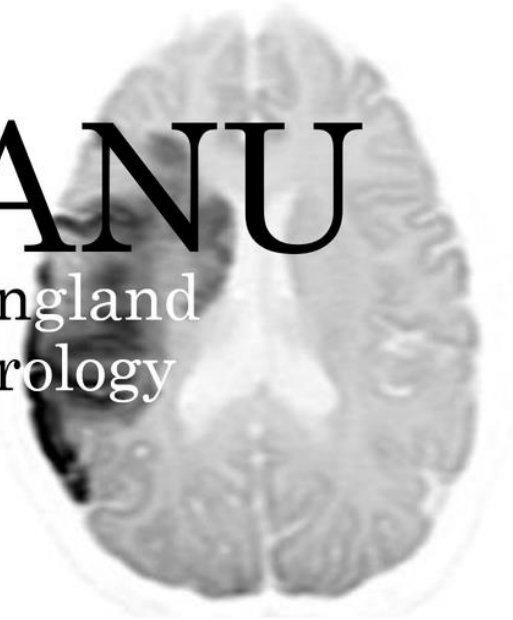
NEANU

North of England Acute Neurology Update



The use (and abuse) of EEG in the acute medical setting

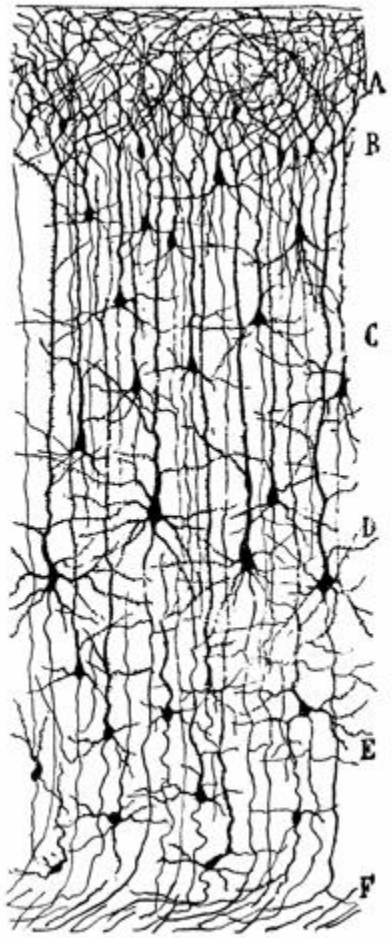
Rajiv Mohanraj

MANCHESTER 1824
The University of Manchester

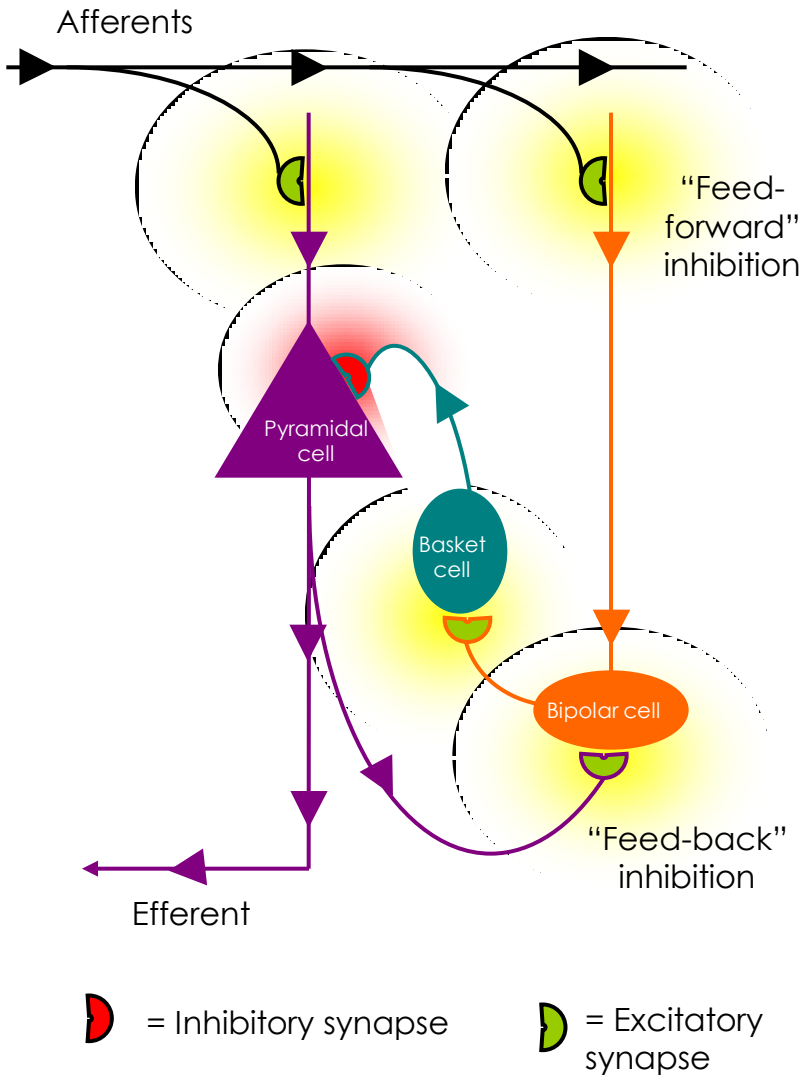
Manchester Centre for Clinical Neurosciences

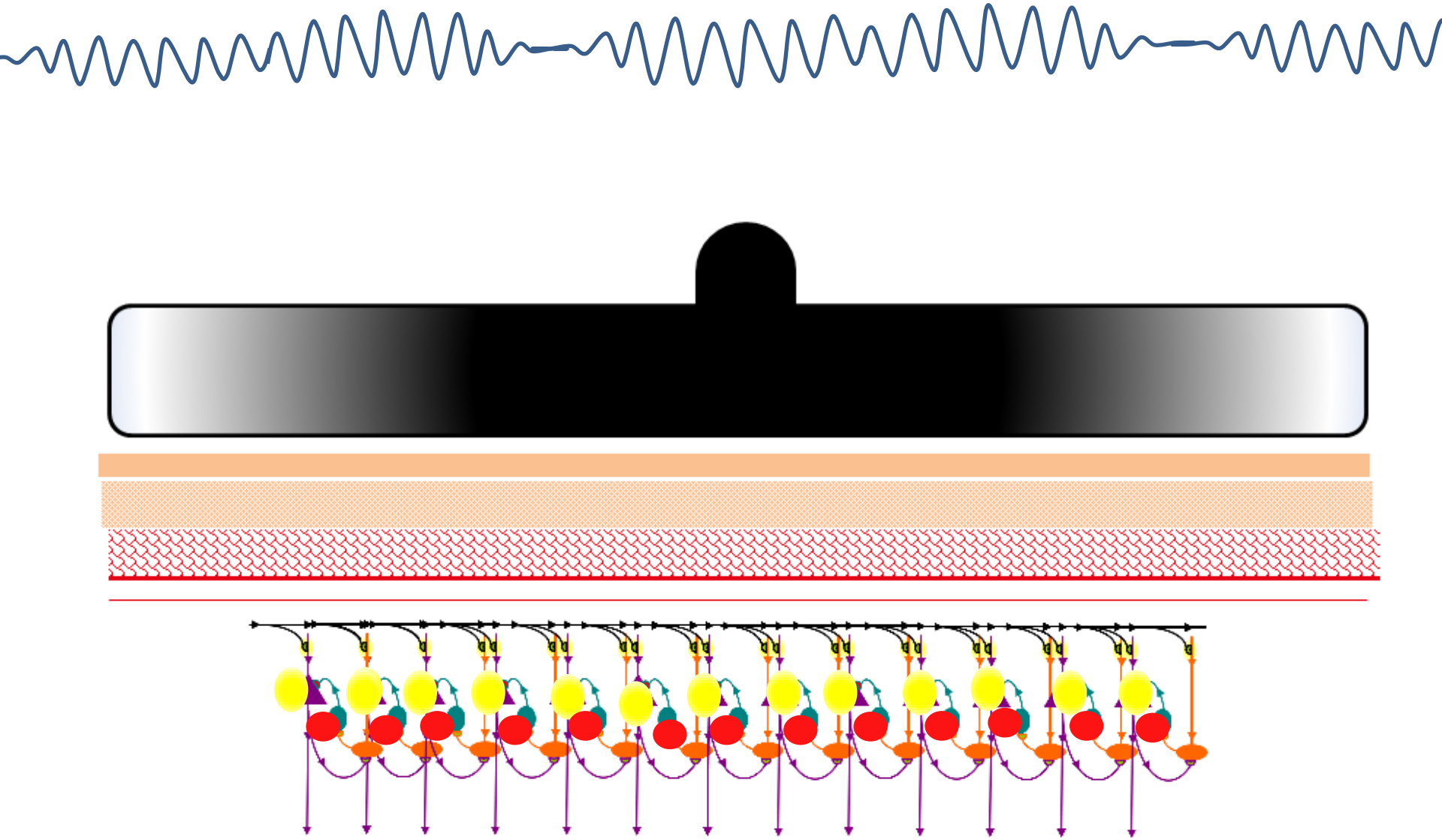
Salford Royal NHS
NHS Foundation Trust

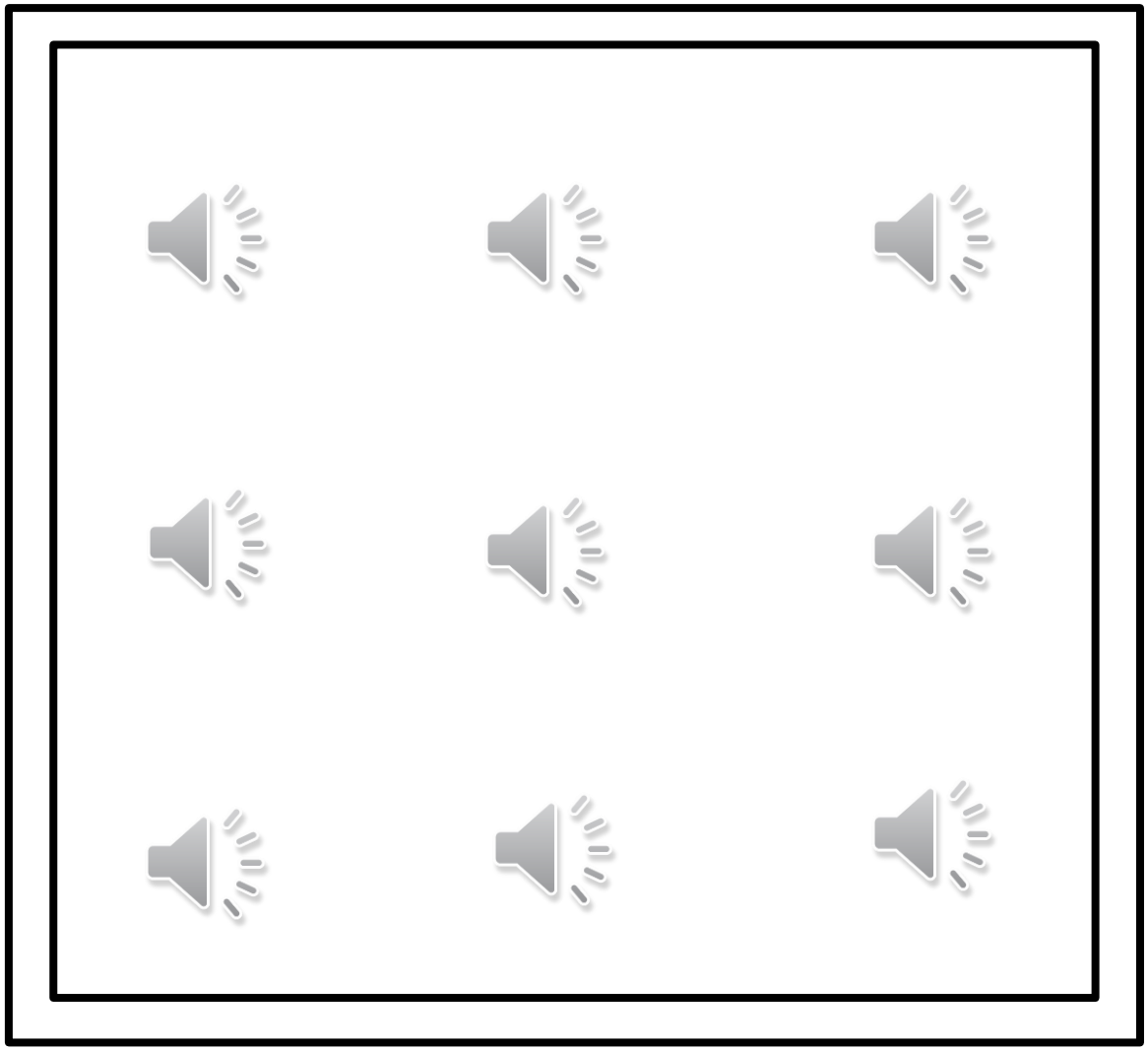
Scheme of cortical connectivity

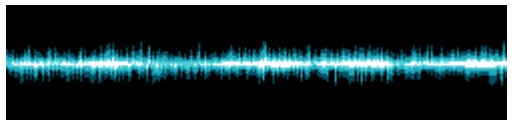


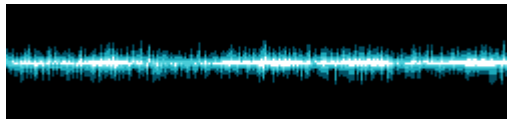
Santiago Ramón y Cajal, Nobel Prize 1906

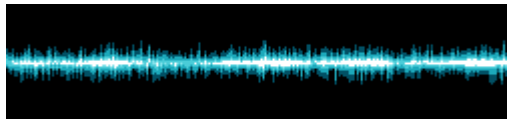




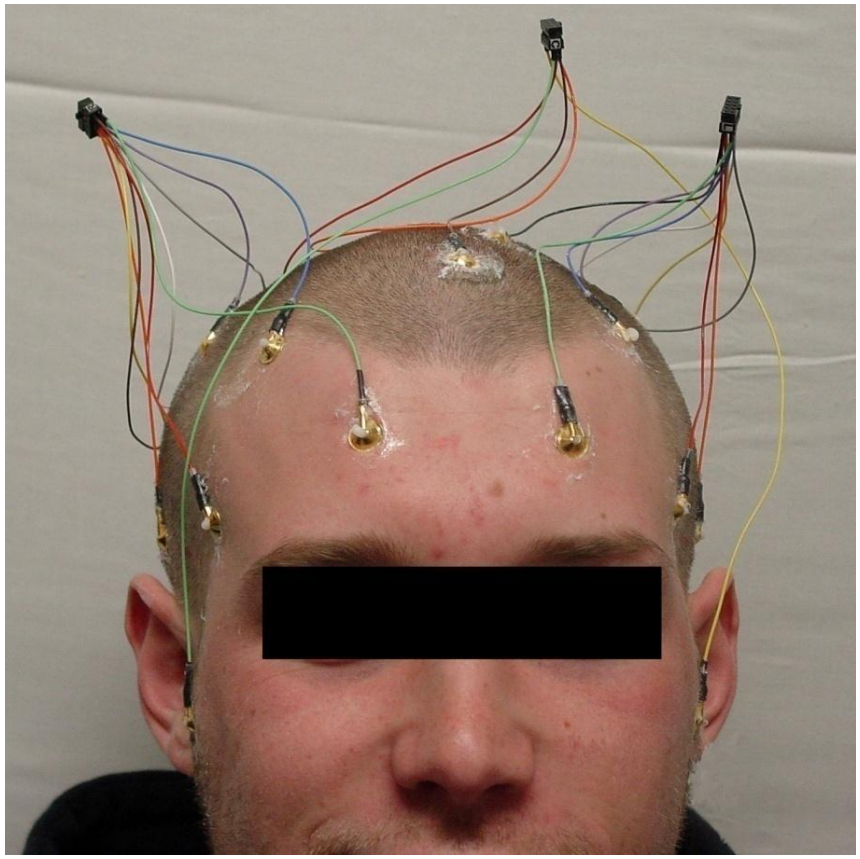


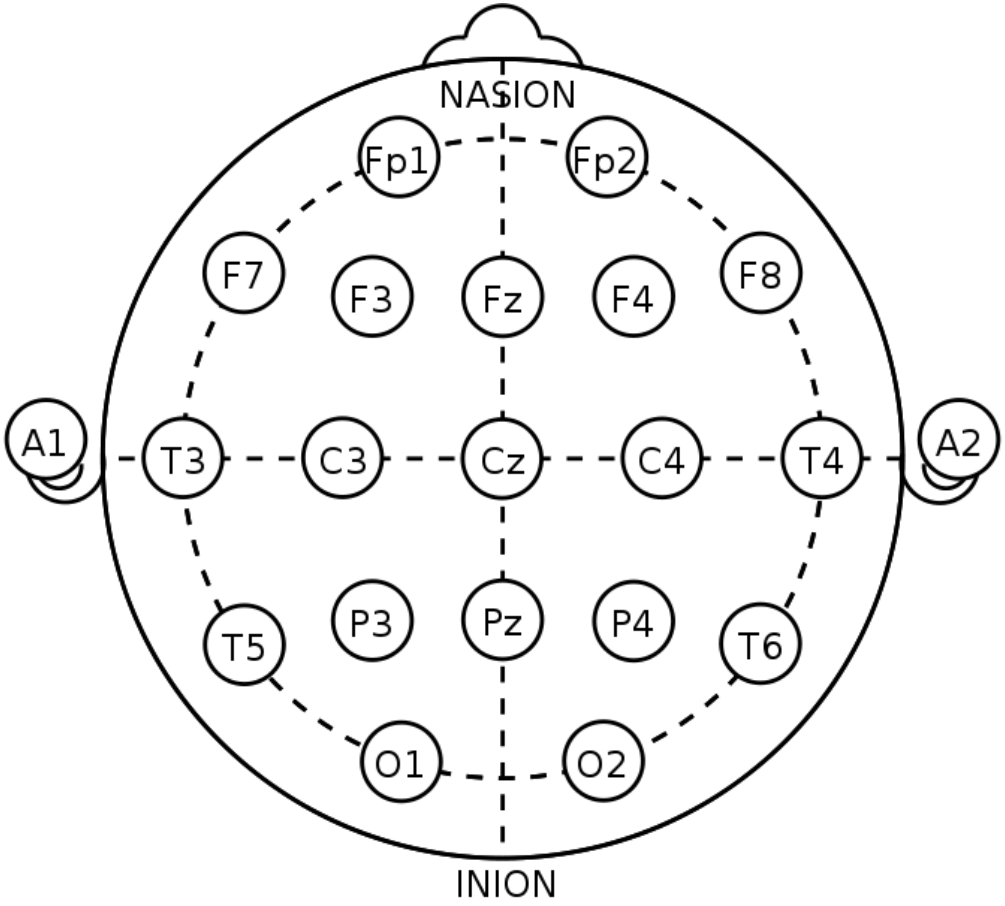






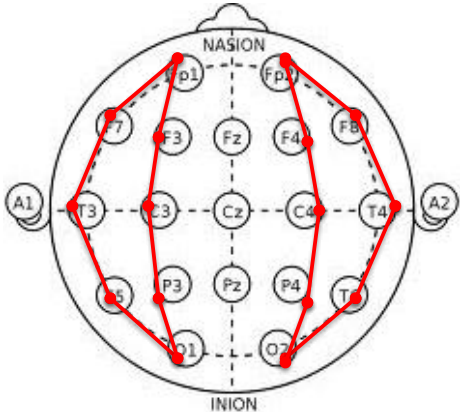
Electroencephalogram (EEG)



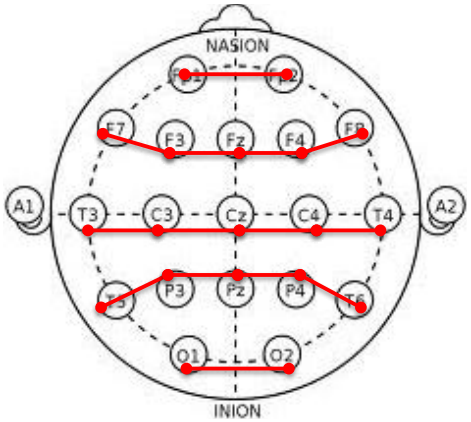


Montages

Bipolar montages

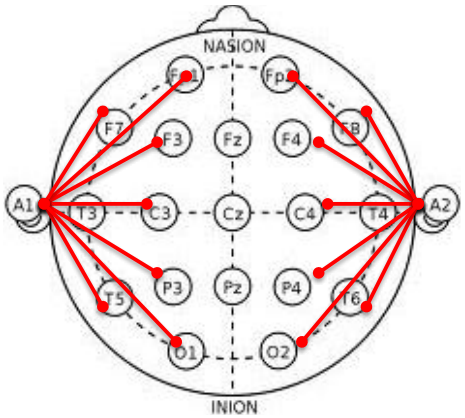


Longitudinal (AP)
'double banana'

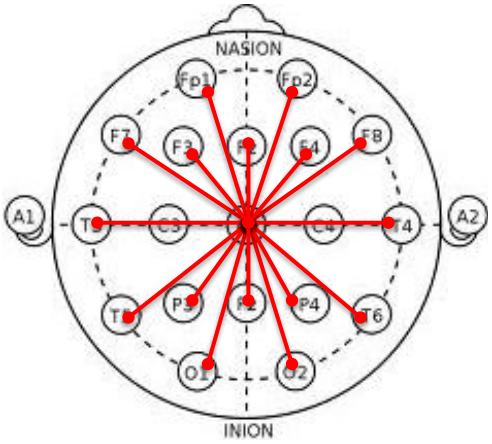


Transverse

Referential montages

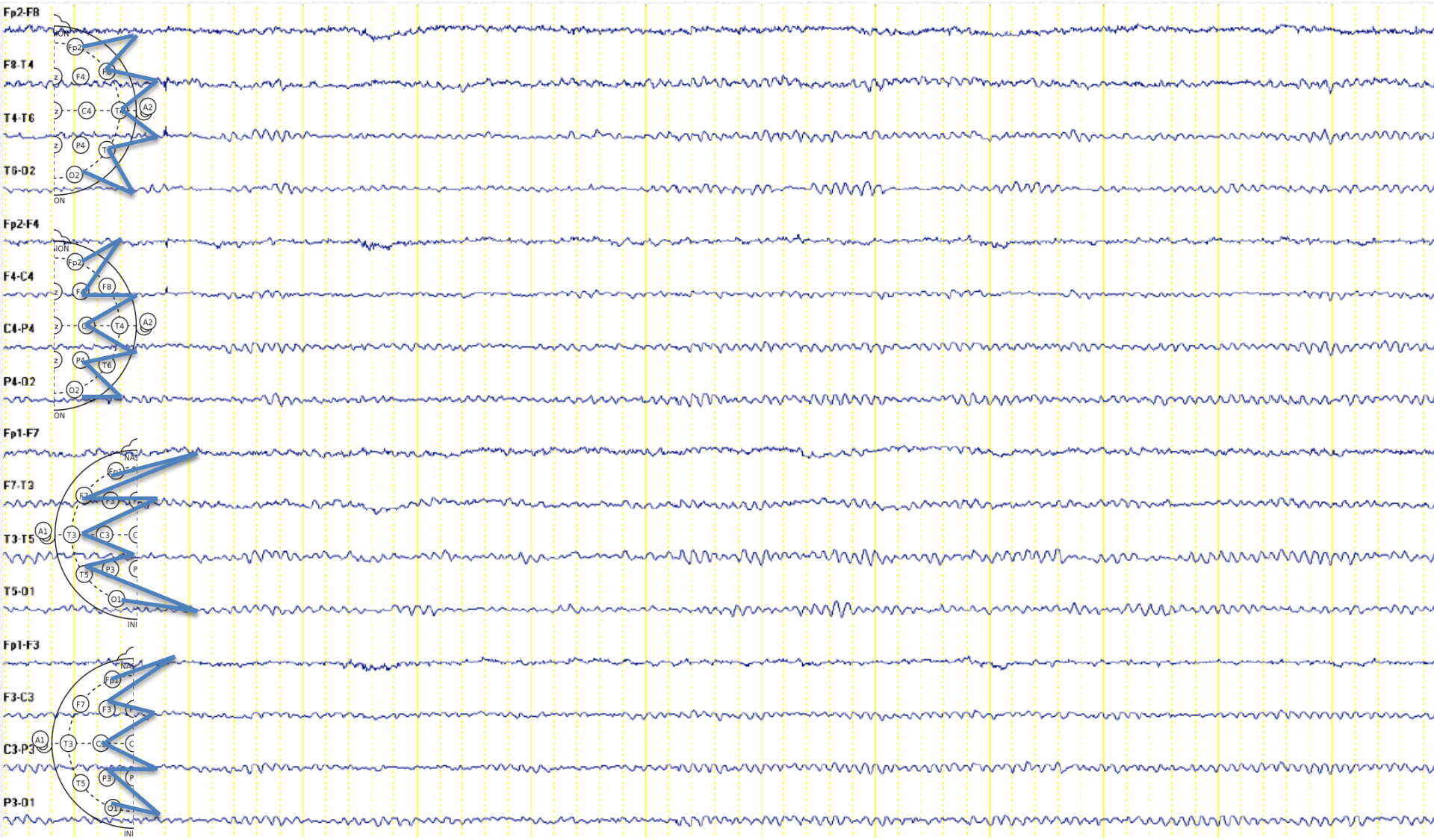


Ipsilateral ear reference



Vertex (Cz) reference

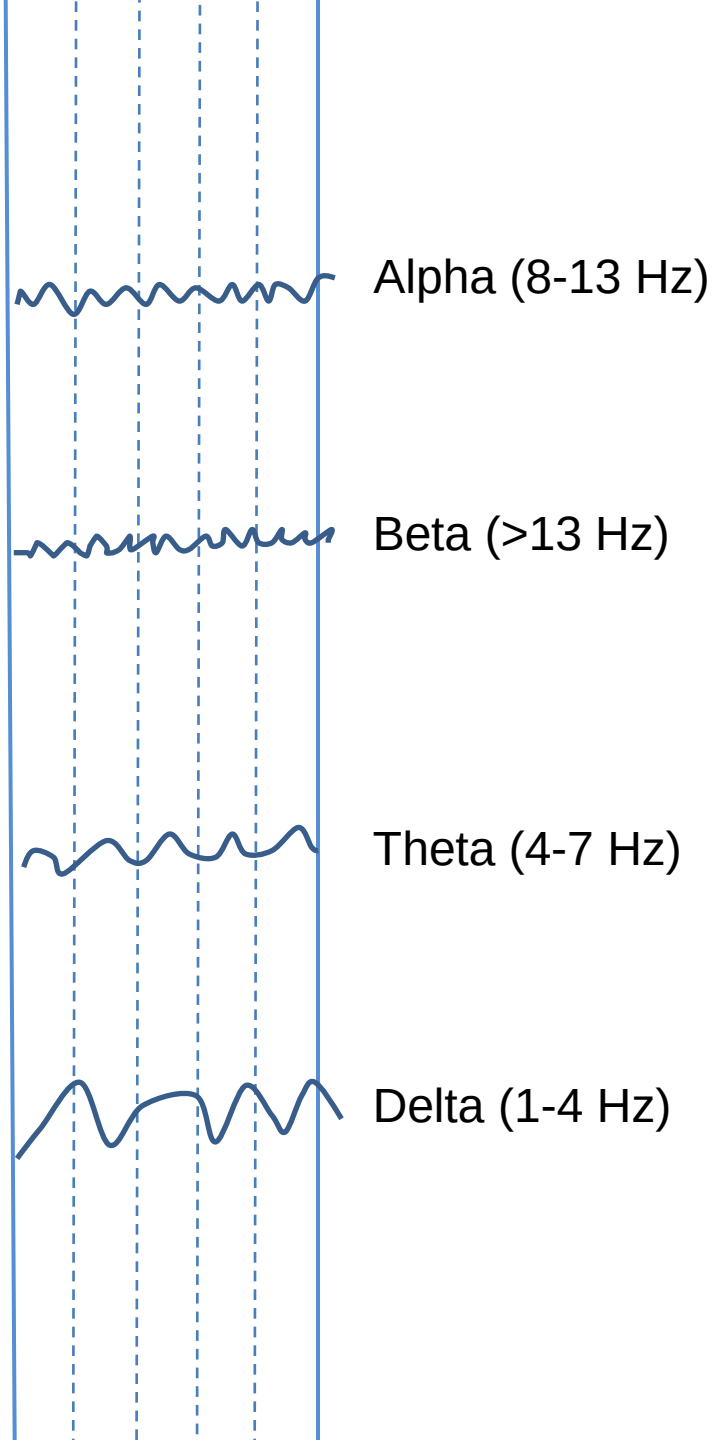
EEG display – double banana montage

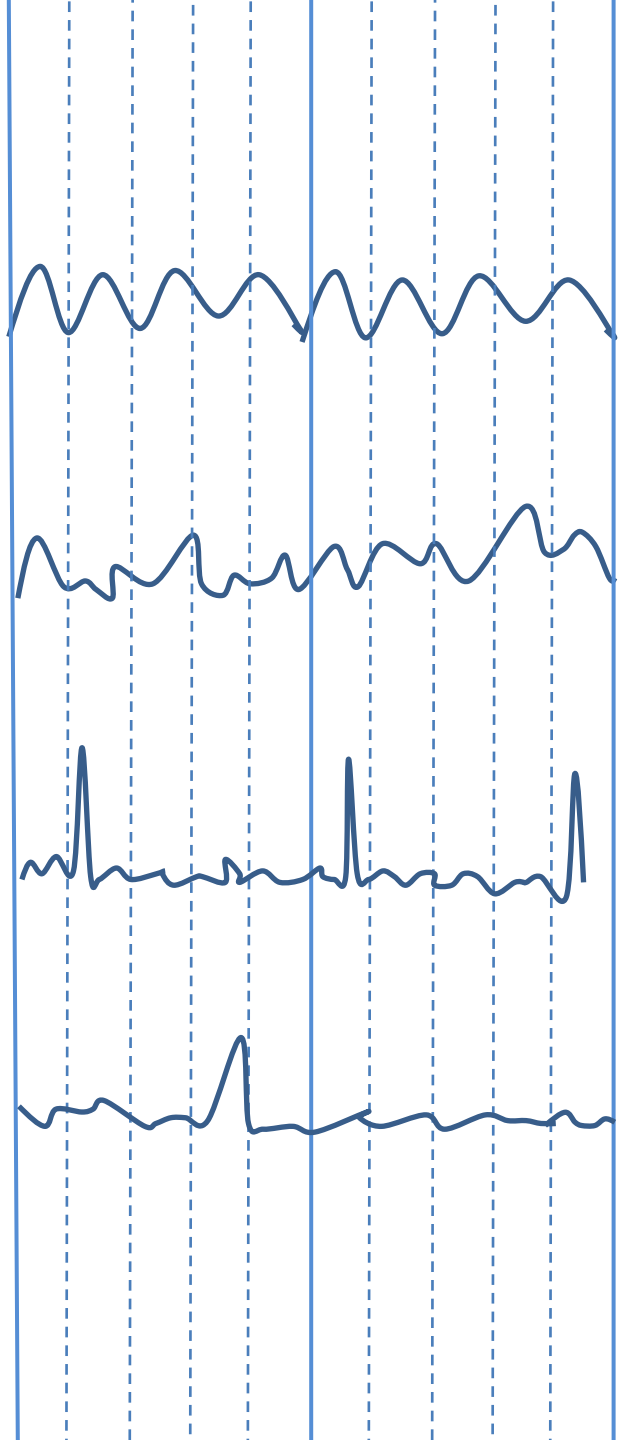


Characteristics of EEG waveforms

- *Frequency*
  - *Beta*
  - *Alpha*
  - *Theta*
  - *Delta*
- *Distribution of the wave form*
  - *Generalised*
  - *Lateralised*
  - *Regional*
- *Repetition pattern (unless isolated)*
  - *Monomorphic / Rhythmic*
  - *Polymorphic / Arrhythmic*
  - *Periodic*
  - *Intermittent*
- *Reactivity*
- *Specific morphology*

Frequency
- Berger Bands





Repetition pattern

Rhythmic (monomorphic)

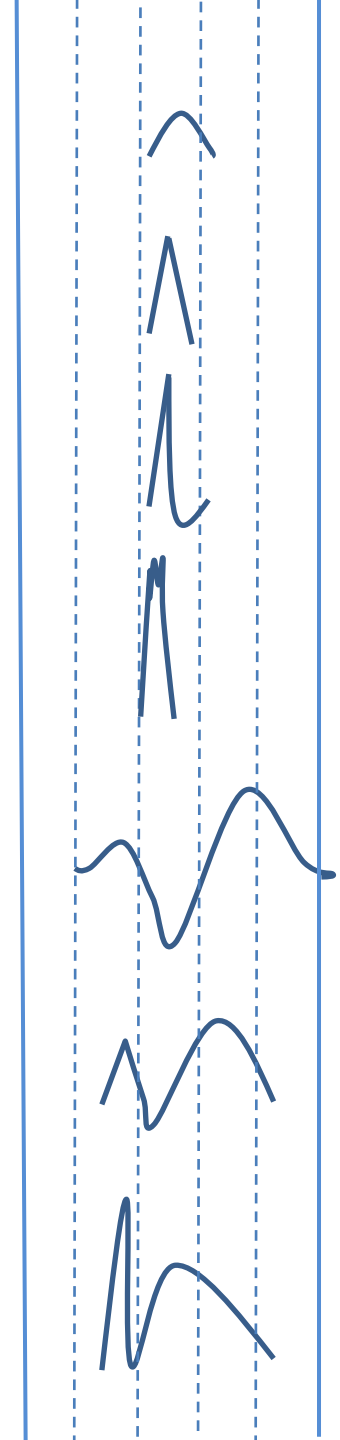
Arrhythmic (polymorphic)

Periodic

Intermittent

- abundant – 50-89% of the record
- Frequent– 10-49% of the record
- occasional– 1-9% of the record
- rare – <1% of the record

Morphology of waveforms



Slow wave

Sharp wave

Spike

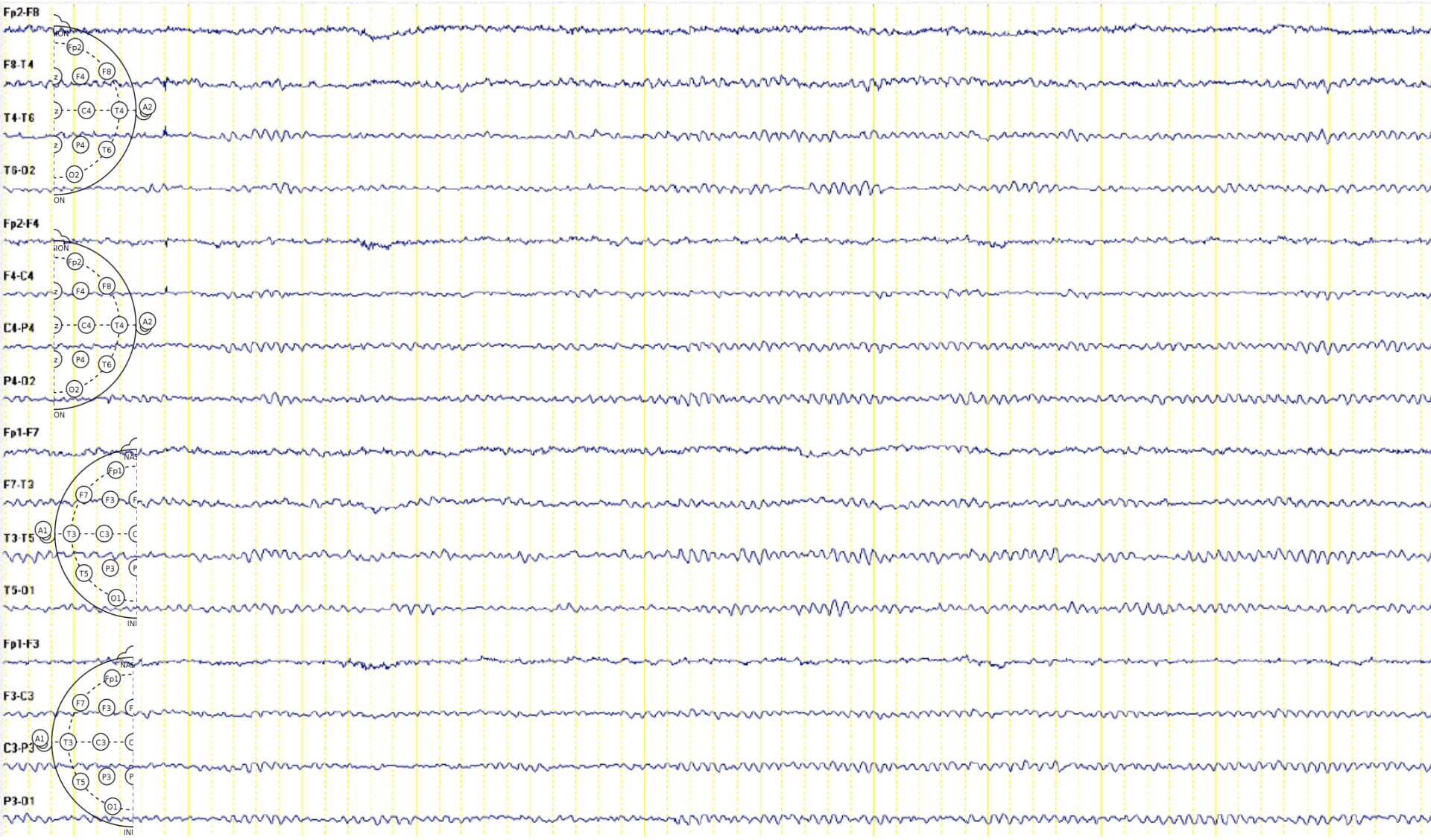
Poly spike

Triphasic wave

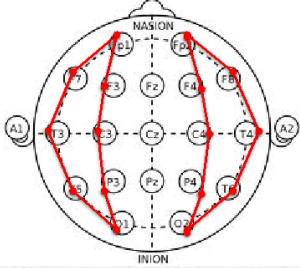
Sharp and slow wave

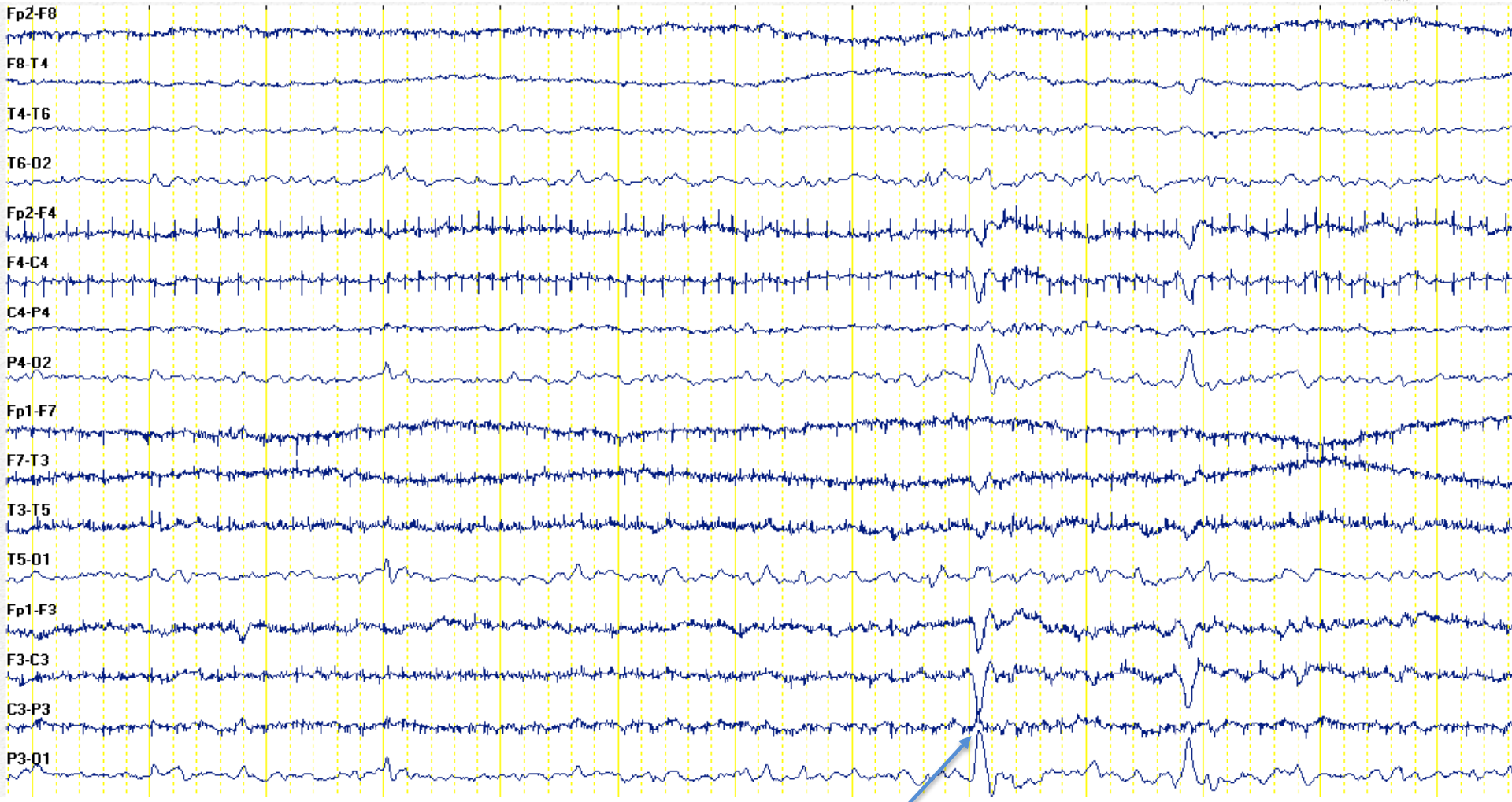
Spike-slow wave complex

Normal adult EEG (awake, eyes closed)



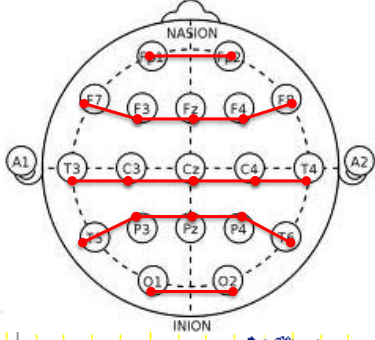
Drowsiness, stage I sleep

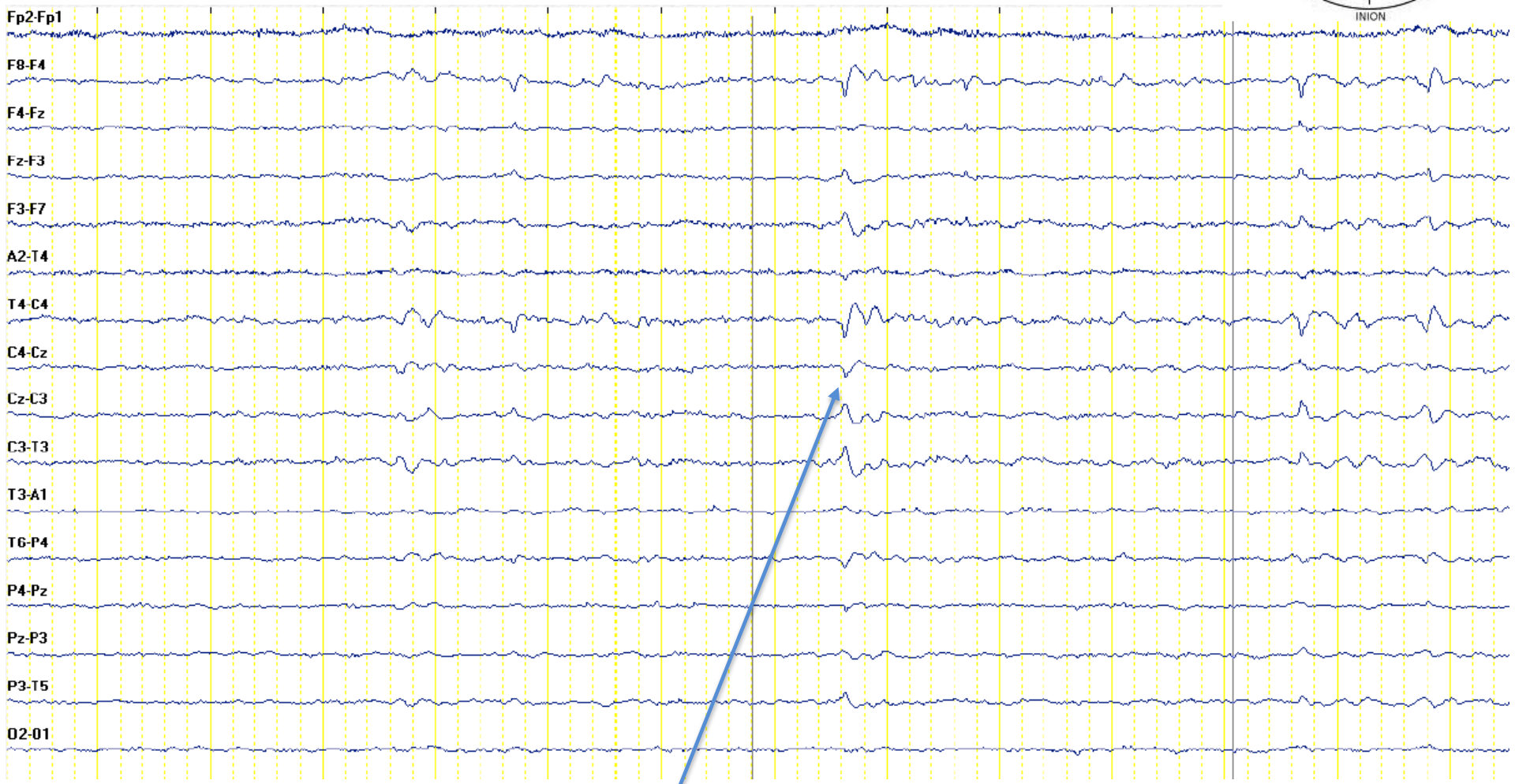




POSTS

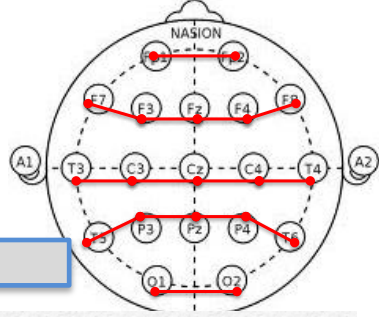
Stage II sleep (note montage)





Vertex (V) waves

Stage II sleep

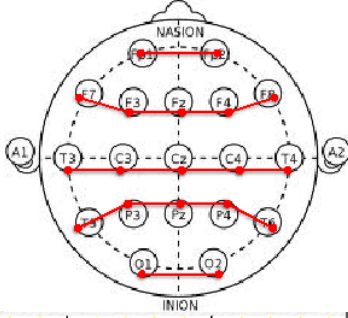


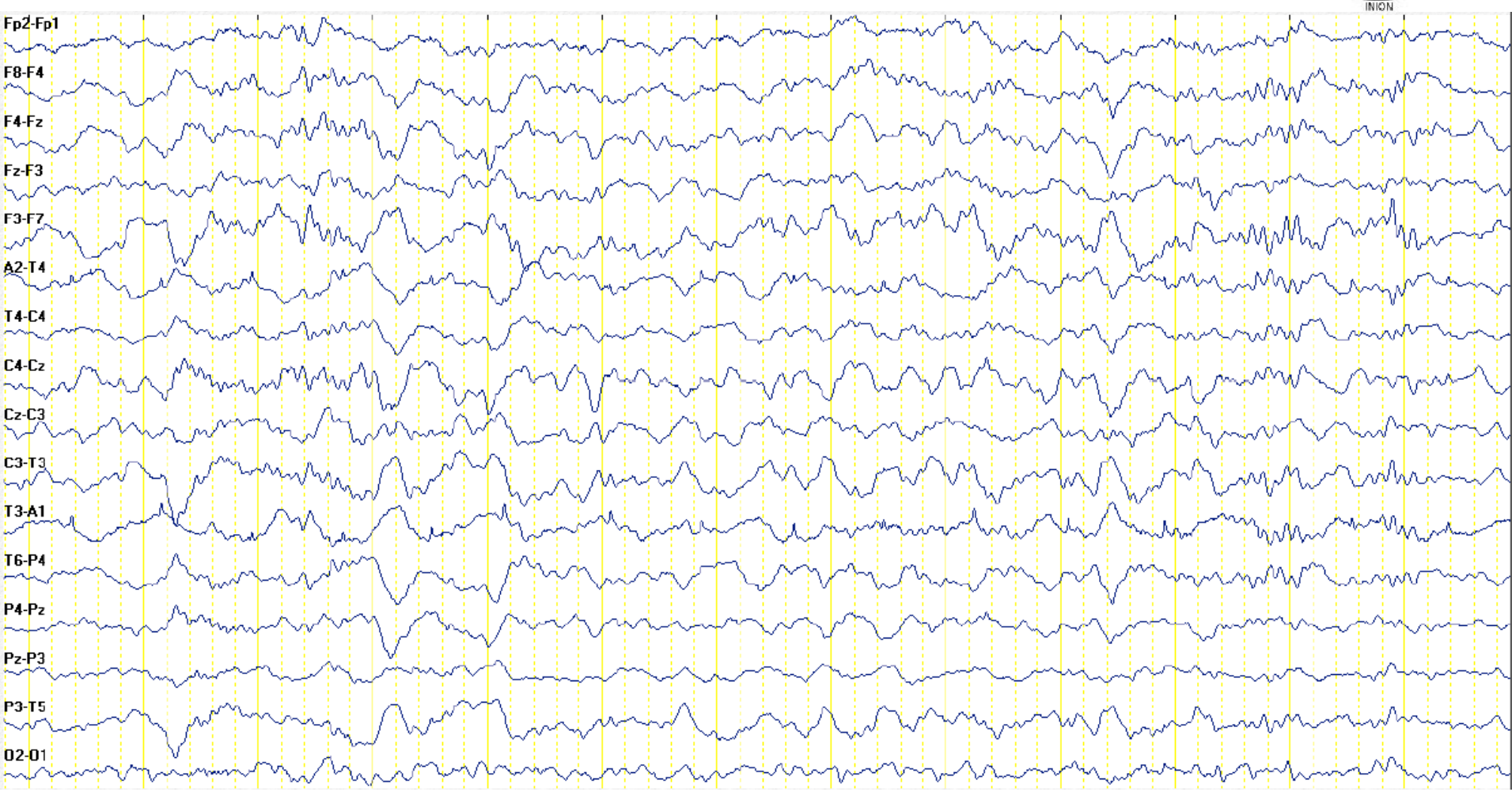
Sleep spindles

Fp2-Fp1
F8-F4
F4-Fz
Fz-F3
F3-F7
A2-T4
T4-C4
C4-Cz
Cz-C3
C3-T3
T3-A1
T6-P4
P4-Pz
Pz-P3
P3-T5
O2-O1

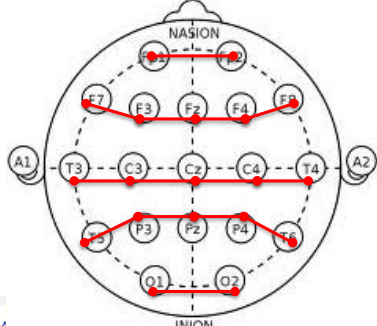
K complexes

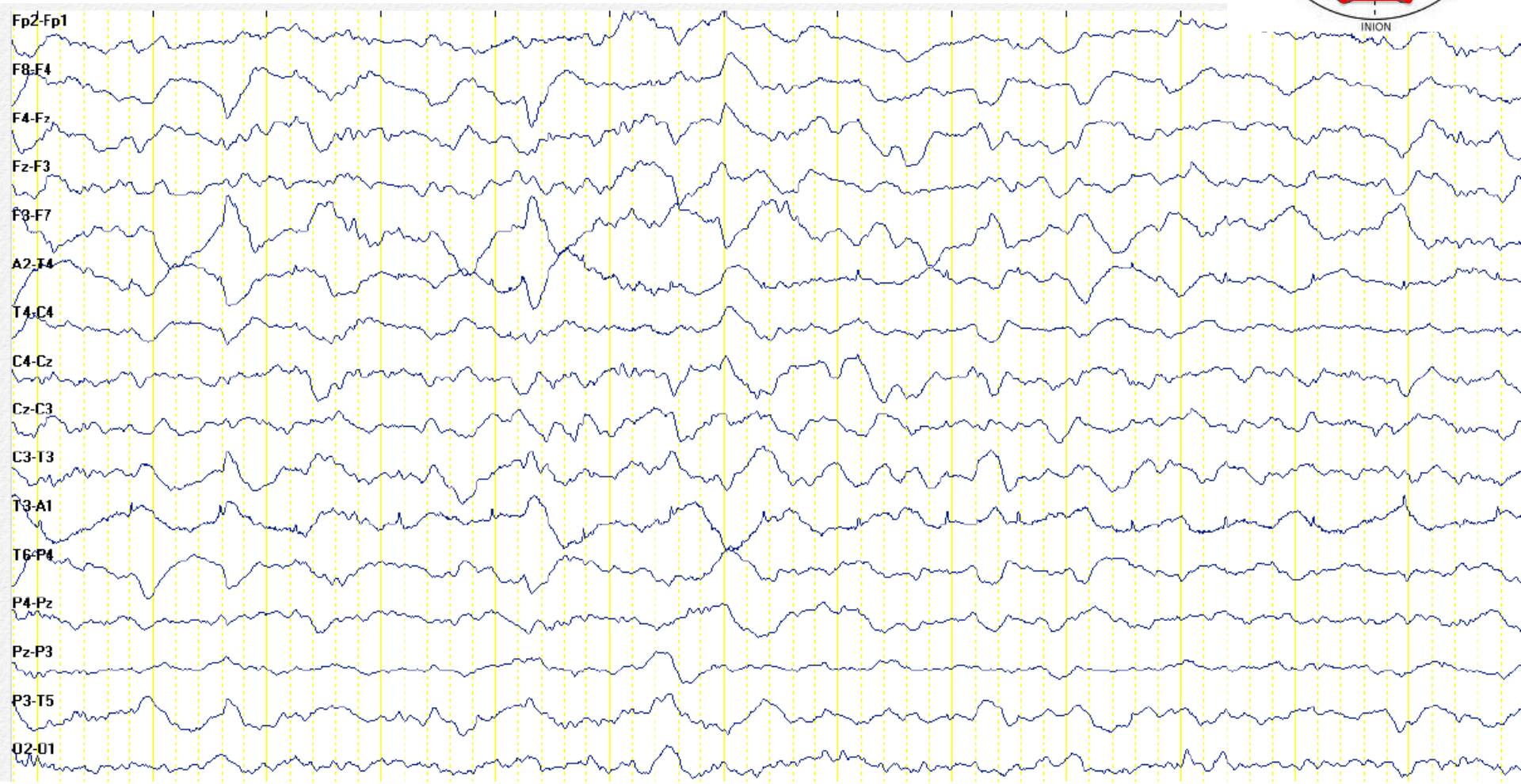
Stage III sleep



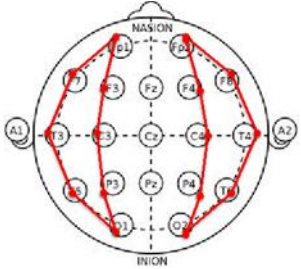


Stage IV sleep





REM sleep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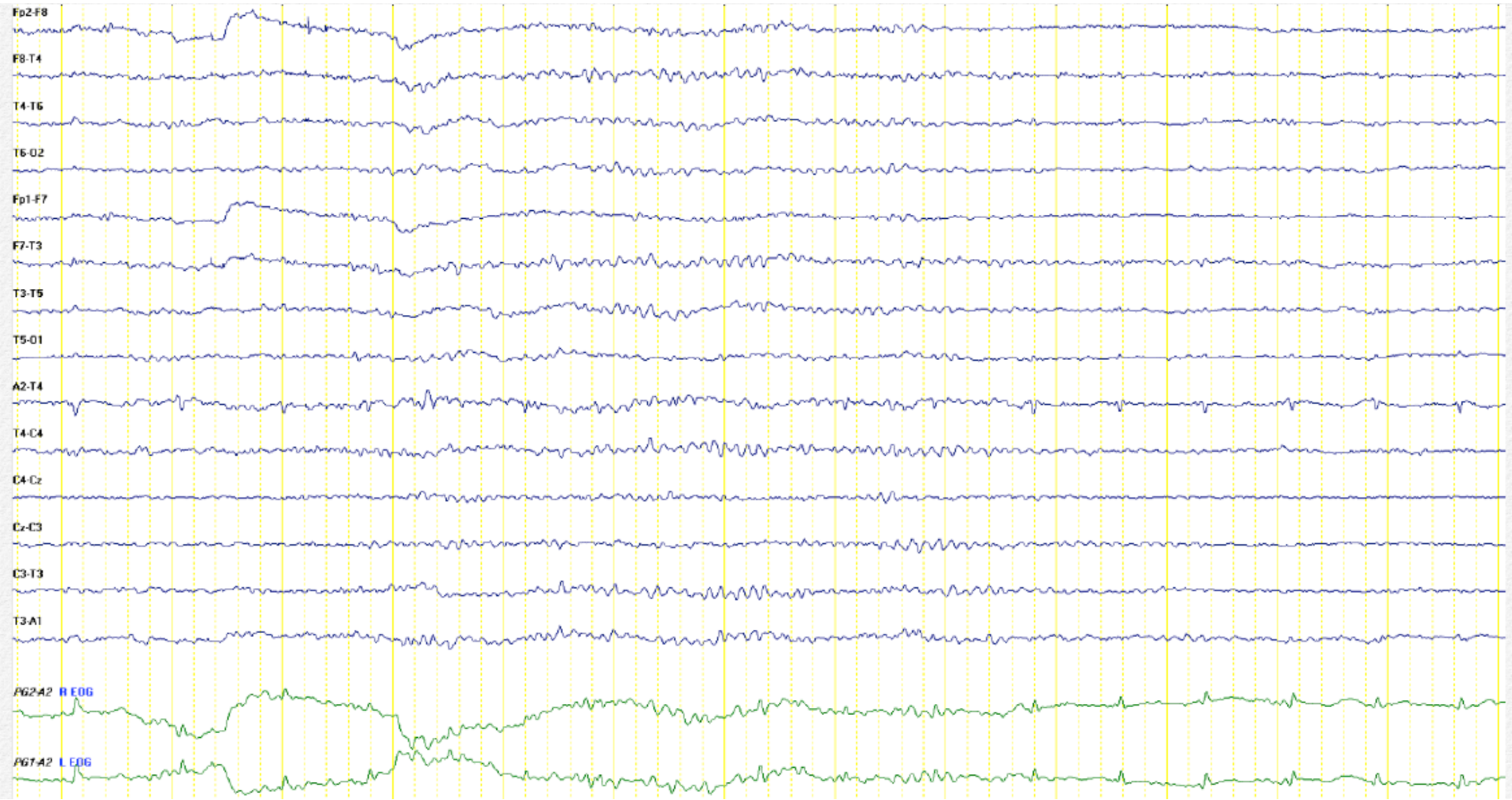
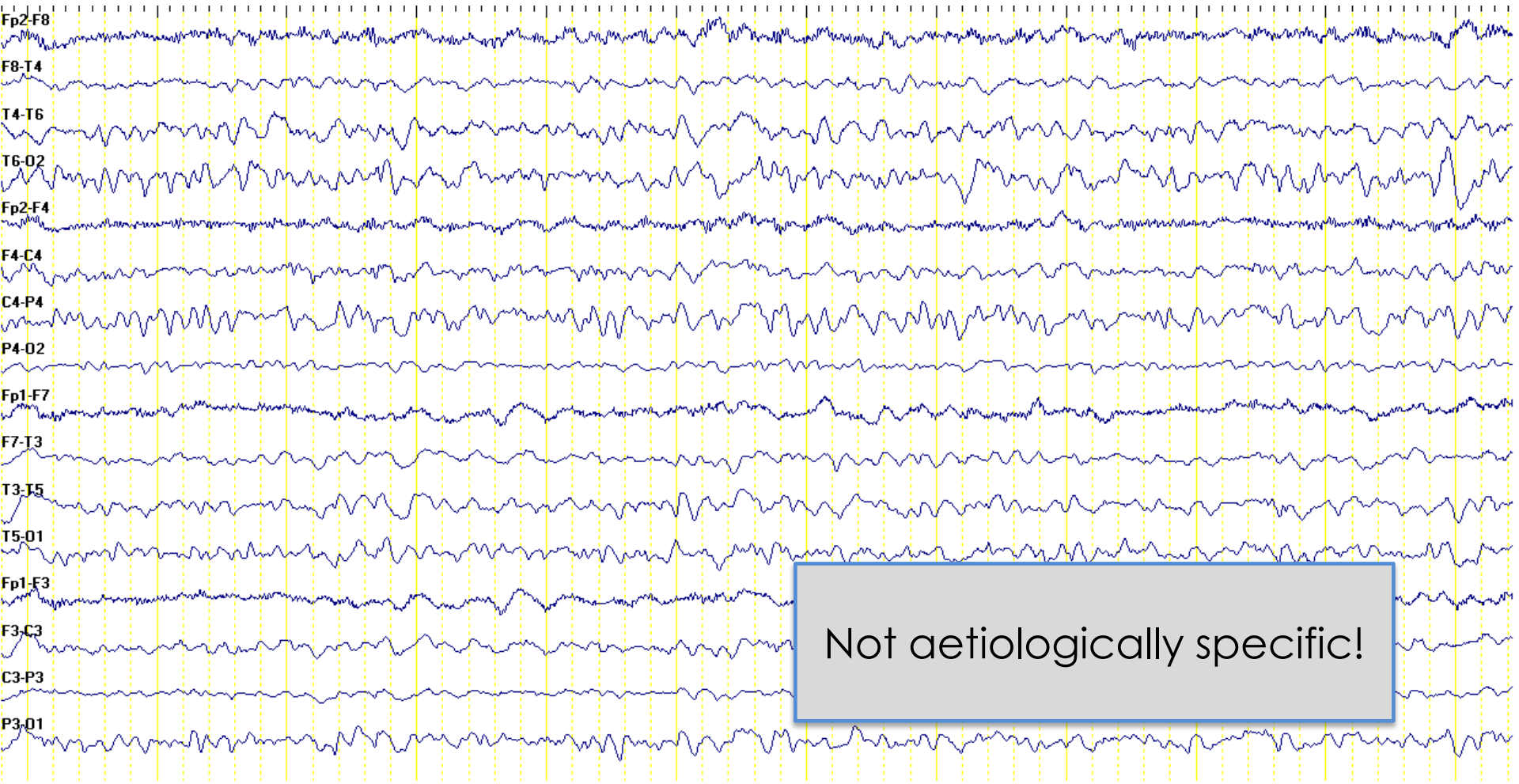
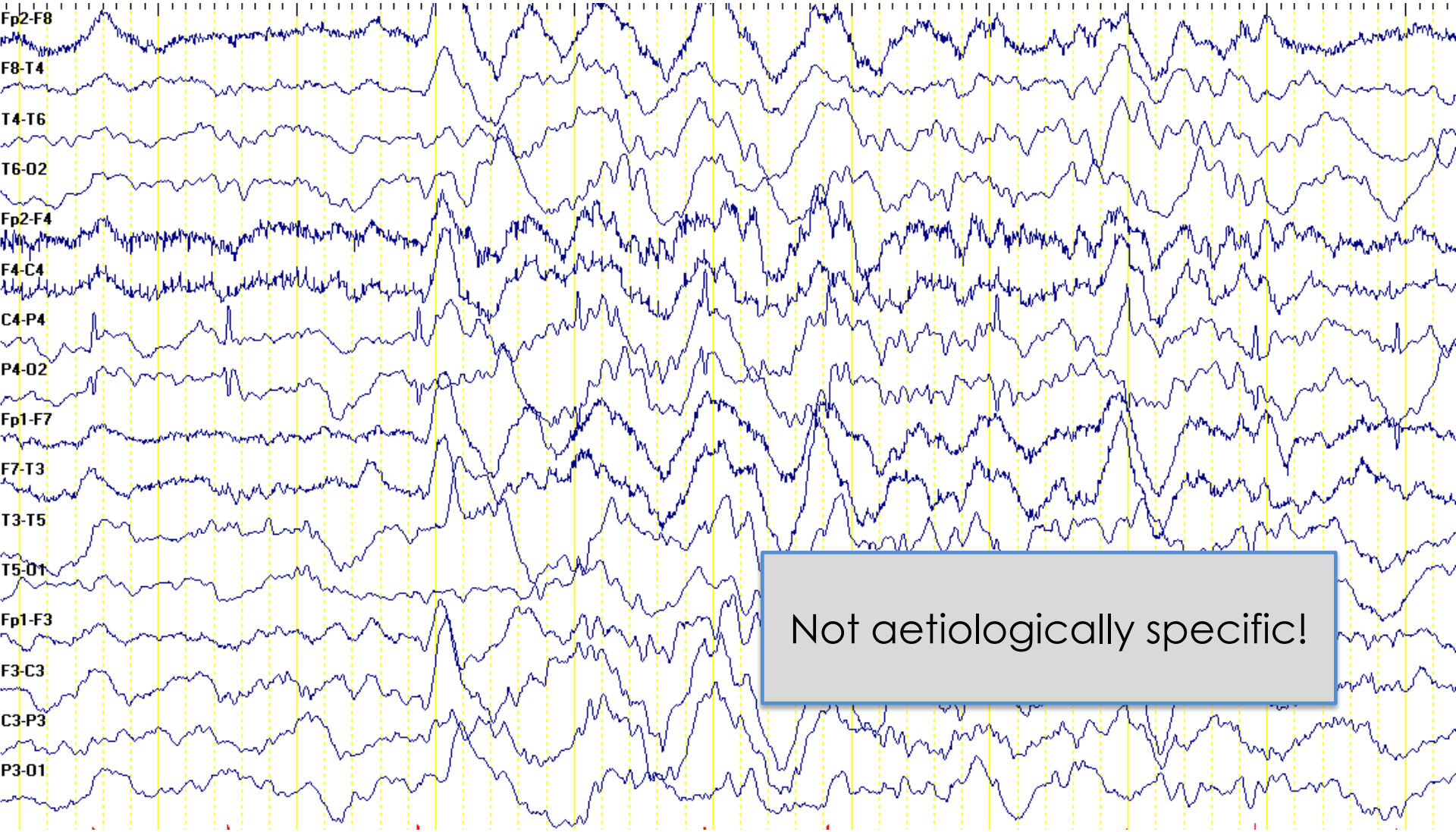

Fp2-F8
F8-T4
T4-T6
T6-O2
Fp1-F7
F7-T3
T3-T5
T5-O1
A2-T4
T4-C4
C4-Cz
Cz-C3
C3-T3
T3-A1
PG2-A2 R EOG
PG1-A2 L EOG

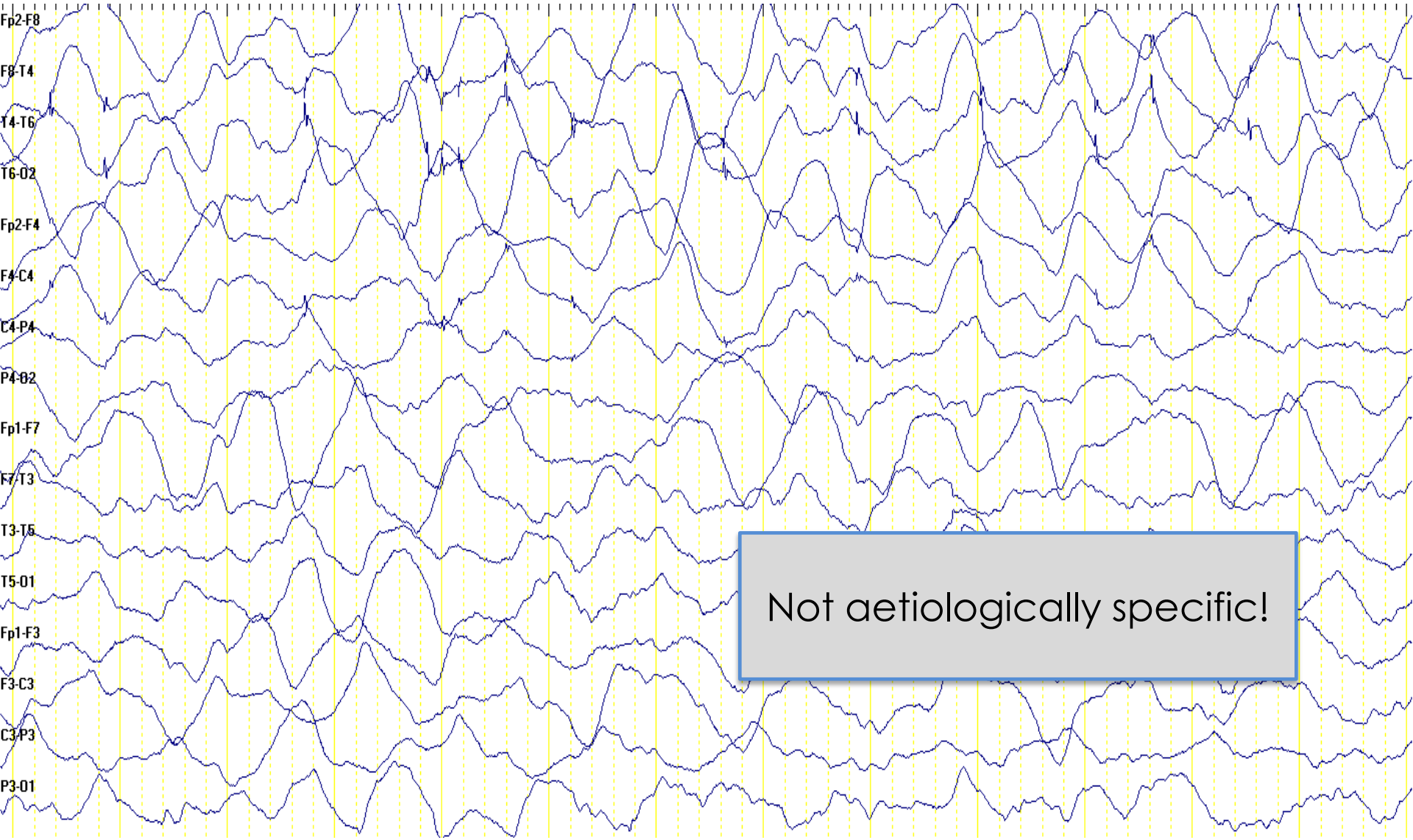
Generalised encephalopathy - mild



Generalised encephalopathy - moderate



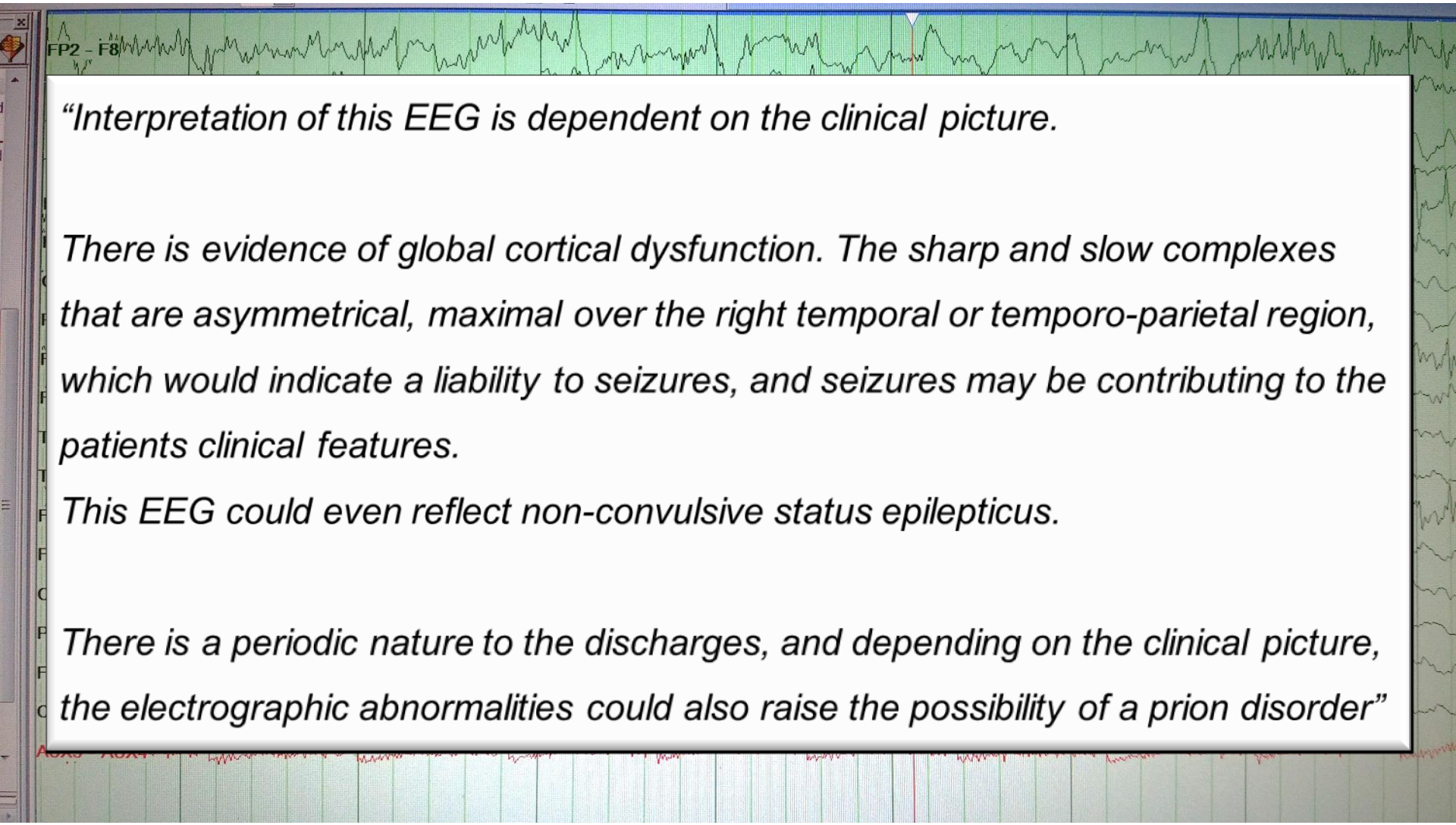
Generalised encephalopathy - severe



Not aetiologically specific!

90 year old F

- Nursing home resident
- Admitted with chest infection
  - Confused
  - CRP 25
- 3 days later
  - Unresponsive
  - Neurology referral
  - EEG requested



"Interpretation of this EEG is dependent on the clinical picture.

There is evidence of global cortical dysfunction. The sharp and slow complexes that are asymmetrical, maximal over the right temporal or temporo-parietal region, which would indicate a liability to seizures, and seizures may be contributing to the patients clinical features.

This EEG could even reflect non-convulsive status epilepticus.

There is a periodic nature to the discharges, and depending on the clinical picture, the electrographic abnormalities could also raise the possibility of a prion disorder"

- Discussed with on call neurology registrar
  - ‘We have a 90 year old in non convulsive status’
  - Loaded with valproate
  - Comatose

- Referred to neurology

CRP 326

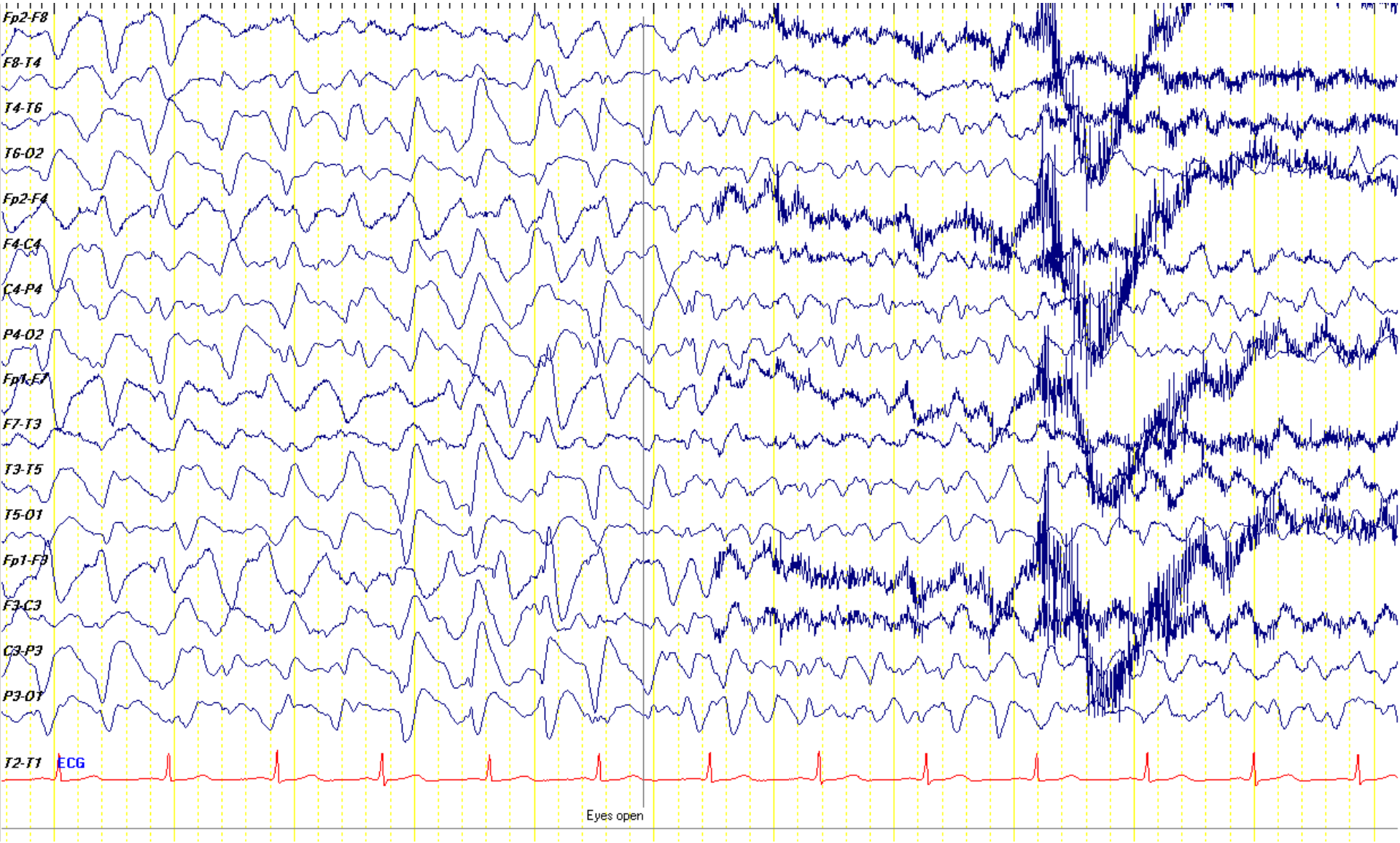
Diagnosis: Septic Encephalopathy due to pneumonia

55 year old F

- Admission to Wythenshawe Dec 2016 – discharge diagnosis ‘encephalitis’
- Neurology clinic six months later
  - Persisting tiredness, sleeps 2 hours during the day
  - Speech slow, word finding difficulty
  - Emotional, cries
- MRI normal
- VGKC Abs 101, NMDA negative

- Neurology ward
  - CSF - WCC 3
    - RCC 2
    - Protein 0.65
    - Glucose 3.8 (plasma 13.9)
  - Hb 8.8
  - Serum iron 5.9 (9.0-30)
  - Ferritin 12 (10- 291)
  - NMDA, TPO Abs negative


Fp2-AV
F8-AV
T4-AV
T6-AV
O2-AV
F4-AV
C4-AV
P4-AV
Fp1-AV
F7-AV
T3-AV
T5-AV
O1-AV
F3-AV
C3-AV
P3-AV
T2-T1


Fp2-F8
F8-T4
T4-T6
T6-O2
Fp2-F4
F4-C4
C4-P4
P4-O2
Fp1-F7
F7-T3
T3-T5
T5-O1
Fp1-F3
F3-C3
C3-P3
P3-O1
T2-T1
ECG
Eyes open

The record is grossly abnormal

The background is dominated by high amplitude delta activity throughout the recording interrupted by periods where eye closure produces attenuation and what looks like recruit rhythm. At the end of the recording triphasic waves, sharps and period of attenuation of activity were seen.

Through the recording the patient was drowsy but fully oriented and no clear behavioural changes.

Overall, these findings are in favour of nonconvulsive status epilepticus. However, encephalitis cannot be ruled out entirely.

A repeat study, after a trial of clobazam, is recommended. An EEG will be arranged for tomorrow

- Started on Clobazam
  - Extremely drowsy
  - Bed bound
  - Barely able to speak

- Repeat EEG

Repeat EEG after three days since she has been started on clobazam

The record is still abnormal

The background is dominated by mixed theta and delta activity and occasionally superimposed by fast activity suggestive of mild to moderate degree of encephalopathy. Infrequent high amplitude delta activity was seen over the frontal regions suggestive of (FIRDA) which is nonspecific.

Infrequent sharp wave activity with phase reversal was seen at P4 suggestive of a focus arising of the right posterior quadrant.

Compared with the previous record, there is neurophysiological response to the clobazam.

A repeat EEG in one week time is recommended.

- Review UHSM notes
- October 2016
  - feeling more tired than normal
  - Speech slurred, balance poor
  - GP – bloods – iron deficiency anaemia – iron sup.
- December 2016
  - Appeared confused at work
  - Seen by GP -> Wythenshawe
  - ?encephalitis – LP normal
  - 5 days IP – IV acyclovir

- Letter from gastroenterology April 2016
  - Referral for investigation of IDA
  - Mentions previous referral in 2015 for ‘mildly deranged LFTs’
  - Gastroscopy ‘3 varices’
  - Started on carvedilol

Serum ammonia 110

Diagnosis – Hepatic encephalopathy due to alcoholic liver disease

34 year old M

- 15-20 episodes of collapsing daily
- Unresponsive for several minutes
- Confused on coming round
- Can hit head, scalp lacerations
- No motor activity, ?some pallor
- ‘Looks like he is asleep’

Video EEG recording

Normal EEG during unresponsive episode

Diagnosis: Psychogenic non epileptic attack

Conclusions

- Interpreting EEG out of clinical context is the single biggest cause of error
  - Clinical context not always available to the neurophysiologist
- Appreciate limitations of EEG
  - In diffuse encephalopathy, EEG does not provide aetiological clues.
  - Periodic triphasic /sharp- slow wave complexes occur in a wide variety of cerebral insults

Take home messages

- In the acute inpatient setting, EEG abnormalities most often reflect systemic disturbances
- A normal EEG can be helpful in the acute inpatient setting
- Beware reports of non convulsive status epilepticus