Non-epileptiform Abnormalities

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September 18, 2022

Outline

- **Abnormal slow pattern**
  - Focal slow pattern
  - Generalized slow pattern
  - Intermittent rhythmic delta activity
- **Amplitude abnormality**
  - Amplitude asymmetry
  - Generalized change of amplitude
- **Other abnormalities**
  - Triphasic wave
  - Abnormal alpha or reactivity of alpha
  - Abnormal beta activity
  - Abnormal sleep potentials

Abnormal Slow Wave

- Focal slow pattern
- Generalized delta slow pattern
- Intermittent rhythmic delta activity

Abnormal Slow Assessment

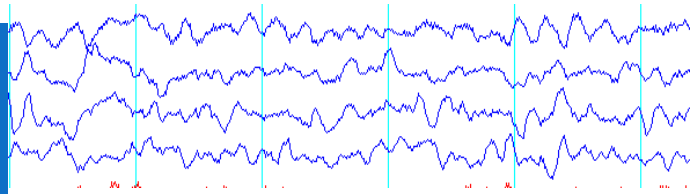
Parameter	Example
Morphology, Rhythmicity	Polymorphic (arrhythmic) Monomorphic (rhythmic)
Amplitude	High, low
Frequency	Delta, theta
Continuity	Intermittent, continuous
Localization	Focal, regional, lateralize, generalize
Reactivity	Reactive, nonreactive
Sleep stage	Seen in awake, drowsiness, sleep

More acute severe pathology

Lower frequency, higher amplitude, greater abundance

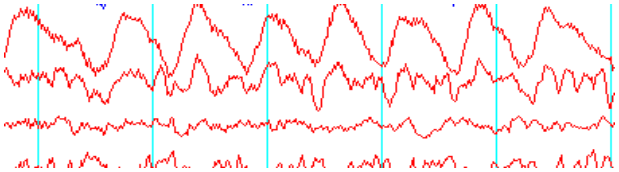
Slow pattern

-Focal
-Generalized
-IRDA



Polymorphic

- Irregular in sharp
- Variable duration and frequency



Rhythmic

- Regular in sharp
- Fairly constant duration, stable predominant frequency

Focal slow pattern

Focal slowing

- Frequency < 8 Hz & limited distribution
- Indicate focal cerebral dysfunction but nonspecific etiology
- Exclude normal physiologic:
  - Midtemporal theta of drowsiness
  - Frontal midline theta (Ciganek)
  - Posterior slow wave of youth (PSOY)

Focal slow Mechanism

- Result of cortex deafferentation from subcortical structure

- **Subcortical white matter lesion**
- Thalamic lesion
- Hypothalamic lesion

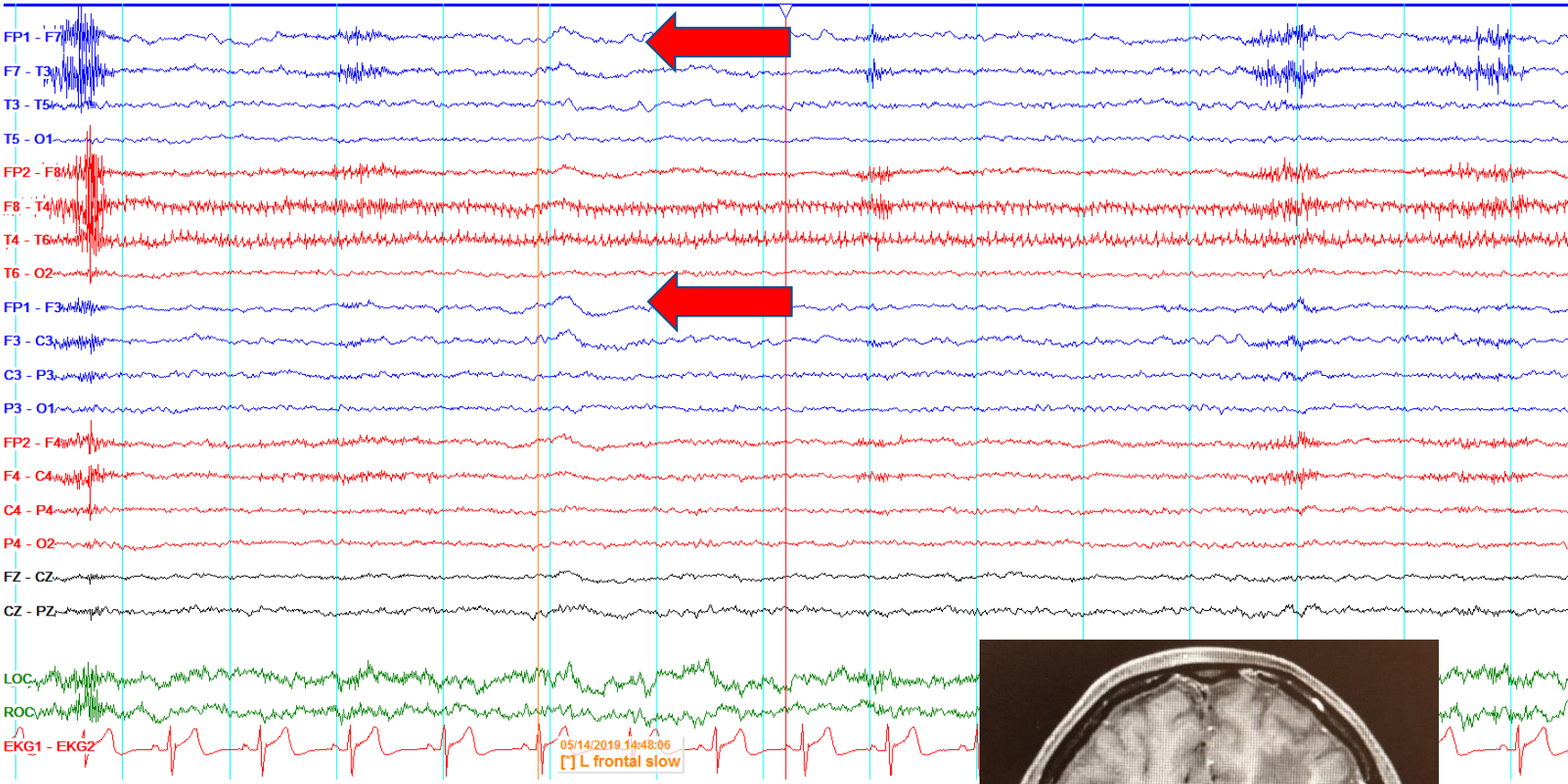
- Not pure cortical lesion, unilateral mesencephalic tegmental lesion, vasogenic edema

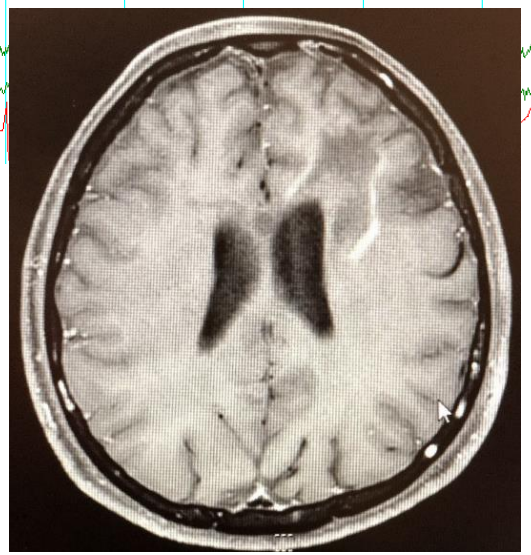
Gloor et al. Neurology 1997;27:326-33.

Focal slow: clinical correlation

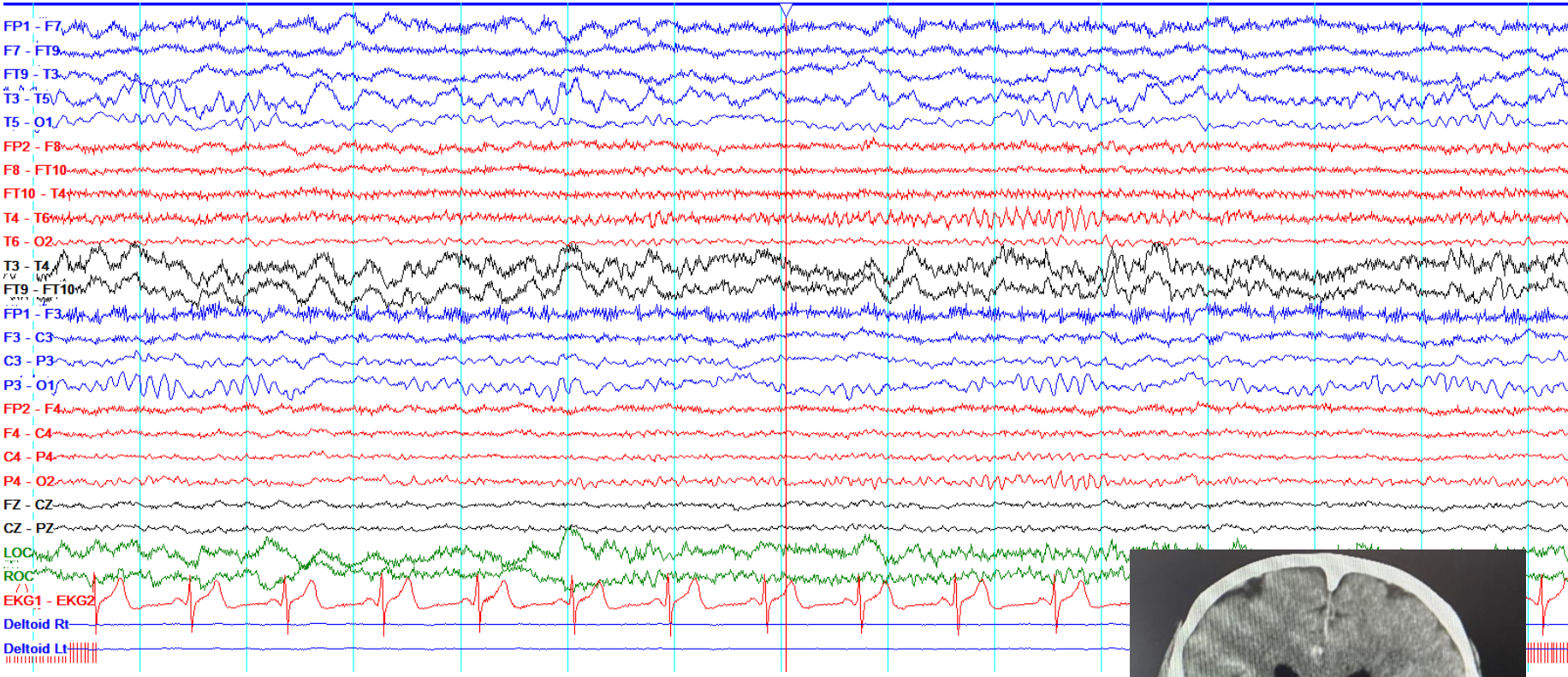
- **Focal arrhythmic polymorphic delta activity**
  - Localized structural lesion in subcortical white matter: continuous slow
    - Stroke, abscess, intraparenchymal hematoma or contusion, tumor
  - Functional: disappears in hours or few days
    - Postictal, complicated migraine, head trauma
  - Rarely: toxic-metabolic (hypo/hyperglycemia)

- **Focal rhythmic monomorphic slowing**
  - More commonly a/w grey matter lesions



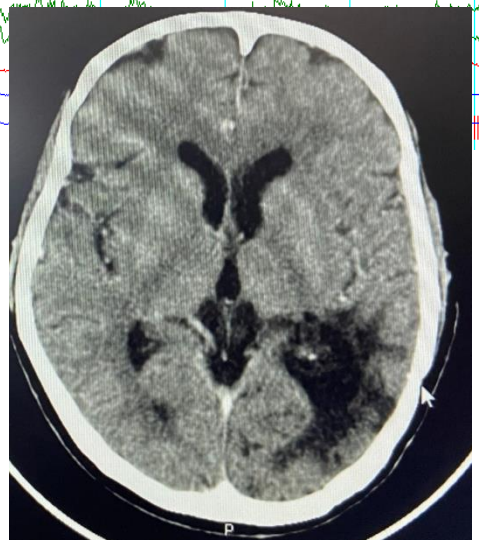


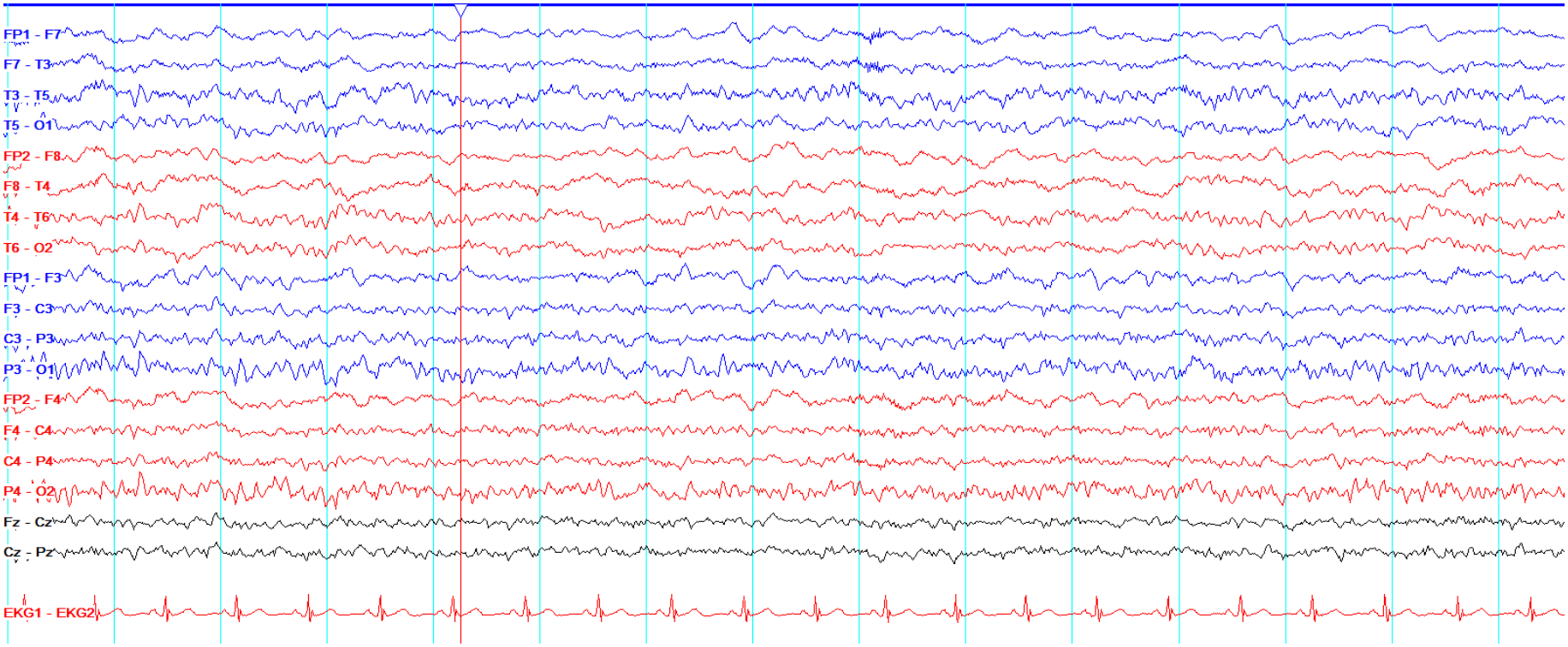
Occasional left frontal slow
Subcortical white matter lesion



Continuous polymorphic slow over left hemisphere predominant at temporal area

Structural lesion:
Encephalomalacia at left temporo-parieto-occipital region





Postictal bitemporal polymorphic slowing

Generalized arrhythmic slow

Generalized Arrhythmic Slow

- Pattern: generalized irregular in shape and variable duration and frequency
- Mechanism:
  - Interfere with structural or function of both hemispheres, often involve subcortical white matter or thalamocortical pathways
  - Widespread interruption cortical input from brainstem reticular formation

Generalized

Arrhythmic

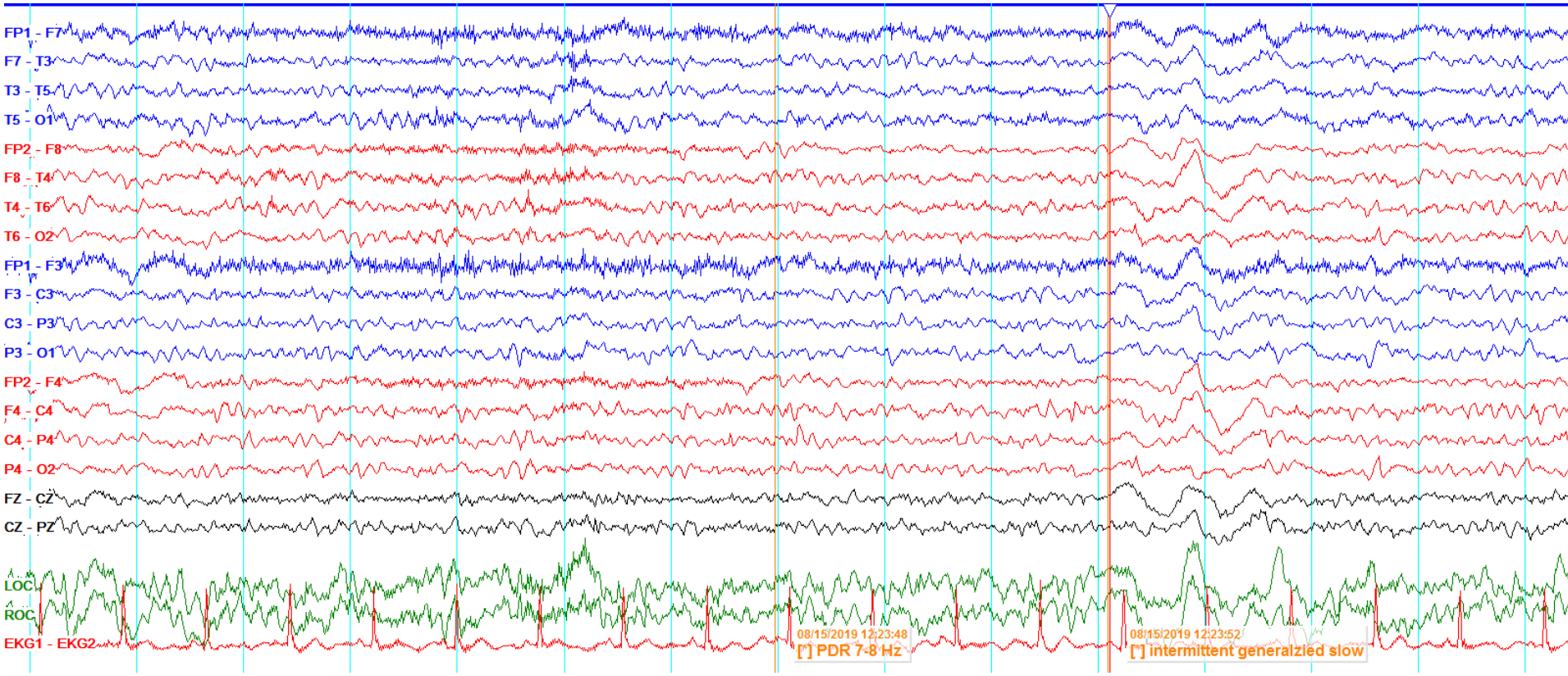
Slow

- Exclude physiologic: drowsiness or sleep
- Diffuse but nonspecific bilateral cerebral dysfunction
  - Most common metabolic and toxic
  - Structural, HIE, infection, neurodegenerative disorder
  - Postictal state
  - Medication effect

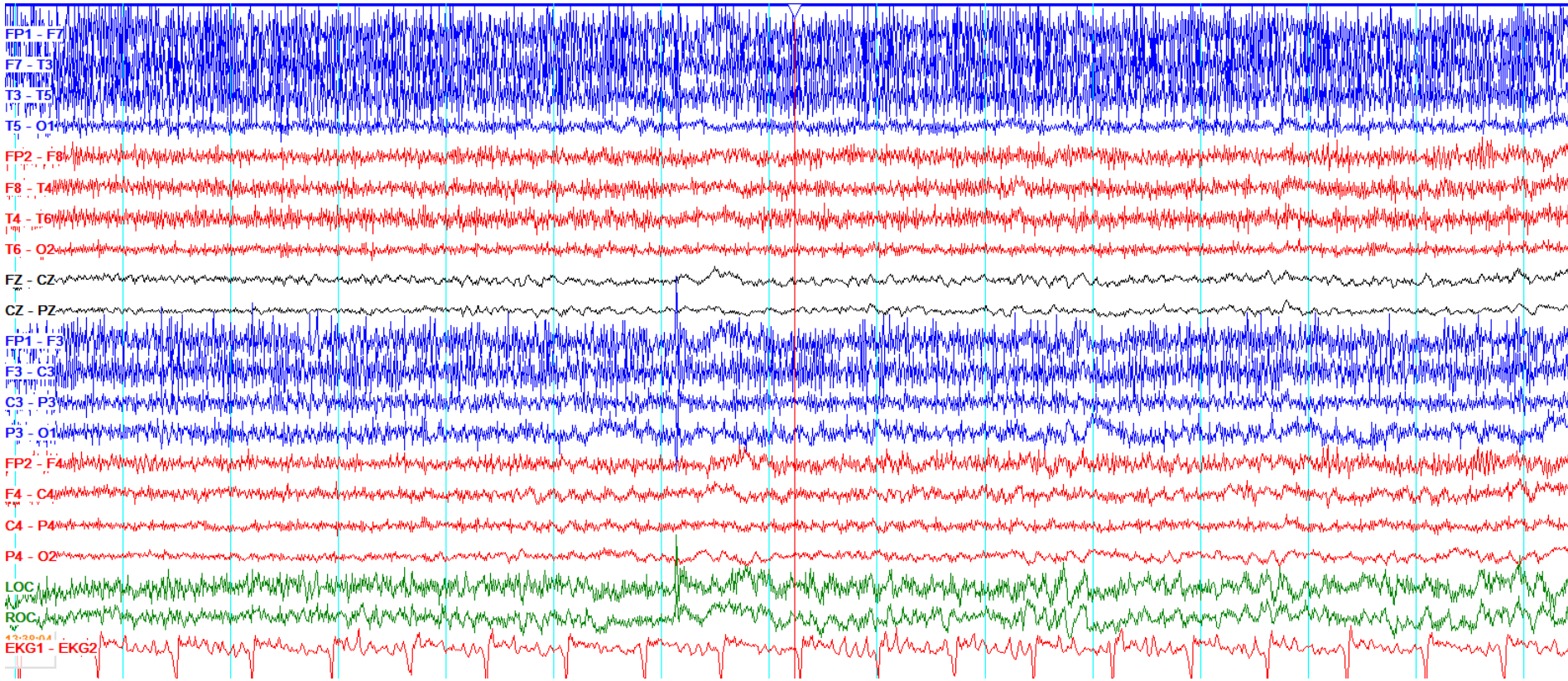
Generalized Arrhythmic Slow

Degree of Encephalopathy & EEG pattern

- **Mild**
  - Slowing of posterior waking background
  - (Intermittent) generalized theta
- **Moderate**
  - No posterior dominant rhythm
  - Predominant polymorphic delta slowing
  - Less reactivity or organization
- **Severe**
  - Continuous polymorphic delta slow
  - Attenuation of BG
  - Not reactive
  - Burst suppression
  - Generalized suppression (< 10 mV)
  - Isoelectric

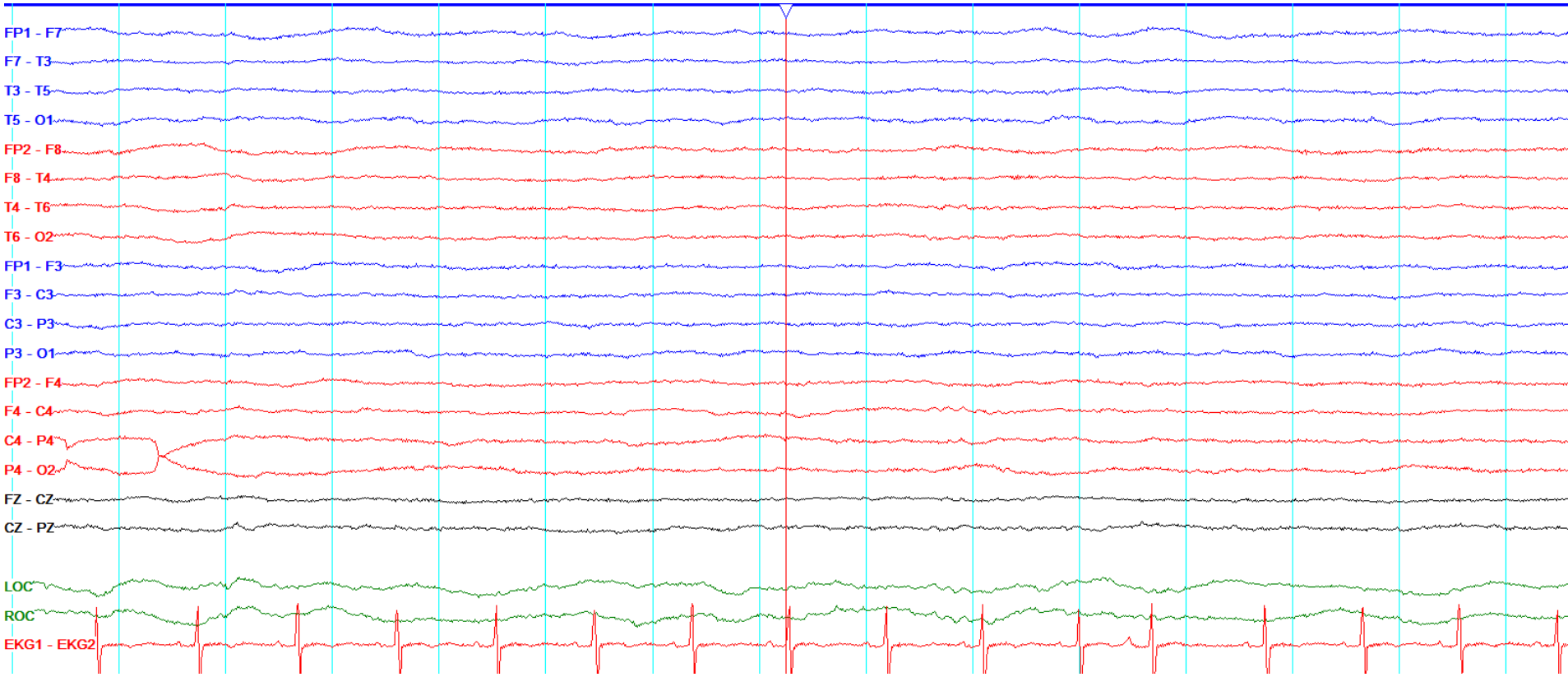


Mild BG slow, PDR 7-8 Hz, intermittent generalized delta
Mild diffuse encephalopathy



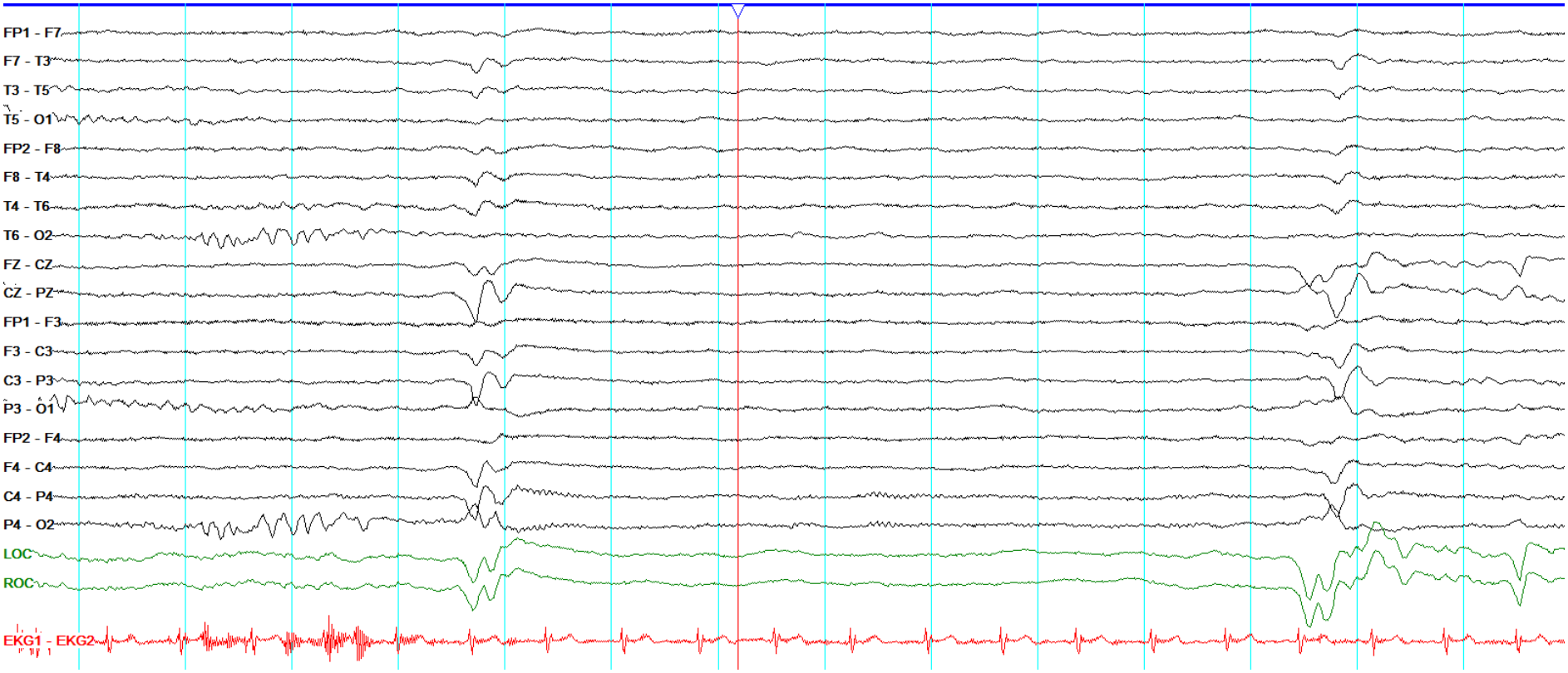
Generalized delta and theta slow, no PDR

Moderate diffuse encephalopathy



Diffuse BG attenuation and delta > theta slowing, mild reactivity

Moderate to severe diffuse encephalopathy



Post cardiac arrest

Discontinuous BG with 7 seconds BG suppression

Severe diffuse encephalopathy

Intermittent rhythmic delta activity

IRDA

- Runs of high voltage rhythmic 2-3 Hz at irregular interval
- Usually bisynchronous and symmetric
- May be asymmetric or unilateral
- Increased during drowsiness and HV
- Reactive/attenuate to eye opening or alerting
- Disappear with onset of stage 2 or REM

IRDA Mechanism

- Increased transventricular pressure within third ventricle → dilatation of third ventricle (Daly et al., 1953, Hess 1975, Klass and Daly 1979)
- Diffuse grey dysfunction either cortical or subcortical (Gloor et al. 1968)
- Focal intracranial lesion → increased ICP → widespread brain dysfunction (Schaul et al. 1981)
- More likely during active fluctuating, progressive or resolving widespread brain dysfunction
- Less likely chronic stable brain dysfunction

Nonspecific, wide variety of pathological process
Systemic toxic metabolic, focal intracranial lesion

IRDA: Physiologic

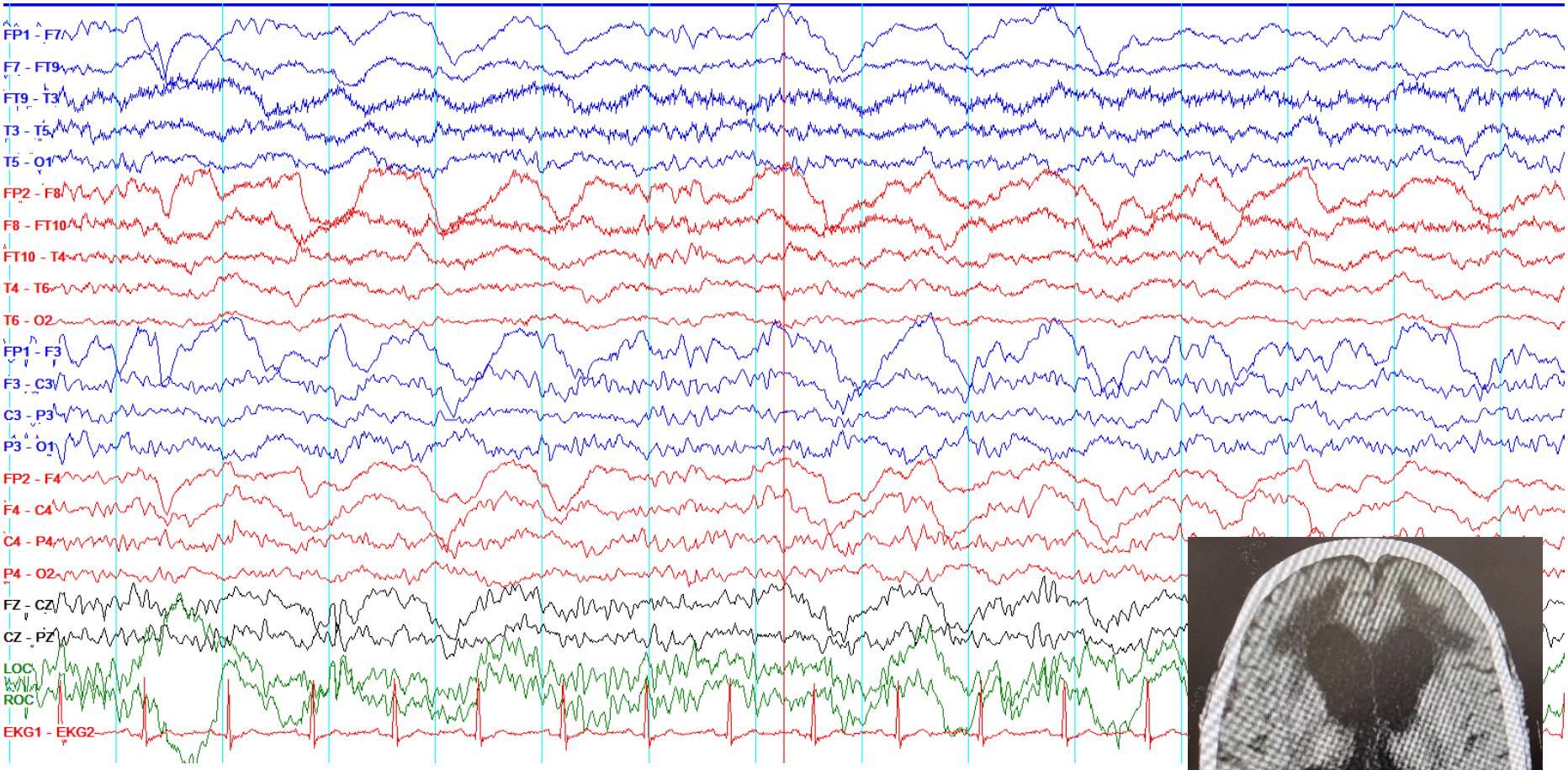
- Normal response to hyperventilation
- Paroxysmal burst of drowsiness in children (hypnagogic hypersynchrony)
- Sleep deprived normal adult
- Occasionally in asymptomatic elderly

IRDA

- Not suggest specific localization, age specific
  - Frontal intermittent rhythmic delta activity (FIRDA): Adult
  - Occipital intermittent rhythmic delta activity (OIRDA): Children
- More specific localization
  - Temporal intermittent delta activity (TIRDA): TLE

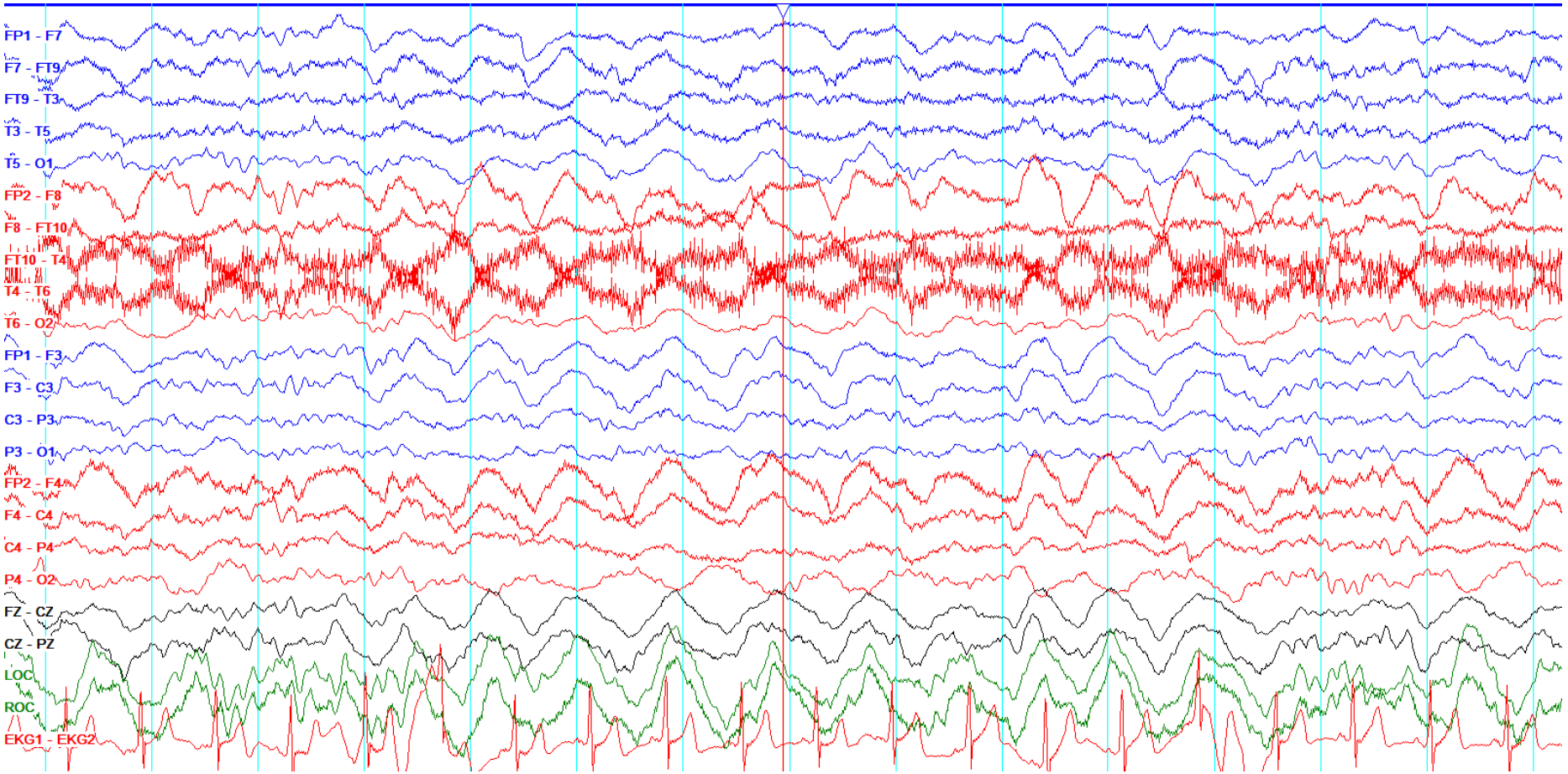
FIRDA

- Bilateral synchronous 2-3 Hz delta with frontal predominance
- Non-specific etiology
  - Early study
    - Tumor of posterior fossa and third ventricle
    - Deep midline lesion
    - Hydrocephalus
  - More recent study
    - Diffuse brain injury, metabolic disturbances, toxic
    - Focal structural lesion: asymmetric FIRDA

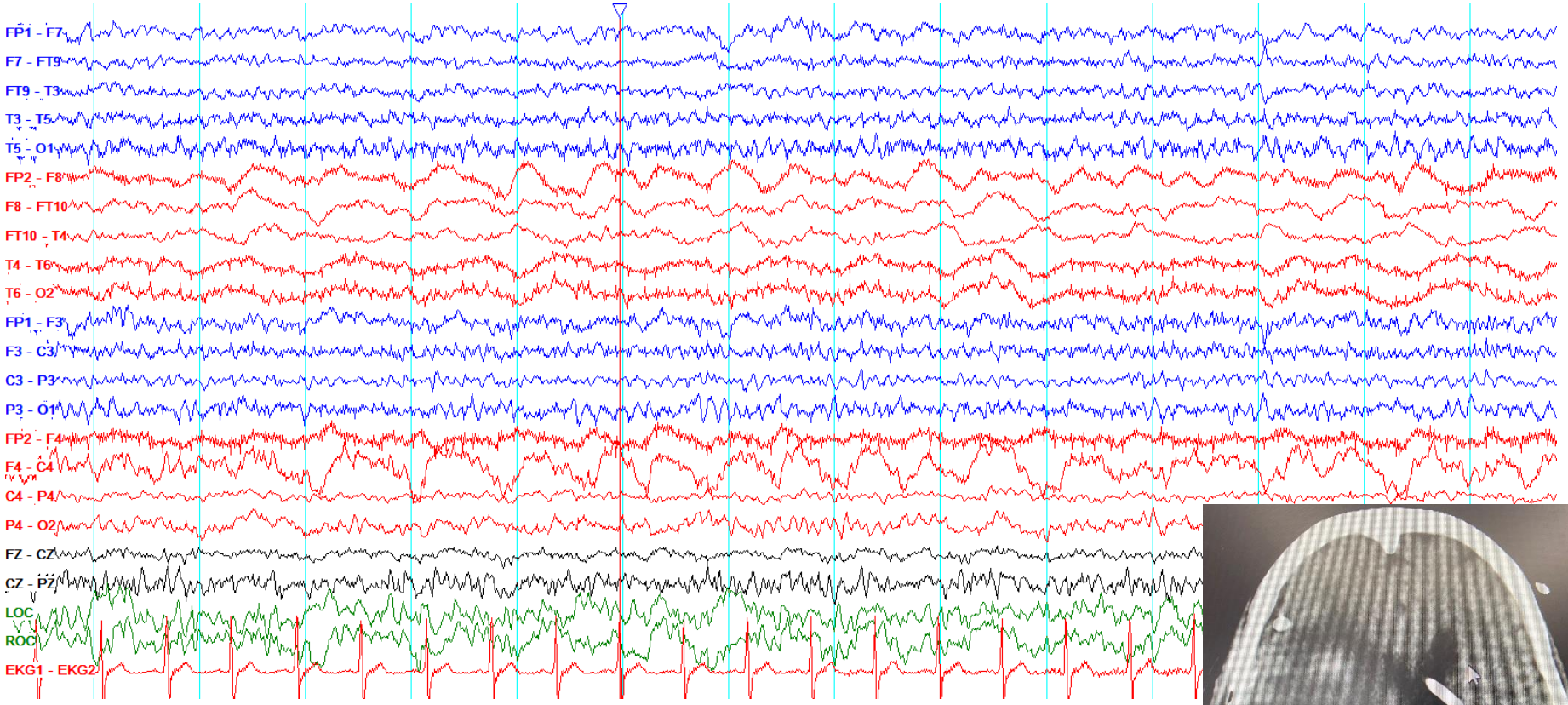


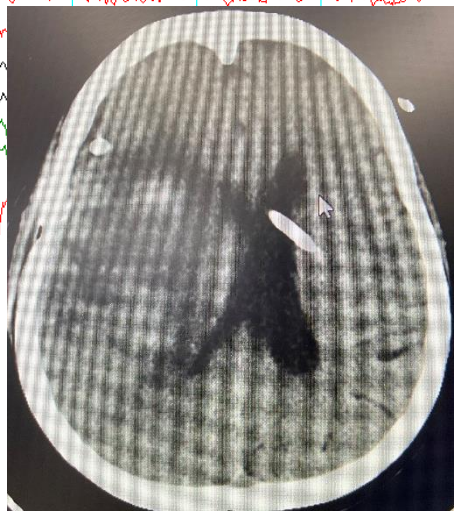
Frontal intermittent semi-rhythmic delta activity

Hydrocephalus



Frontal predominant generalized semi-rhythmic delta activity
LGI1 autoimmune encephalitis



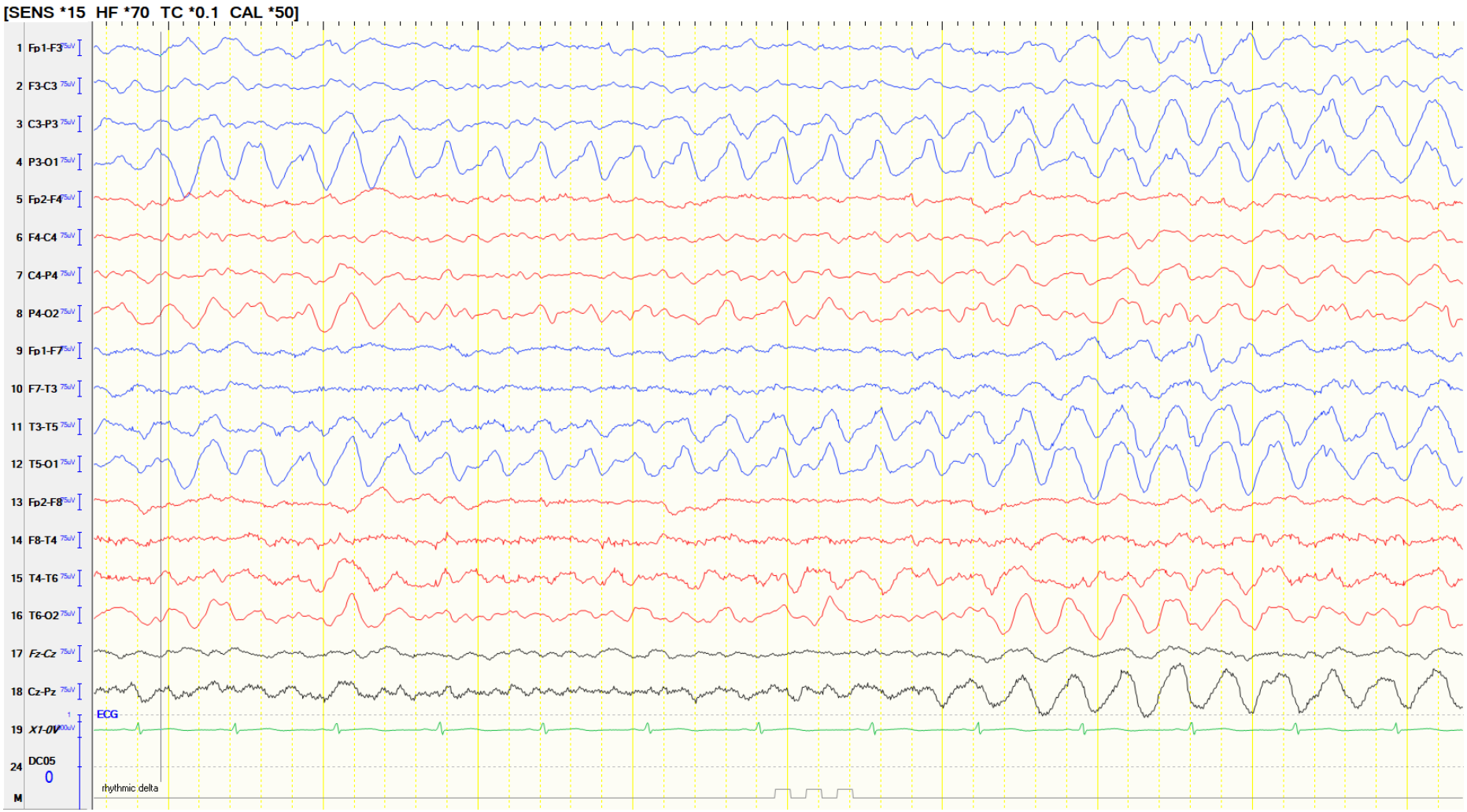


Asymmetric FIRDA

Ruptured aneurysm right MCA bifurcation s/p clipping

OIRDA

- Bi-posterior predominant rhythmic delta activity
- Occurred primarily in children with absence epilepsy
- Lesion in occipital lobe with involvement of posterior lateral ventricle: unilateral OIRDA

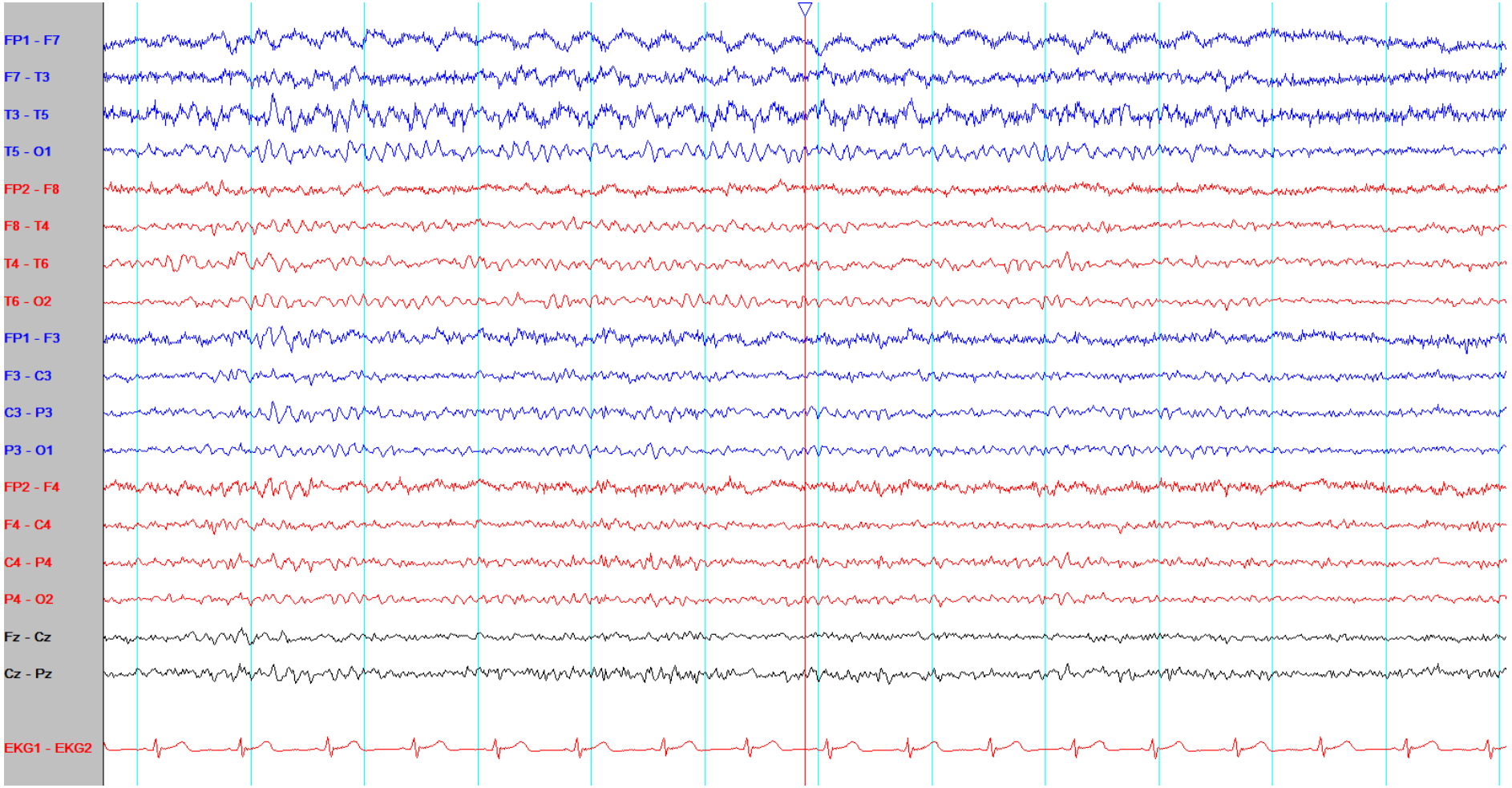


Occipital intermittent rhythmic delta activity

Absence epilepsy

TIRDA

- Bursts or train of >/=3 sec of repetitive, rhythmic, saw-toothed or sinusoidal 1-4 Hz, 50-100 mV
- Predominant over anterior temporal region
- Potential epileptogenicity in ipsilateral temporal lobe
  - Same meaning as epileptiform activity
- May relate with structural change affect lateral ventricle
  - Projected or distance rhythm over temporal lobe



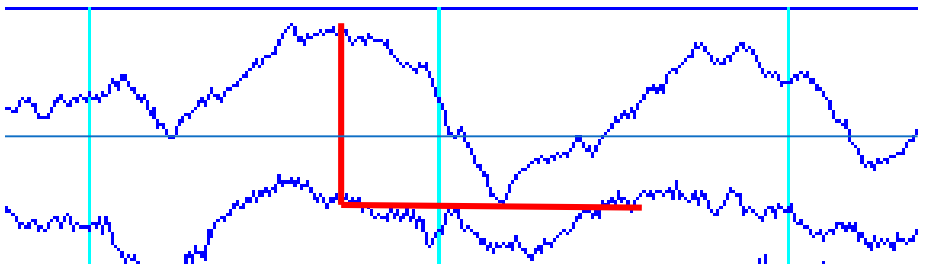
Left temporal intermittent rhythmic delta activity
Patient with left hippocampal sclerosis

Outline

- Abnormal slow pattern
  - Focal slow pattern
  - Generalized slow pattern
  - Intermittent rhythmic delta activity
- **Amplitude abnormality**
  - **Amplitude asymmetry**
  - **Generalized change of amplitude**
- Other abnormalities
  - Triphasic wave
  - Abnormal alpha or reactivity of alpha
  - Abnormal beta activity
  - Abnormal sleep potentials

Amplitude

- Peak to trough
- Standard 10-20 electrodes
- Montage: longitudinal bipolar



Amplitude asymmetry

- **Normal asymmetry**
  - Some asymmetry of alpha and beta may be due to difference in skull thickness
  - Asymmetry of photic driving, POST, mu rhythm in absence of other abnormalities
- **Abnormal asymmetry**
  - Consistent asymmetry of beta > 35%
  - Consistent asymmetry of alpha > 50%, 35-50% when lower on right side

Cause of Amplitude asymmetry

- **Decreased amplitude (focal attenuation)**
  - Impaired/damage cortical generators:
    - Unilateral lesion
    - Transient: focal seizure, TIA, migraine
  - Change in conducting medium between cortex and recording electrode (SDH, skull defect, local scalp edema)

- **Increased amplitude** of all type of activities: local skull defect

Amplitude abnormality

- Amplitude asymmetry

- **Generalized change of amplitude**
  - **Low voltage and suppressed**
  - **High voltage**

Generalized Change of Amplitude

- Low voltage and suppressed patterns
  - Low voltage: <20 µv
  - Generalized suppression: <10 µv
  - Burst suppression
  - Electro-cerebral inactivity
- High voltage pattern

Abnormal Low Amplitude

- Low amplitude normal adult subjects 5-10%
- Normal low amplitude < 20 µV
  - Occurs transiently from eye opening, mental effort, anxiety, alerting or drowsiness
- Moderate or marked reduction amplitude <10 µv → abnormal at any age
- Generalized attenuation/suppression
  - Generalized cortical injury or transitory dysfunction
  - Excessive fluid or tissue over cortex

Burst Attenuation/ Suppression

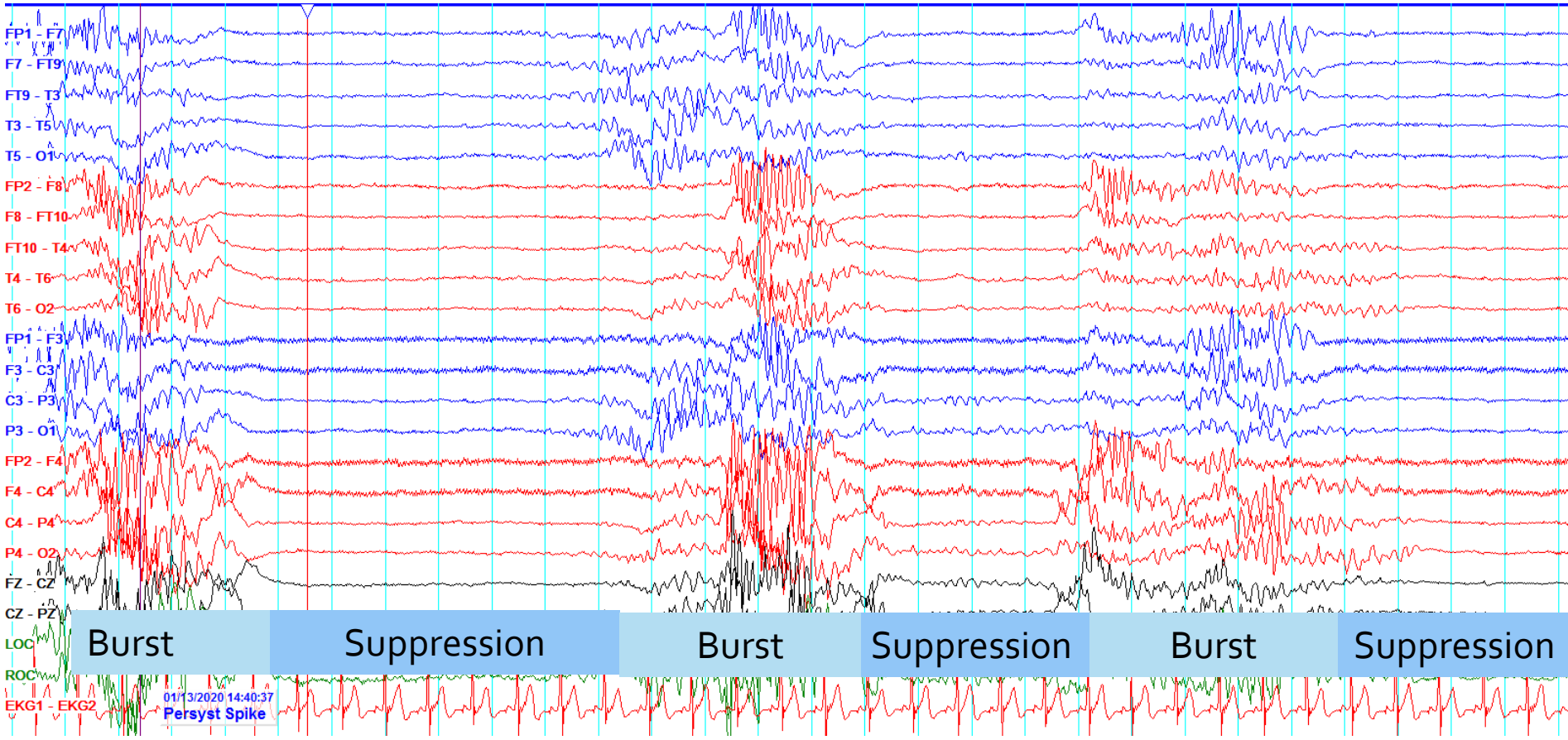
Bursts of high voltage 50-100 µV, mixed frequency, usually with sharply contoured/spike waves at least 0.5 sec (typical 1-3 sec)

Alternate with **attenuation** < 20 µV or **suppression** < 10 µV, **50-99%** of recording

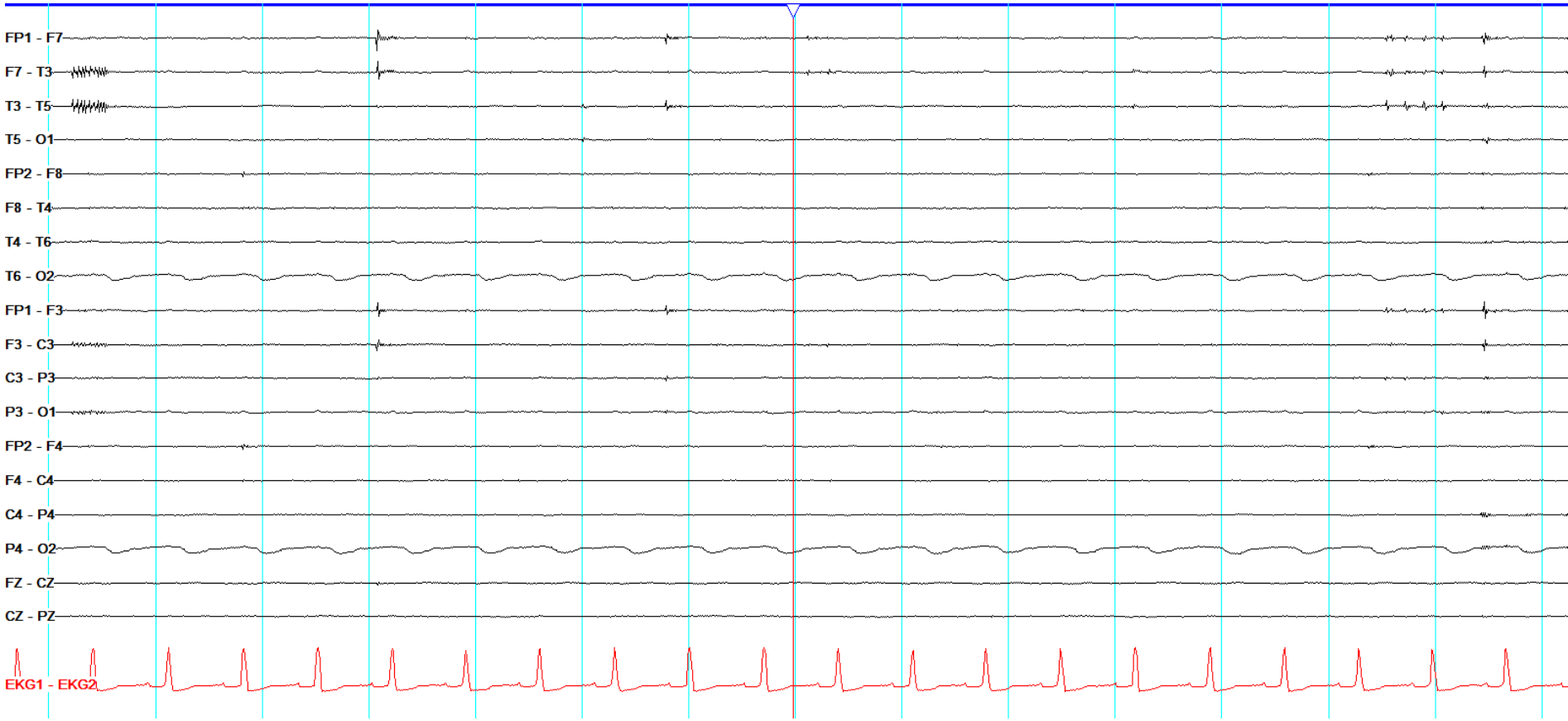
(**Burst: >/= 0.5 seconds, at least 4 phases**)

Etiology

- Anoxic
- Intoxication with sedative drugs
- Anesthetic use
- Hypothermia



97 year-old woman s/p cardiac arrest
Burst suppression pattern



S/P cardiac arrest

Generalized background suppression, pulse artifact

Severe diffuse encephalopathy

High amplitude

- Normal high amplitude in waking record of children and deep stage of sleep at any age

- Abnormal high amplitude
  - Amplitude >/= 150 µv
  - Amplitude > 100 µv is uncommon in wakeful adults, only considered abnormal on basis of frequency, morphology, or distribution rather than amplitude alone

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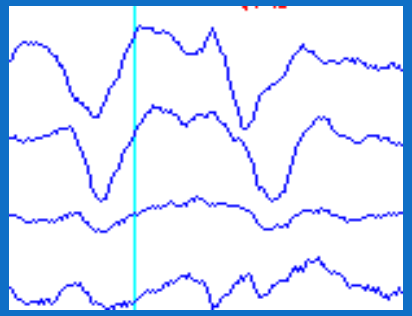
- **Other abnormalities**
  - **Triphasic wave**
  - **Abnormal alpha or reactivity of alpha**
  - **Abnormal beta activity**
  - **Abnormal sleep potentials**

Triphasic wave

Triphasic Wave

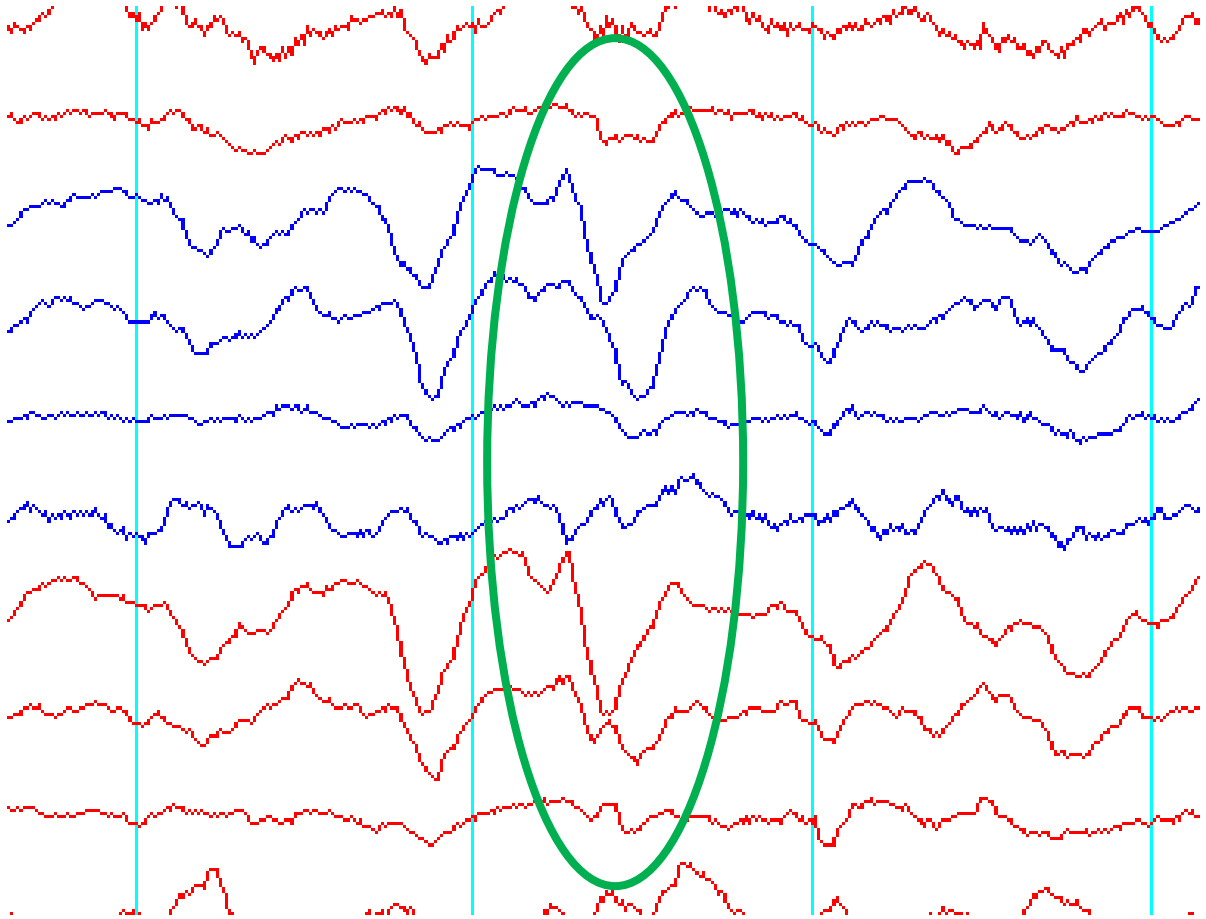


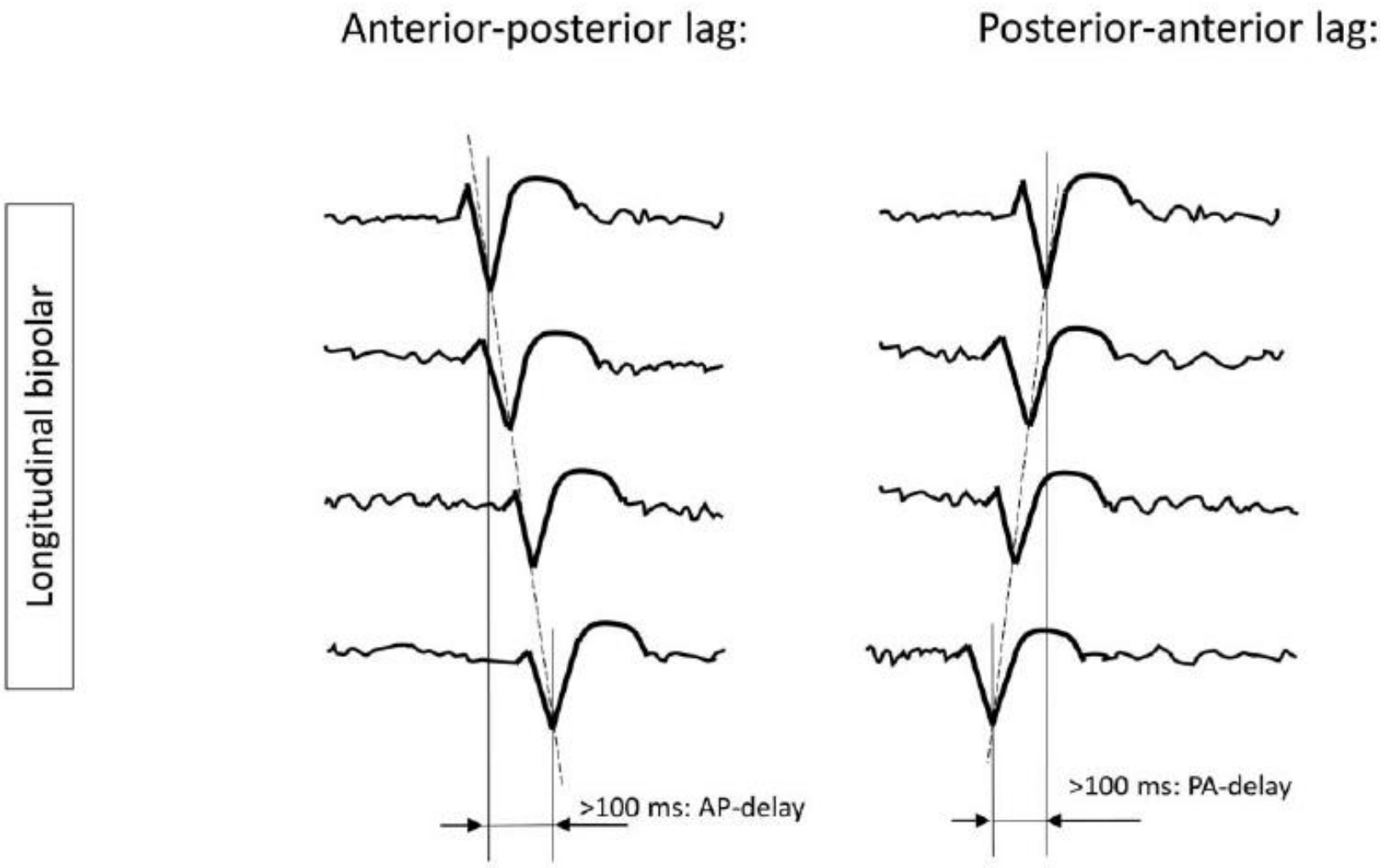
- Periodic short interval diffuse discharges
- Bilaterally synchronous and symmetrical waves
- Three phases:
  - Initial small negative wave (up)
  - Larger positive phase (down)
  - Followed by negative phase, longer duration than other 2 phases
- Bi-frontal predominant, 1.5-2.5 Hz, 70-300 µV
- May have >100 ms anterior–posterior or posterior-anterior lags
- May increase or attenuate with stimulation
- Intermittently in brief runs or continuous pattern

Triphasic wave

Negative-
Positive-
Negative

1. Sharpest
2. Highest
3. Longest





Antero-posterior lag (AP lag)

L. J. Hirsch et al. J Clin Neurophysiol 2021;38: 1–29.

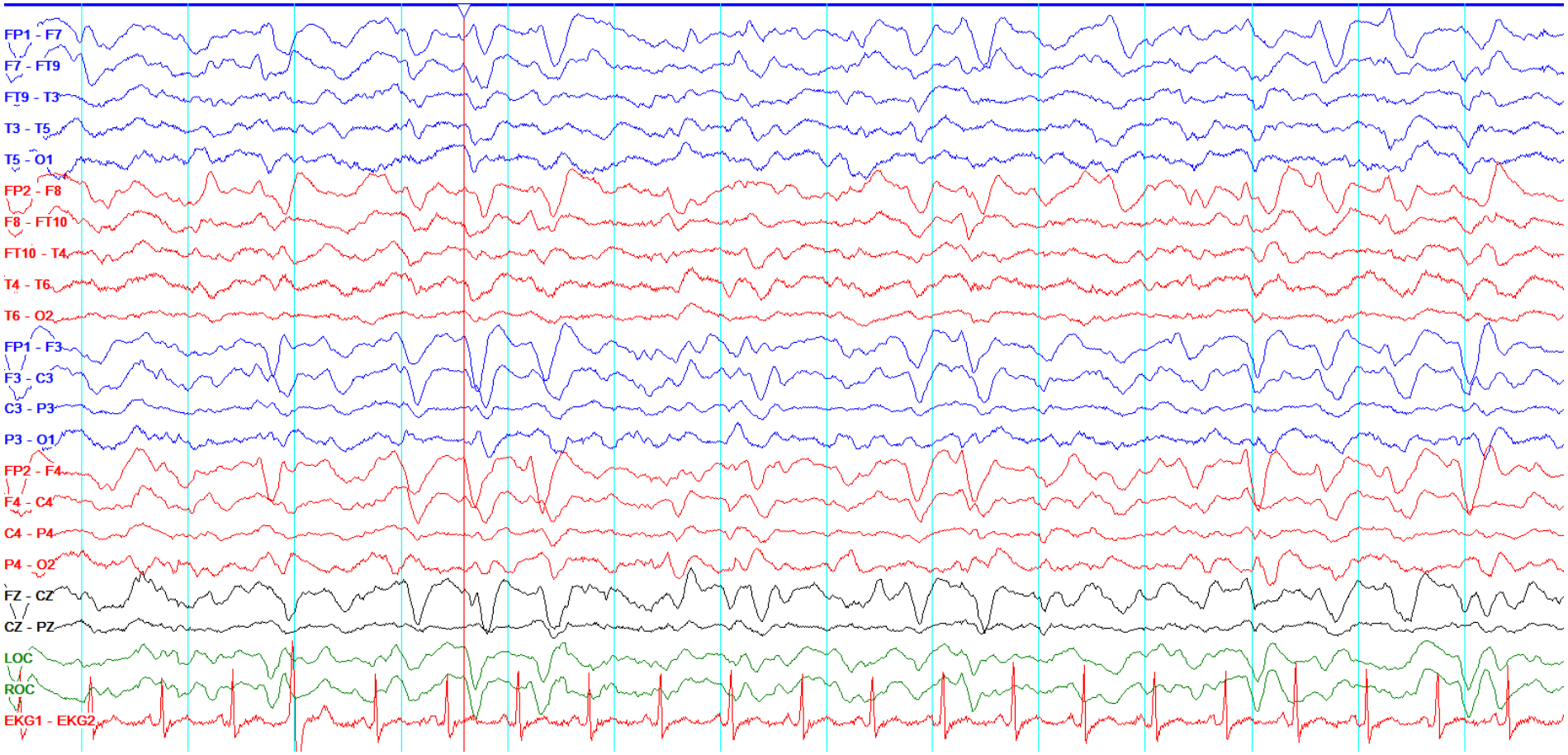
Triphasic waves

Historical Blunt spike and slow waves in hepatic coma

- **Etiology: wide variety**
  - Renal failure, hyponatremia, metabolic encephalopathy
  - Hepatic encephalopathy
  - Hypothyroidism
  - Encephalitis
  - Post-anoxic encephalopathy
  - Sepsis
  - Dementia: in rapidly progressive dementia→ CJD
  - Drug intoxication
  - Structural lesion: subdural hematoma, stroke

Correlate closely with severity of encephalopathy

- *Longer in duration and more widespread when became less responsive*
- *Usually absent in alert or deeply comatose patient*



Triphasic wave

Man with DM, HTN, CKD presented with altered mental status

Abnormal alpha or Reactivity of alpha rhythms

Abnormal frequency of alpha rhythm

Unilateral decrease in alpha frequency

- Mild asymmetry (consistent asymmetry 0.5 - 1 Hz)
- Marked asymmetry (>1 Hz)
- Unilateral functional or structural damage of occipital cortex or thalamus

Bilateral decrease of alpha frequency

- Abnormal <8 Hz
- Generalized disturbance of cerebral function
- Bilateral structural damage to occipital cortex or thalamic input
- Change of rhythmical input to cortex

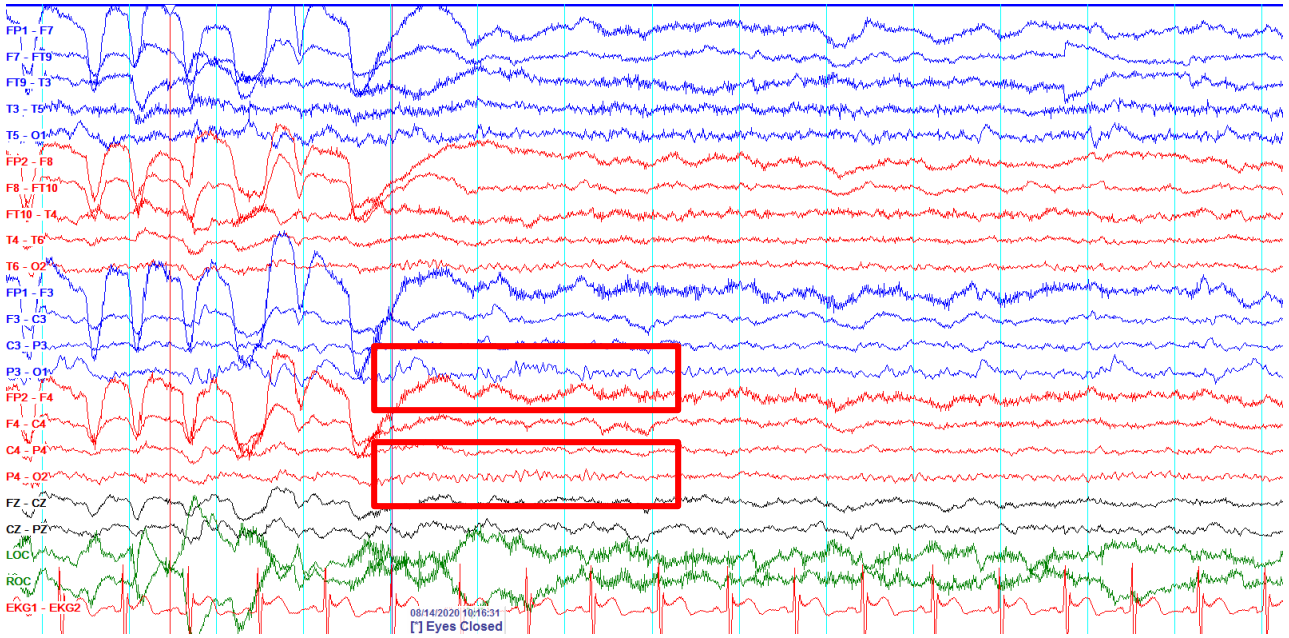
Bilateral increase of alpha frequency

- Metabolic disorder: fever, hyperthyroid

Abnormal reactivity of alpha

- **Bancaud's phenomenon**
  - Unilateral failure of alpha rhythm to attenuate with eye opening
  - Abnormal hemisphere is the side alpha does not attenuate
- **Alpha frequency coma**
  - Central pontine lesion
  - Widespread cerebral damage

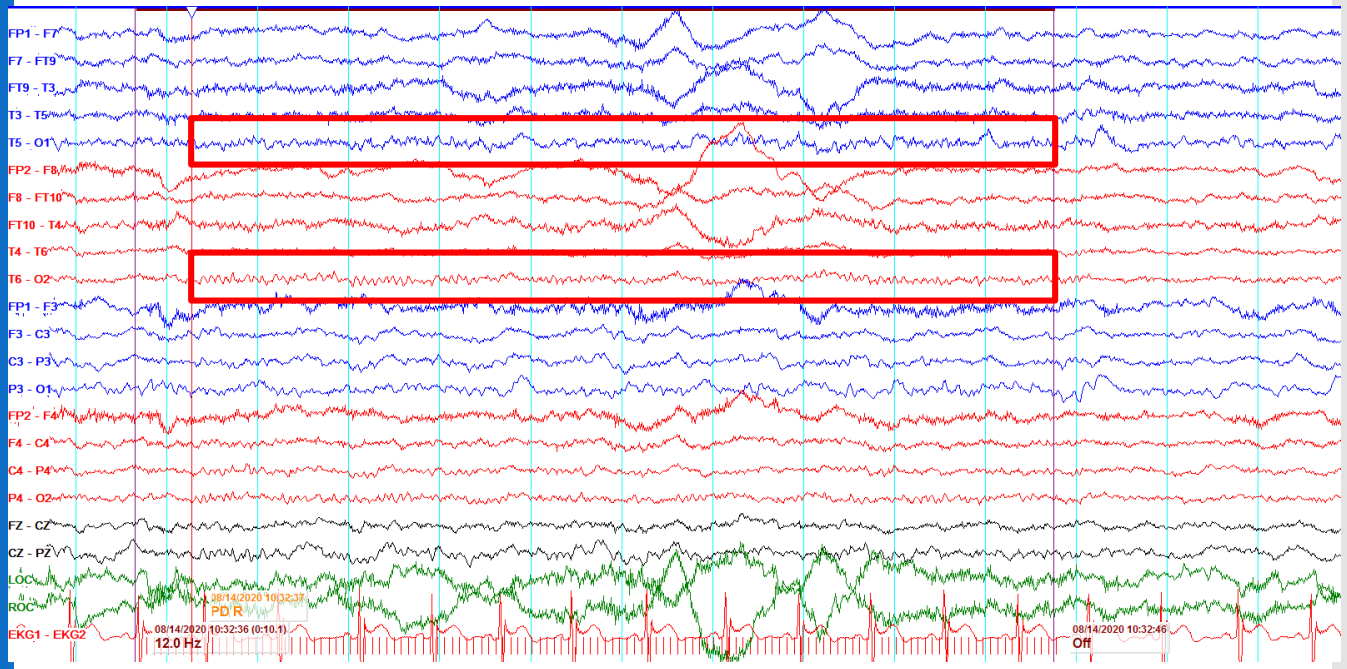
Eye closure PDR better over right



Asymmetry Response to Photic Stimulation

- Lesions on the side of lower voltage
- Can consistently lateralized in some normal individual
  - *Should not be interpreted as abnormal in absence of corroborative findings*
- Rarely epileptogenic lesions result in **higher** voltage photic stimulation on the side of lesion

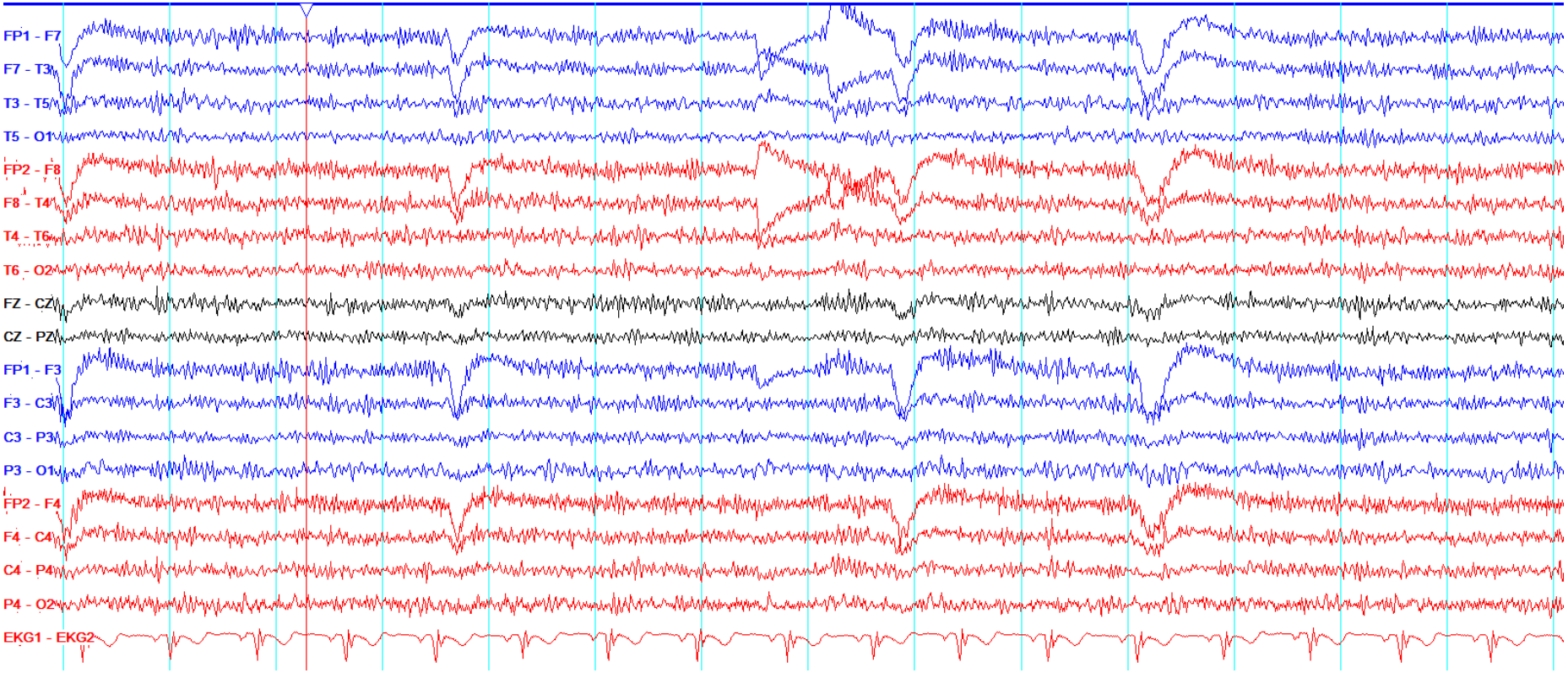
Asymmetrical Photic driving response



Abnormal beta pattern

Symmetrical Excessive Beta Activity

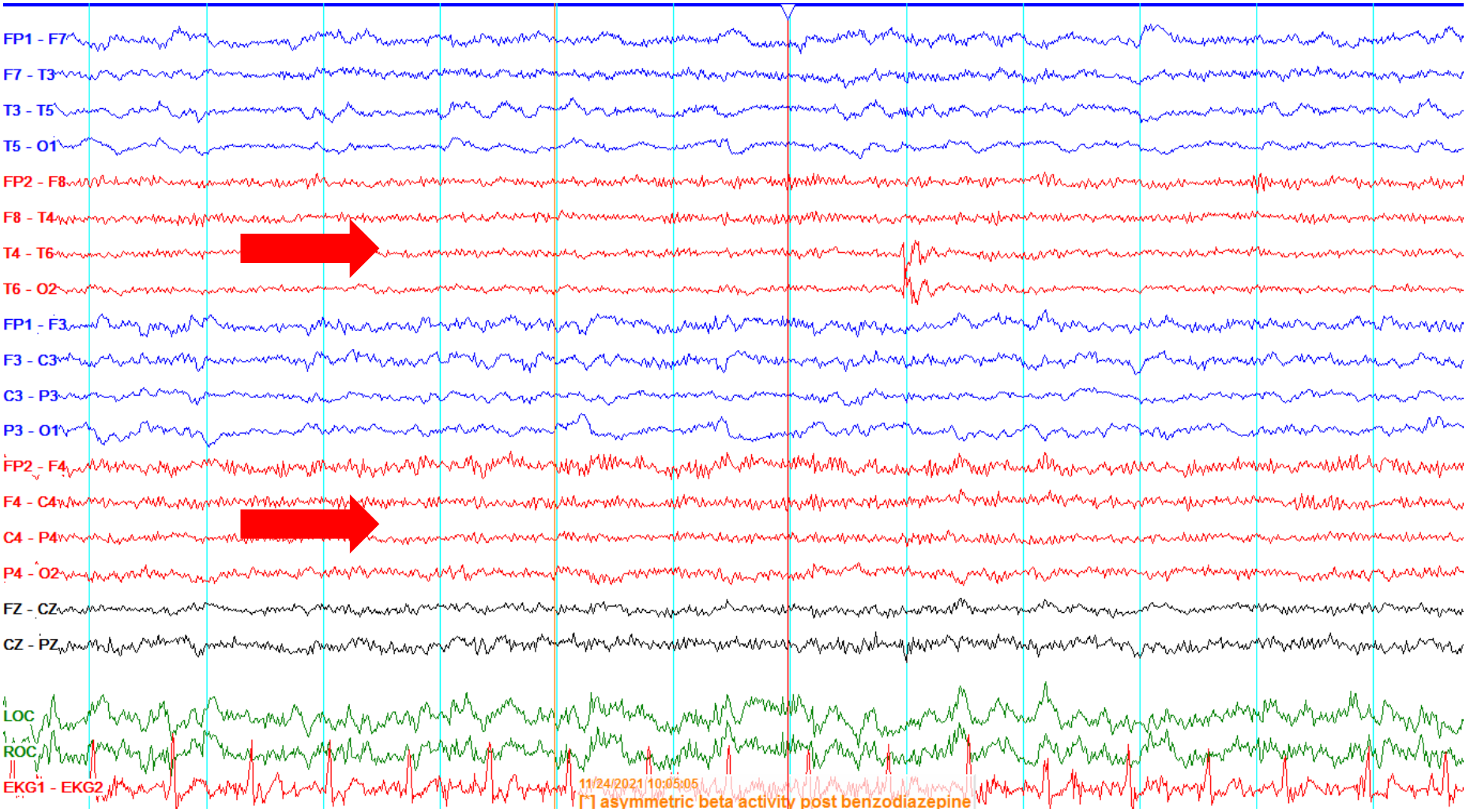
- Sedative, hypnotic, anxiolytic
  - Usually diffuse beta, maybe frontally predominant
  - *Should not be interpreted as abnormal*
- Diffuse lesion
  - Lissencephaly, pachygyria
- Localized or regional fast
  - Focal dysplastic lesion
- Hyperthyroidism: central or wide distribution
- Acute and chronic anxiety



Diffuse excessive beta activity
S/P benzodiazepine

Asymmetric beta activity

- Abnormal if persistent voltage difference >/= 35%
- Marker of cortical damage
- Lower voltage fast
  - Area of abnormality e.g. cortical stroke
  - Subdural, epidural or subgaleal fluid collections
  - Barbiturates → bring out a beta asymmetry
- Localized or regional abnormal fast → regional dysplastic lesion



Benzodiazepine bring out fast activity over right hemisphere
Abnormal slow over left hemisphere

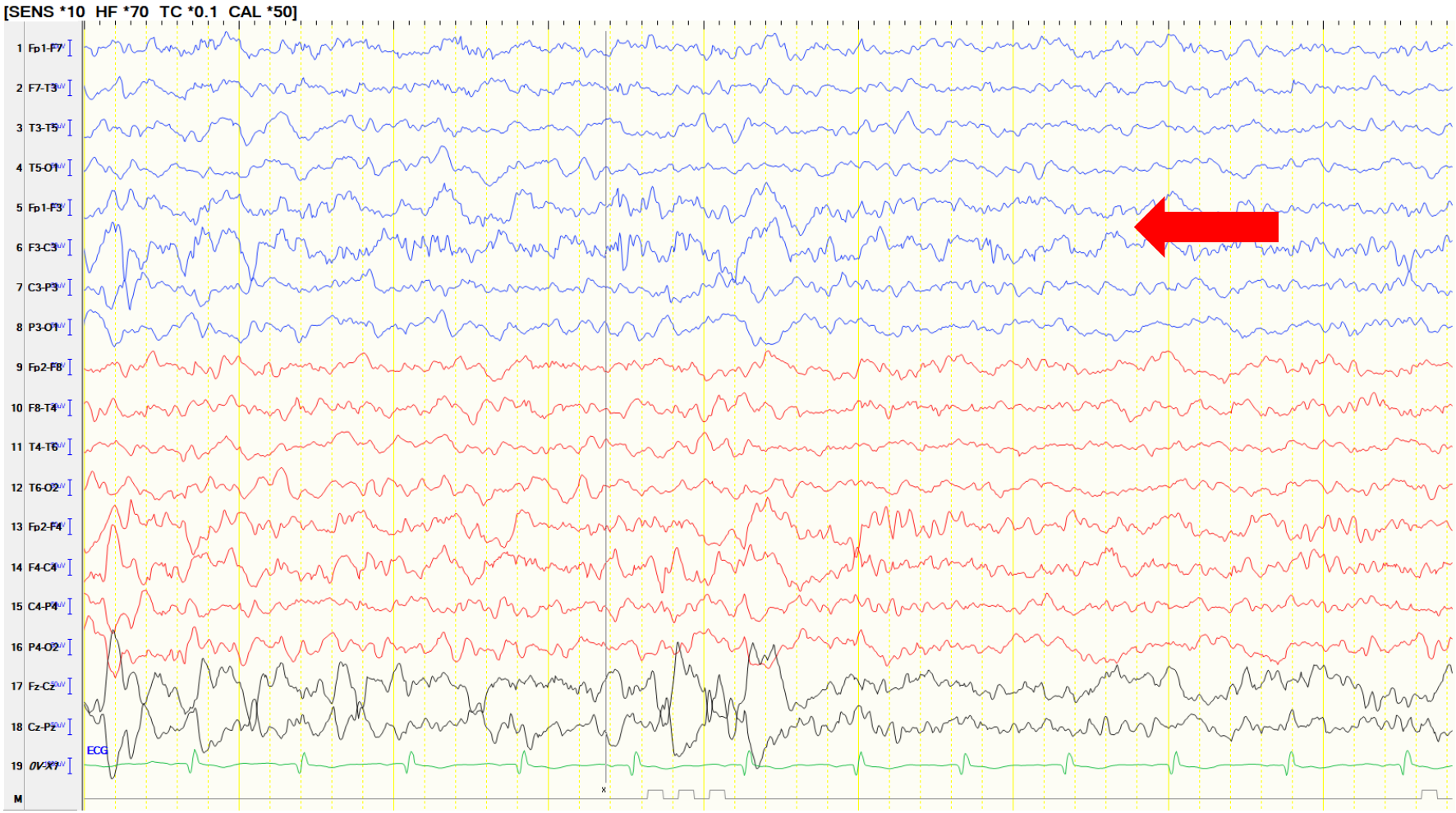
Breach Rhythm

Breach Effect

Consequence of intracranial surgery requiring craniotomy or burr hole

Less often from skull fracture

- ✓Higher voltage activity
- ✓Enhance fast frequency
- ✓Sharply contoured or spiky morphology



11 year-old girl s/p left frontal FCD resection

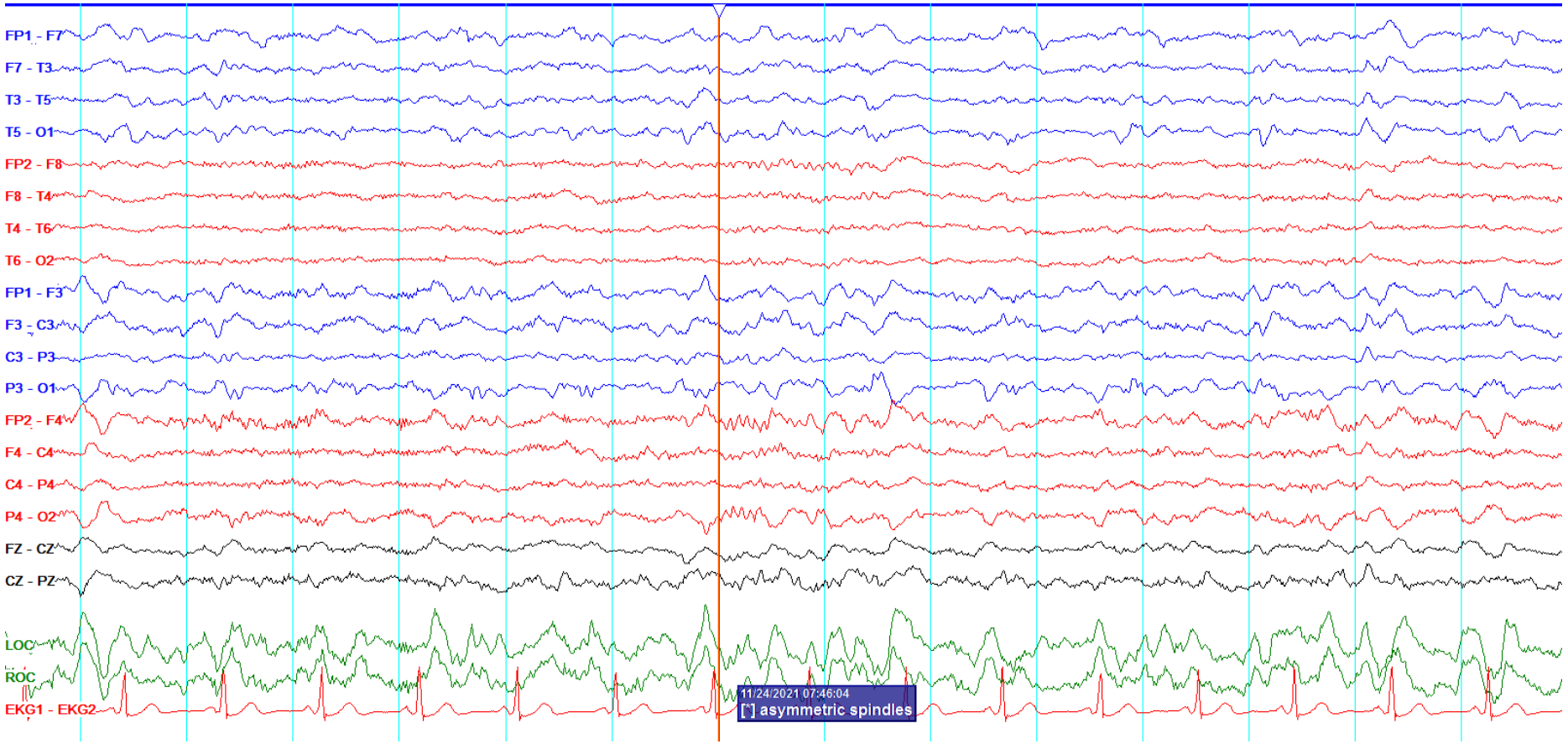
EEG: spiky appearance, increased fast, higher voltage

Breach effect left frontal region

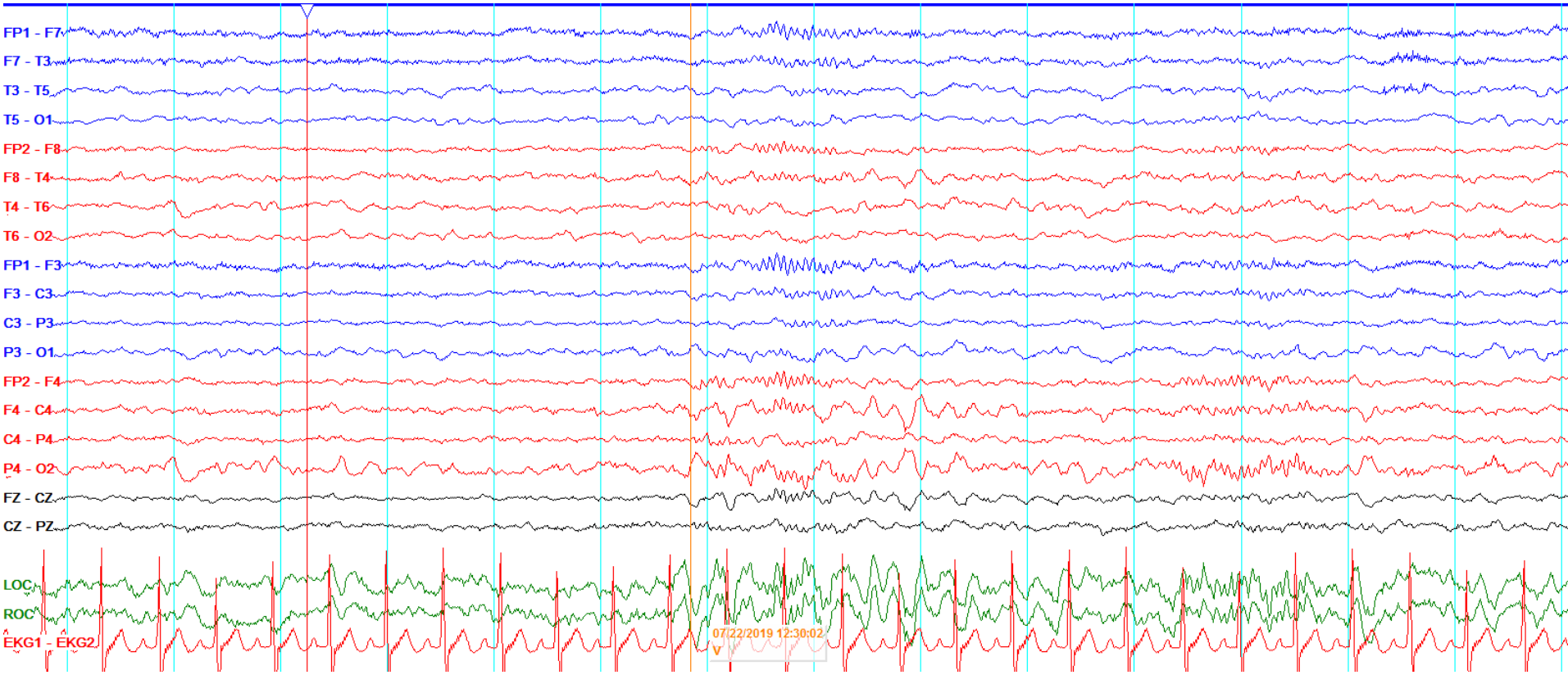
Abnormal sleep potential

Abnormal Sleep Potentials

- Abnormal sleep structure
  - Decrease sleep potential, spindle
    - Lesion involved thalamus, thalamocortical projection or cortex
  - Enhanced voltage of sleep architecture
    - Skull defect
- Abnormal ordering of sleep stage



Asymmetrical sleep spindles
Structural abnormality over left hemisphere



Asymmetric vertex wave

18 year-old ALL with behavioral change

MRI bilateral subcortical white matter change & old hemorrhage left thalamus

Extreme spindles

- Widespread, high voltage 100-400 µV and persistent than normal spindles
- Diffuse encephalopathy of childhood, mental retardation

Conclusion

- ✓Non epileptiform abnormality provide evidence of brain dysfunction
- ✓Mostly not specific to particular etiology
- ✓Can help in diagnostic possibility by combining with history and other findings and guide the treatment