



EEG reviewing, BCNEPS tutorial

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www.bcneps.org

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Rev.class

BCNEPS 1 yr certification course 2020-21

- What is our aim?
 - A Clear idea about cl NP, EEG
 - Able to perform a routine test using 10-20 electrode placement technique.
 - Able to identify normal background activity for the age and state
 - Able to identify cerebral reactivity to external stimuli
 - Able to identify common epileptogenic discharges
 - Able to understand a report while you treat a child
- What is Expected at the end of LI ?
 - Perform a routine EEG record in children and adult independently
 - Communicate with referring doctor about the test and the patient
 - Write a descriptive review
 - Understand a report made by an expert neurophysiologist
- What next?
 - SKY IS THE LIMIT
 - LIFE TIME IS THE LIMIT

How much we achieved?

What are the Clinical Neurophysiology tests?

NeuroInvestigative tests

- **EEG:** Routine and special
- Polysomnography (PSG), Actigraphy
 - **Epilepsy, Encephalopathy**, sleep disorders
- **EVOKED potentials:** VEP, ERG, **AEP**, SEP
- **EMG, NCV studies**

EEG in Therapy: Neurofeedback, BCI

- ADHD
- ASD

What CL NP tests can do



	Confirms	Exclude	Helps
EEG	Seizure & Epilepsy syndrome	Non-epileptic Paroxysms Epileptic cause for psychomotor deficit	Metabolic dis. Inf Battnes dis
	Epileptic encephalopathy	Epileptic cause for Transient Behavioral disorder by special technique	Non-Ketotic hyperglycemea
	Encephalopathy / Encephalitis	Paroxysmal event	Structural anomaly
	Acute stroke syndrome Retts syndrome		Predict Neurodev. Outcome
	Localized cerebral dysfunction		Underlying cause
	SSPE at early stage	Myoclonic epilepsy syndrome	
VEP	Maturation		Neurometabolic
ERG	Ant .Chamber defect		Neurometaboic dis
EMG, NCV	Neurogenic	Pathology at Peripheral Nervous system	Disorder of PNS
	Myogenic, Myopathic	Neuro-muscular junction	Muscle and
	Neuro –muscular junction defect	Muscle	Neuromascular junction



What does the EEG record?

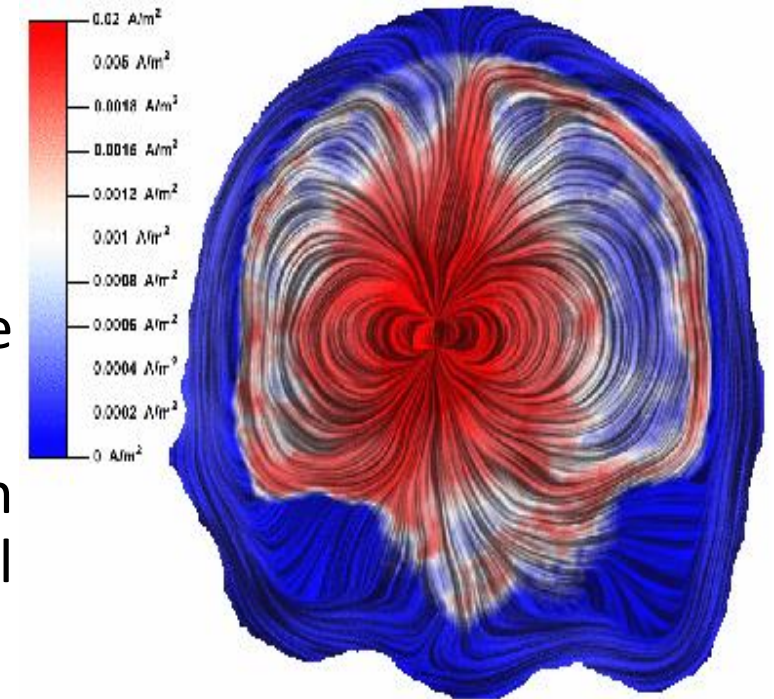
Mainly NOISE!!

Volume Conduction

The electrical activity flows through the tissue between the electrical generator and the recording electrode.

Thus, the EEG is a 2-D representation of a 3-D reality, which poses a problem in localizing the sources of the electrical activity

Inverse problem: Simulating the potentials at the electrode positions from current sources inside the brain is known as the EEG forward problem; **inference of the position of the current sources from electrode potentials is known as the EEG inverse problem or the neural source imaging problem** (Grech et al., 2008, Brannon et al., 2008)





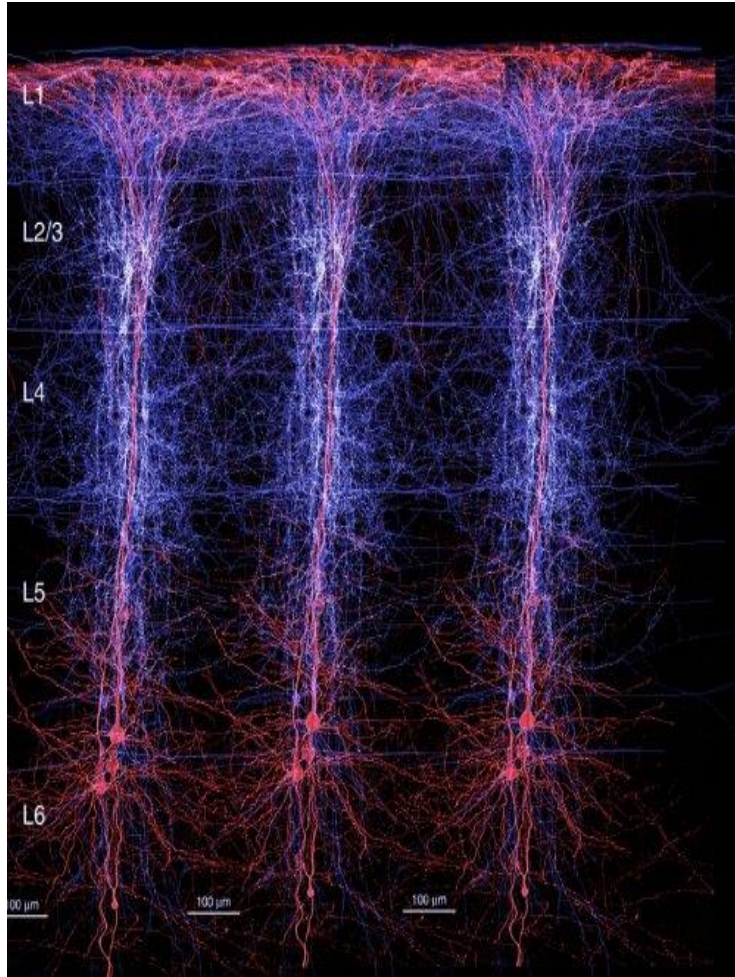
NP Basis of EEG

The neurophysiological basis of EEG can be summarized conveniently as follows at present

1. Repeated waves recorded from scalp or from brain surface are summated **Synaptic potential** generated by the **pyramidal cells**
2. **Synaptic Potentials are the response of the cortical cells to the rhythmic discharges from thalamic nuclei**
3. Frequency and size of the thalamic discharges (hence the cortical potentials) are determined by the special arrangement of excitatory and inhibitory interconnections among the thalamic cells
4. During 'activation' inputs from reticular formation abolish the rhythmic discharges in the thalamic nuclei and cause the cortical potentials to become desynchronized.



Neural basis of the EEG



The **principal generators of EEG fields** measured at the scalp are graded **synaptic potentials**; i.e., EPSPs and IPSPs of the **pyramidal neurons**.

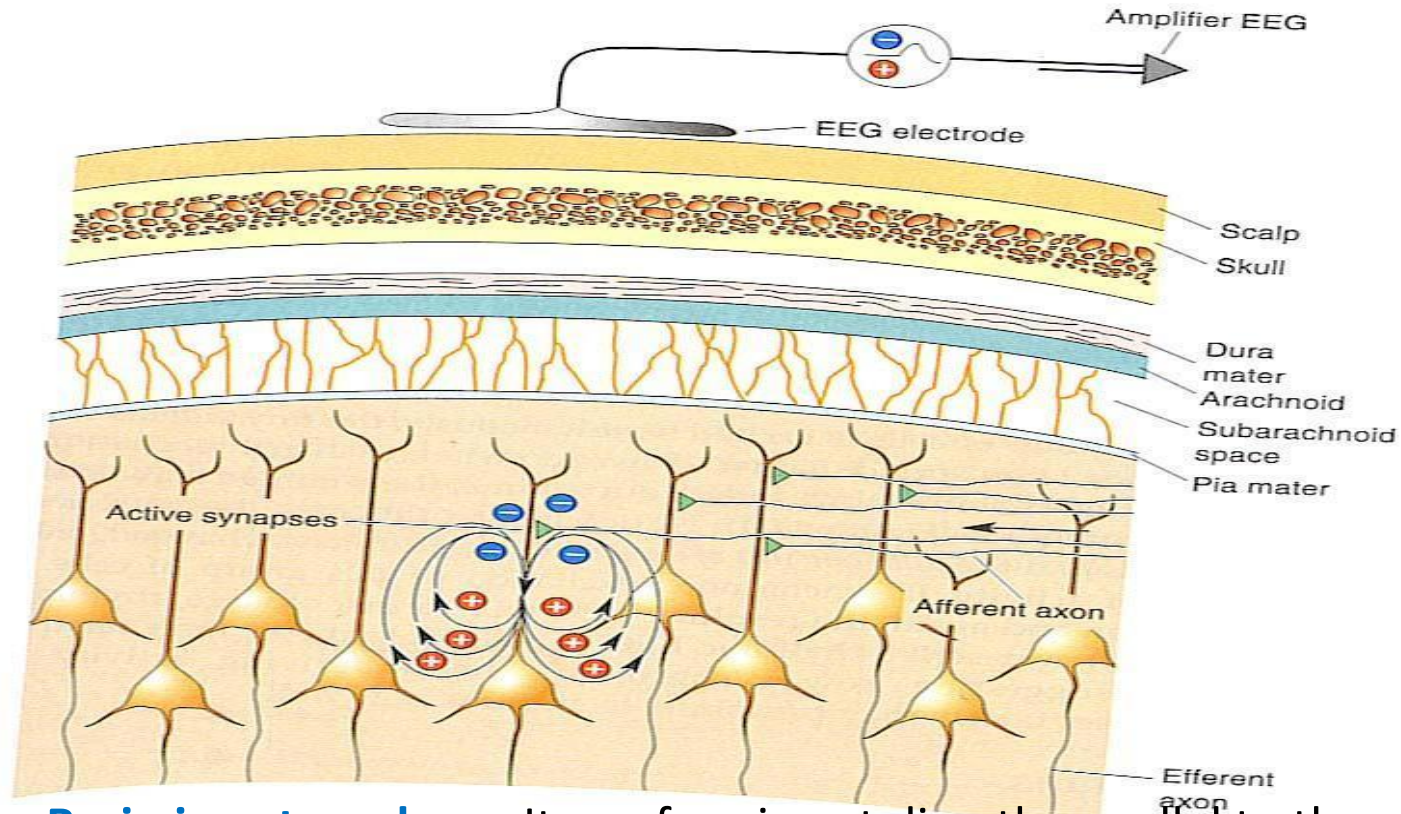
At the synaptic site of an EPSP there is an **active current sink (extracellular negative field)**.

Positive ions migrate to the cell and depolarize the membrane. At the distal part of the cell (body and distal dendrites) a passive current source out of the cell is associated with **extracellular positive field**.

EEG fields are primarily generated by the large, vertically oriented pyramidal neurons located in cortical layers III, V, and VI.

The electrical activity from deeper generators gets dispersed and attenuated by volume conduction effects.

EPSPs and IPSPs are response of the cortical cells to the rhythmic discharges from thalamic nuclei



EEG, fallacy

1. **Brain is not a sphere**, Its surface is not directly parallel to the overlying structure where electrodes are placed
2. **Each small electrode is averaging the activity within 1inch** area, limit of such region depends on many factors
3. **Slight alteration of electrode placement would result in the electrode averaging the activity from a different region of the brain**
4. **Appropriate electrode placement and Montage setting is important for localizing the disturbed area**
5. **All EEG record are practically bipolar event when theoretically unipolar**



How to collect and organize the data

- **During recording:**
 - Machine calibration,
 - Impedance checking,
 - removing artifacts,
 - patient care & activation procedure **during data collection**
- Montage: To organize data for reviewing

Commonly used montages

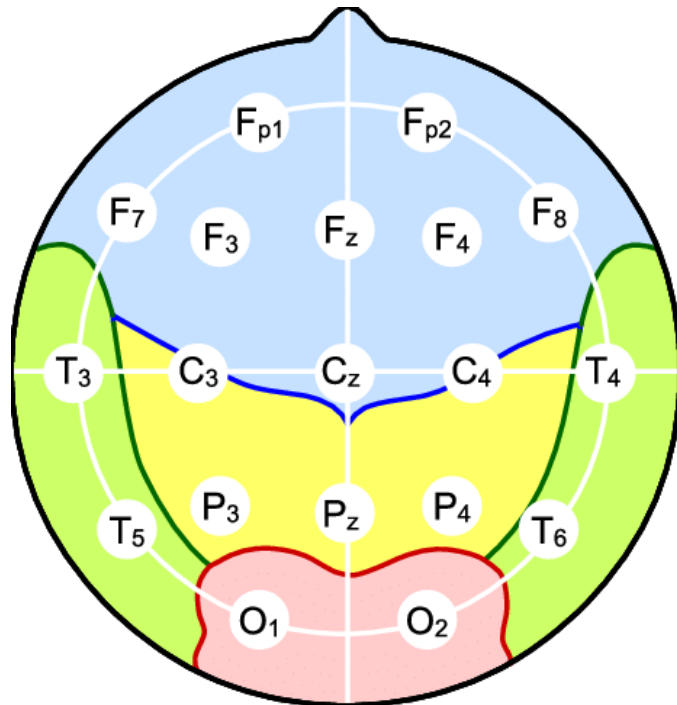
- Longitudinal OR anterior posterior (M1)
- Transvers (M2)
- Multiphotic (M3)
- Average (M4)
- Referential (M7)



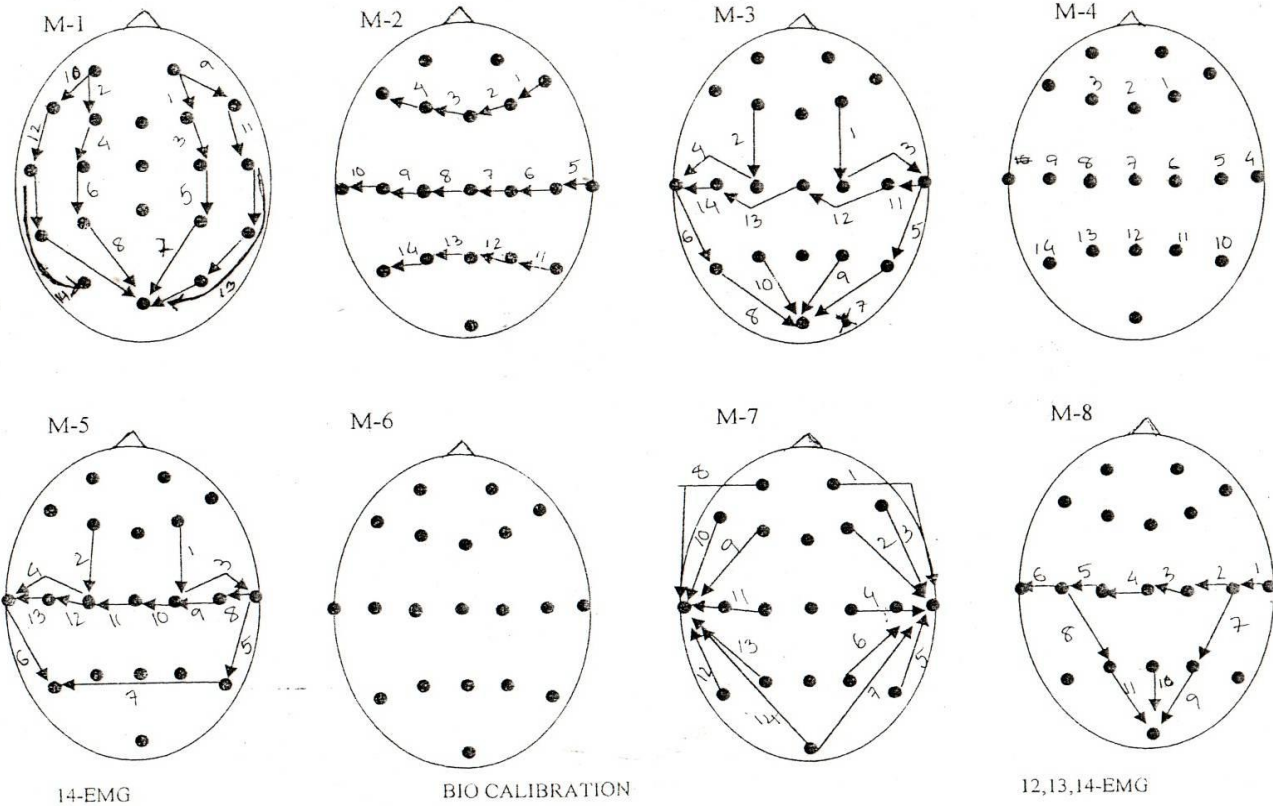
The letters used are:

- F - Frontal lobe
- T - Temporal lobe
- C - Central lobe
- P - parietal lobe
- O - Occipital lobe

"Z" refers to an electrode placed on the mid-line



Reviewing: Montage we use





Musts: for EEG reviewing

- Patient's Age, sex, state
- Equipment Setting
- Knowledge about
 - Recording technique
 - Normal BG. activity for the age and state (normative data and their variations)
 - Cerebral reactivity to activation procedure
 - Artifacts
 - Well established/described cerebral dysfunction

**Repeated reviewing
use all the montage**

EEG reviewing: organize your description

- Background activity
- Reactivity to eye closure
- Effect on PS
- Hyperventilation
- Drowsy state
- Sleep stages:
 - Early sleep stage/ drowsy state
 - Stages 1st, 2nd, 3rd, 4th
 - REM sleep
- On awakening
 - Arousal phenomena

WHAT TO LOOK FOR IN EEG RECORD?

Background ACTIVITY (*spontaneous and reactivity to stimuli*)

- **Symmetry:** IN voltage/ amplitude, IN frequency/ Hz / ____ c/sec

- Mild asymmetry
- Marked asymmetry

Reactivity

- to eye closure
- Photic stimulation
- Hyperventilation
- Activities in Drowsy state
- Sleep state

- **Discharges** of **spike**, **polyspikes**, **sharp** waves/transients, **polyspike-wave complexes**

- Transient (focal, multifocal, bursts) /paroxysms/ persistent/ runs
- Localization, describe the appearance of discharges
- Repeated, periodic, Persistent
- Synchrony / asynchrony

- Bursts (transient bursts/ recurrent/ periodic/ repeated
- Stretches of attenuation, periods of attenuation, Suppression
- Discontinuous/ Continuous
- Specific pattern (Hypsarrhythmic/ B-S/ PLED/CSWSS



EEG Rhythms

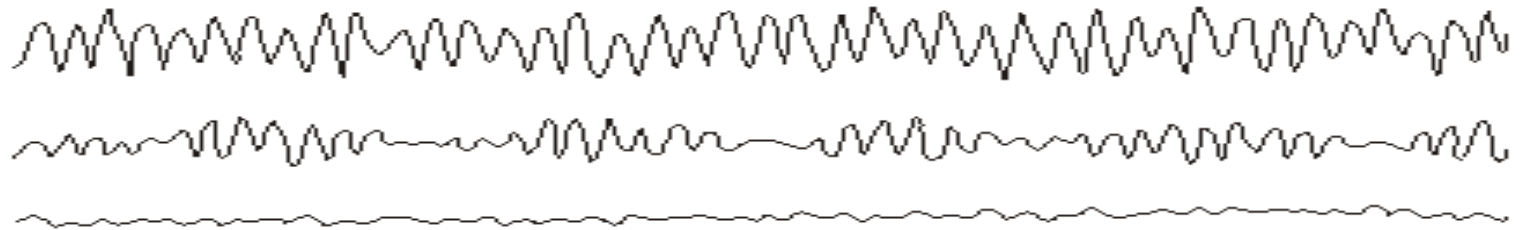
Beta (β) 13-30 Hz

Parietally and frontally



Alpha (α) 8-13 Hz

Occipitally



Theta (θ) 4-8 Hz

Children, sleeping adults



Delta (δ) 0.5-4 Hz

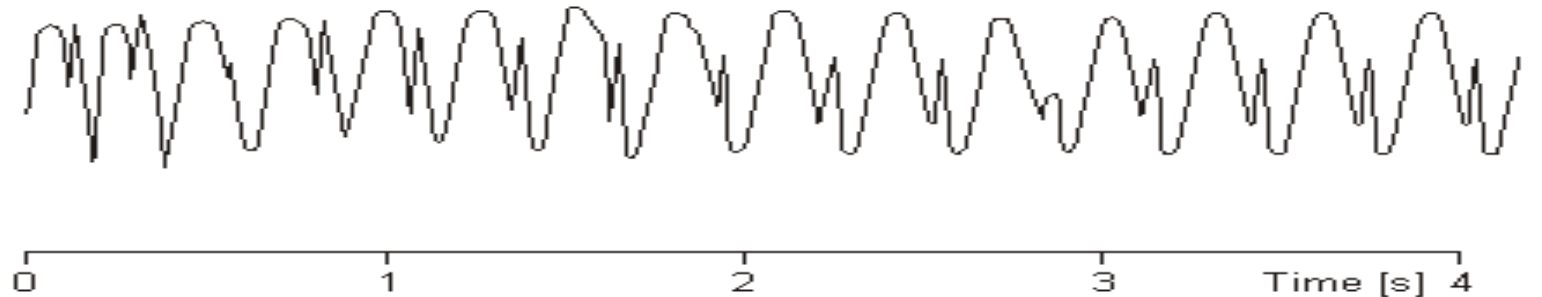
Infants, sleeping adults



Spikes 3 Hz

Epilepsy - petit mal

200
100
0
V [μ V]



Spikes range from 20 to less than <70 milliseconds (ms) High-amplitude!

Sharp waves pointed peak and duration of about 70 to 200 ms

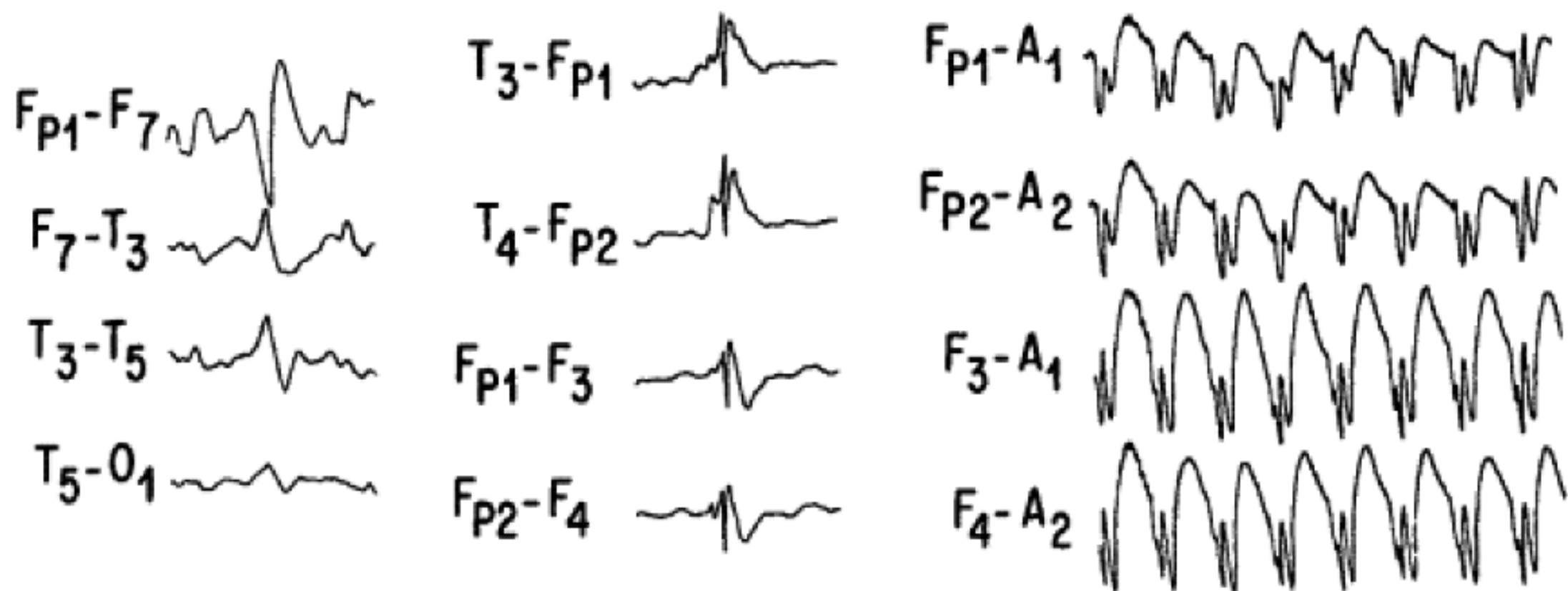
Spike-wave complexes (SWCs) are the repetitive occurrence of a *Spike* followed by a *Slow Wave*, although a run of 3 seconds is required to classify a record as SWC.

Polyspikes are multiple repetitive spikes occurring at about 20 Hz

Hypsarrhythmia is defined as continuous (during wakefulness), high-amplitude (>200 Hz), generalized polymorphic slowing with no organized background and multifocal spikes

Abnormalities	
Spike	
Sharp wave	
Spike-and-wave	
Polyspike	
Polyspike-wave	

EPILEPTIFORM DISCHARGES



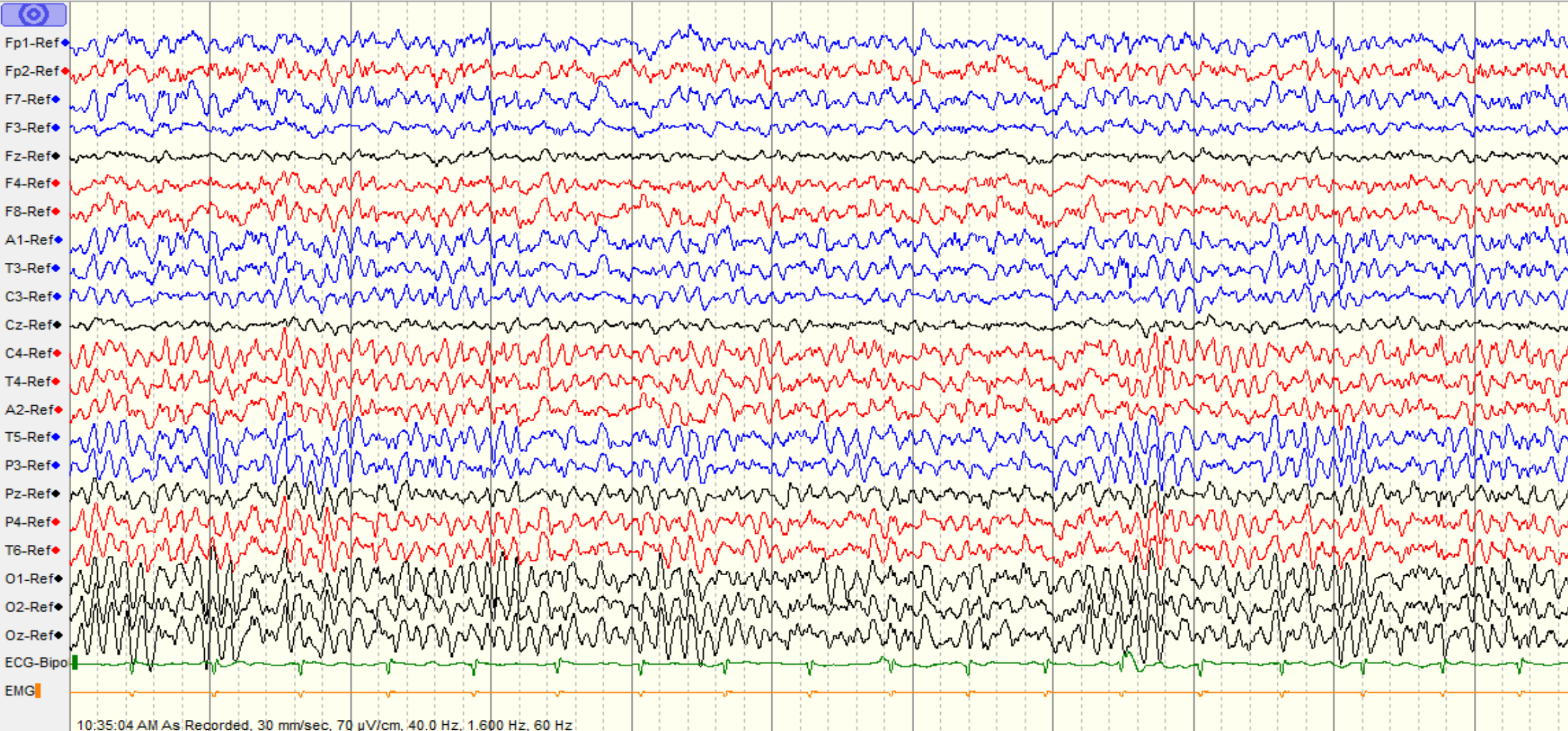
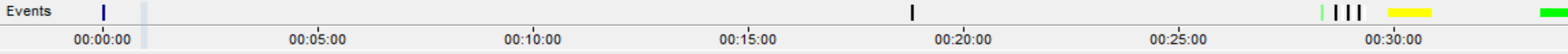
Sharp wave
(> 70 msec)

Spike
(< 70 msec)

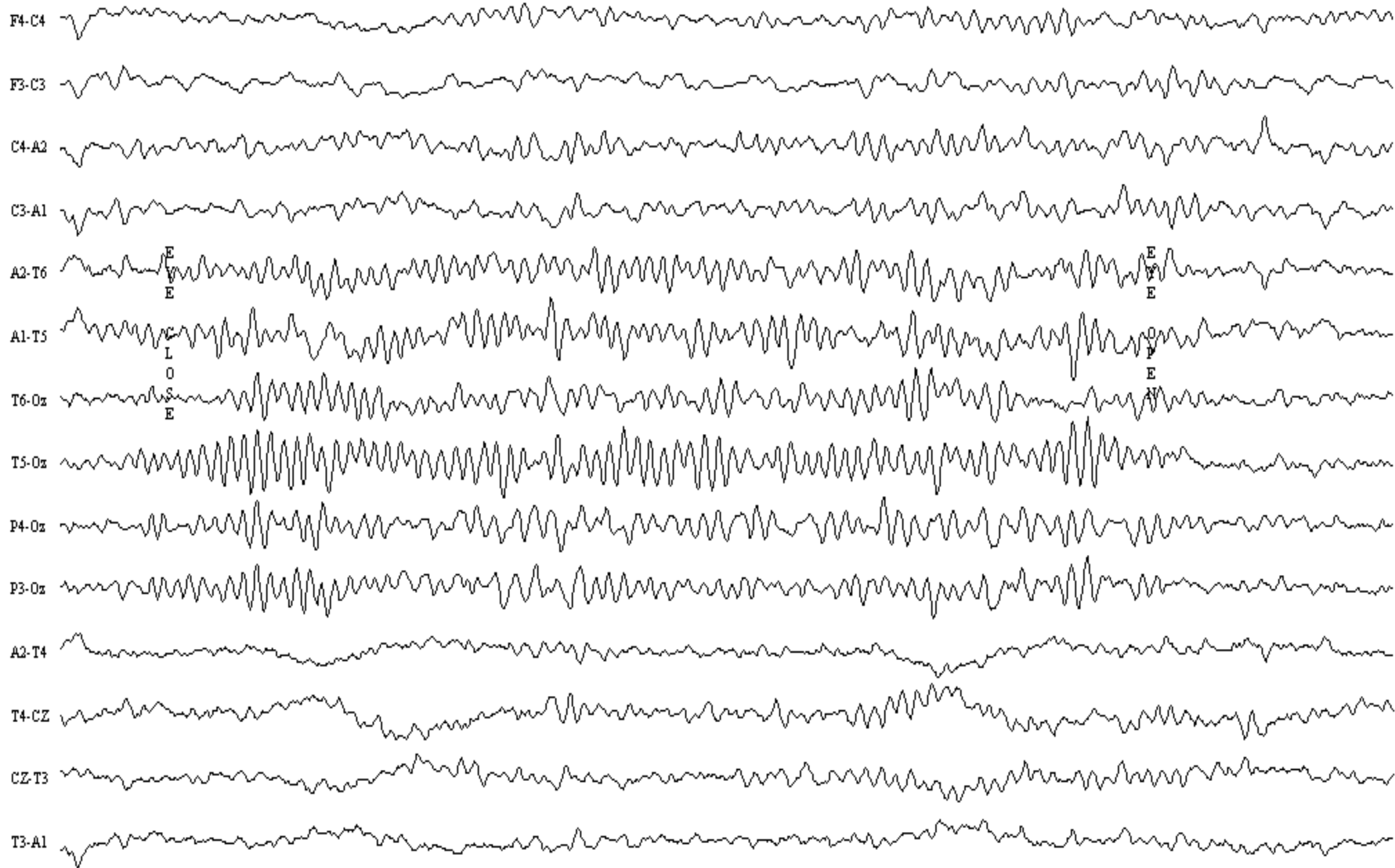
Spike and wave

100 μV
1 sec

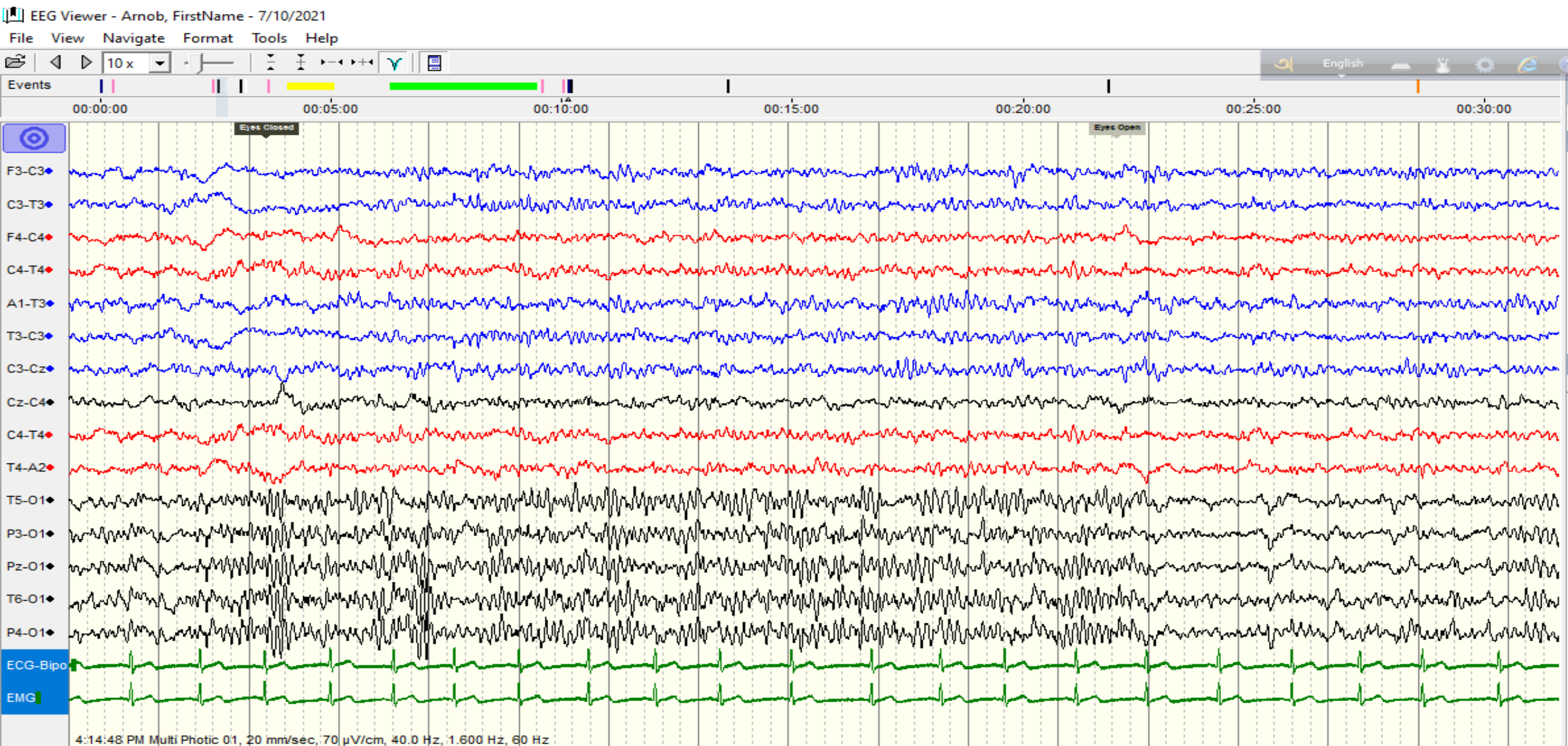
What this slide tells?



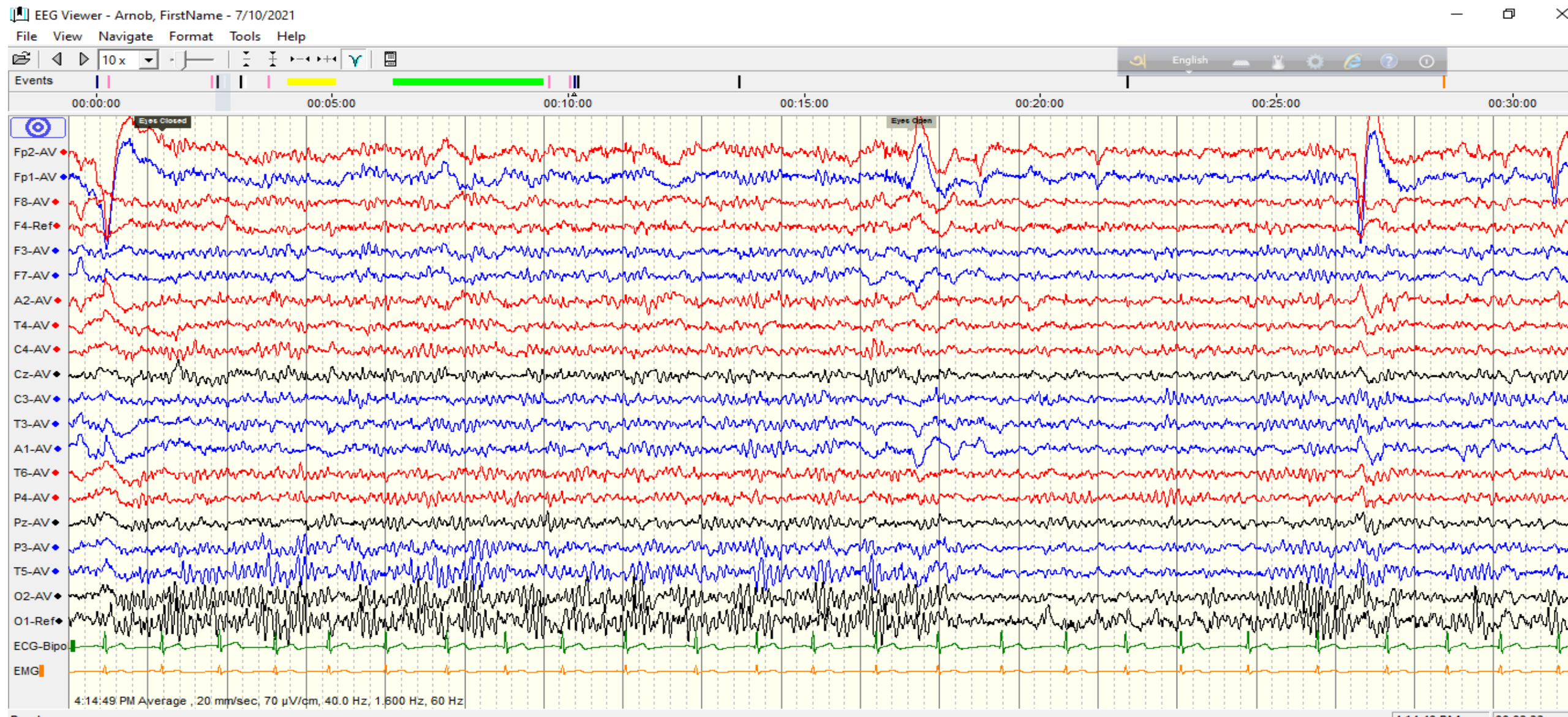
10:35:04 AM As Recorded, 30 mm/sec, 70 μ V/cm, 40.0 Hz, 1.600 Hz, 60 Hz



14 yr boy, EC effect on montage MF O1



14 yr boy, EC effect on montage Av, same page



Photic stimulation

- A series or train of Photic Strobe 3-4 upto 20-30/sec without interruption
- IPS
- **Effect?**
 - **Photic driving response** represents repetitive visual evoked potentials produced in response to the photic flash. The response occurs in a time-locked fashion
 - A photoparoxysmal response (PPR), formerly known as photoconvulsive response, occurs when IPS generates bilaterally synchronous epileptiform discharges that may outlast the stimulus by several seconds. At times, this may precipitate a seizure (see Fig. 7.7).
 - **PPRs may occur as one of two subtypes:**
 - (a) limited to the stimulus train (self-limited), or
 - (b) self-sustained (nonself-limited). The latter is more likely to be associated with clinical seizures (Kasteleijn-Nolst Trenité et al., 2012).

Other responses (physiological) to PS

- that resemble pathological findings may be identified during IPS.
- **A photomyogenic response** consists of repetitive contractions of the [frontalis muscle](#) synchronized to the light flash at a delay of 50–60 ms.
- **A photoelectric response** occurs as the result of a photochemical response generated by the electrodes, and can coexist with other waveforms, such as eye movement artifact, to mimic a PPR.
- **A physiologic electroretinal response** generated by retinal ganglion cells may occur in the frontal leads, mimicking an abnormality. Psychogenic nonepileptic attacks may at times be induced by HV and IPS together (Benbadis et al., 2000; Bodde et al., 2009).

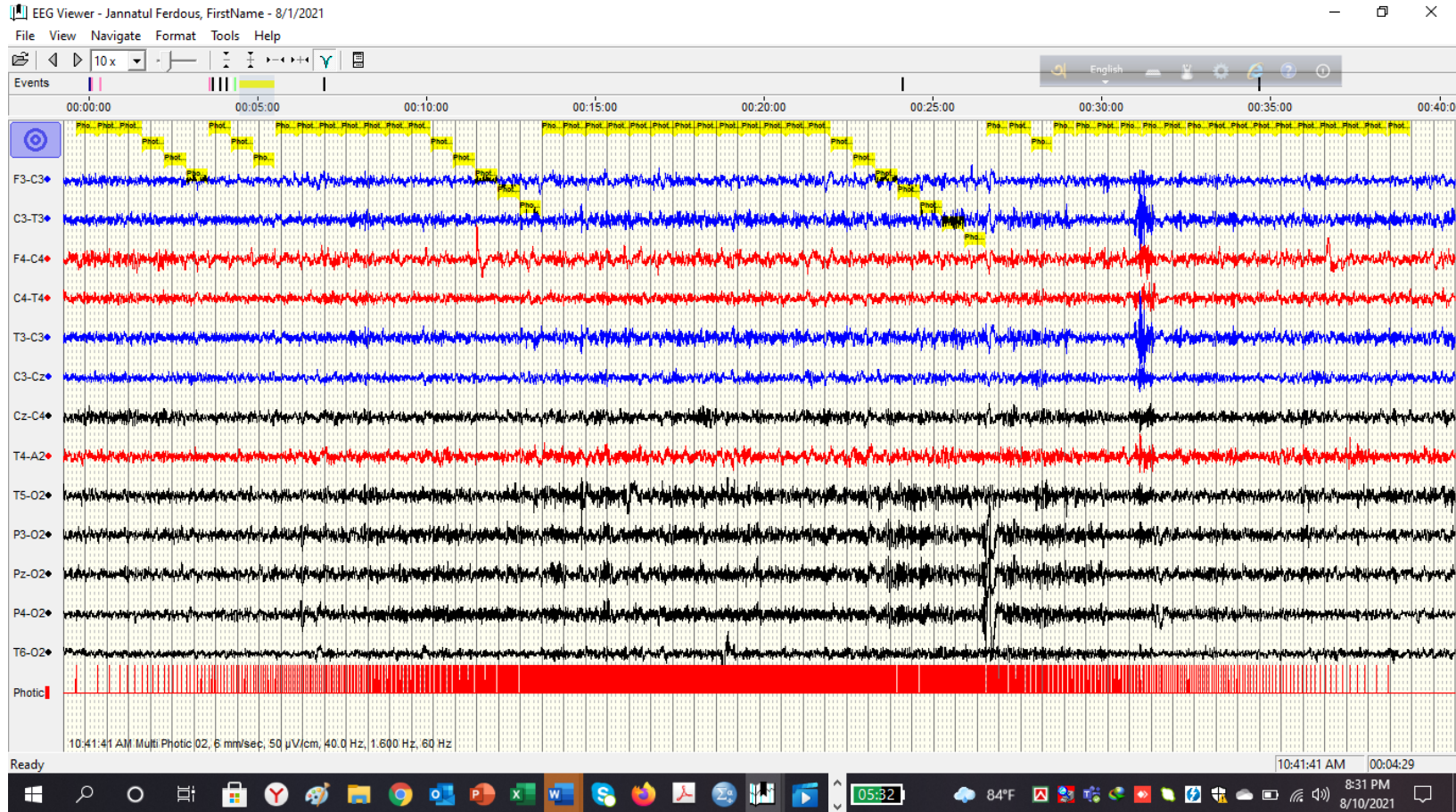
Diagnostic importance of PS

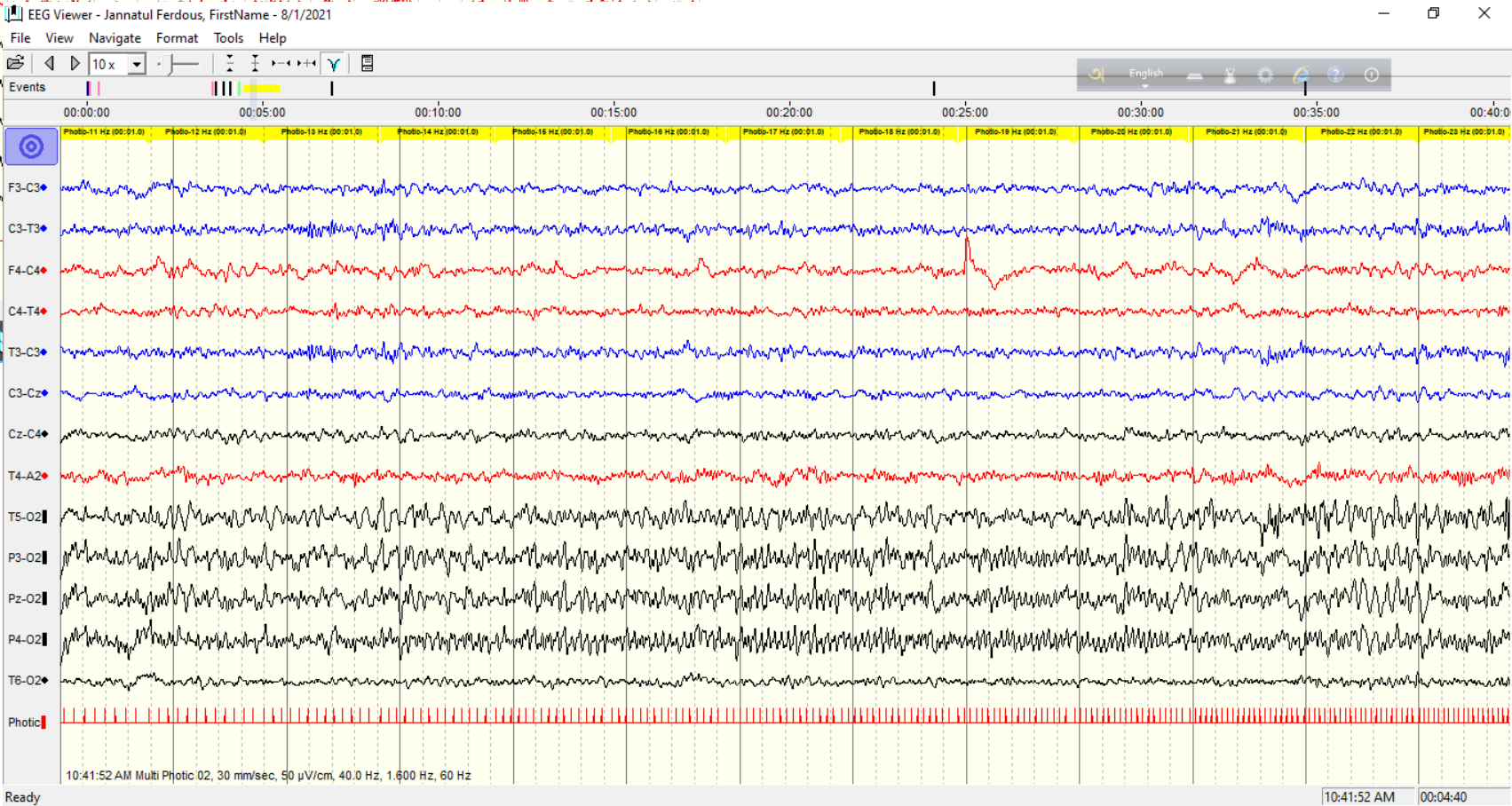
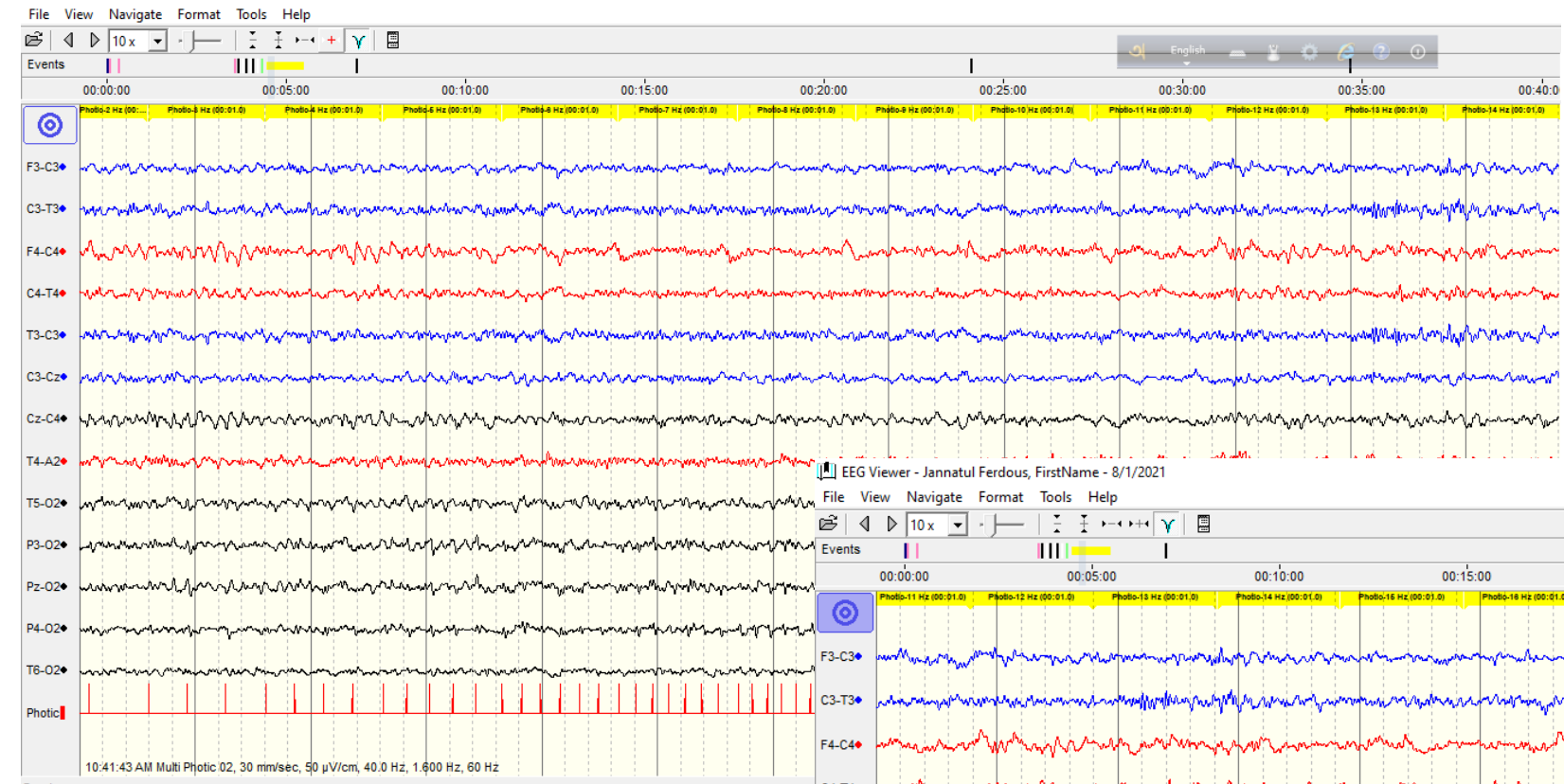
- Diagnostic
 - Photo-sensitive epilepsy
 - IGE
 - Certain metabolic and degenerative diseases
- Prognostic evaluation
- Management
 - Appropriate AED selection
 - Use dark glass

More clinical importance: [Unverricht–Lundborg's Disease](#)

- *A. Magaudda, in [Encyclopedia of Movement Disorders](#), 2010*
- **EEG**
- At the disease onset, background activity (BA) is normal or mildly slow (BA at 6–7 Hz).
- Superimposed slower activity at 4–5 Hz can be present, resulting in an irregular appearance of BA. BA keeps stable during the course of the disease.
- During the initial years of the disease, EEGs show spontaneous brief bursts of GSWD, characterized by very rapid spikes (**Figure 1**). Focal epileptiform abnormalities can be observed over the central and posterior regions of the scalp. Intermittent photic stimulation (IPS) provokes the appearance of GSWD and increases myoclonus, both generalized and focal.

Train of photic strobe without interruption





Photic stimulation, intermittent PS

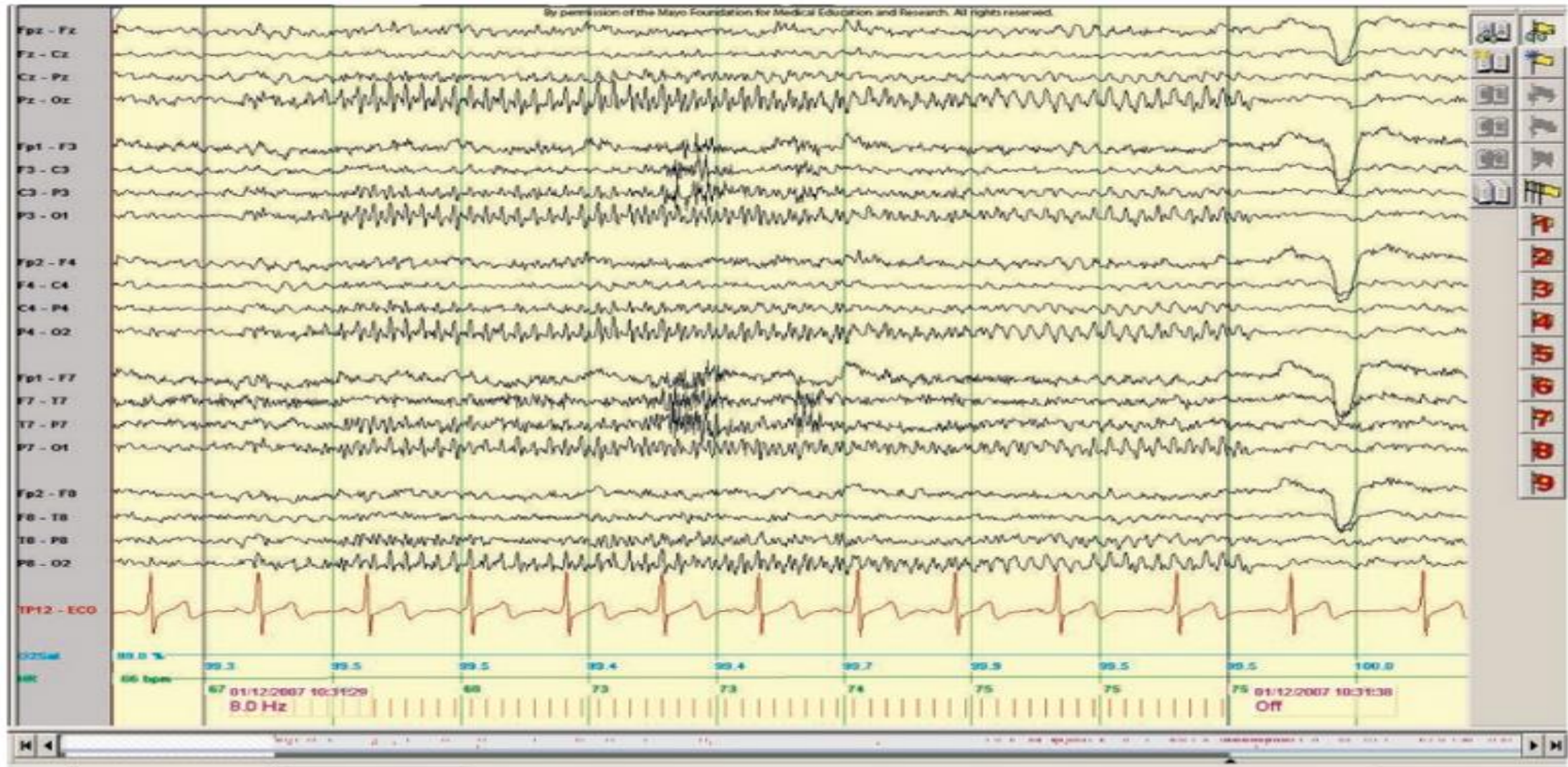




Fig. Photoparoxysmal response (PPR) during 30-Hz photic stimulation in an 18-year-old girl with photosensitive genetic generalized epilepsy. Note the anterior-dominant spike and slow wave discharge outlasted the stimulation (nonself-limited PPR).

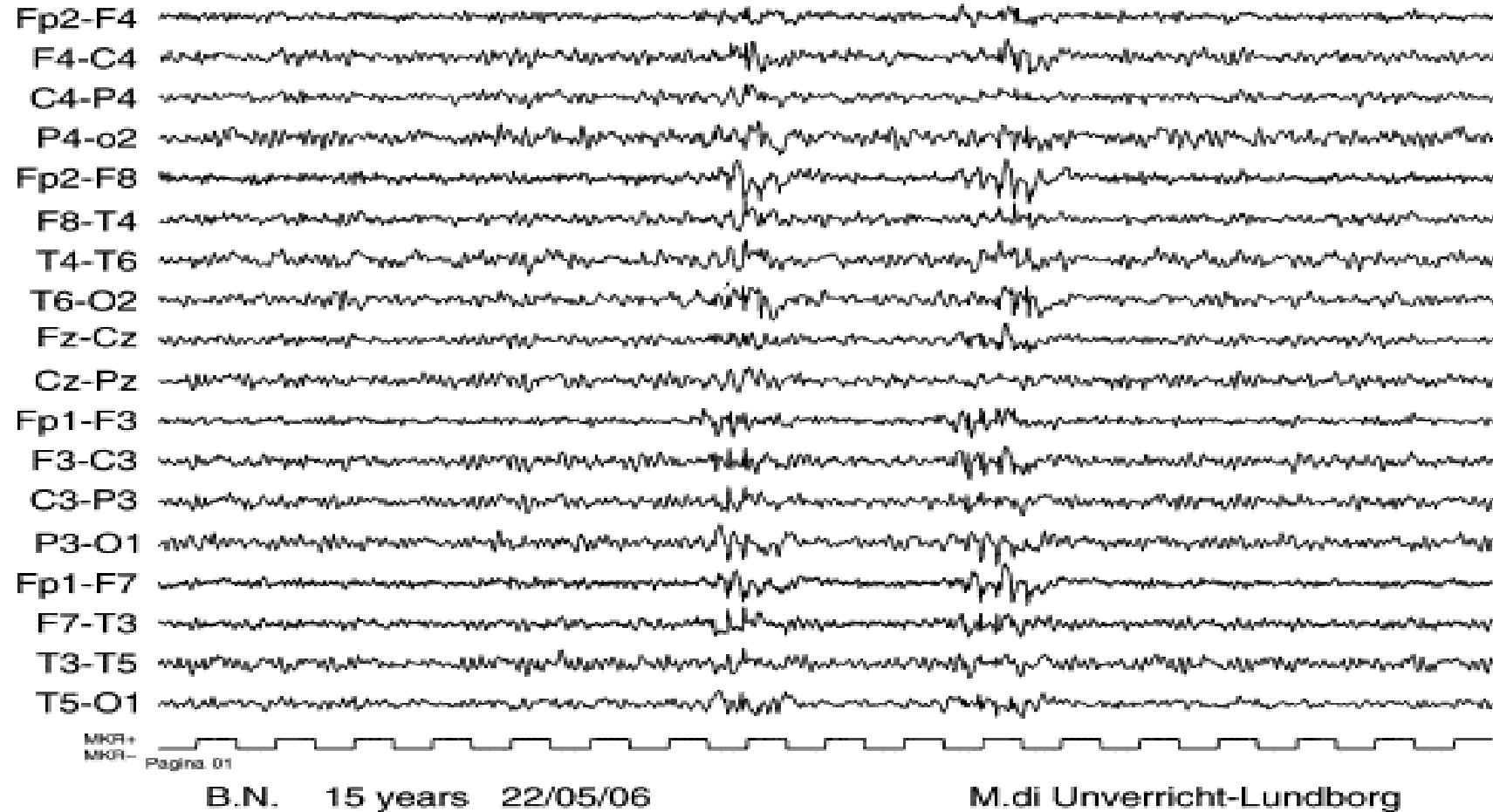


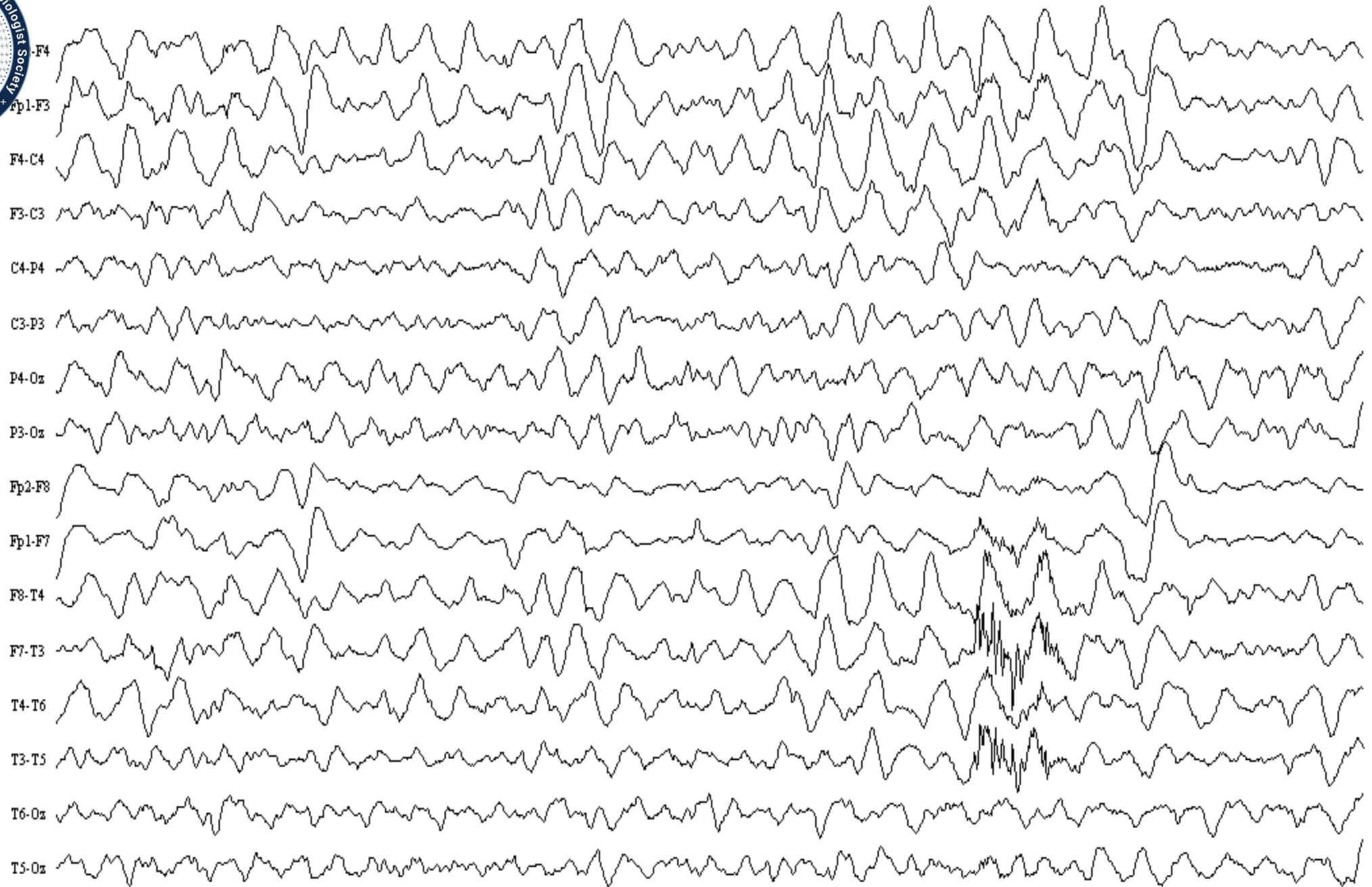
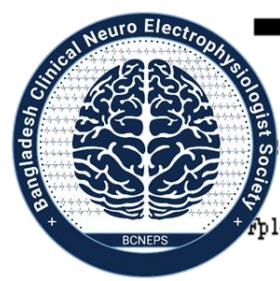
Figure EEG in a 15-year-old ULD patient, showing normal background activity and brief bursts of irregular generalized spike – wave discharges, characterized by very rapid spikes.

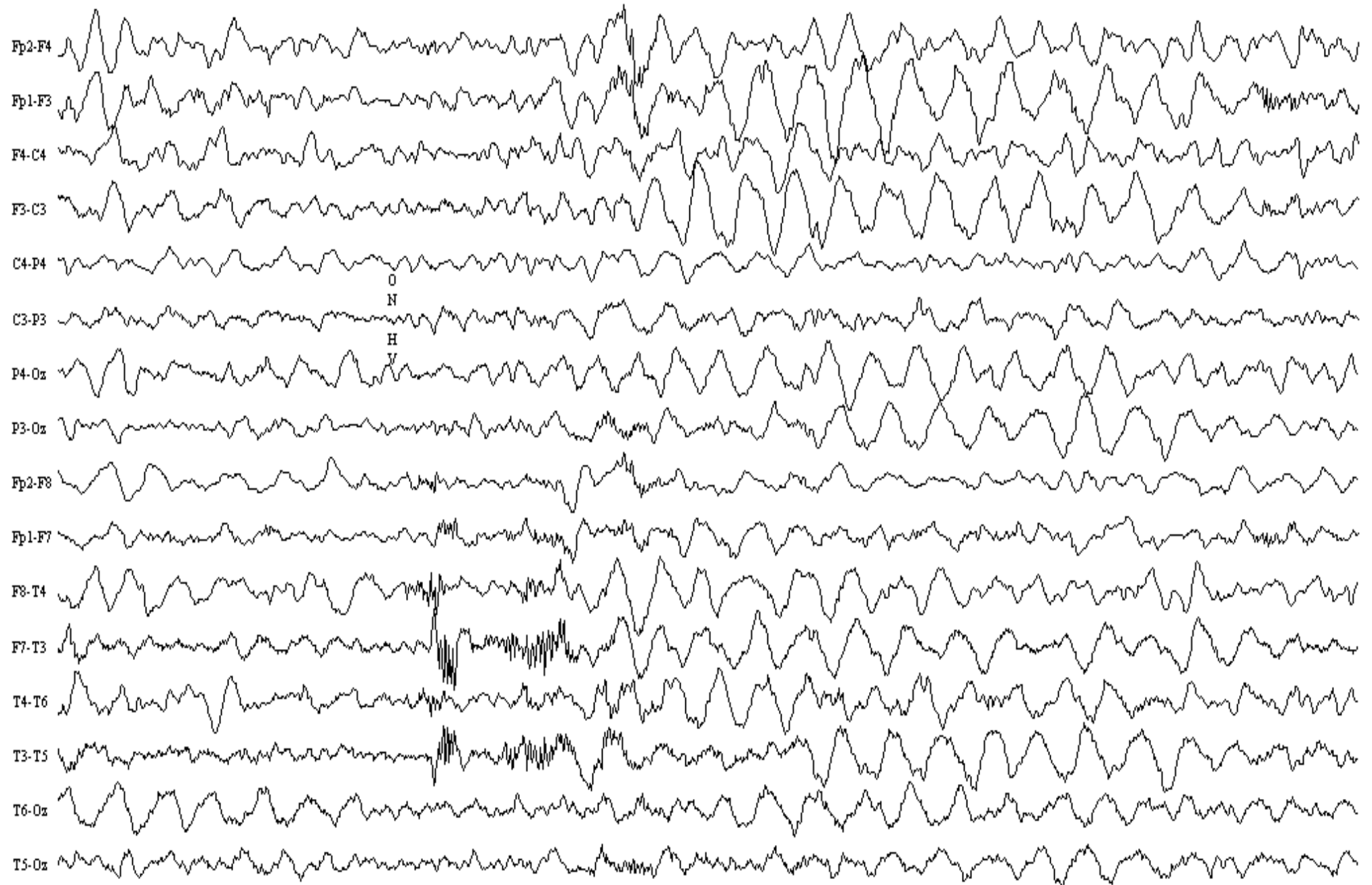
During sleep, a normal representation of physiological sleep patterns and a reduction of GSWD during nREM and [REM sleep](#), along with the presence of fast spikes and polyspikes over the central and the vertex regions during REM sleep, were observed.

Long-term evolution of EEG in ULD is characterized by no relevant deterioration of BA, a gradual reduction of GSWD and PPR, correlating with good seizure outcome, and a progressive disappearance of physiological sleep patterns 10–20 years after the disease onset.

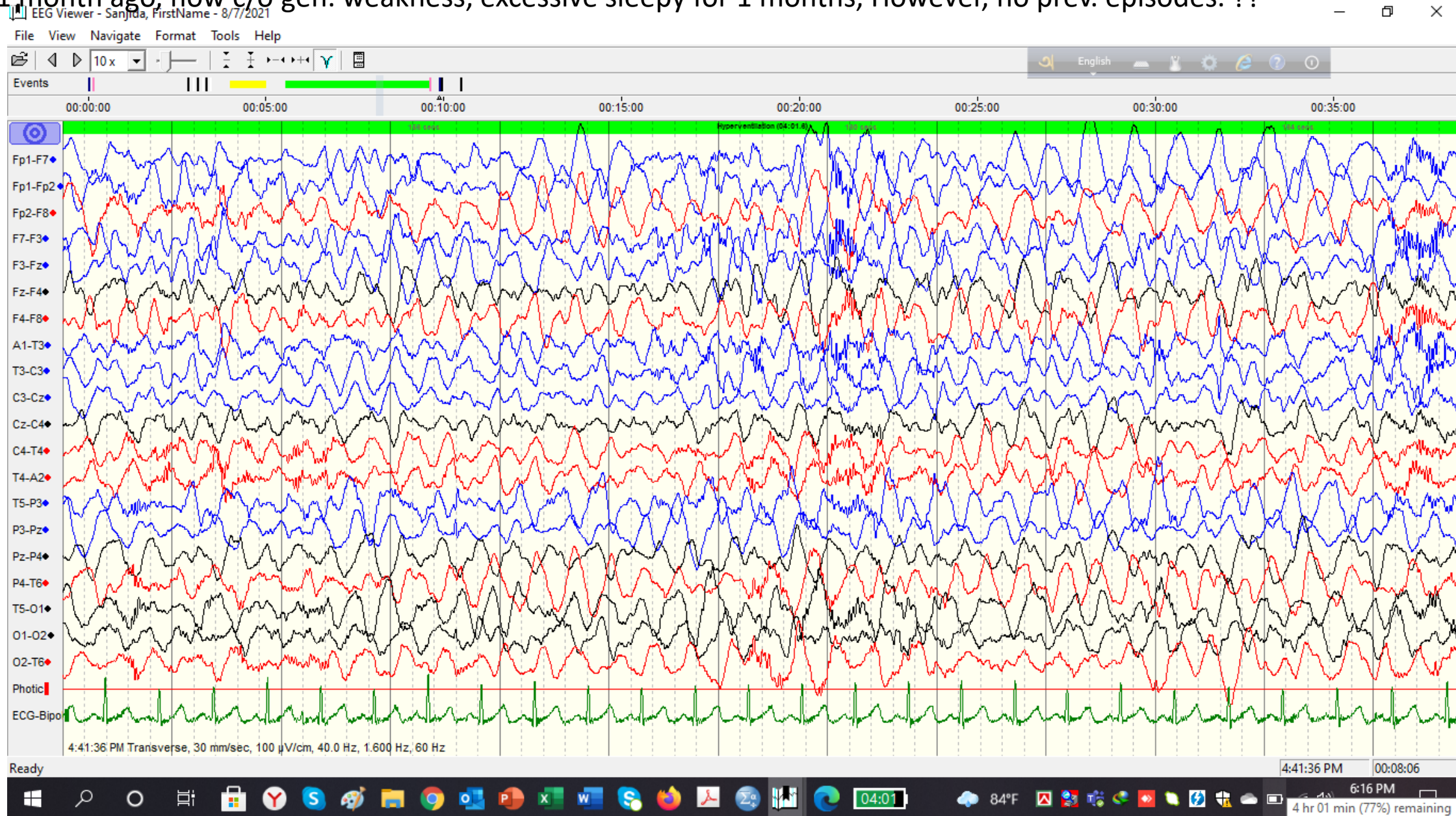
Hyperventilation

- Purpose?
- Process?
- Response?

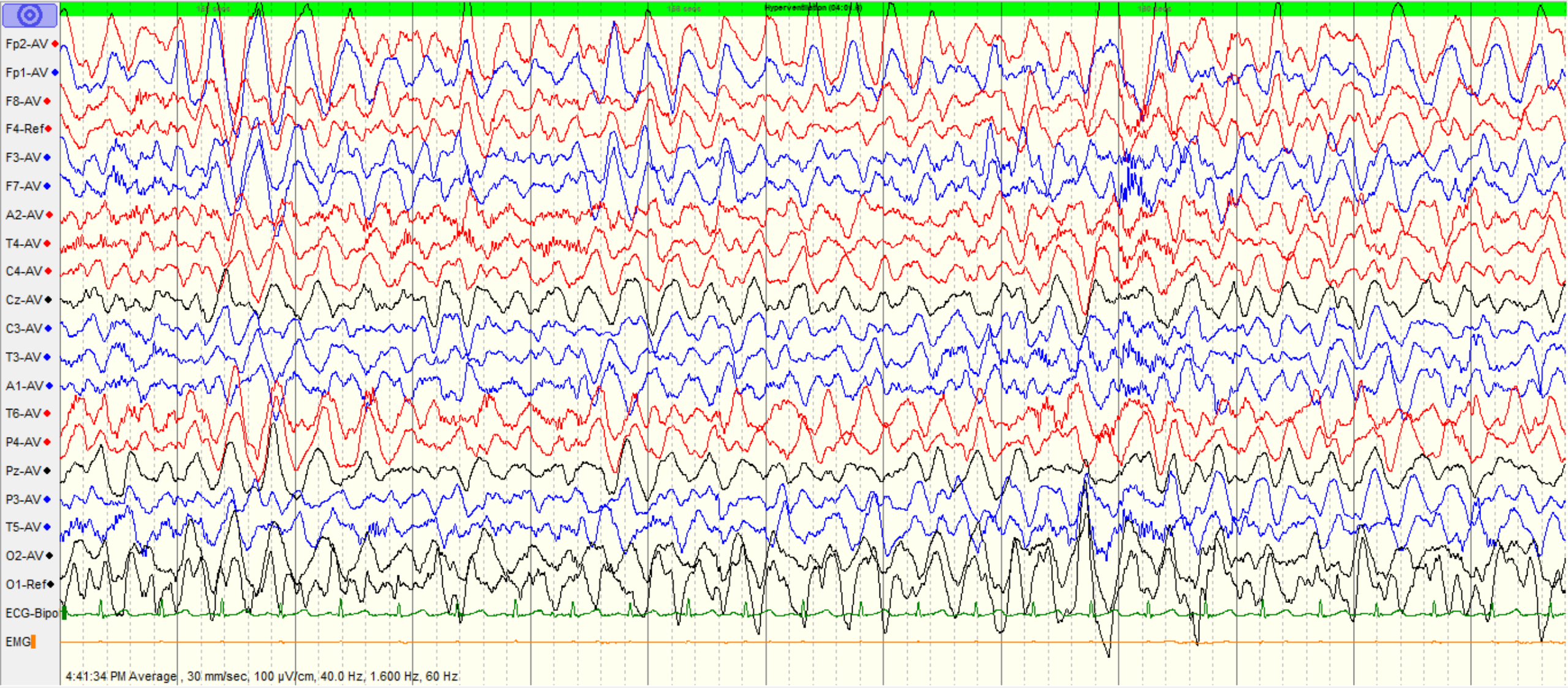




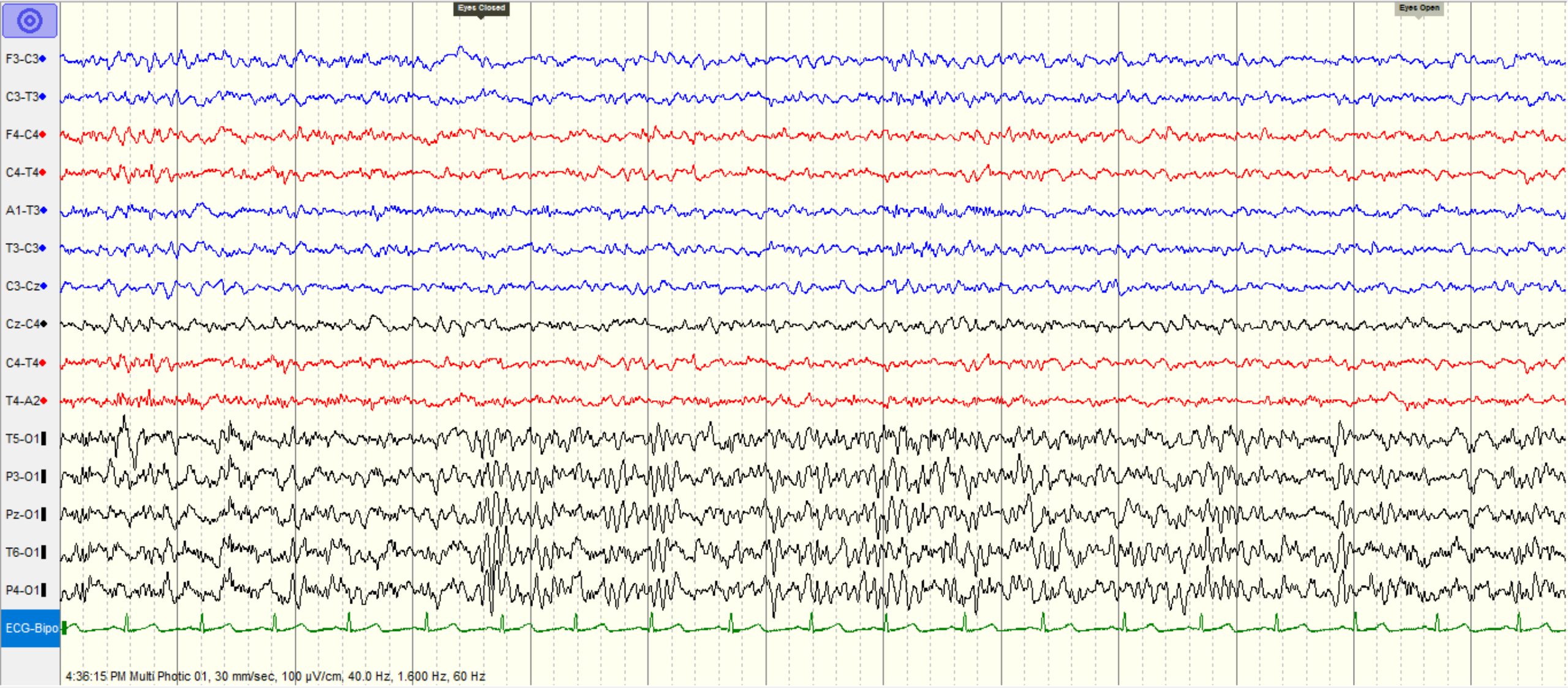
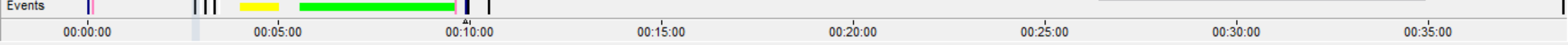
11 yr F, ?Epilepsy, had episodes of transient loss of awareness 3 months ago, went to a neurologist, Rx with VPA (no EEG) 1 month ago, now c/o gen. weakness, excessive sleep for 1 months, However, no prev. episodes. ??



Navigation and utility icons including zoom (10x), pan, and search. A search bar contains the text "English".



4:41:34 PM Average, 30 mm/sec, 100 µV/cm, 40.0 Hz, 1.600 Hz, 60 Hz

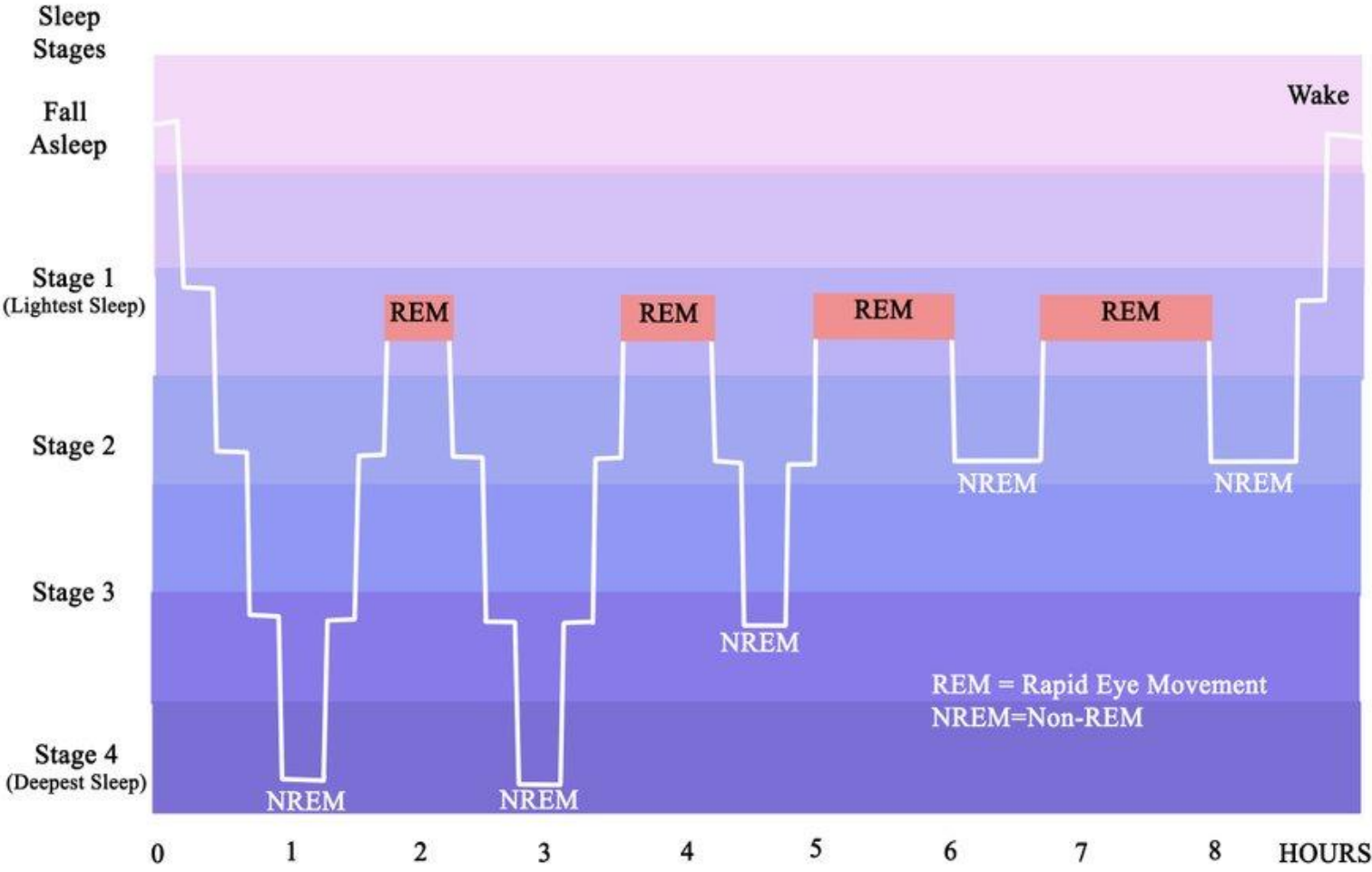


Sleep

- Normal sleep stages
 - Non Rapid Eye Movement sleep (NREM)
 - Rapid Eye movement Sleep (REM)
- Clinical importance
- Normal variants (age dependent)
- **Misconception results in wrong interpretation**

Normal human sleep cycle, 1 cycle = 90 min.

Sleep Cycles



Stage 1

lightest (1-7 min)

- Heartbeat slows down
- Breathing slows down
- Eye movements slow down
- Muscles relax, and might occasionally twitch
- Brain waves begin to slow down

Stage 2

light (10-25 min)

- Heartbeat and breathing slow down even more
- Muscles relax even more
- Body temperature drops
- Eye movements stop
- Brain wave activity slows

Stage 3

deep sleep (20-40 min)

- Heartbeat and breathing slow to the lowest levels they will reach during sleep
- Muscles stay relaxed
- Brain waves slow down even more

Stage 4

REM (20-40 min)

- Behind your eyelids, your eyes move rapidly from side to side
- Breathing speeds up and can become irregular
- Heart rate increases
- Blood pressure increases

EEG activities in different stages

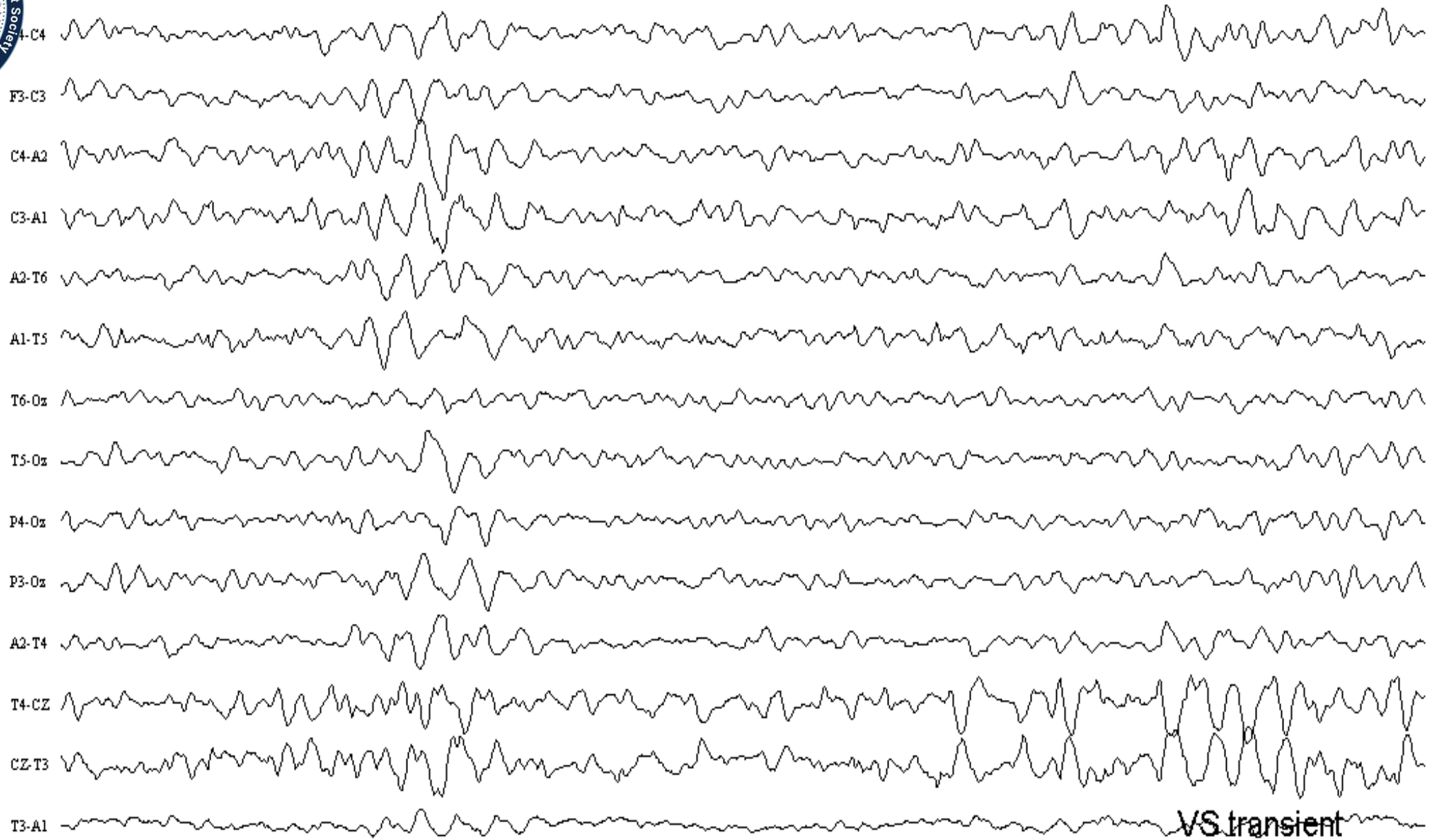
Alpha rhythm on EC disappears, Central theta W, Hypnagogic hypersynchrony appears

Vertex Sharp Trans K complexes and sleep spindles appear successively

Sleep spindles **starts reducing**, background slowing starts
V. Deep sleep= **slow background**

Mixed activity in the BG almost like awake stage

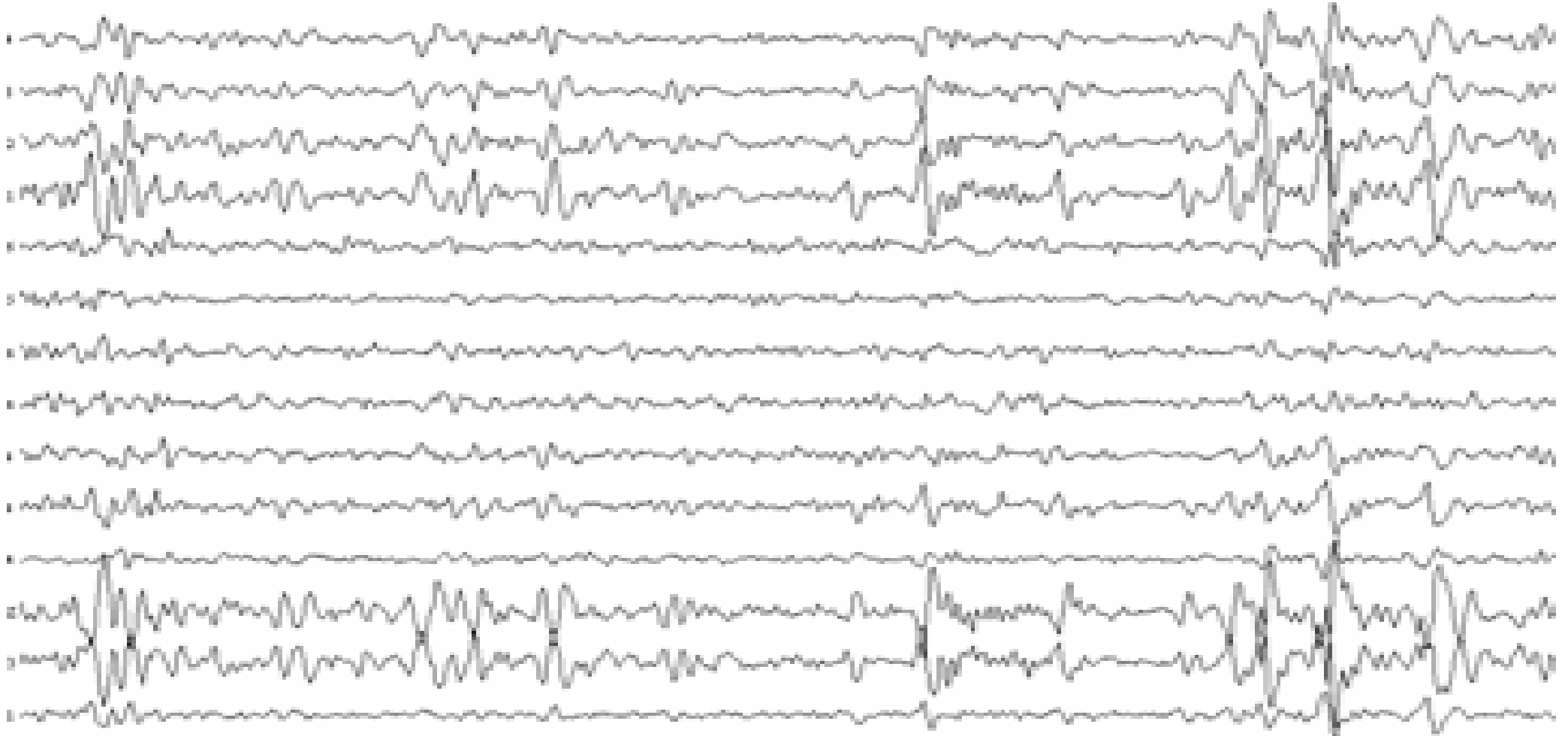




Hypnagogic hypersynchrony & vertex Sharp tr. in early sleep

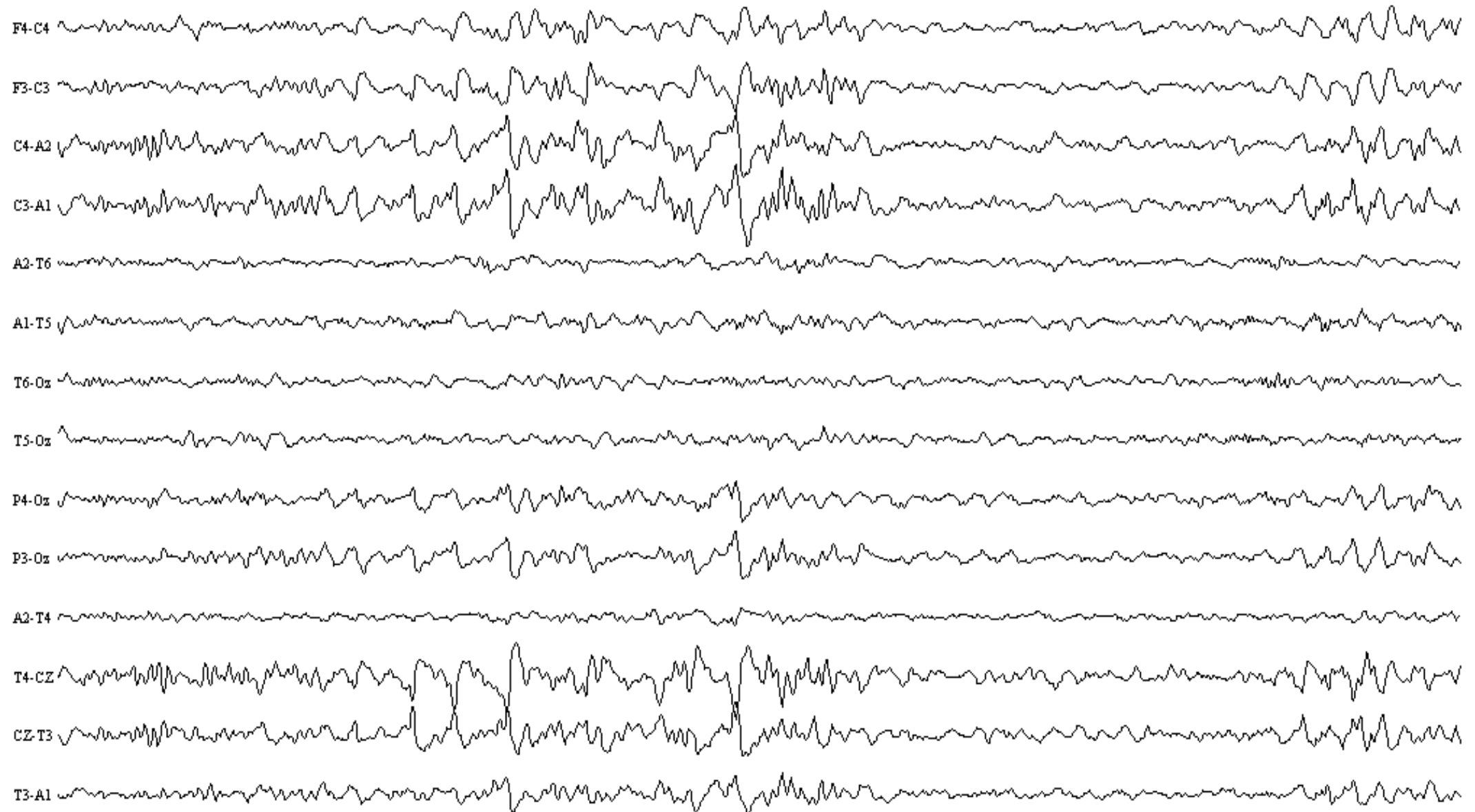


Vertex sharp transients/K complexes: normal sleep stage 1 and 2 (*drowsy and early sleep*), mistaken for epileptiform discharges



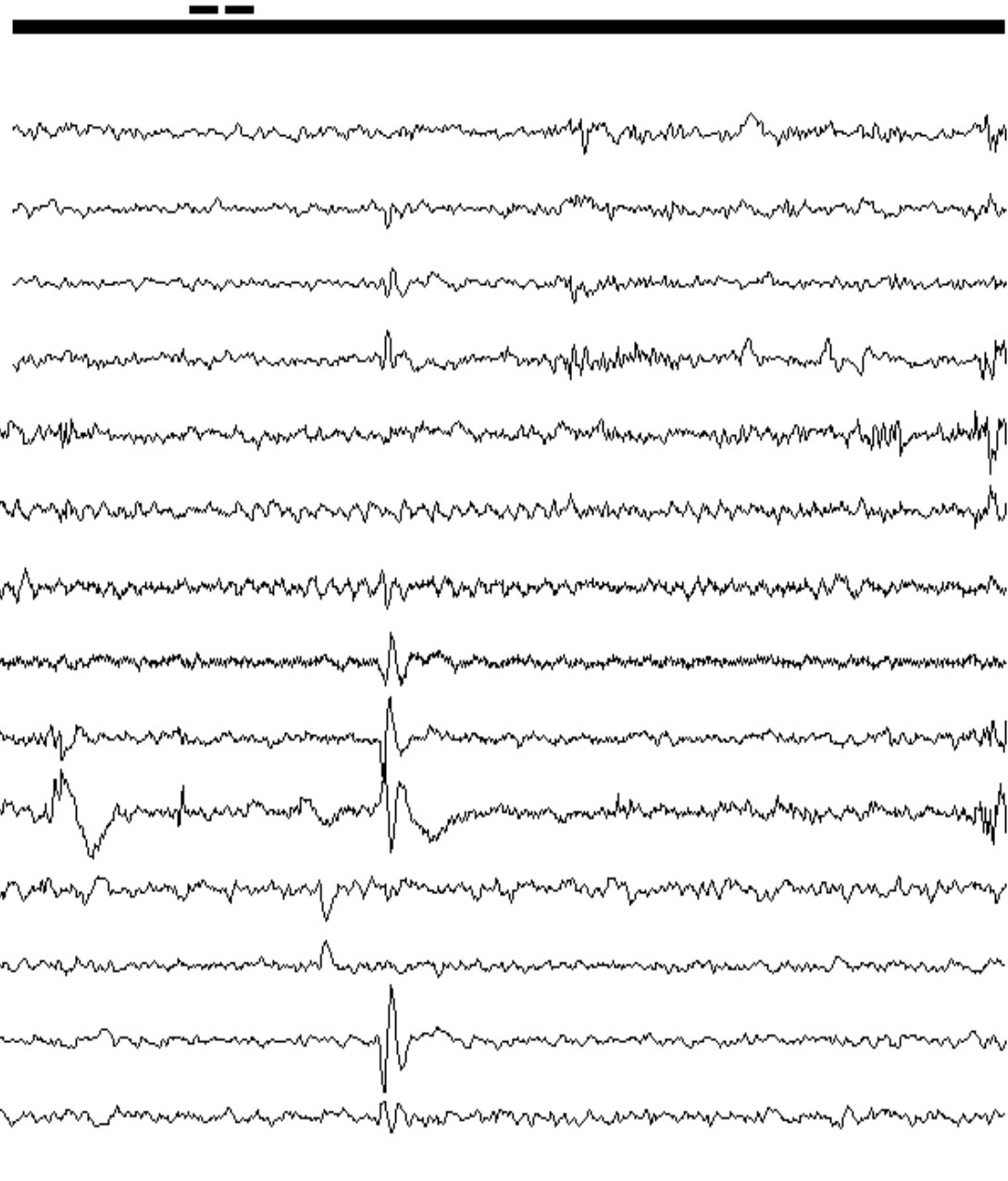


sleep stage2, Sen-14, Hf-30Hz, Tc- 0.03s



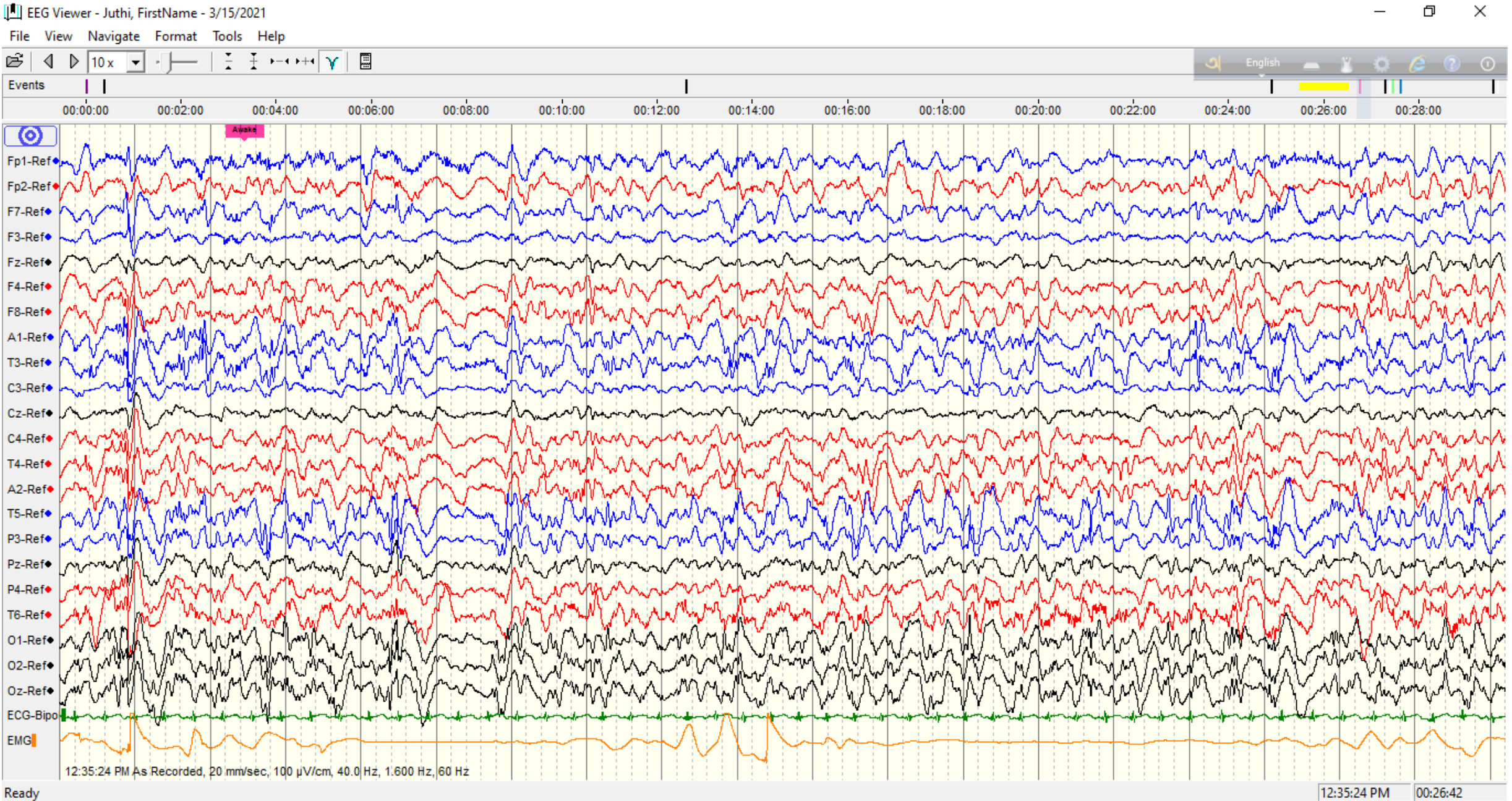


Mu rhythms in alert state, lateralized,
discharges over left side

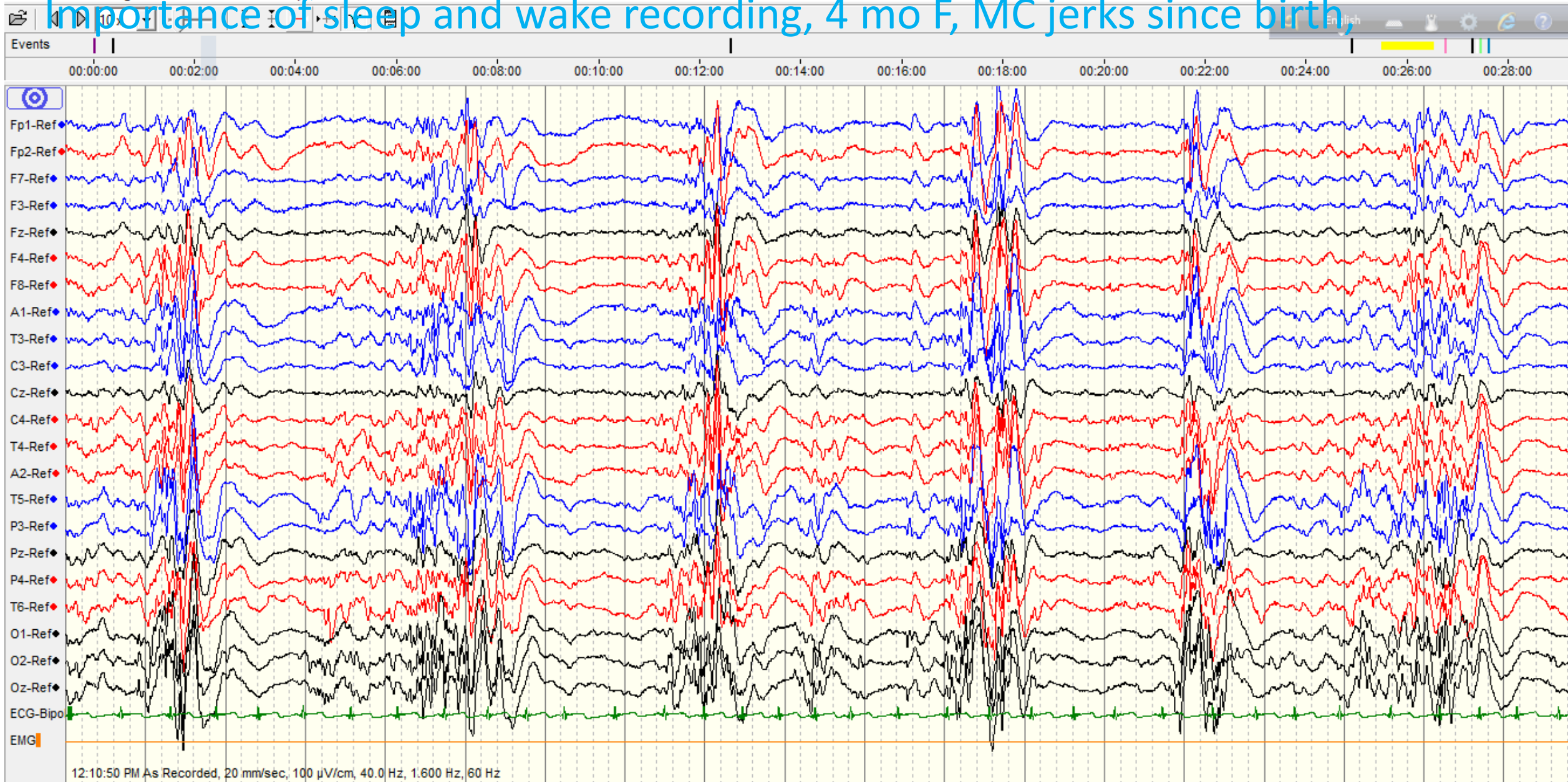




Importance of sleep and wake recording, 4 mo F, MC jerks since birth,

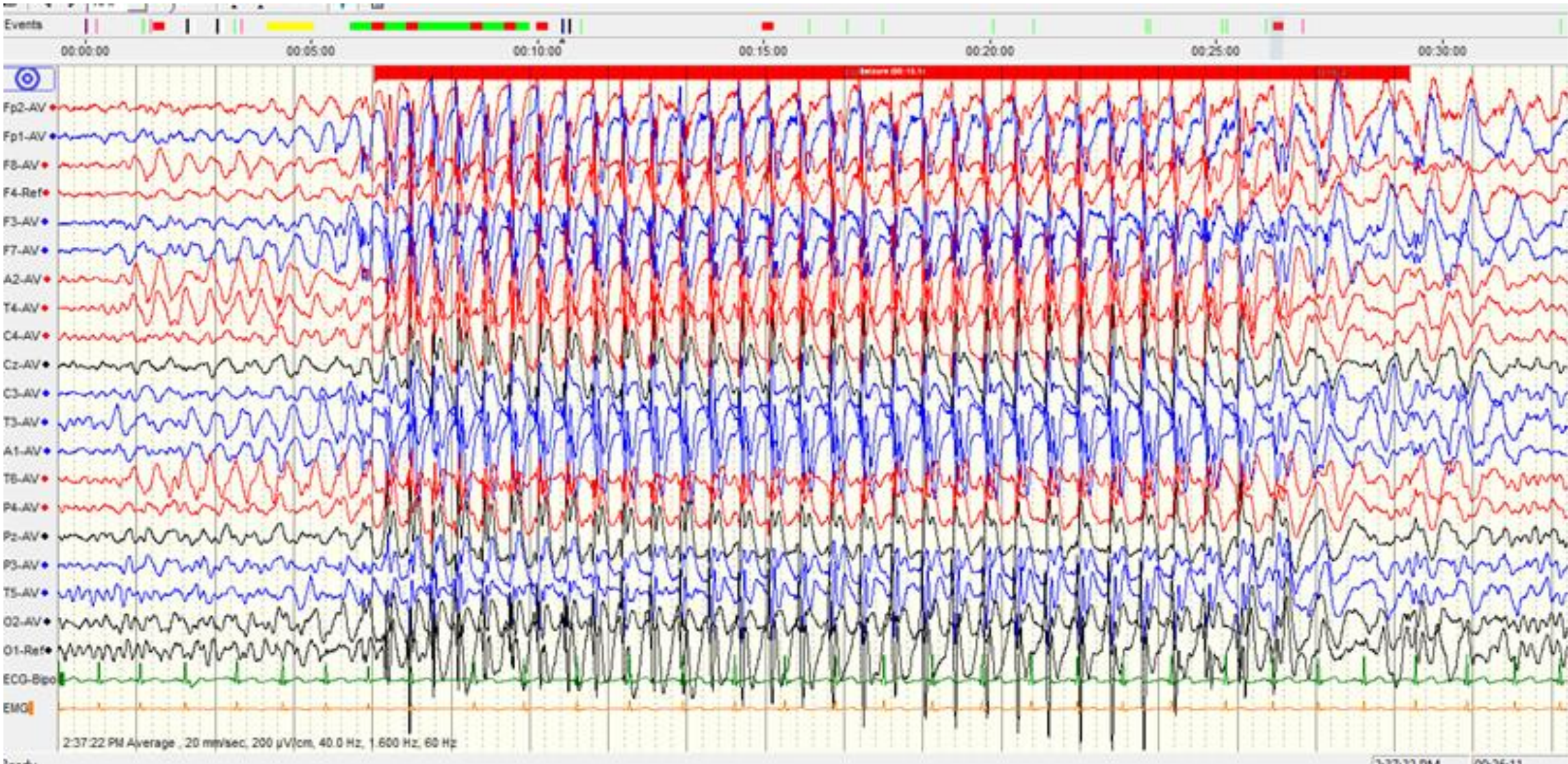


Importance of sleep and wake recording, 4 mo F, MC jerks since birth,

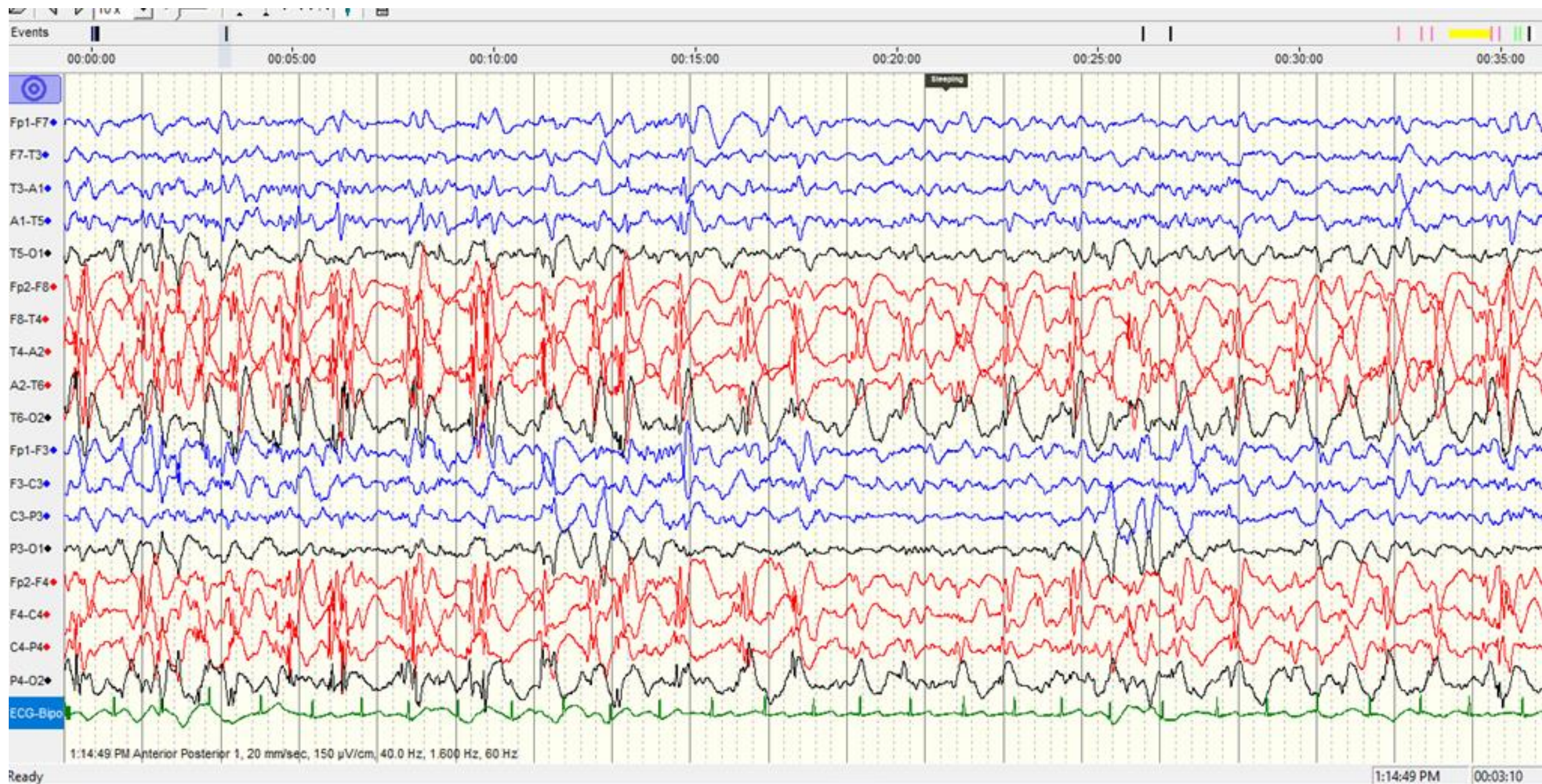


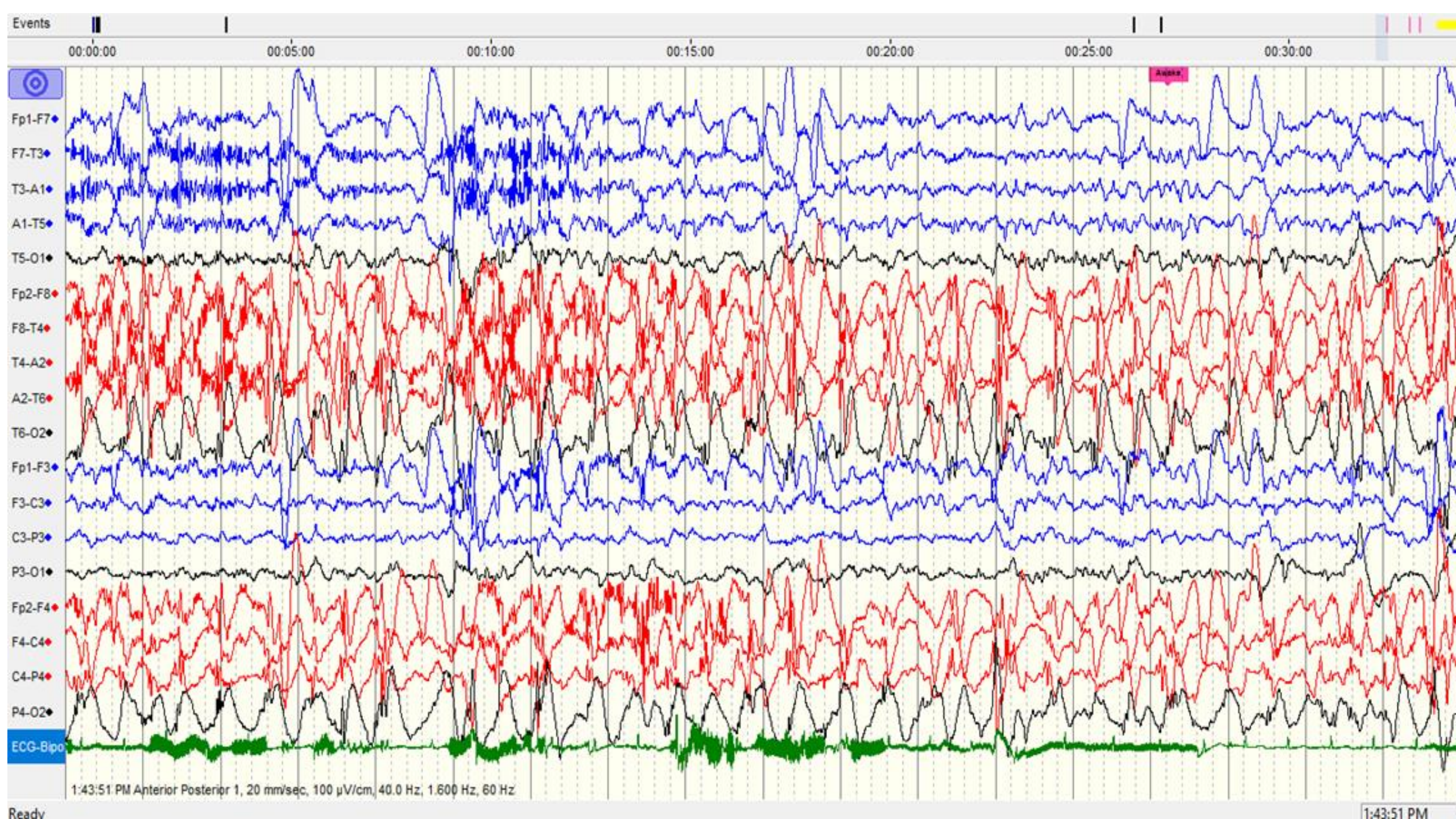


4yr, F, ?Absent minded for a few seconds daily for last 2 months, h/o 8 episodes of Febrile szs during 3mo till 12 month age, FT delivery by CS, NC
Suggestive of generalized epilepsy with absence seizures.

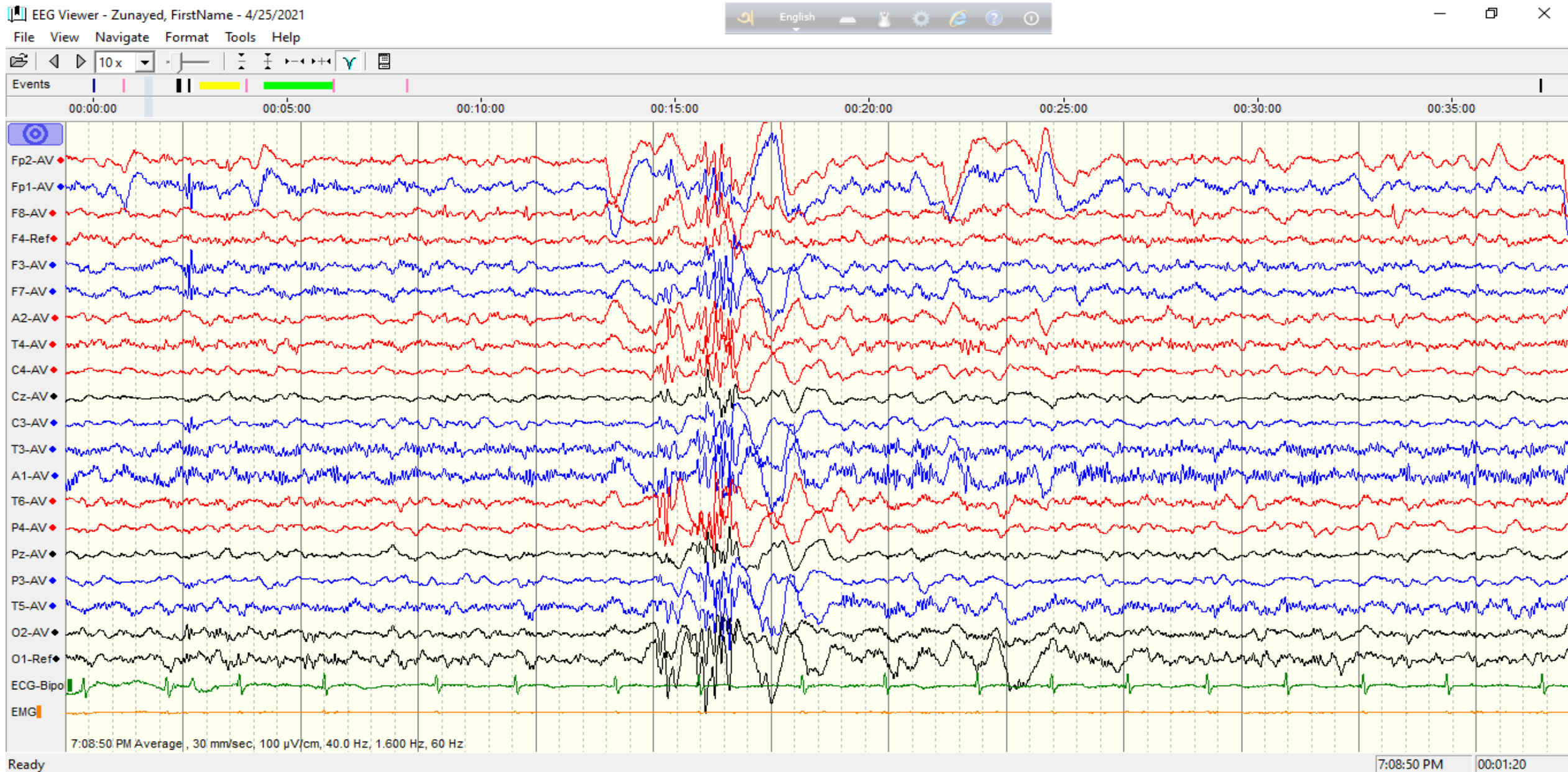


8yr, F, c/o sudden screaming- gen sz for >60 minutes, ended with vomiting, headache+ vomiting for 3 yrs, student.

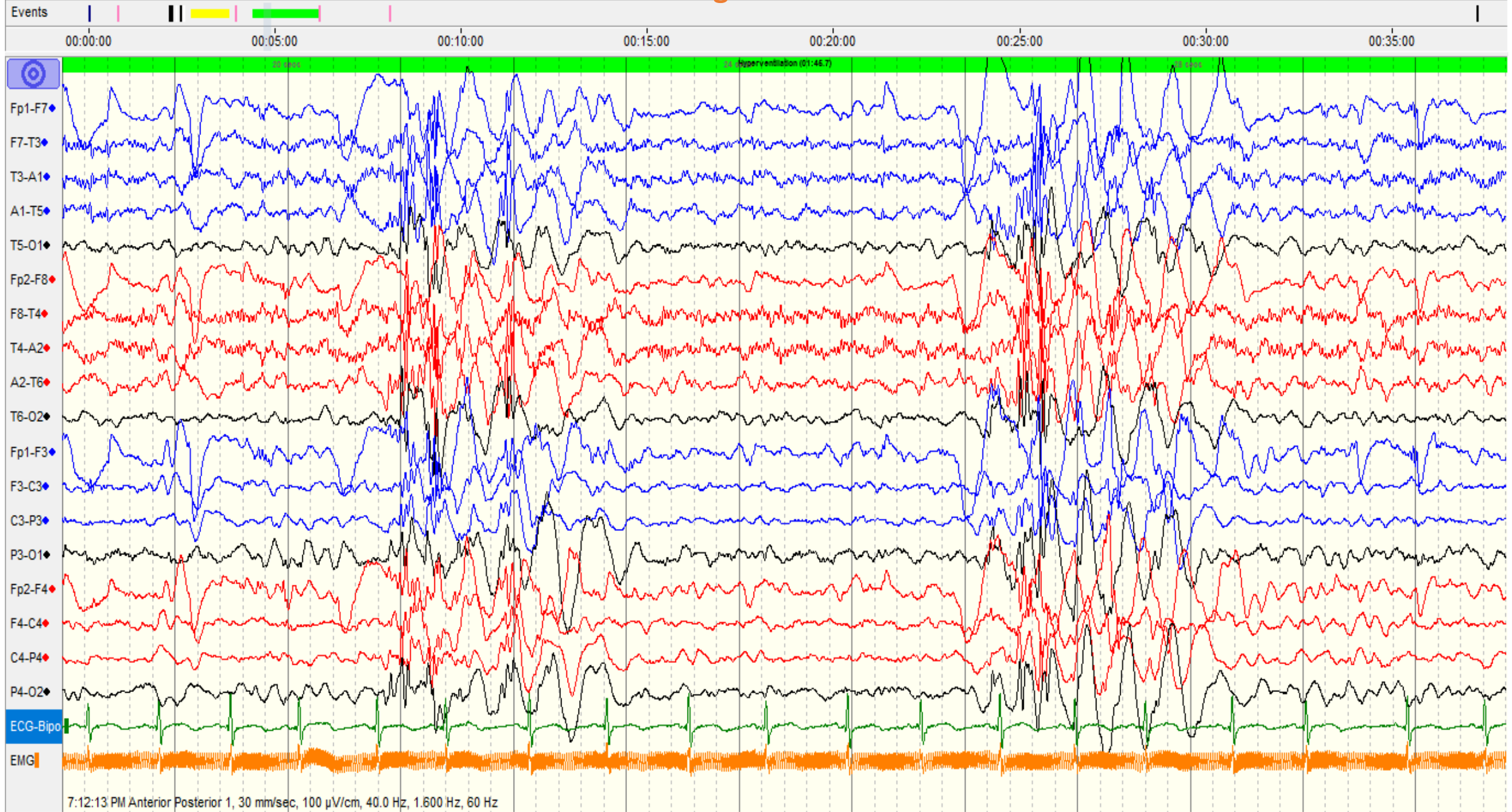




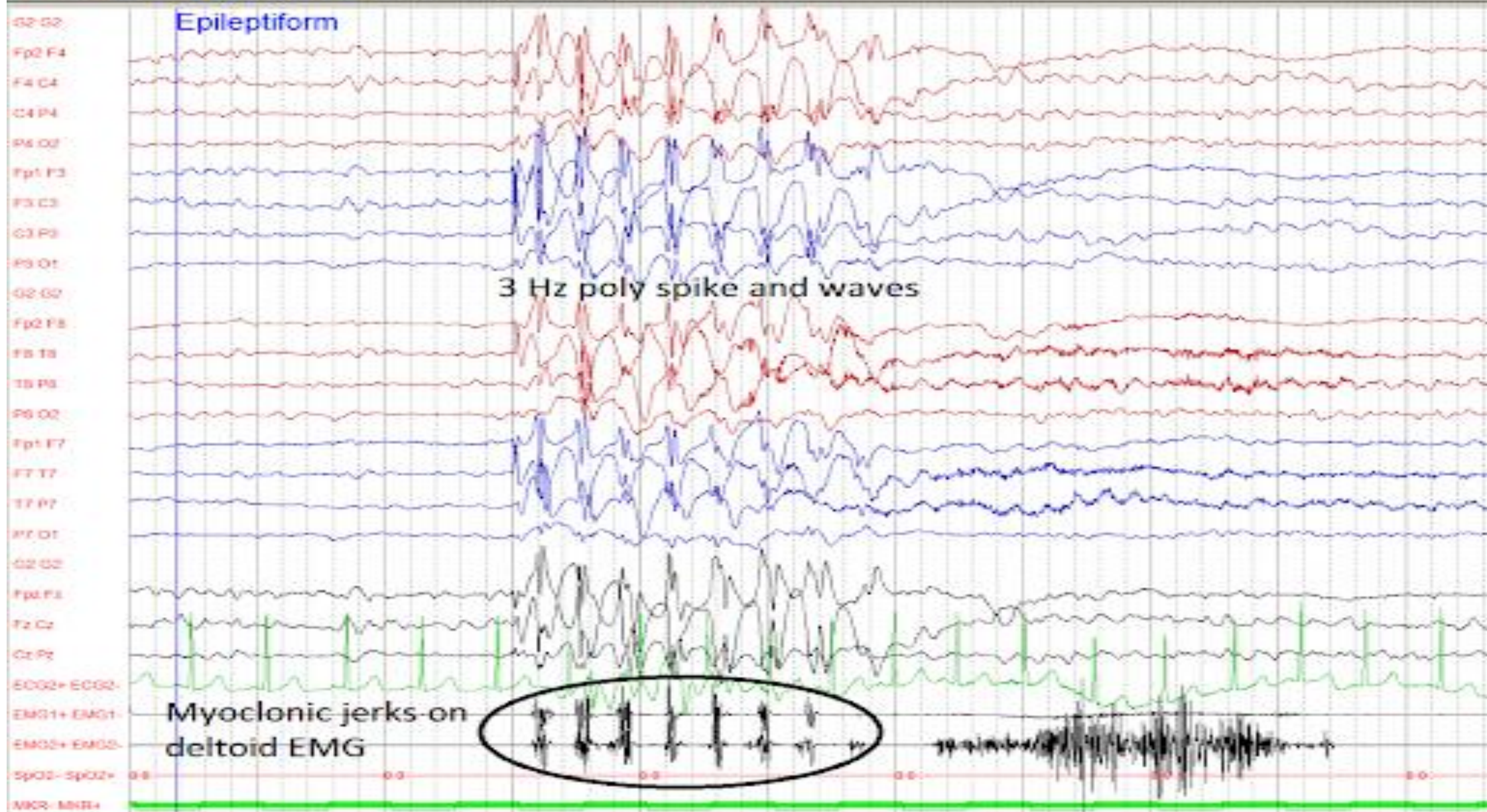
8 Yrs boy, c/o GTCSz on the previous day, has eye blinking, transient discrete jerks for last 6 months, no recognizable comorbidity. **What is missing here?, Technician's job?**



What is missing here?



7:12:13 PM Anterior Posterior 1, 30 mm/sec, 100 μ V/cm, 40.0 Hz, 1.600 Hz, 60 Hz



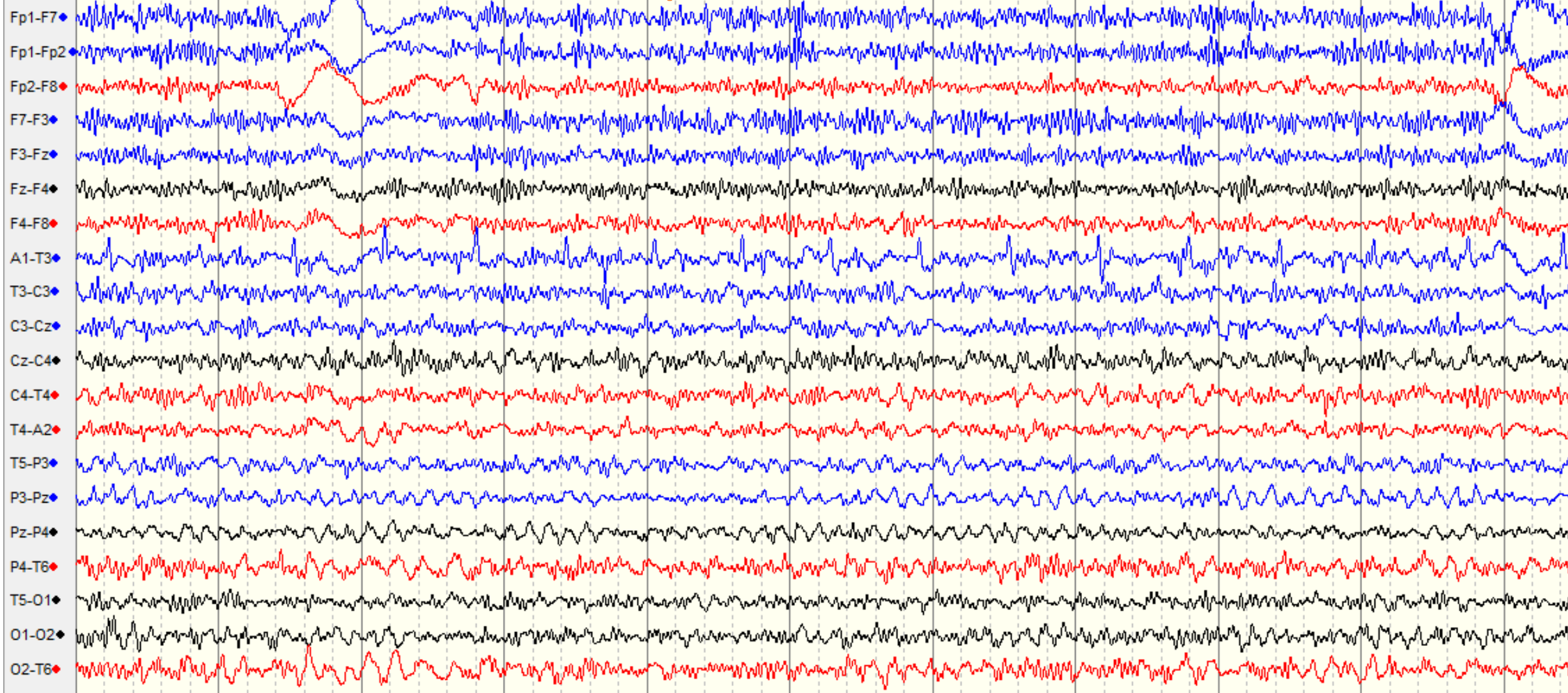
Epileptiform

3 Hz poly spike and waves

Myoclonic jerks on deltoid EMG

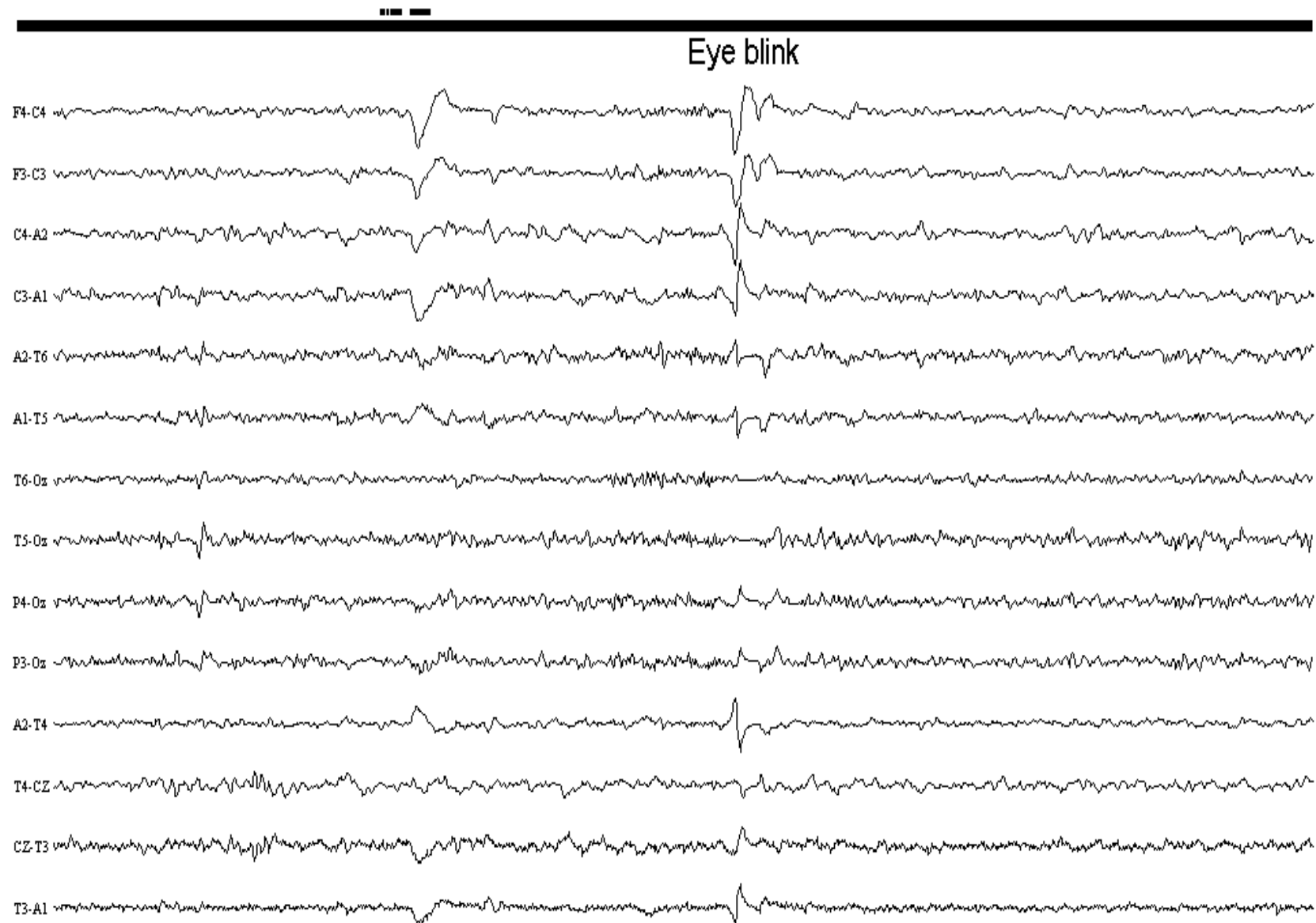


Focal spikes over T3 ?

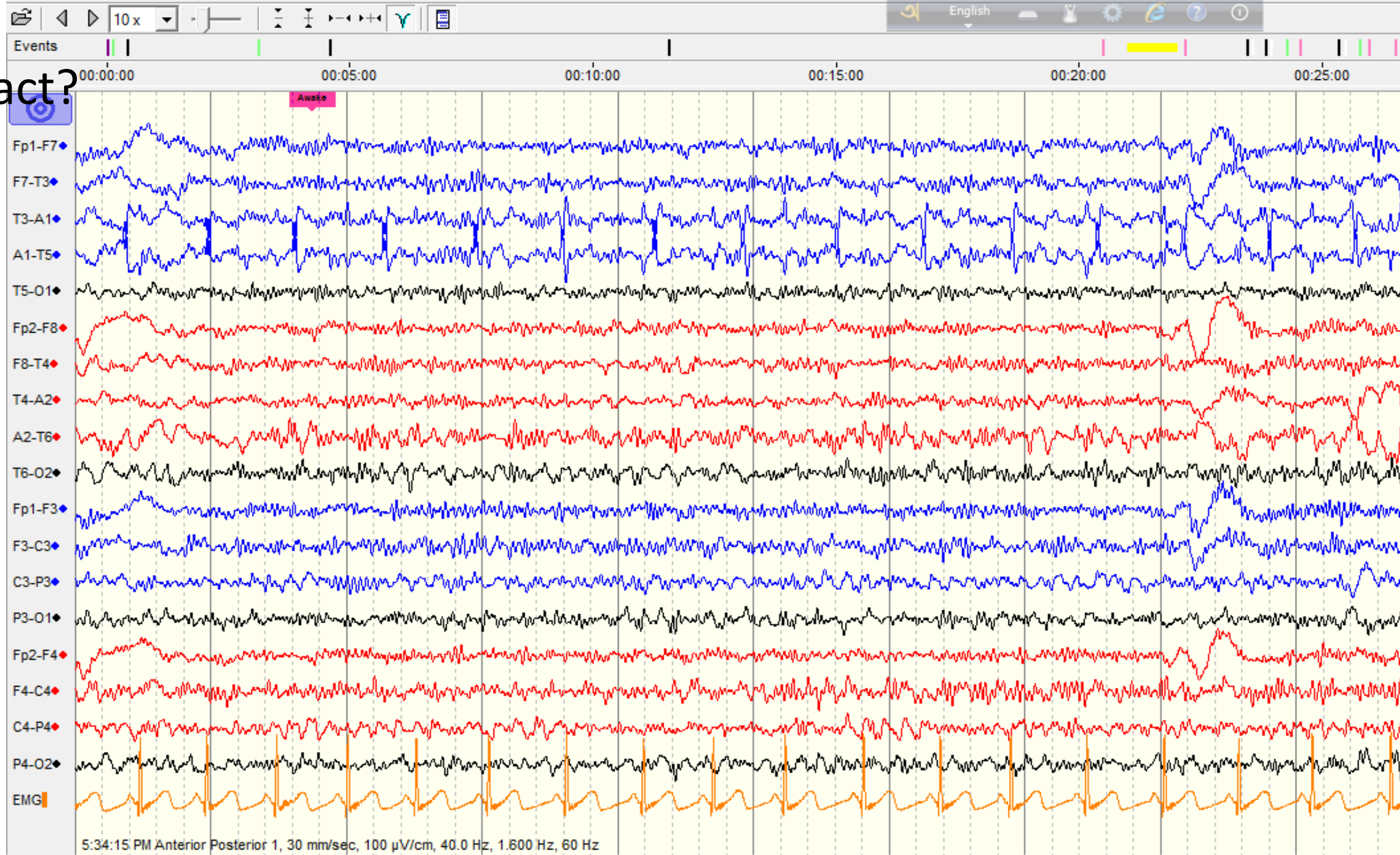


5:33:15 PM Transverse, 30 mm/sec, 100 μ V/cm, 40.0 Hz, 1.600 Hz, 60 Hz

Artifact?

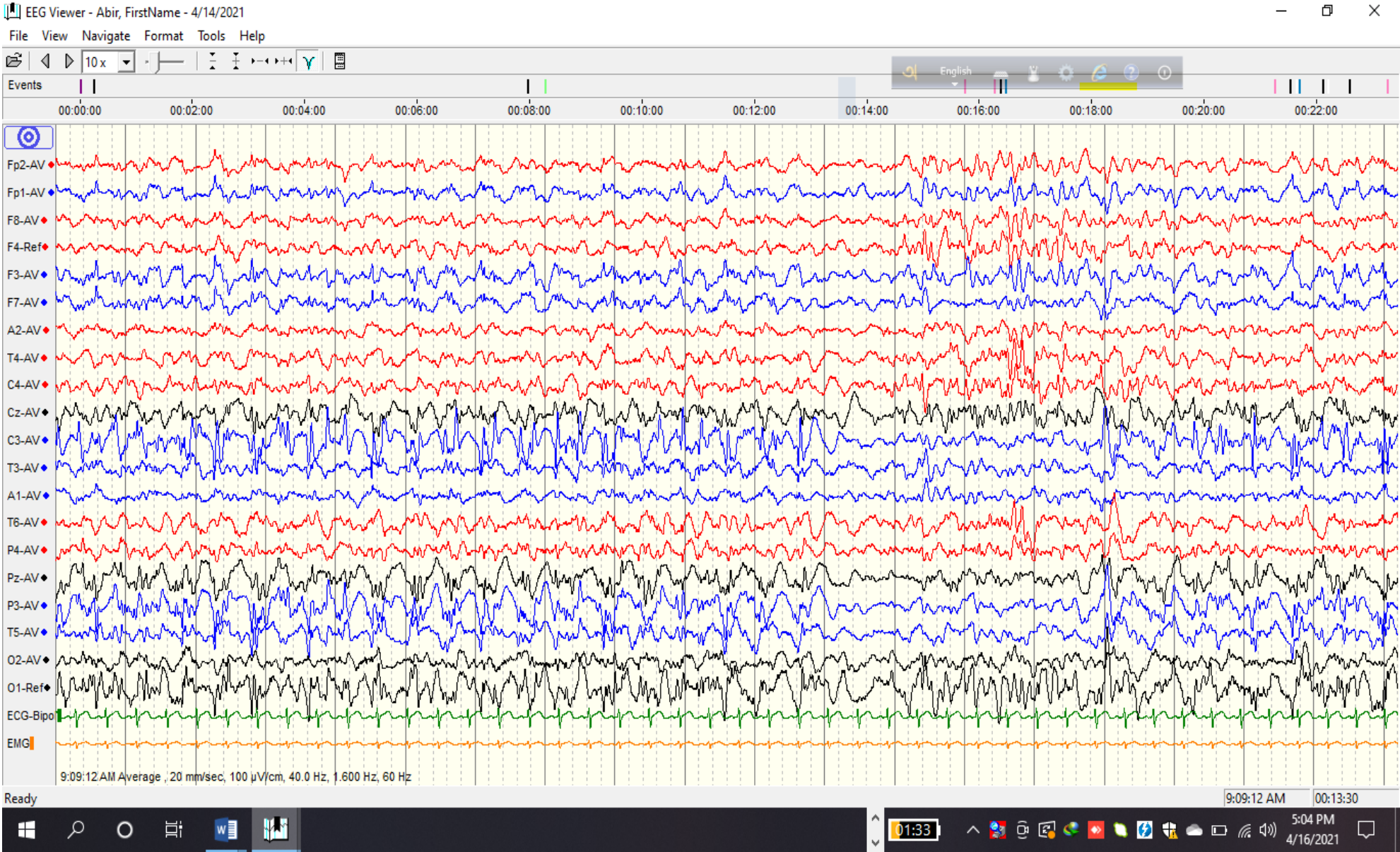


Artifact?



Ready

Localized dysfunction? Describe this



Description of epileptiform discharges

➤ Morphology

- Size & shape/ amplitude, frequency, distribution, duration

➤ How they are arising

- In relation to time, state of the patient
- Spontaneous or Provoked by activation process
- Synchrony of firing
- Spatial, Temporal distribution
- In short or prolonged runs
- Arising as periods

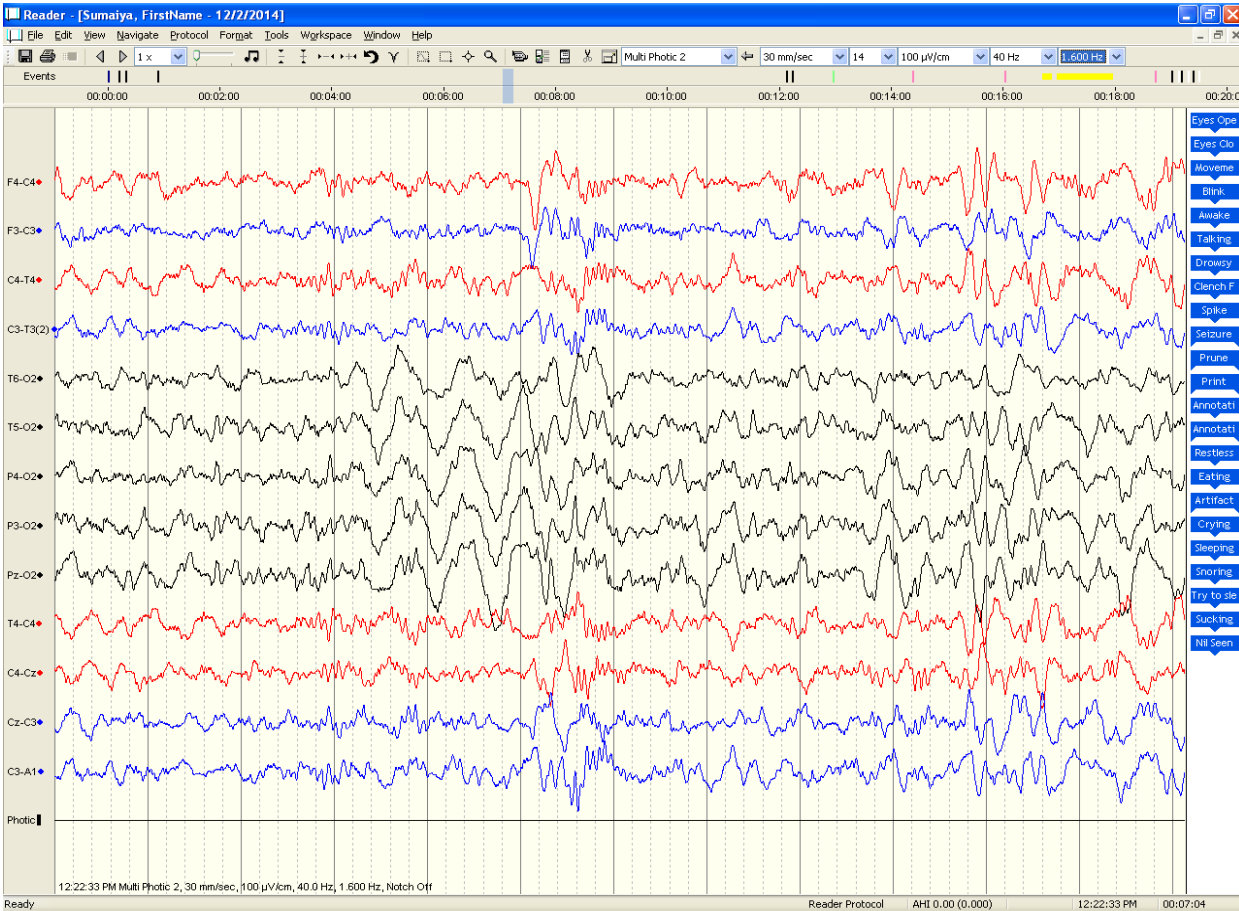
Type of discharges

- **GENERALIZED**
- **FOCAL / LOCALIZED**
- LATERALIZED
- BILATERAL
- SYNCHRONIZED/ ASYNCHRONIZED
- ISOLATED
- REPEATED
- PERIODIC
- Specific pattern of discharges
 - Burst suppression
 - Continuous / discomipis
 - Hypsarrhythmic



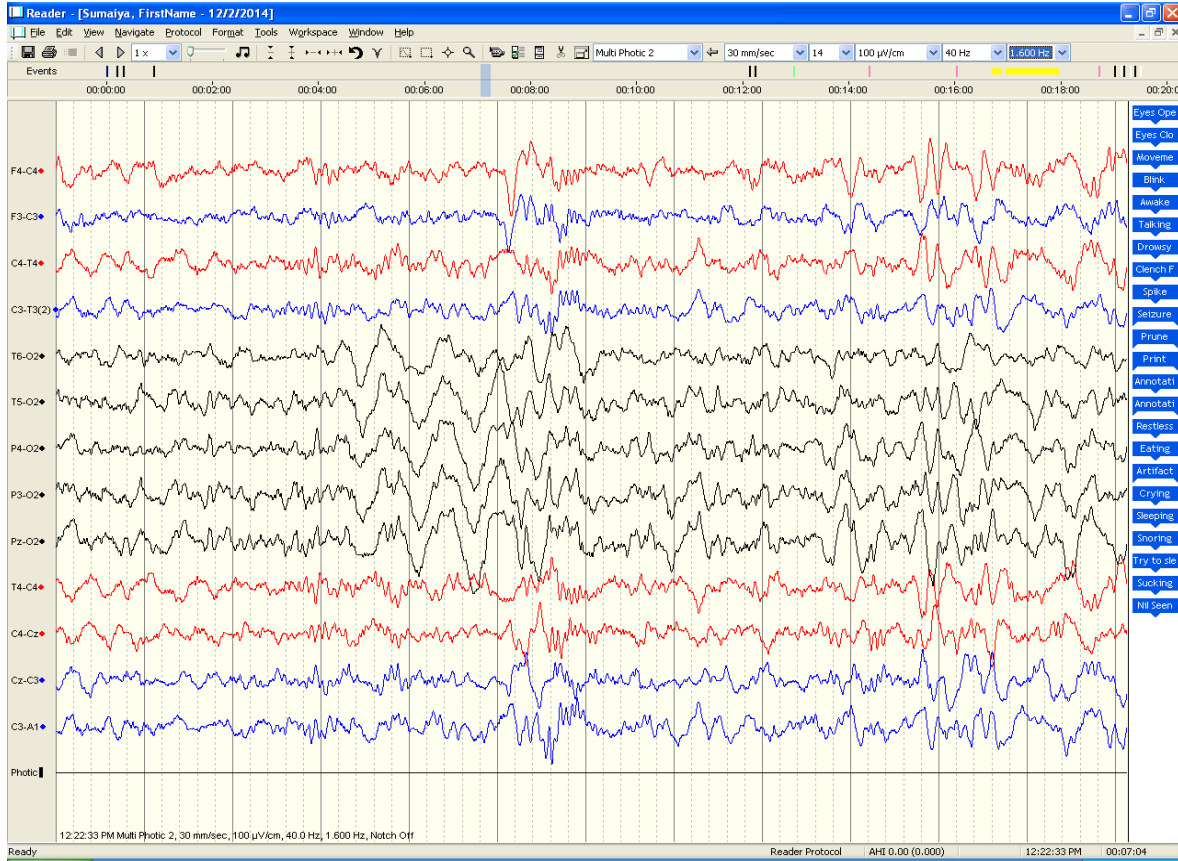
Effect of Machin setting

Off Low cut filter

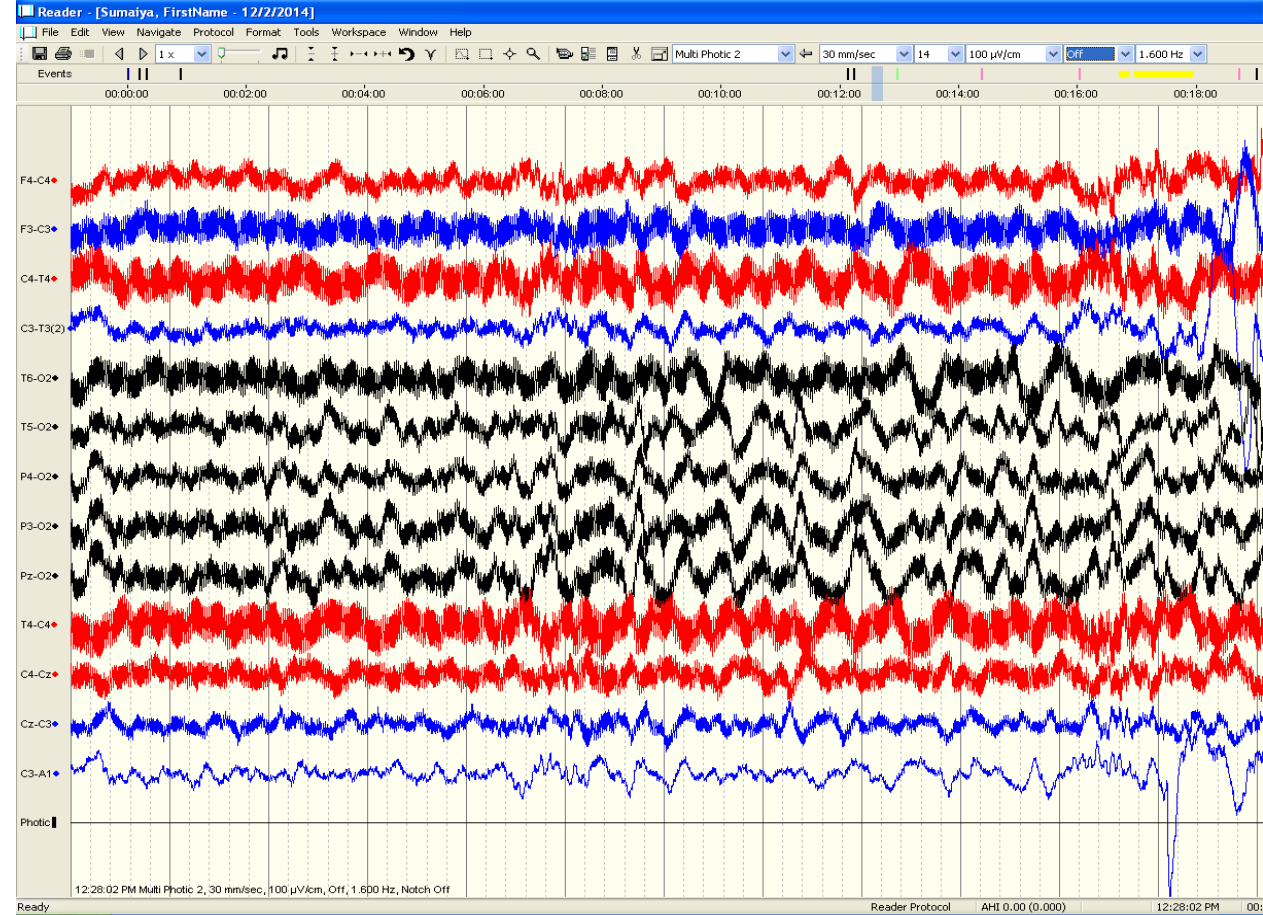




Off HFF

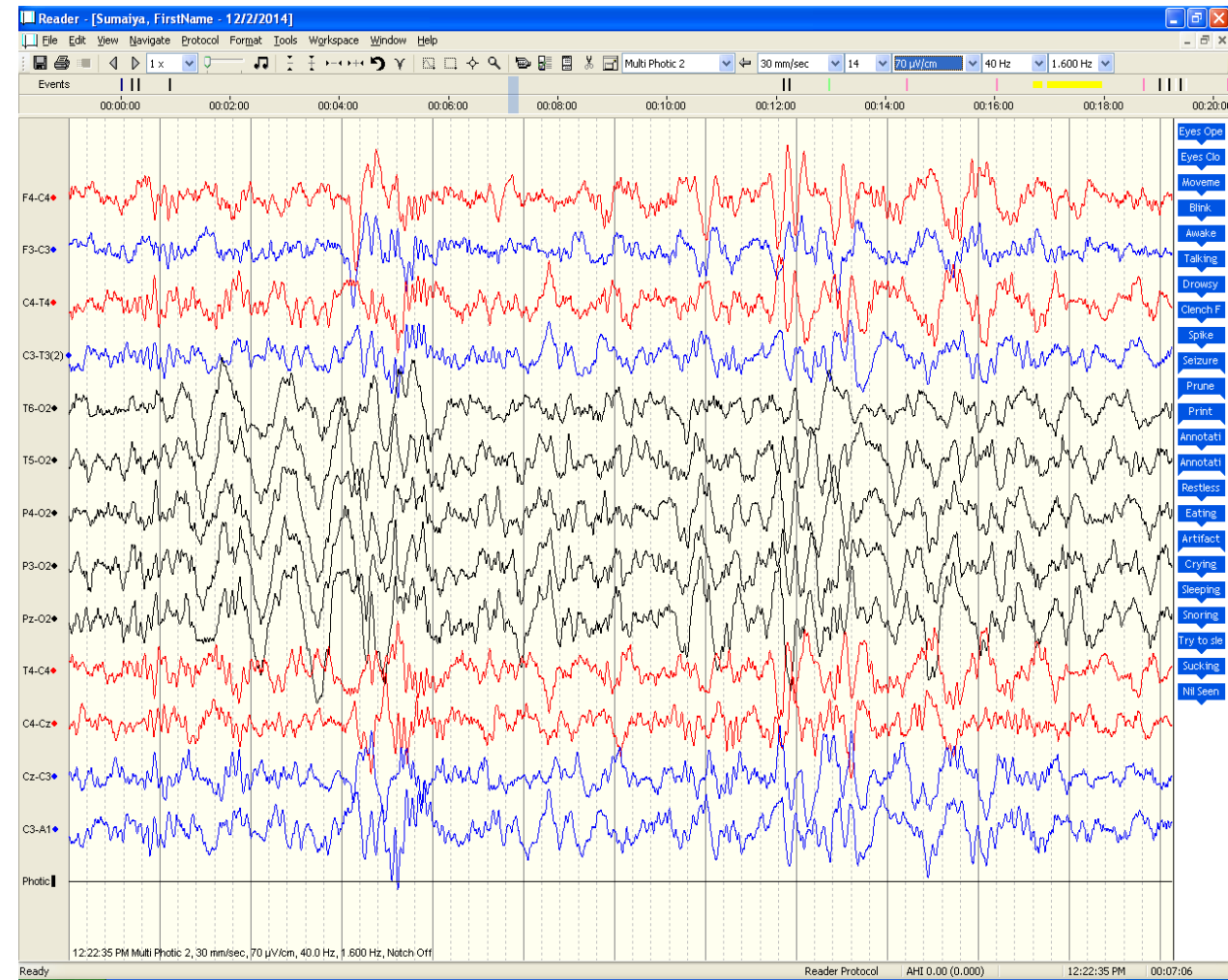
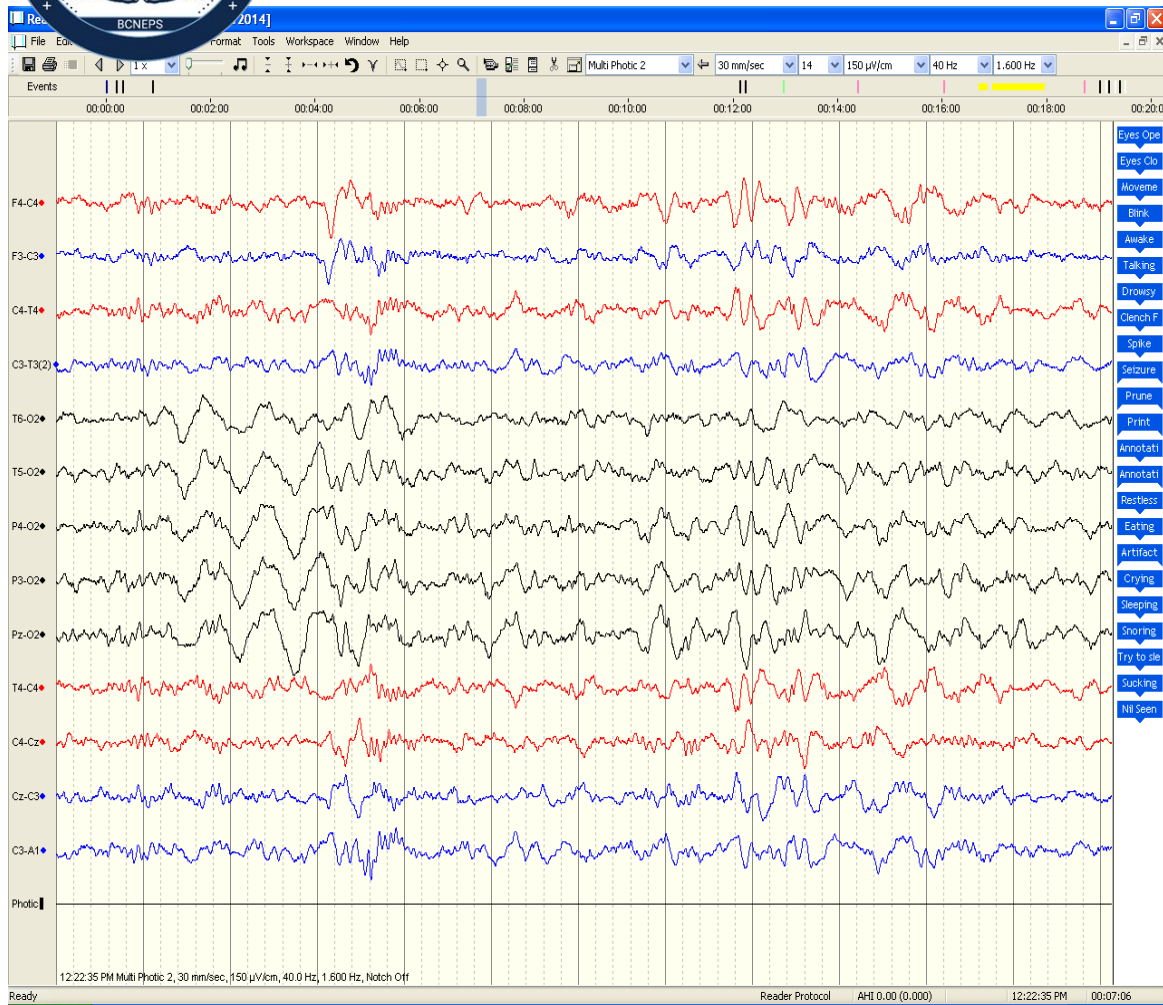


Power line interference



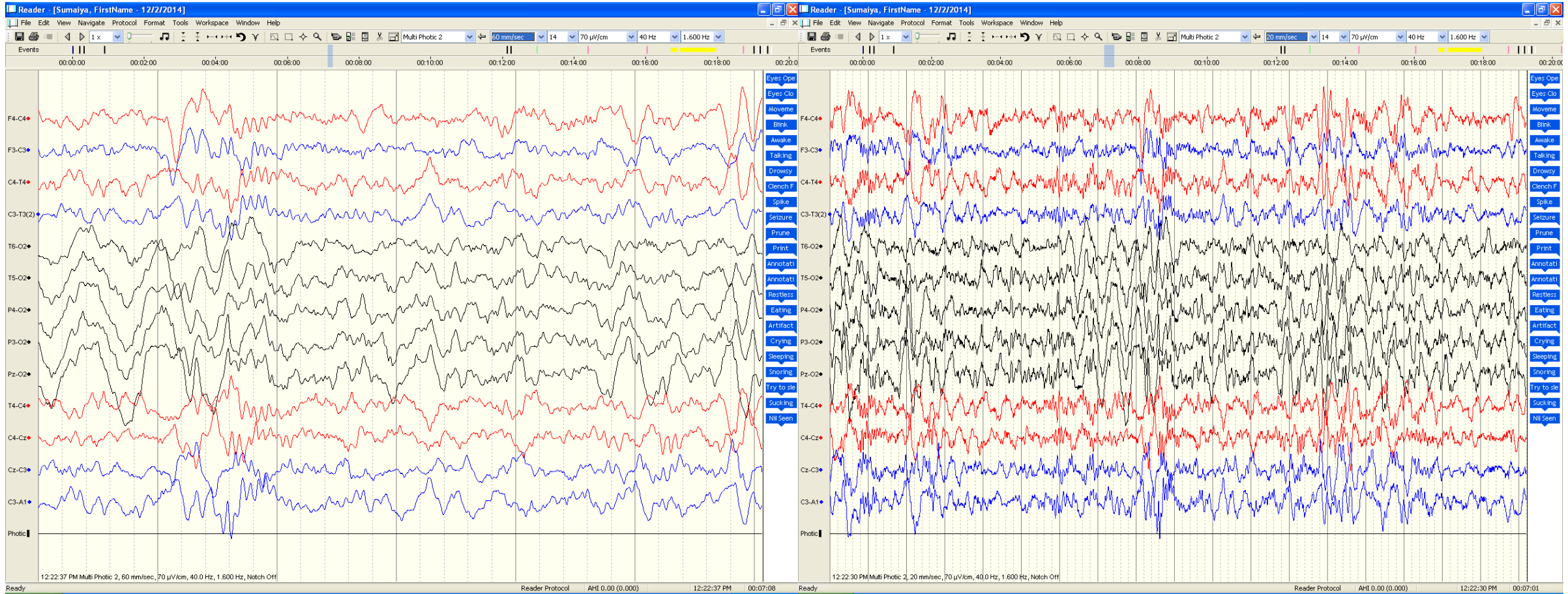


Sensitivity: $100\mu\text{V}/\text{cm}$ to $70\mu\text{V}/\text{cm}$





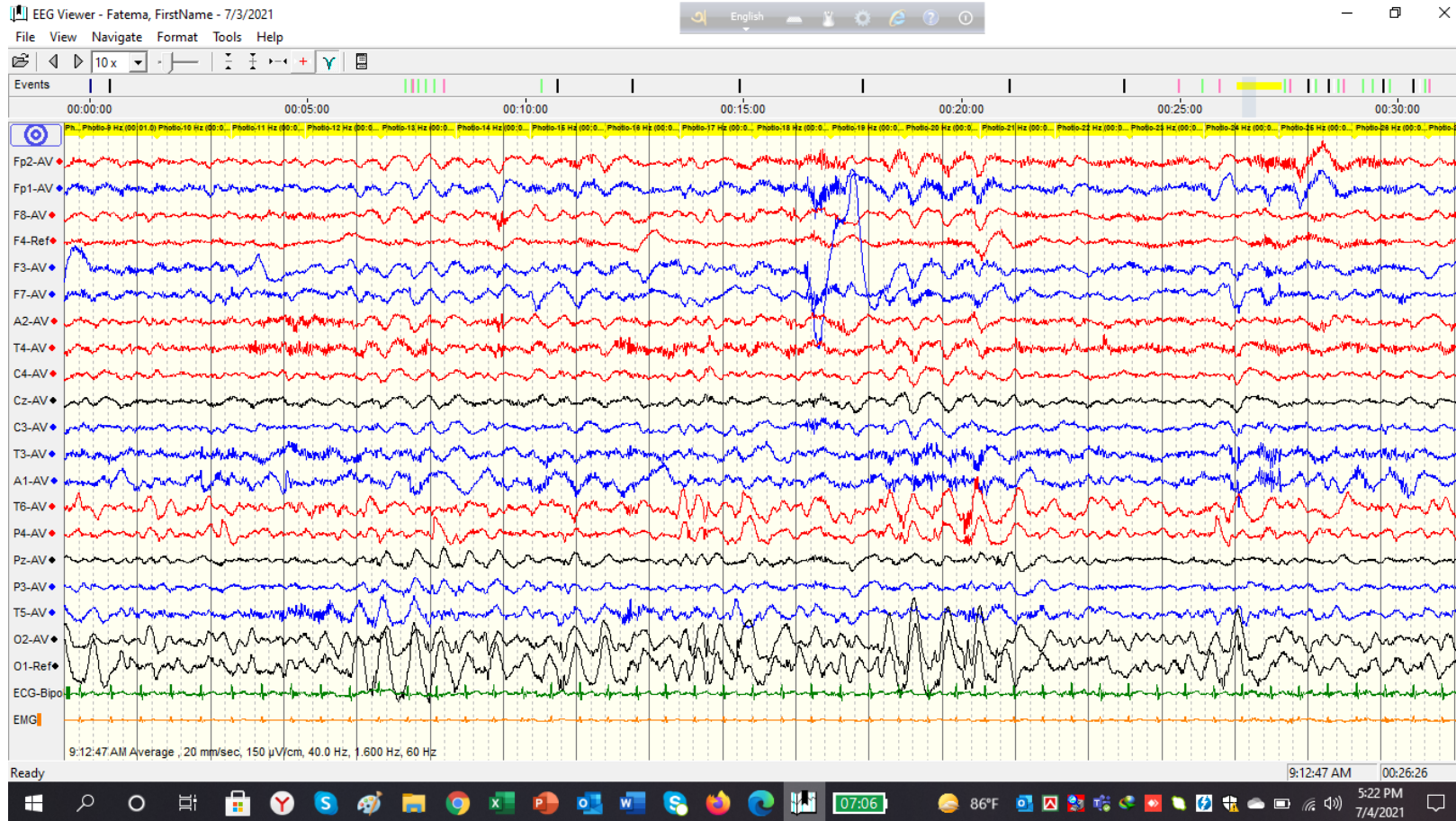
Changing paper speed



Thank you

Q & A

09/08/2021



**PATIENT
INFORMATION:**

PATIENT NAME: Fatema
DATE OF BIRTH:
AGE: 9m **SEX:** F
GESTATIONAL AGE:

P ID : D- 1443147
REC. DATE: 03/07/2021
OFC: 41cm **ENO:** 31517
HAND DOMINANCE: Rt

CLINICAL SUMMARY:

REFERRED BY: Prof. Dr. Md. Mizanur Rahman
Poor eye contact, can't sit, no clinical sz, FT hospital delivery by CS, N.
Jaundice.

**CURRENT
TREATMENT:**

Trihexy, Sirdalud.

**PATIENT STATE
DURING RECORDING:**

The child was sleeping at the beginning later awake during recording

PREVIOUS EEG

Nil

DESCRIPTION: Irregular 4-5 c/s 30-45 μ V activities are seen over both the hemispheres mixed with slower components in the background. Repeated complexes mixed with polymorphic activity, 1-2 c/s 70-200 μ V are noted over the temporo-parietal and occipital regions predominantly involving the right hemisphere from beginning to end. No cerebral reactivity is observed over the posterior region on **eye closure**. Frequent sleep spindles are noted over the fronto-central area during **sleep state**.

COMMENT: This record shows high amplitude epileptogenic complexes mixed with polymorphic slow waves over the posterior regions. Other activities in asleep state are within normal limit for the age and state of the child. The EEG feature is suggestive of epileptogenic cerebral lesion. Please correlate.