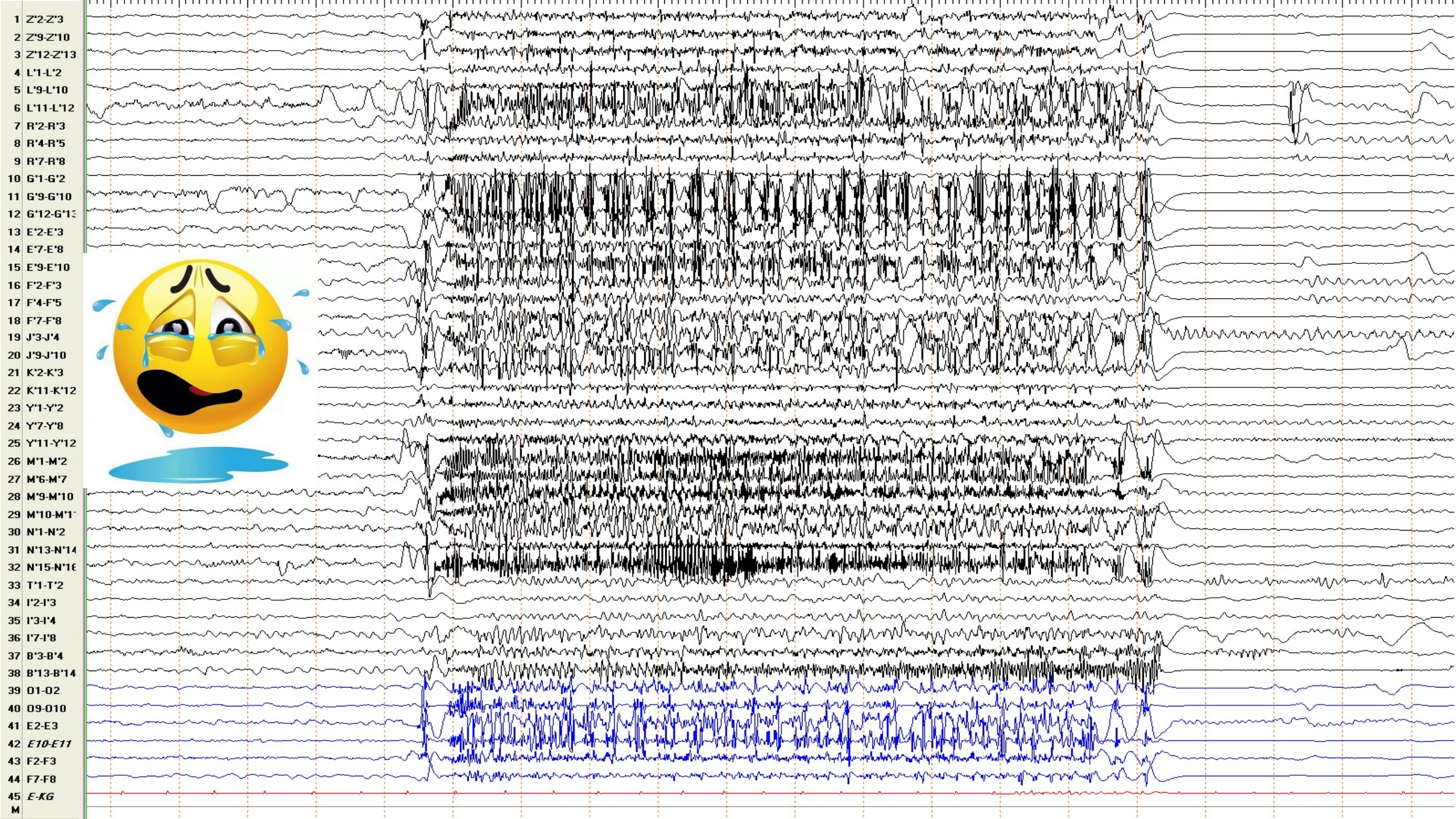


F: ref_plus_seeg (50%)
B: ref





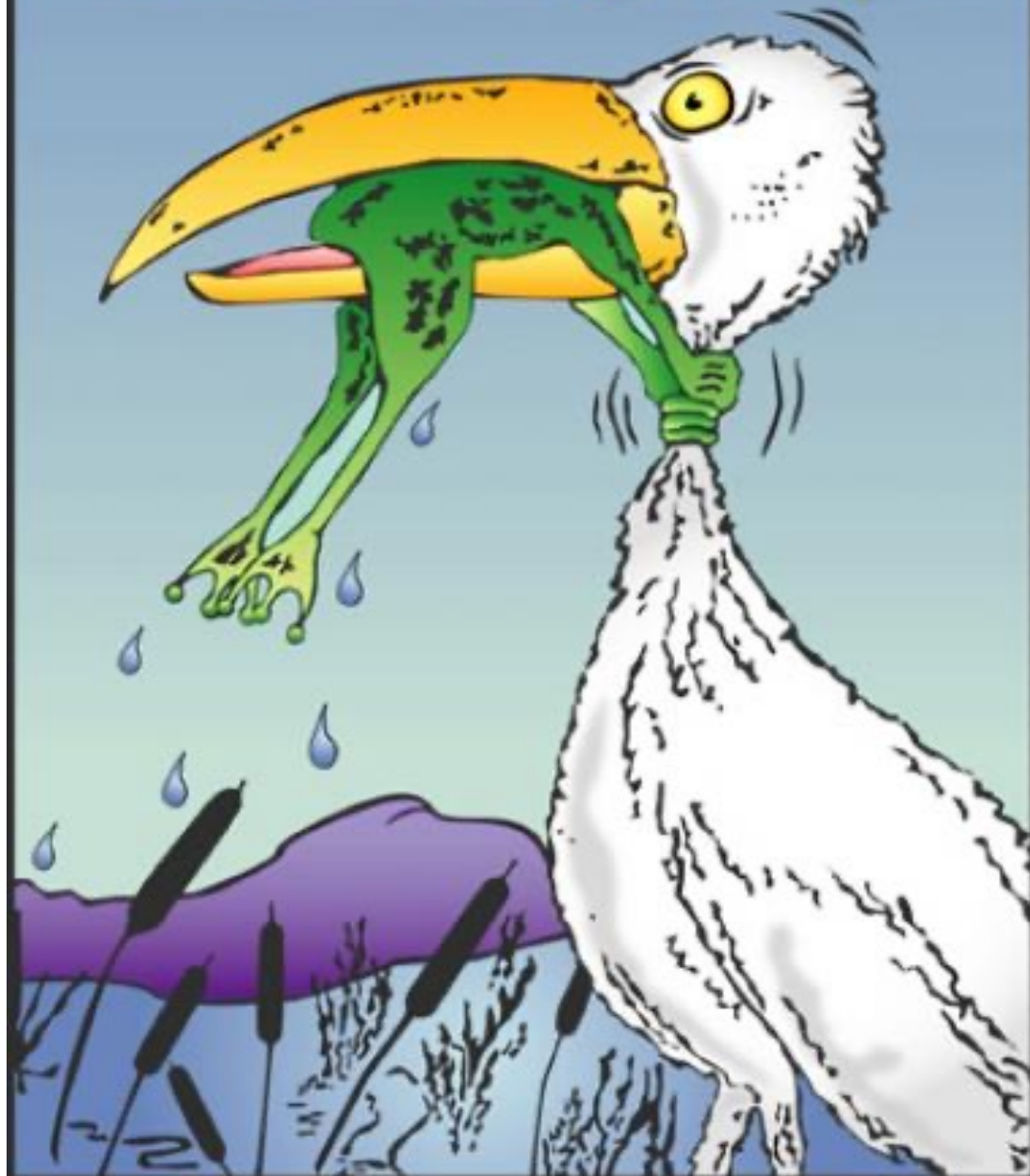




CONCLUSIONS

- **Huge lobe, very well connected, mimicking semeiology.**
- **Ictal symptoms depend on etiology, localization, age.**
- **The localizing diagnosis is based on anatomico-electro-clinical data.**
- **Clinical manifestations chronology is mandatory.**
- **Very difficult interictal and ictal EEG.**

Never ever give up!





Clinical Neurophysiology

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Ivana Sartori



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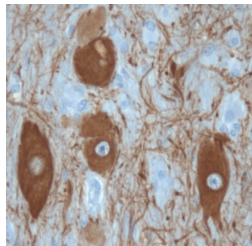
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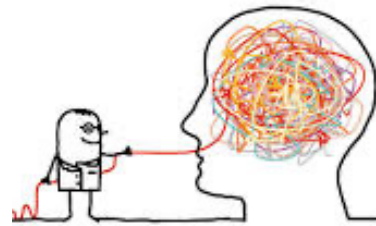
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