NON EPILEPTIFORM ABNORMALITY

Suda Jirasakuldej, MD.

Comprehensive Epilepsy Center of Excellence

King Chulalongkorn Memorial Hospital

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NON EPILEPTIFORM ABNORMALITY: OUTLINE

- Frequency abnormality
  - Slow pattern
  - Fast frequency pattern
- Amplitude abnormality
  - Amplitude asymmetry
  - Generalized change of amplitude
- Abnormal frequency or reactivity of alpha
- Abnormal sleep potentials

FREQUENCY ABNORMALITY
ABNORMAL SLOW ACTIVTY

Focal, generalized, triphasic

ABNORMAL SLOW: ASSESSMENT

- Arrhythmic polymorphic or rhythmic monomorphic
- Persistence: intermittent or continuous
- Reactivity
- Amplitude
- Frequency
- Distribution: localize, unilateral, generalize

MORE ACUTE OR SEVERE PATHOLOHY

- Lower Frequency
- Higher amplitude
- Greater abundance

FOCAL SLOWING

- Frequency < 8 Hz and limited distribution
- Indicate focal cerebral dysfunction but nonspecific etiology
- Result of cortex deafferentation from subcortical structure

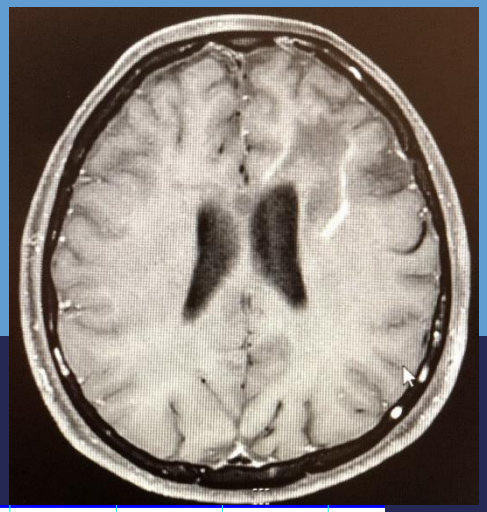
FOCAL SLOW: CLINICAL CORRELATION

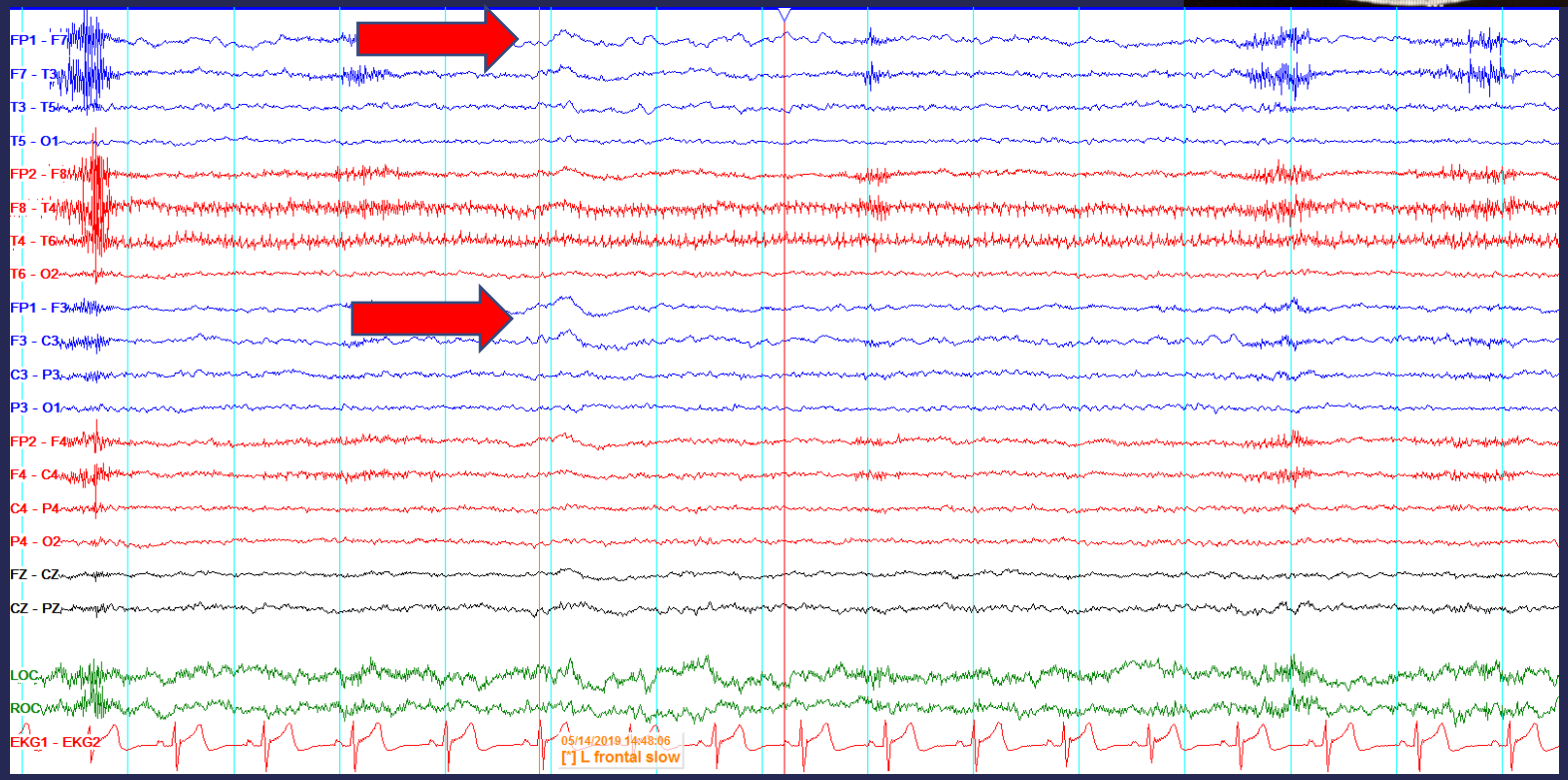
- Focal arrhythmic polymorphic delta activity
  - Localized structural lesion in subcortical white matter
    - Stroke
    - Abscess
    - Intraparenchymal hematoma or contusion
    - Tumor
  - Functional: postictal, complicated migraine, head trauma
  - Rarely: toxic-metabolic → hypoglycemia, hyperglycemia

- Focal rhythmic monomorphic slowing
  - More commonly associated with grey matter lesions

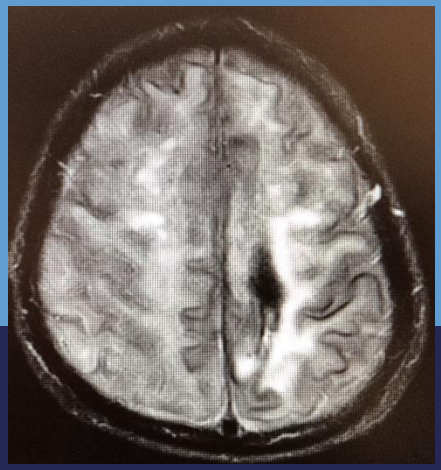
Focal slow disappear within hours or a few days of acute transient disorder

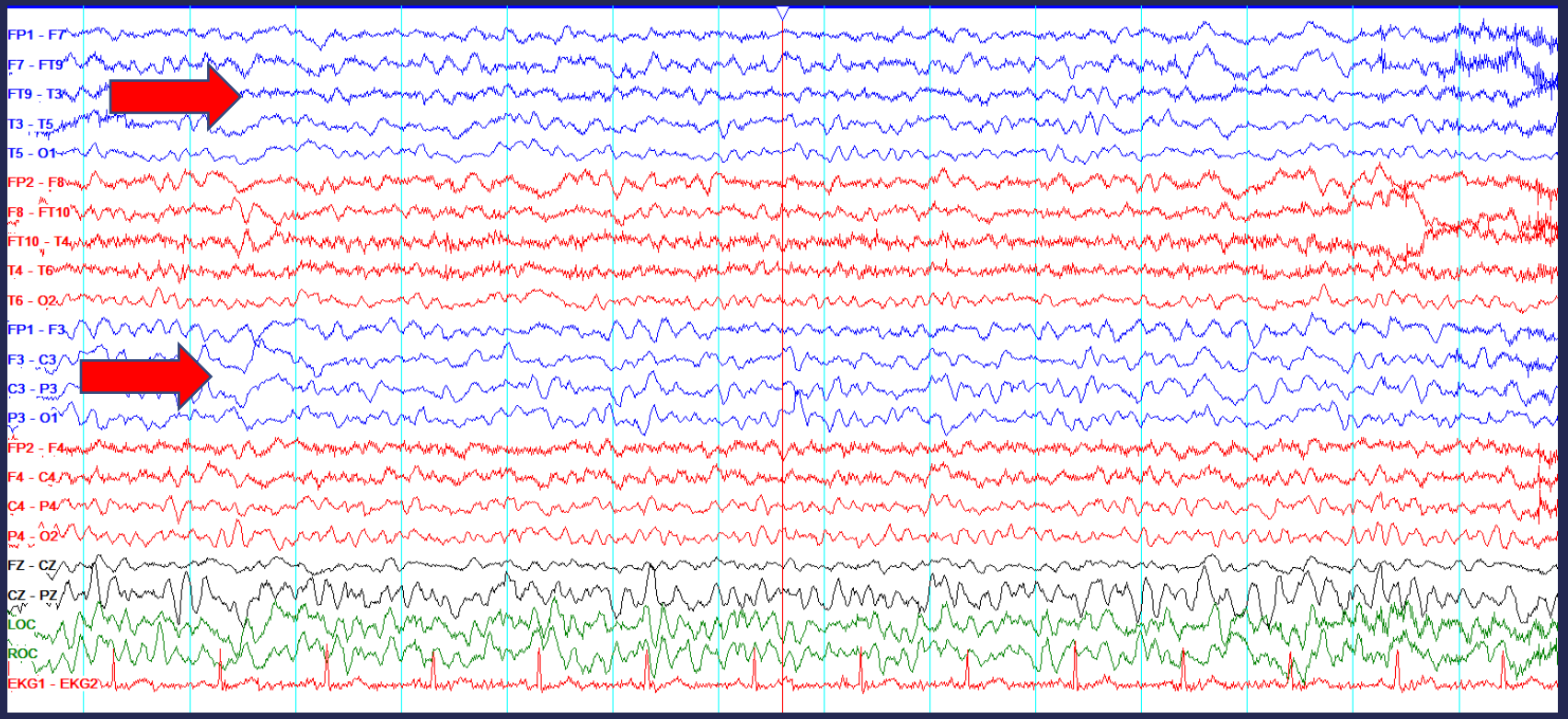
LEFT FRONTAL POLYMORPHIC SLOW





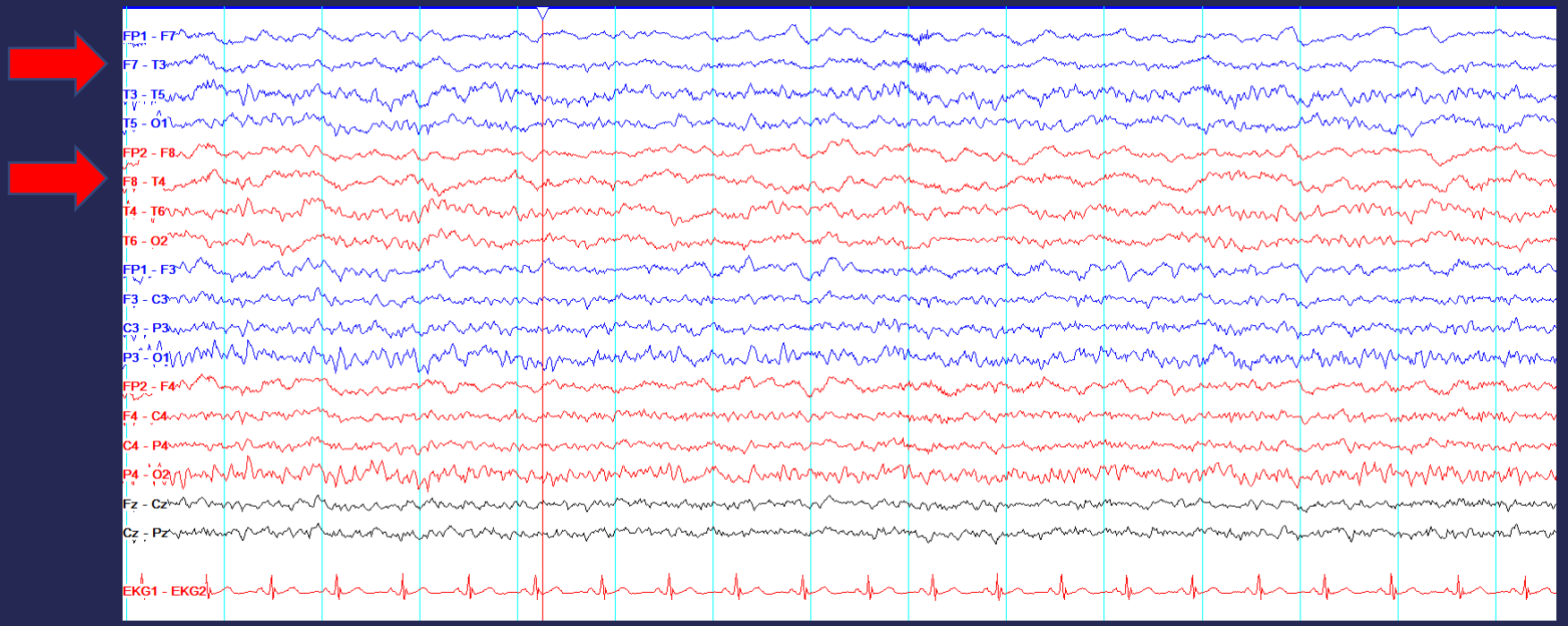
81 YEAR-OLD MAN WITH OLD CVA AND TRANSIENT EPISODE OF DYSPHASIA





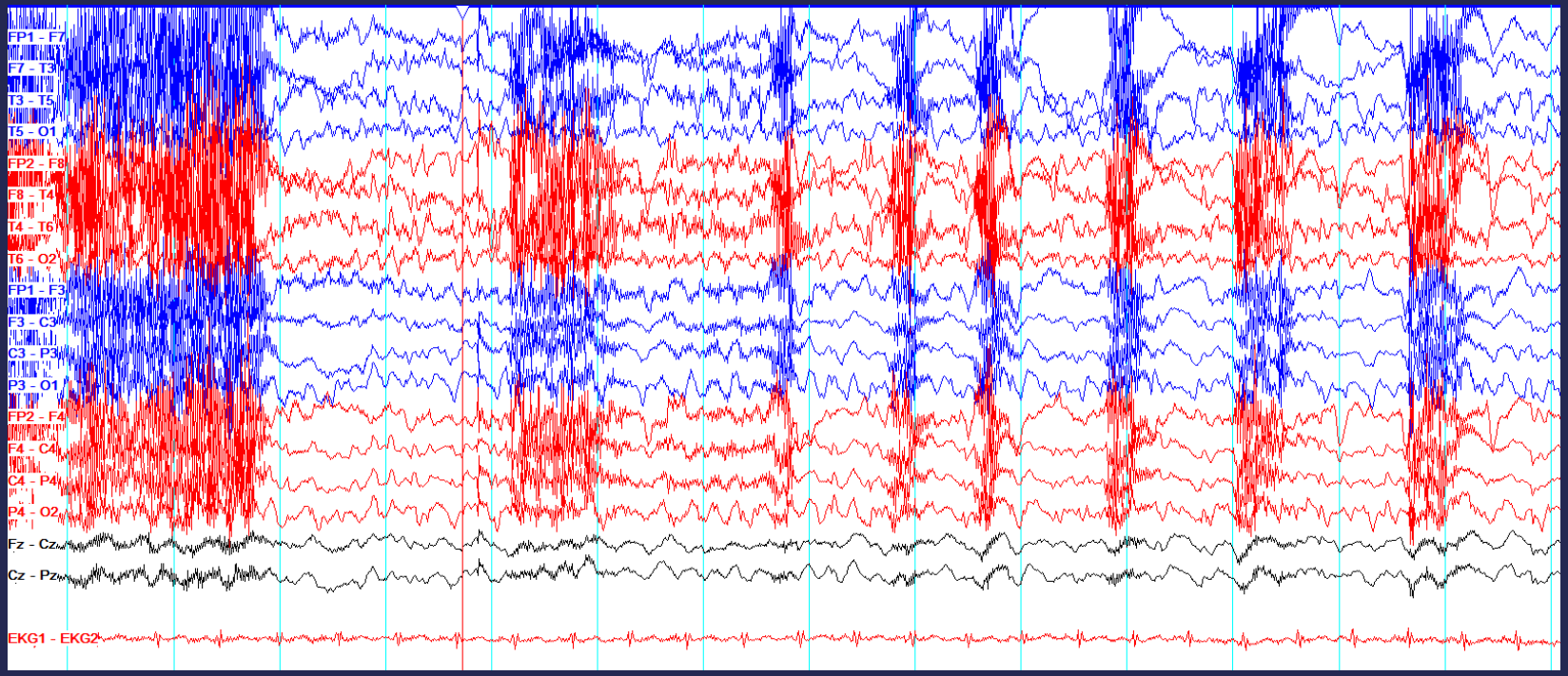
Continuous polymorphic slow left >> right hemisphere

BILATERAL TEMPORAL R > L POLYMORPHIC DELTA SLOWING



Postictal focal slowing

EEG BEFORE


FP1 - F7
F7 - T3
T3 - T5
T5 - O1
FP2 - F8
F8 - T4
T4 - T6
T6 - O2
FP1 - F3
F3 - C3
C3 - P3
P3 - O1
FP2 - F4
F4 - C4
C4 - P4
P4 - O2
Fz - Cz
Cz - Pz
EKG1 - EKG2

GENERALIZED ARRHYTHMIC SLOW

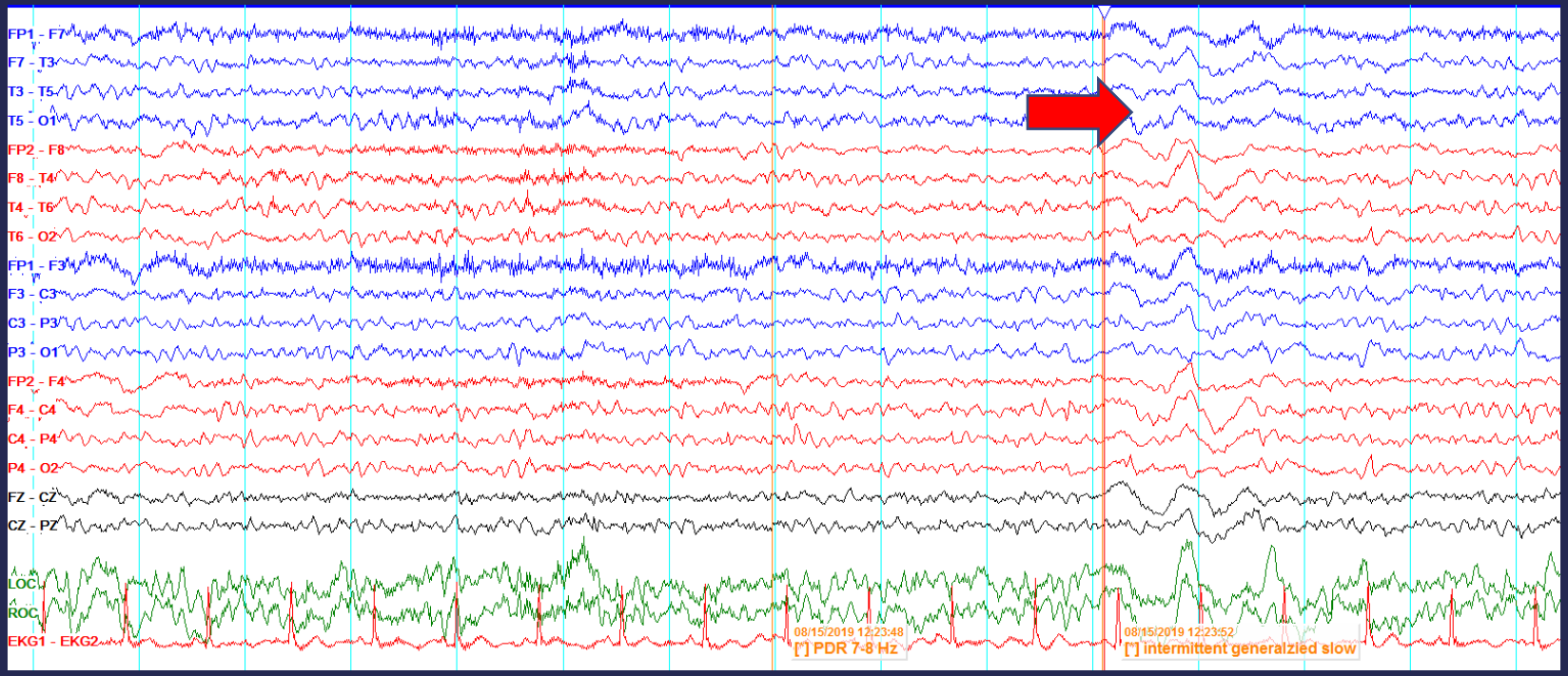
GENERALIZED ARRHYTHMIC SLOW

- 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
- Etiology
  - Exclude physiologic!!! Drowsiness or sleep
  - Diffuse but nonspecific disturbance of cerebral activity
  - Most commonly metabolic and toxic encephalopathy

CONTINUOUS GENERALIZED ARRHYTHMIC SLOW DEGREE OF ENCEPHALOPATHY

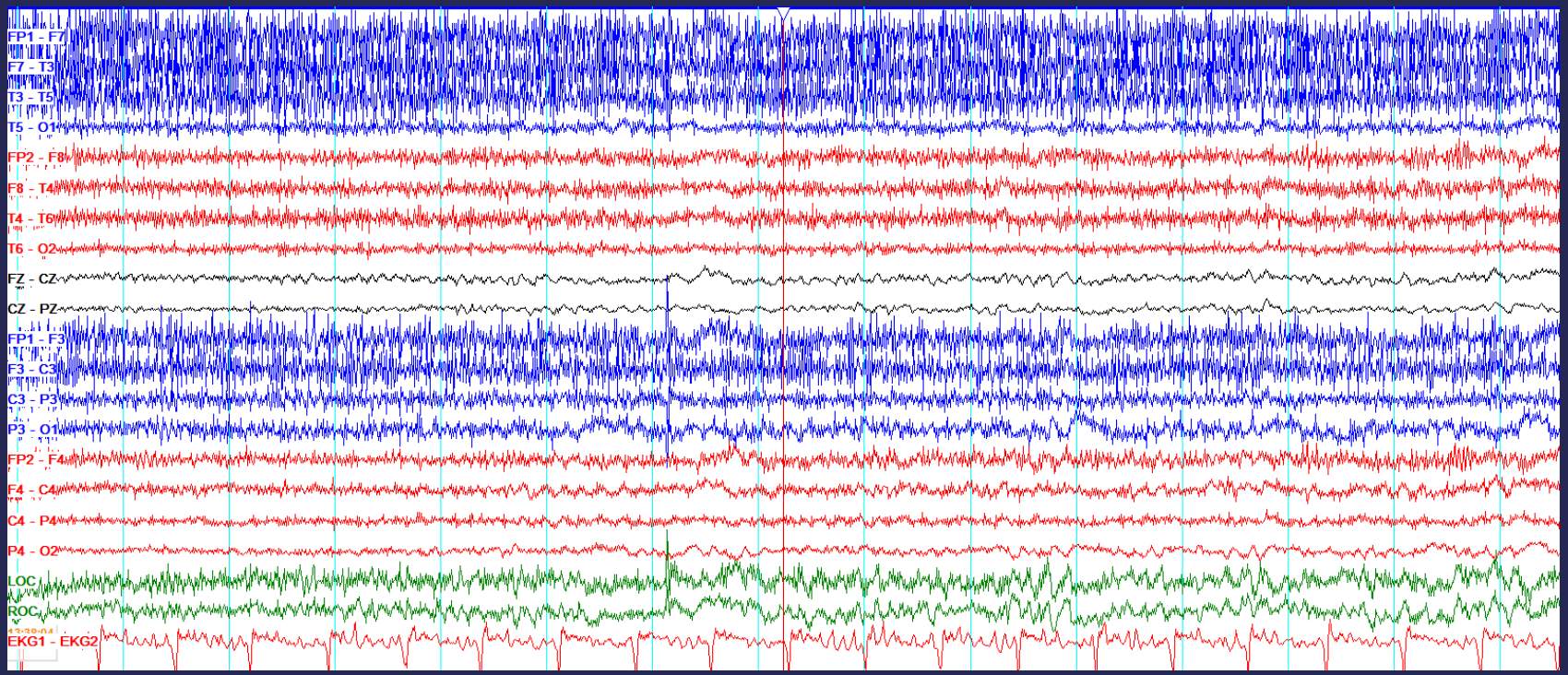
- Earliest sign
  - Slowing of posterior waking background
  - Generalized theta
- Encephalopathy deepens
  - Slow of anterior rhythm
  - Predominant polymorphic delta slowing
  - Less reactivity or organization
- Deep coma
  - Burst suppression
  - Generalized suppression (< 10 mV)
  - Isoelectric

62 YEAR OLD WOMAN WITH HISTORY OF SEIZURE



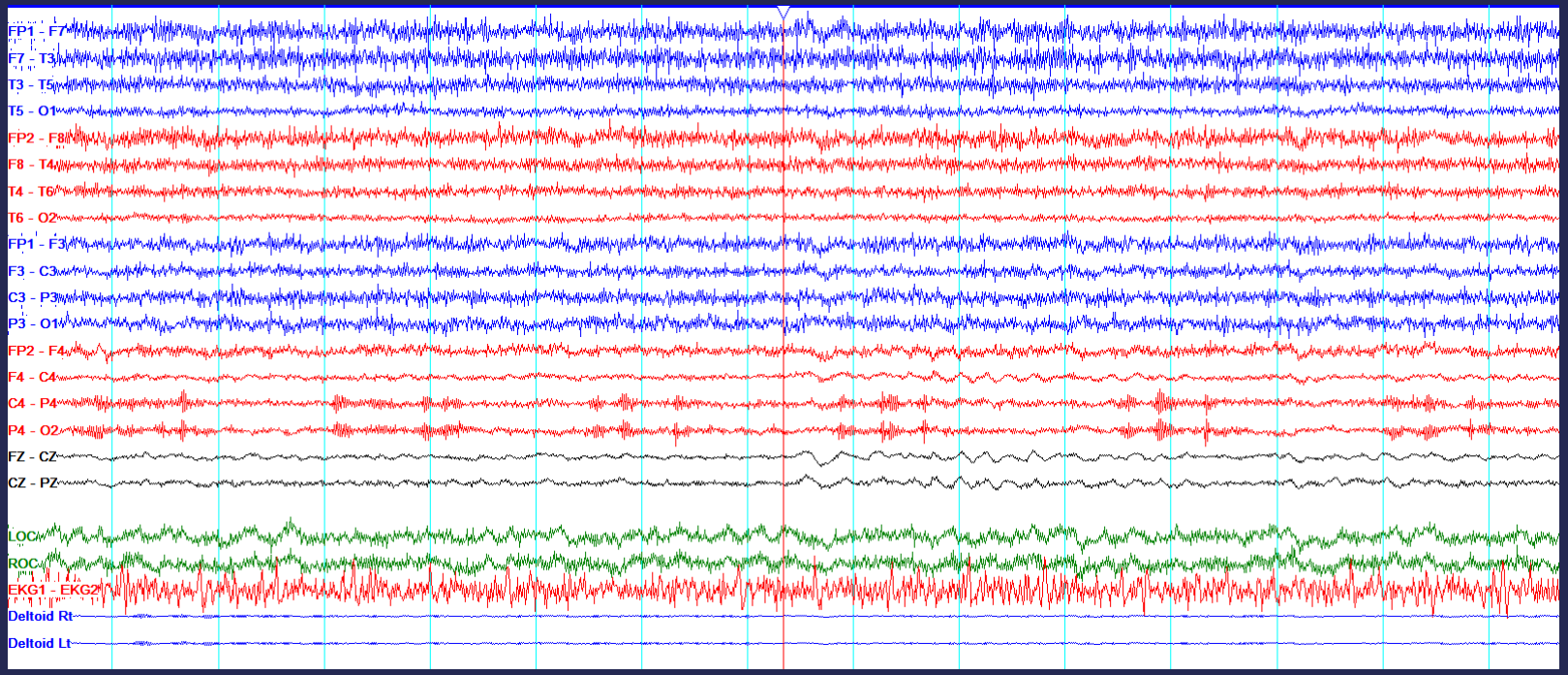
Mild slow BG, PDR 7-8 Hz, intermittent generalized delta → mild diffuse encephalopathy

71 YEAR-OLD ESRD, STEMI WITH VT S/P CARDIAC ARREST



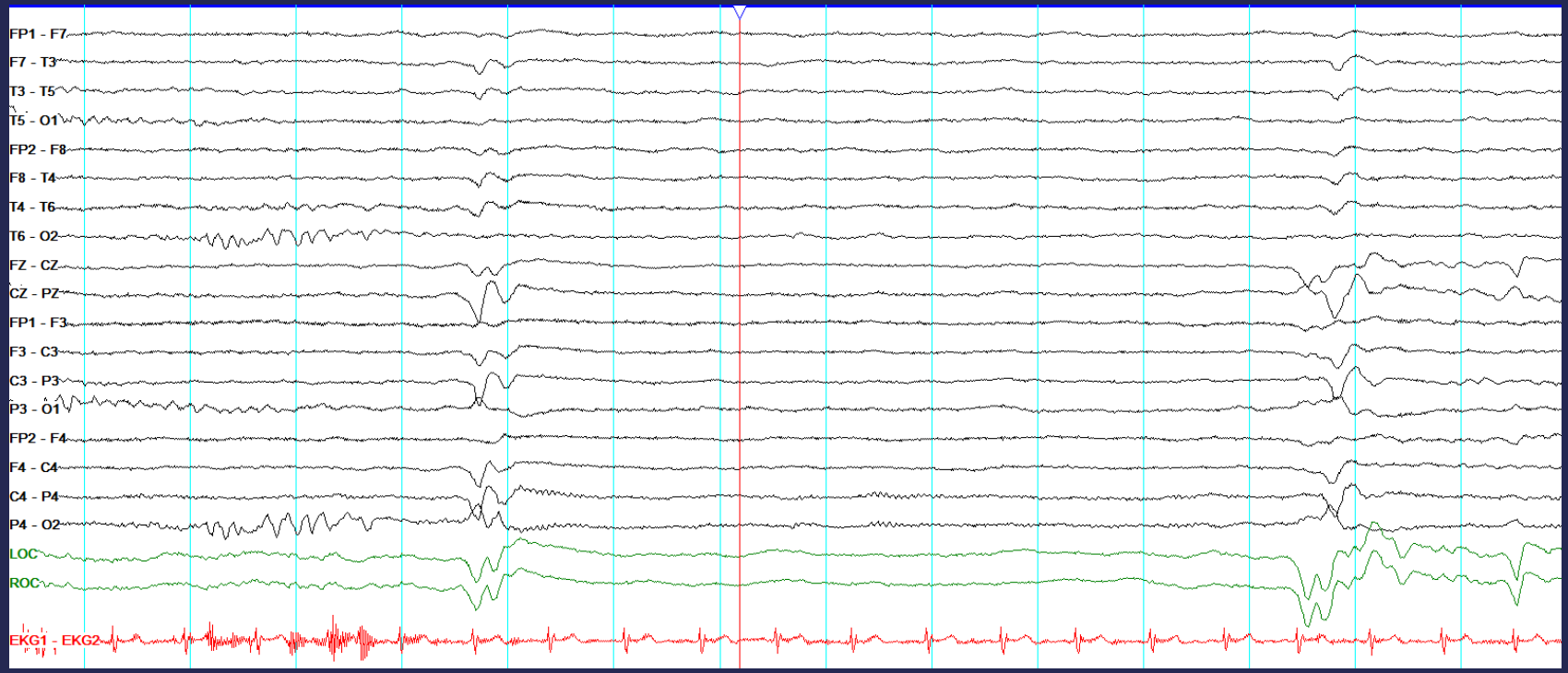
Generalized delta and theta slow, no PDR → moderate diffuse encephalopathy

54 YEAR-OLD MAN WITH CAR ACCIDENT AND CARDIAC ARREST



Generalized low amplitude delta-theta slow, no reactivity, no PDR, no sleep feature
Severe diffuse encephalopathy (delta-theta coma)

88 YEAR-OLD WOMAN S/P CARDIAC ARREST



Discontinuous BG with 7 seconds BG suppression → severe diffuse encephalopathy

INTERMITTENT RHYTHMIC DELTA ACTIVITY

IRDA

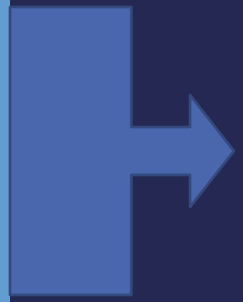
- Runs of high voltage rhythmic 2-3 Hz at irregular interval
- Usually bisynchronous and symmetric
- May be asymmetric or unilateral
- Reactive/attenuate to eye opening or alerting
- Adult usually frontal
- Children usually occipital
- Considered a projected rhythm from deep structure, may reflect diffuse grey dysfunction either cortical or subcortical

IRDA: PHYSIOLOGIC

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

IRDA

- Frontal intermittent rhythmic delta activity (FIRDA)
- Occipital intermittent rhythmic delta activity (OIRDA)
- Temporal intermittent delta activity (TIRDA)



Not suggest specific localization

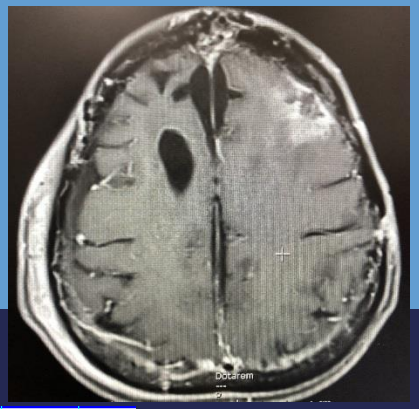
Age specific

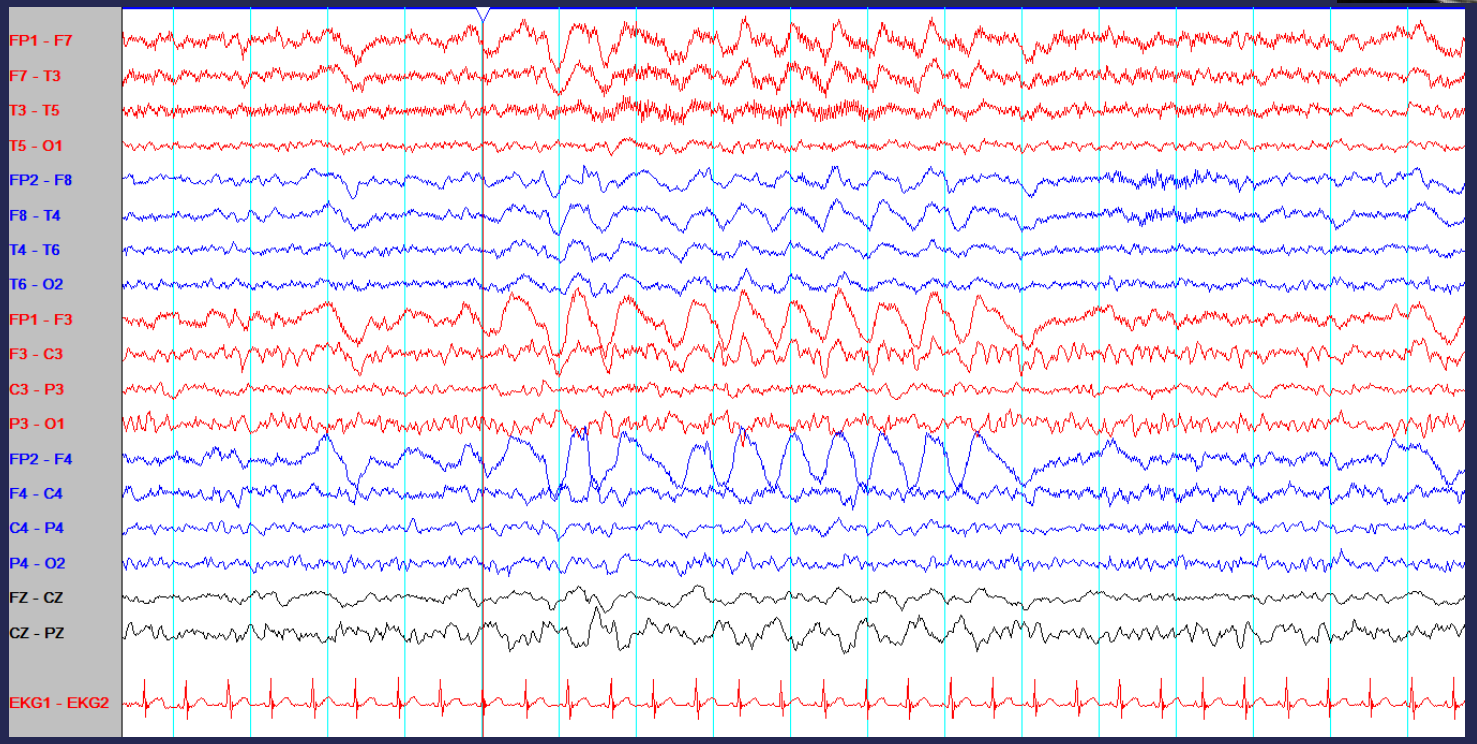
More specific localization

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

57 YO ANAPLASTIC MENINGIOMA ACUTE SDH AND ICH





Frontal intermittent rhythmic delta activity

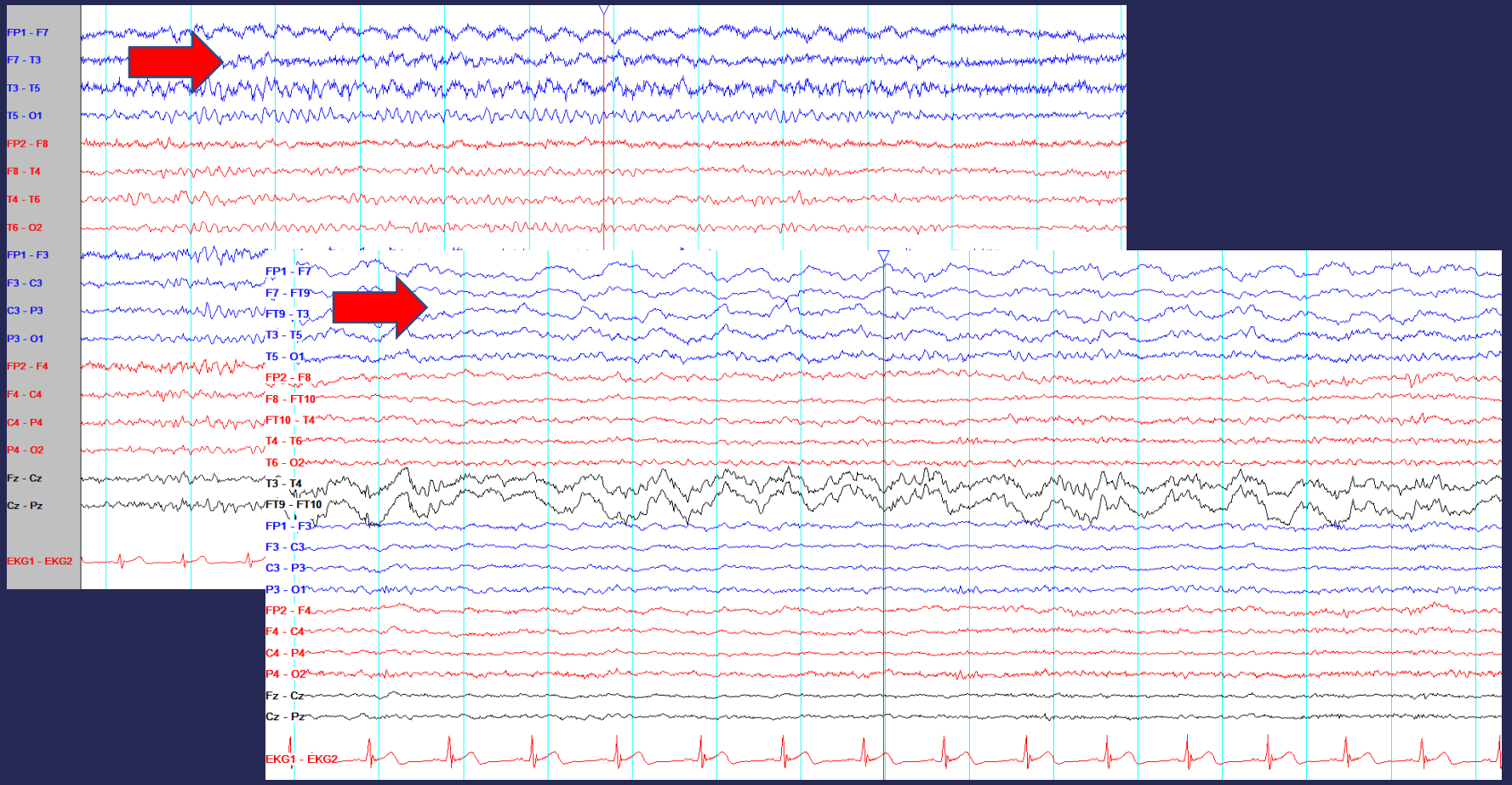
OIRDA

- 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

TIRDA

- Bursts or train of 3 seconds or more 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 interictal epileptiform activity
  - Associated with temporal lobe epilepsy
- May relate with structural changes affecting lateral ventricle
  - Projected or a distance rhythm over temporal lobe

LEFT TEMPORAL INTERMITTENT RHYTHMIC DELTA ACTIVITY (TIRDA)



TRIPHASIC WAVE
SPECIAL TYPE OF GENERALIZED SLOW WAVES

TRIPHASIC WAVE
FIRST DESCRIPTION : BLUNT SPIKE AND SLOW WAVES IN HEPATIC COMA

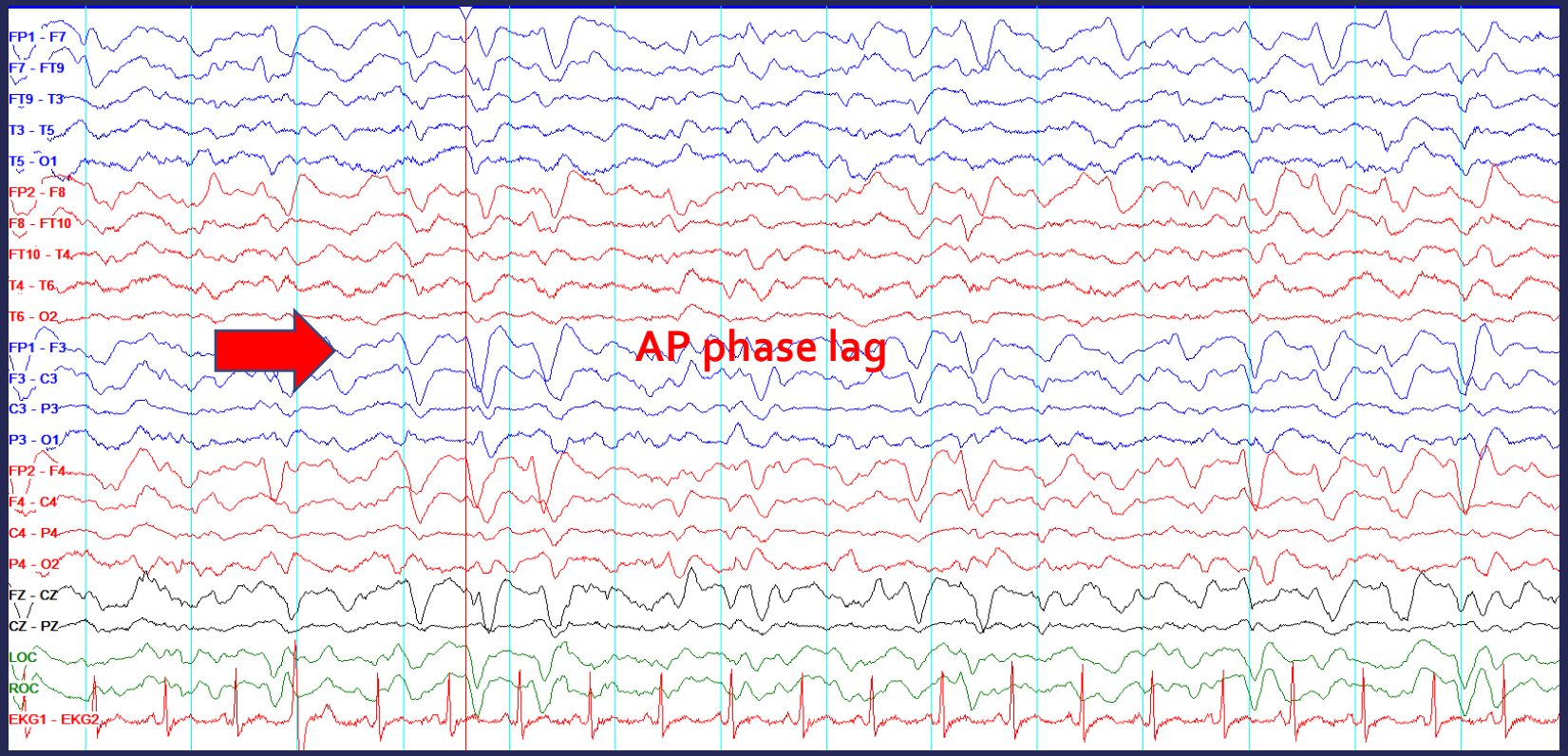
- Bilaterally synchronous and symmetrical waves
- Three phases:
  - Initial small negative wave (up)
  - Larger positive phase (down)
  - Followed by negative phase, longer duration than other 2 phases
- Bi-frontal predominant, 1.5-2.5 Hz, 70-300 µV
- May have 25-140 ms anterior–posterior or posterior-anterior lags
- May increase or attenuate with stimulation
- Intermittently in brief runs or continuous pattern

TRIPHASIC WAVES: CLINICAL CORRELATION

- 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*

MAN WITH DM, HTN, CKD, AMS



Triphasic wave

FAST FREQUENCY PATTERN

Abnormal beta activity
Breach rhythm

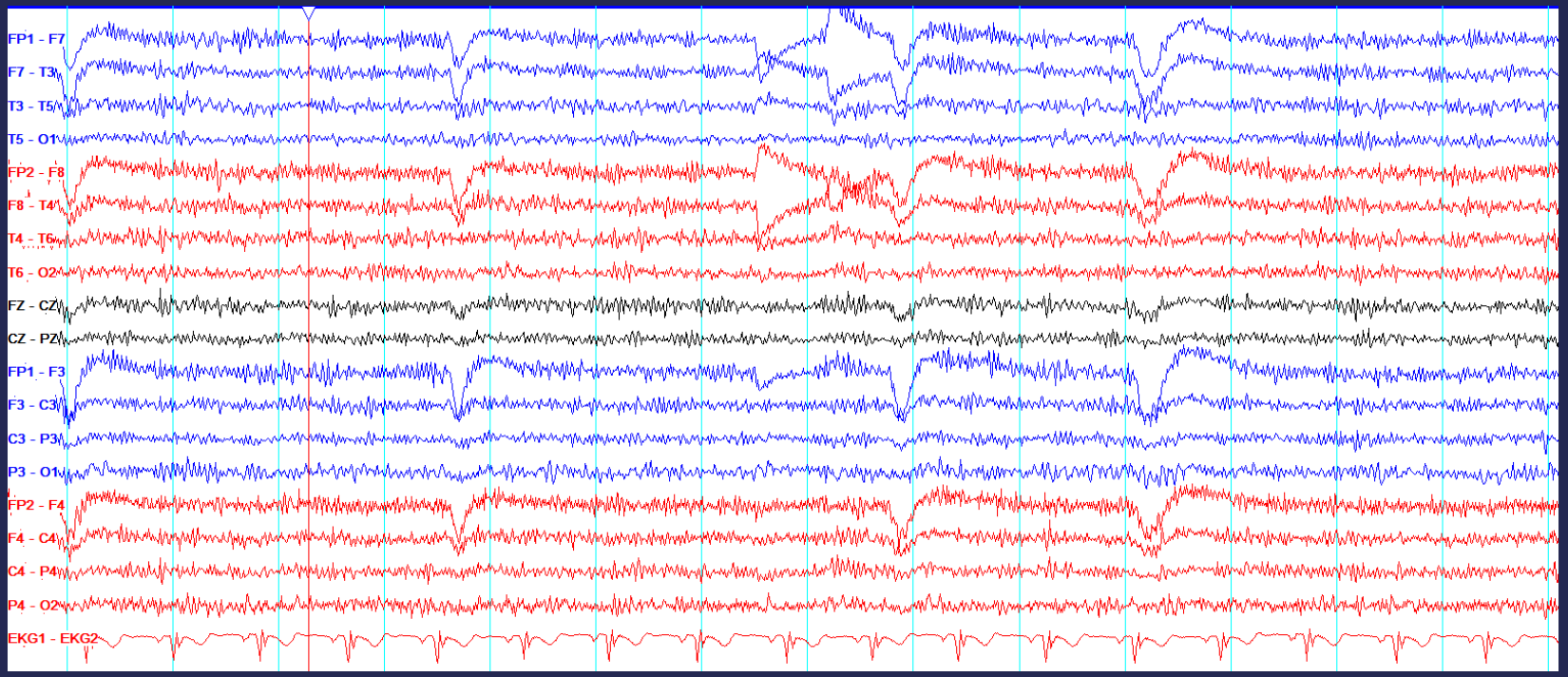
ABNORMAL BETA RHTYHMS

Beta waves believed to be generated at level of cortex

SYMMETRICAL BETA

- Sedative, hypnotic, anxiolytic
  - Usually diffuse beta, maybe frontally predominant
- Diffuse lesion: lissencephaly or pachygyria
- Hyperthyroidism: central or wide distribution
- Acute and chronic anxiety

SEIZURE S/P BENZODIAZEPINE



Diffuse excessive beta activity

ASYMMETRIC BETA ACTIVITY

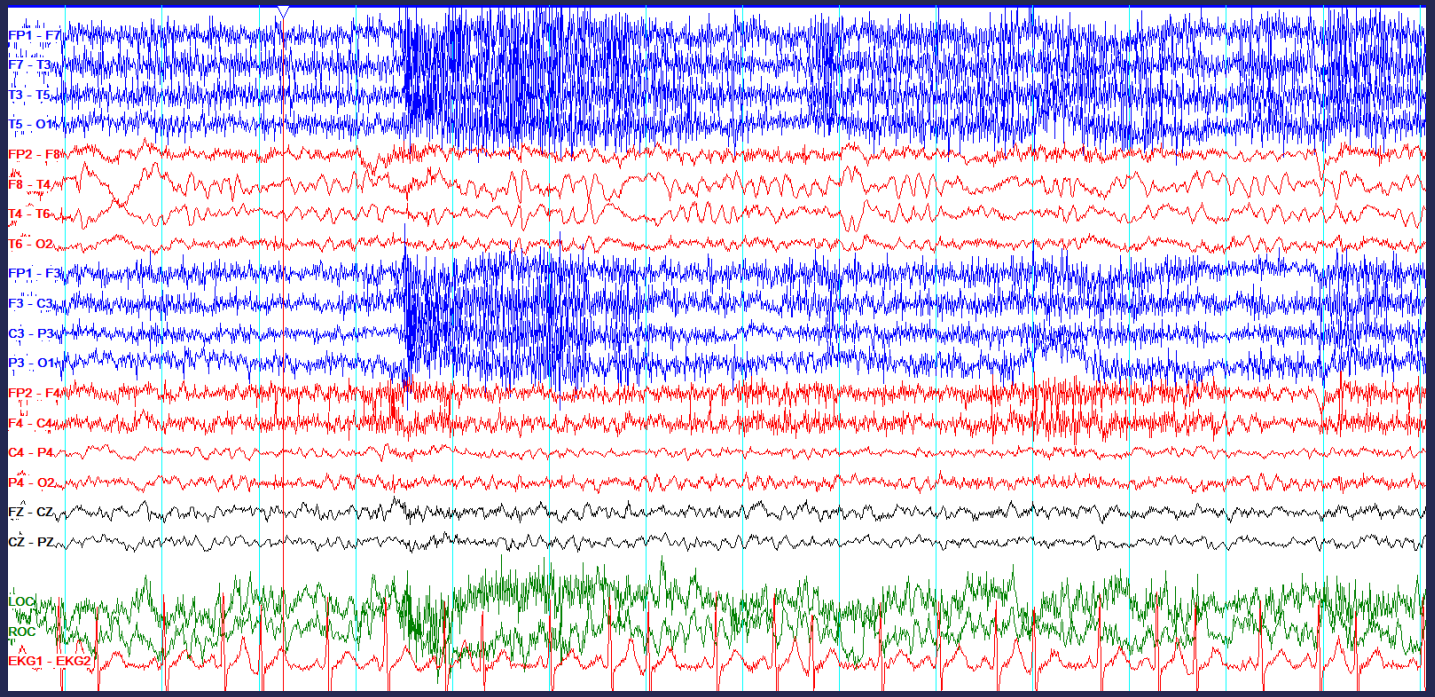
- Abnormal if persistent voltage difference of 35% or more
- Marker of cortical damage
- Lower voltage fast
  - Area of abnormality e.g. cortical stroke
  - Subdural, epidural or sub-galeal fluid collections
  - *Barbiturates → bring out a beta asymmetry*
- Localized or regional abnormal fast → regional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

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

66 YEAR-OLD WOMAN NCSLC WITH LEPTOMENINGEAL METASTASIS, BRAIN BIOPSY RIGHT TEMPORAL



Focal slow right temporal, breach artifact right temporal

NON EPILEPTIFORM ABNORMALITY

- Frequency abnormality
  - Slow pattern
  - Fast frequency pattern
- **Amplitude abnormality**
  - **Amplitude asymmetry**
  - **Generalized change of amplitude**
    - **Low voltage and suppressed**
    - **High voltage**
- Abnormal frequency or reactivity of alpha
- Abnormal sleep potentials

AMPLITUDE ASYMMETRY

AMPLITUDE ASYMMETRY

- Differences in amplitude from two sides of head

- Normal asymmetry
  - Asymmetry of photic driving, POSTs, mu rhythm in absence of other abnormalities
- Abnormal asymmetry
  - Consistent asymmetry of beta > 35%
  - Consistent asymmetry of alpha > 50%

CAUSE OF AMPLITUDE ASYMMETRY

- Decreased amplitude
  - 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

GENERALIZED CHANGE OF AMPLITUDE

GENERALIZED CHANGE OF AMPLITUDE

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

- High voltage pattern

ABNORMAL LOW AMPLITUDE

- Low amplitude in normal subjects seen in 5-10% of adults
- Normal low amplitude < 20 microvolts occurs transiently as a result of eye opening, mental effort, anxiety, alerting or drowsiness
- Moderate or marked reduction amplitude <10 microvolts → abnormal at any age

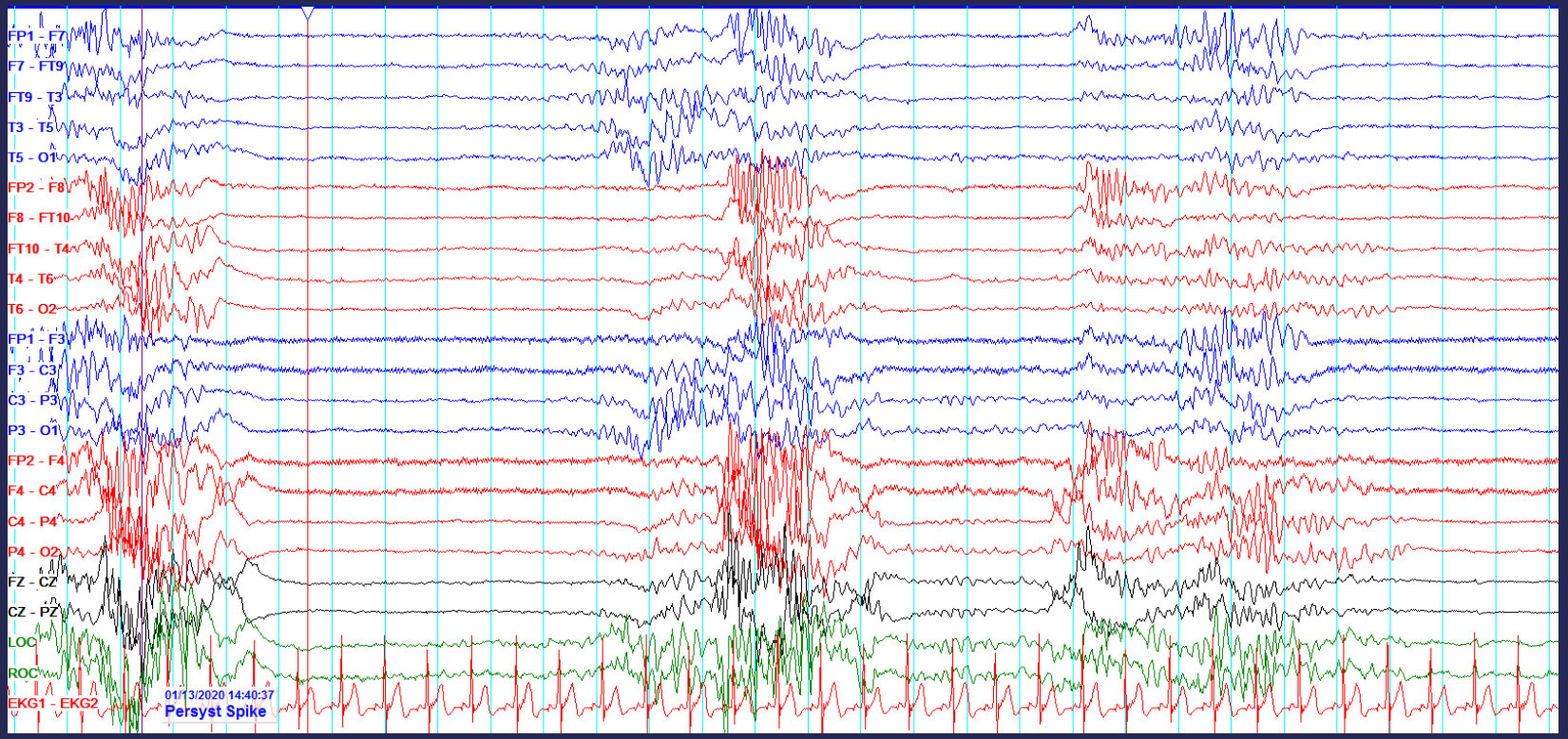
BURST SUPPRESSION

Presence of brain activity bursts of high voltage, mixed frequency activity, usually with sharp and spike waves lasted at least 0.5 sec, alternate with attenuation or suppression >50% of recording

Etiology

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

97 YEAR-OLD WOMAN S/P CARDIAC ARREST

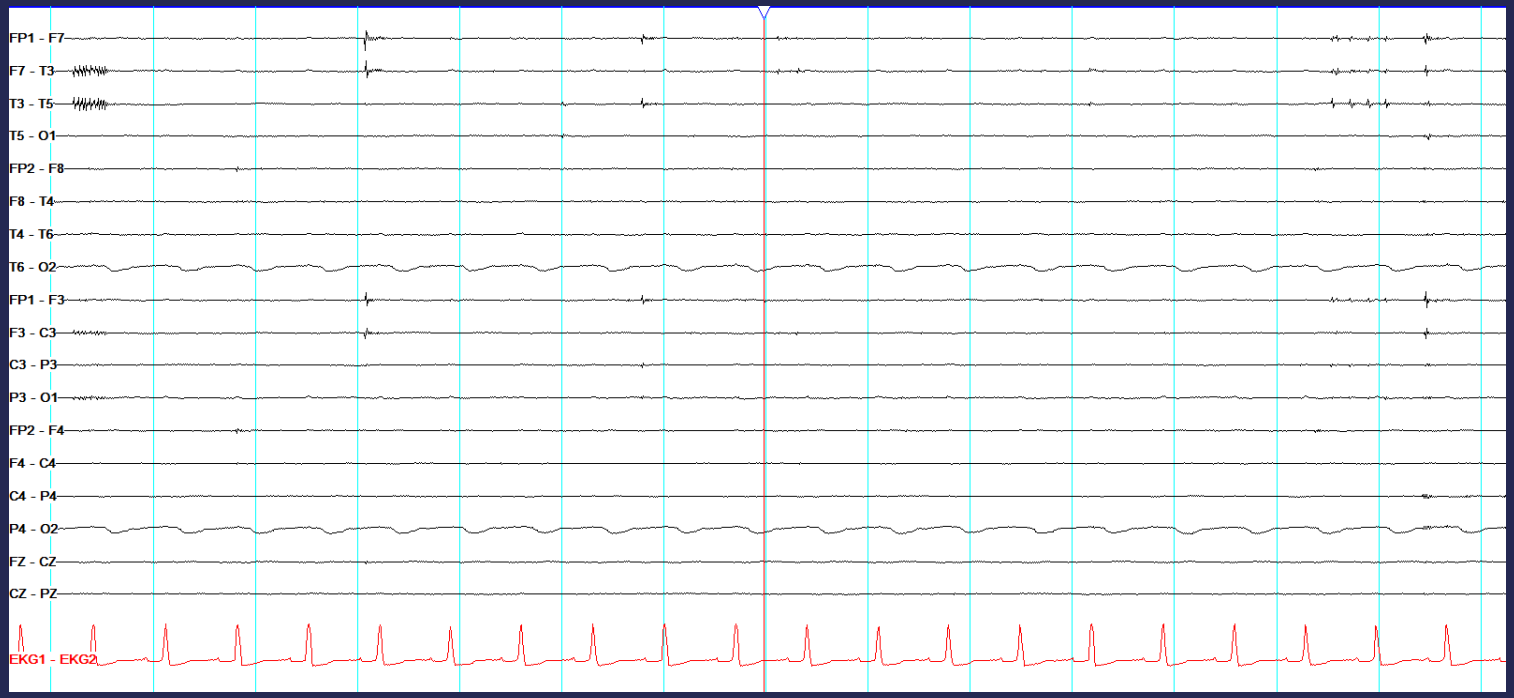


Burst suppression pattern

ELECTRO-CEREBRAL INACTIVITY

- Isoelectric, nonreactive EEG, flat EEG
- Severe and widespread cerebral dysfunction in which EEG activity is undetectable (amplitudes of < 2 μV)
- Conventional scalp electrodes placed at double the routine international 10-20 electrode distances
- Body core temperature above 34 degrees centigrade
- No CNS depressant medications
- At least 30 minutes of continuous recording

S/P CARDIAC ARREST



Generalized background suppression → severe diffuse encephalopathy

HIGH AMPLITUDE

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

- Abnormal high amplitude
  - Not well defined
  - Amplitude > 200 microvolts
  - Amplitude > 100 microvolts is uncommon in wakeful adults, only considered abnormal on basis of frequency, morphology, or distribution rather than amplitude alone

NON EPILEPTIFORM ABNORMALITY

- Frequency abnormality
  - Slow pattern
  - Fast frequency pattern
- Amplitude abnormality
  - Amplitude asymmetry
  - Generalized change of amplitude
- **Abnormal frequency or reactivity of alpha**
- Abnormal sleep potentials

ABNORMAL ALPHA RHYTHMS

ABNORMAL FREQUENCY OF ALPHA

Unilateral decrease in alpha frequency

- Abnormal <8 Hz
- Mild asymmetry (consistent asymmetry 0.5 - 1 Hz)
- Marked asymmetry (>1 Hz asymmetry)
- Causes:
  - Unilateral disturbance of function or structural damage of occipital cortex or its thalamic input

ABNORMAL FREQUENCY OF ALPHA

Bilateral decrease of alpha frequency

- Generalized disturbance of cerebral function
- Bilateral structural damage to occipital cortex or its thalamic input
- Change of rhythmical input to cortex

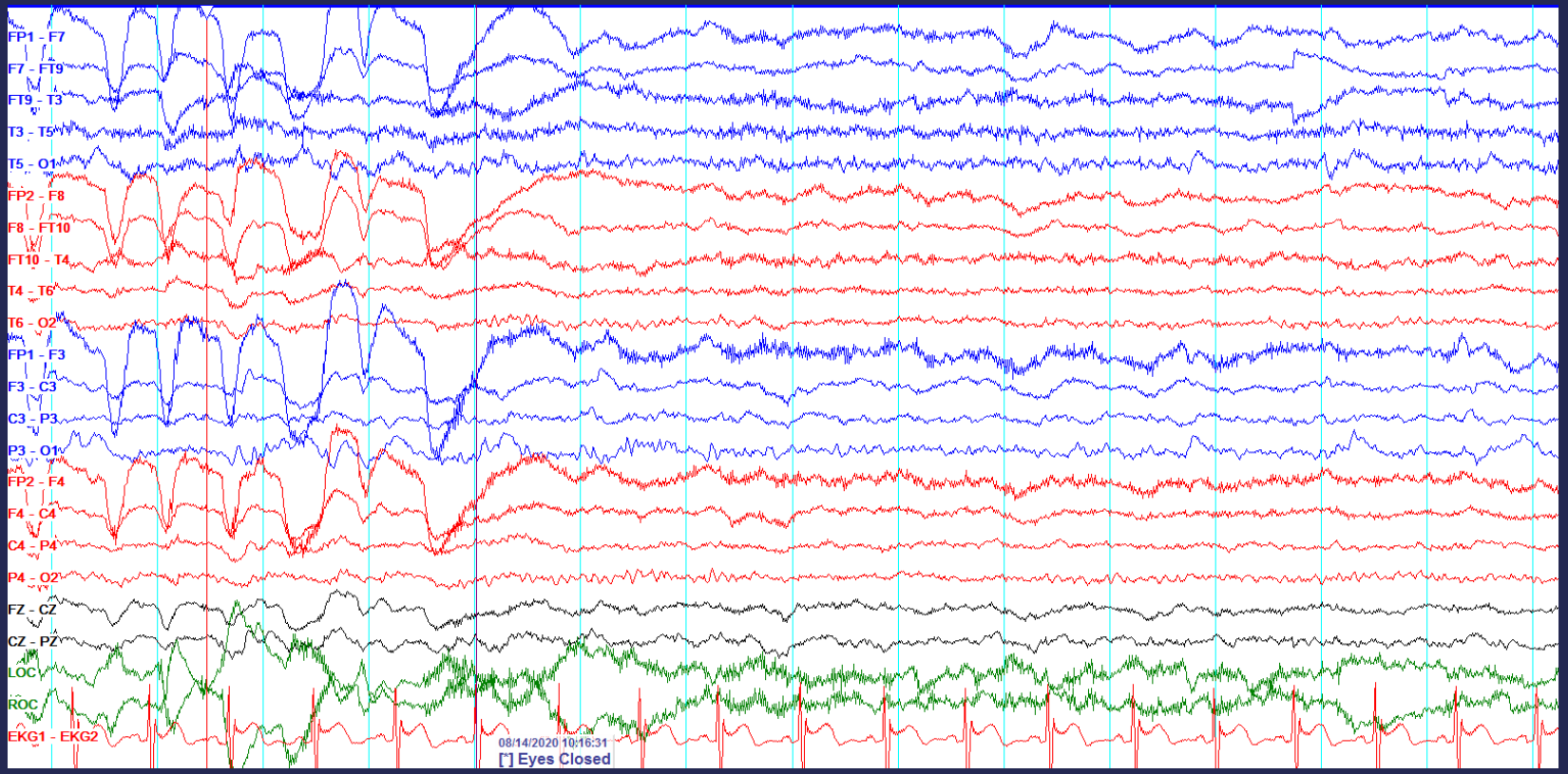
Bilateral increase of alpha frequency

- Metabolic disorder: fever, hyperthyroid

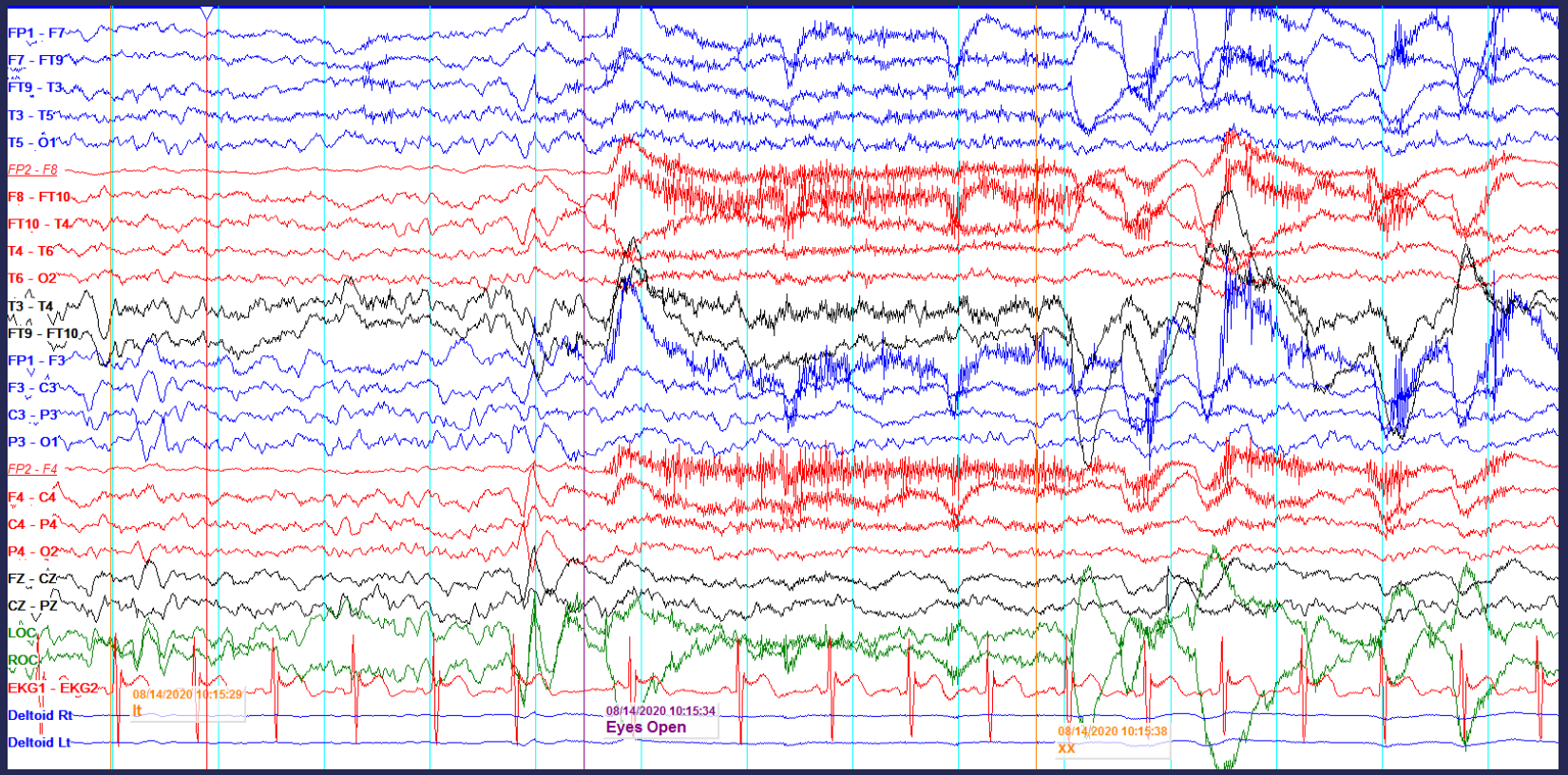
ABNORMAL REACTIVITY OF ALPHA

- Unilateral failure of alpha blocking (attenuation with eye opening): Bancaud's phenomenon
  - Posterior subcortical lesion
  - Parietal or temporal lesions
- Bilateral failure of alpha blocking in monocular input
  - Disorder of one eye or optic nerve
- Absence of alpha rhythm, presence of occipital spikes
  - Long standing diseases of both eyes or central visual pathway
- Alpha frequency coma
  - Central pontine lesion
  - Widespread cerebral damage

EYE CLOSED
PDR BETTER OVER RIGHT



DECREASED REACTIVITY OF ALPHA RHYTHM TO EYE OPENING OVER LEFT



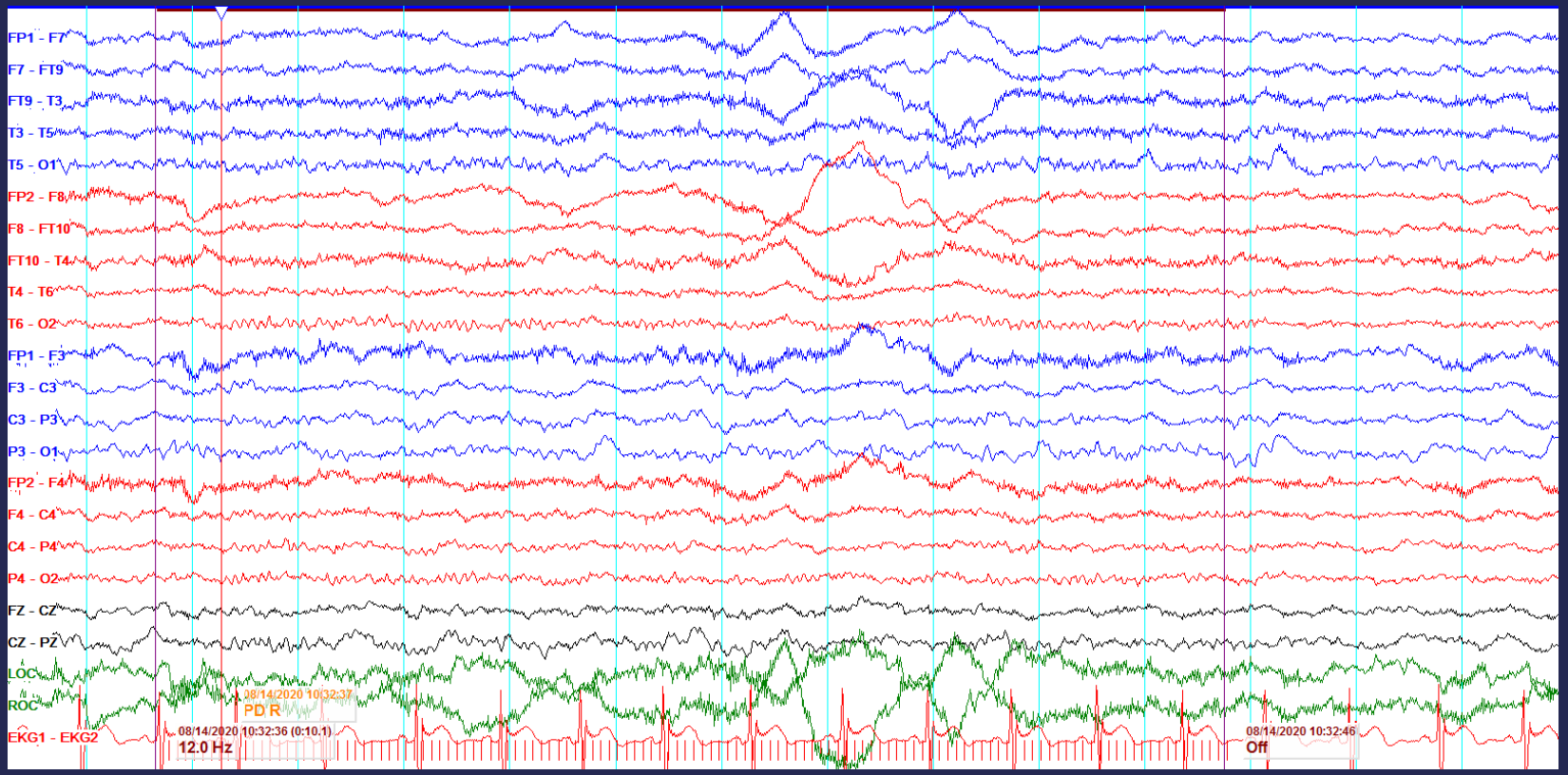
Bancaud's phenomenon

ASYMMETRY RESPONSE TO PHOTIC STIMULATION

- May result from 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 responses to photic stimulation that are of higher voltage on the side of lesion*

ASYMMETRICAL PHOTIC DRIVING RESPONSE

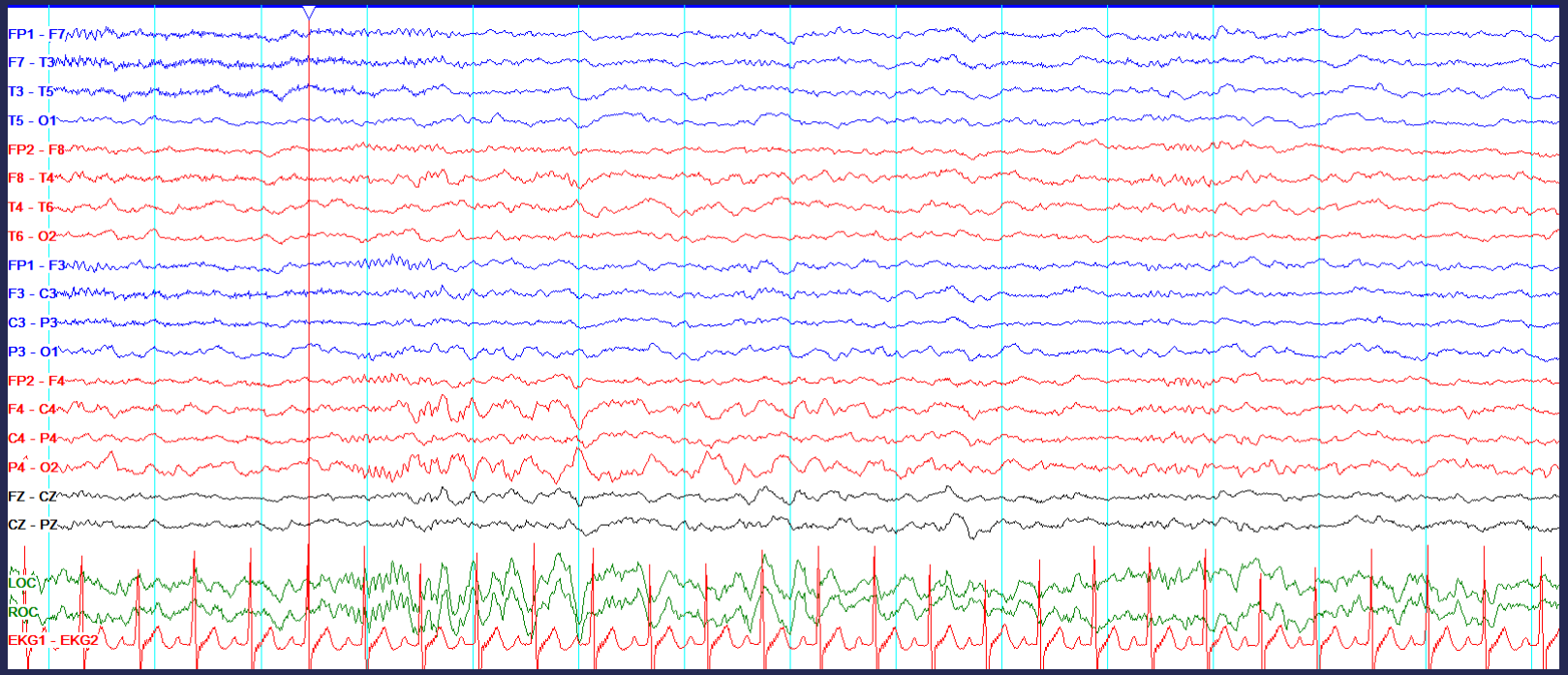


ABNORMAL SLEEP POTENTIAL

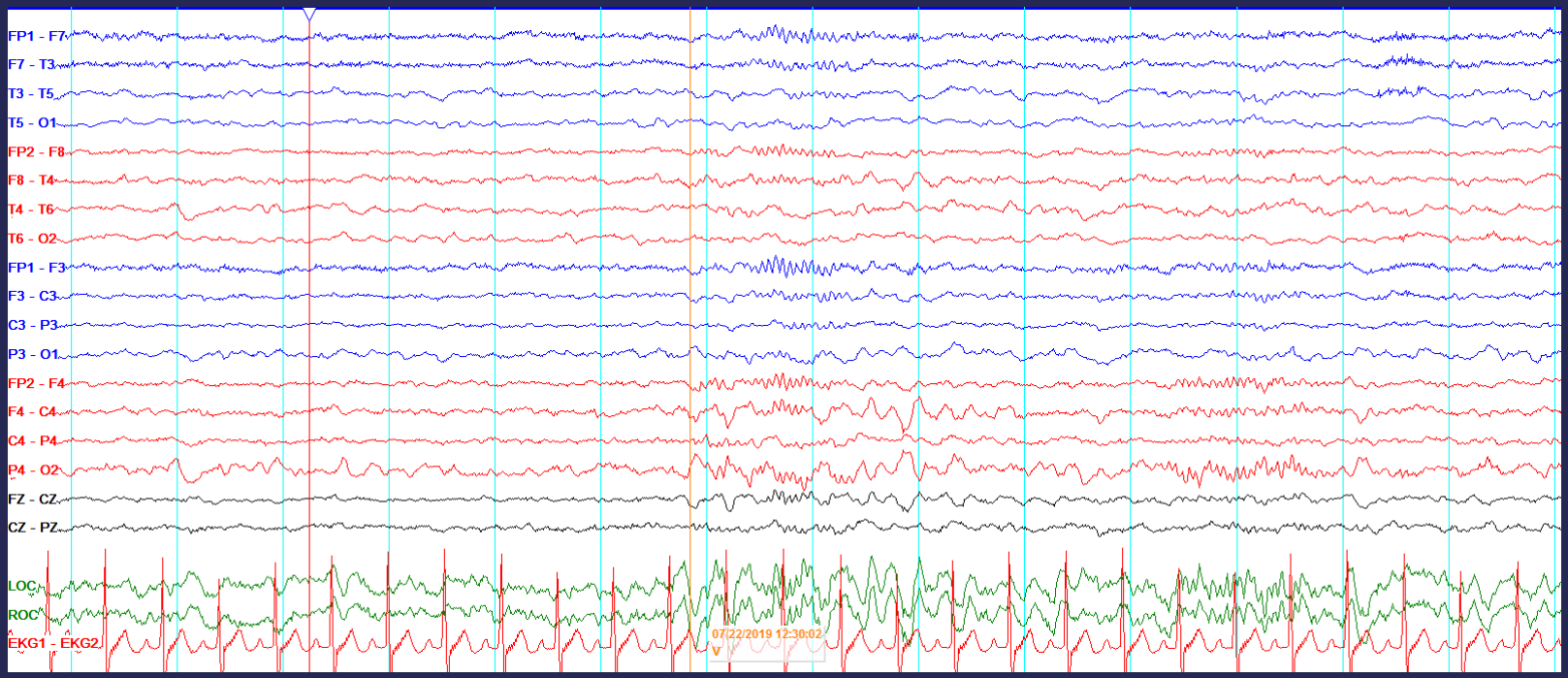
ABNORMAL SLEEP POTENTIALS

- Normal sleep patterns, sleep spindle, vertex can be affected by cerebral lesion
- Decrease sleep potential, spindles, if involved either thalamus, thalamocortical projection or cortex
- Skull defects enhance scalp-recorded voltage of sleep spindles and vertex

18 Y/O ALL, BEHAVIORAL CHANGE



ASYMMETRIC VERTEX



MRI bilateral subcortical white matter change
Old hemorrhage left thalamus

EXTREME SPINDLES

- Wide spread 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

THANK YOU