The use of EEG in Epilepsy, Encephalopathy and Coma in Adults

The Basic Principles

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Electroencephalography (EEG)

- Epilepsy and encephalopathy are clinically-based diagnoses but EEG is an extremely useful tool when used to bolster these clinical diagnoses.

- In good hands, EEG can be of immense benefit but, where used by inexperienced electroencephalographers, it may result in misdiagnosis and serious harm.

Reading an EEG

Effective reading of an EEG requires three elements:

1. Analysis of waveforms
2. Pattern recognition, and
3. Interpretation of these waveforms within the clinical context.

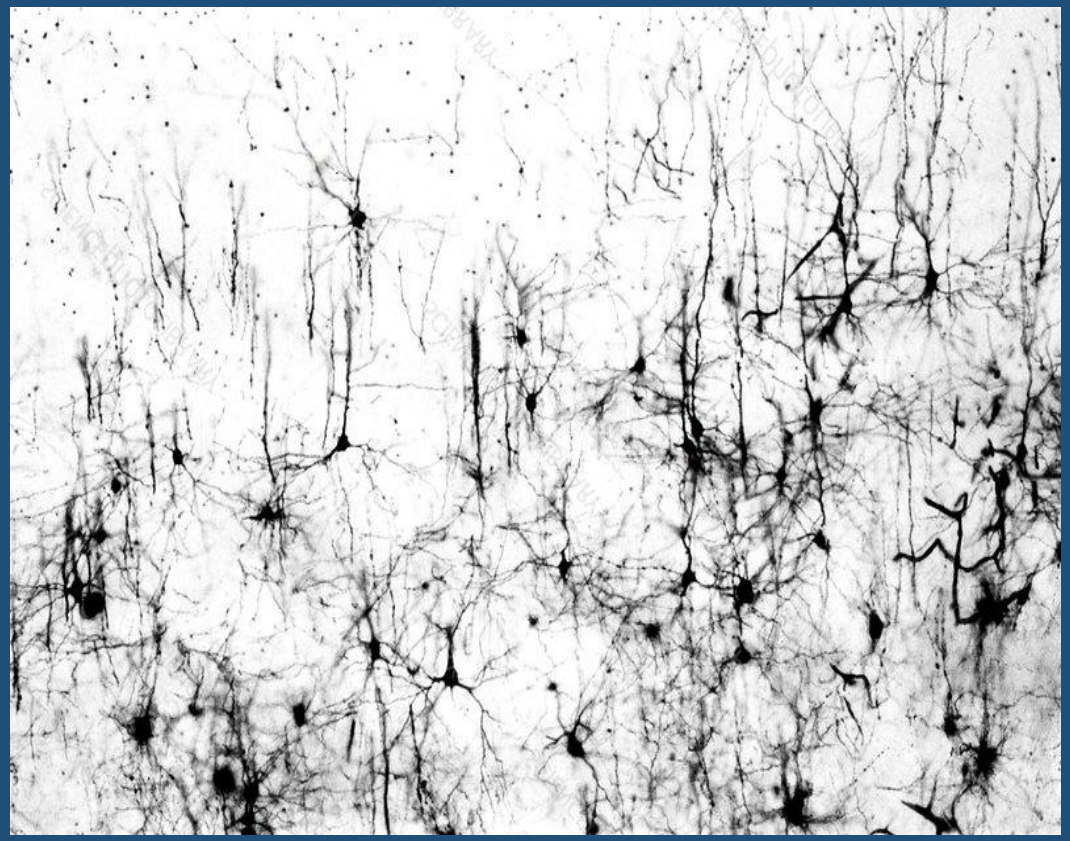
Brain Signal Generation

Origin of voltage changes within the cortex

The voltage changes within the cortex which are recorded on the scalp are primarily derived from the excitatory and inhibitory post-synaptic potentials **(EPSPs and IPSPs)** and *not* from the neuronal action potentials.

This is because, although action potentials are of much greater voltage, they are very brief, whereas the EPSPs and IPSPs are of much longer duration and are able to **summate** to create a signal capable of being detected on the surface of the scalp

The Role of Dendritic Palisades



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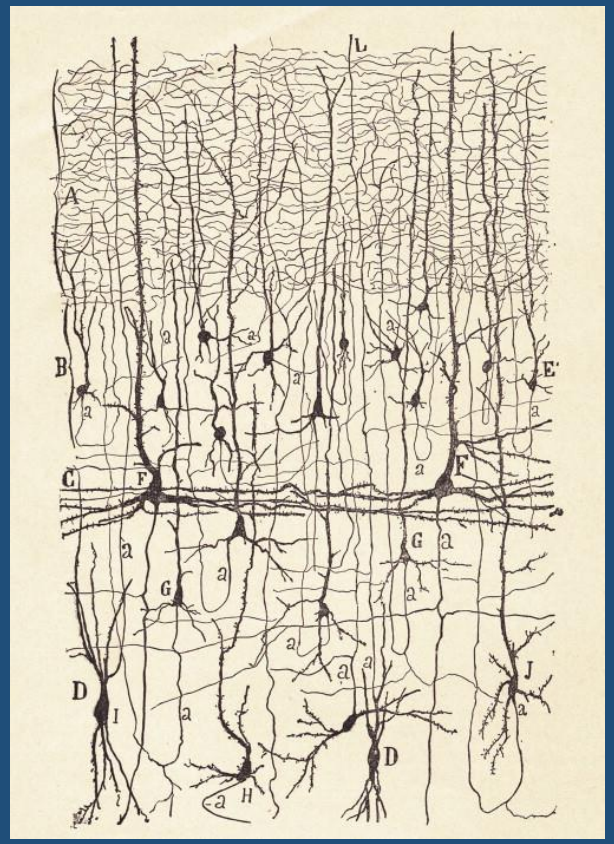

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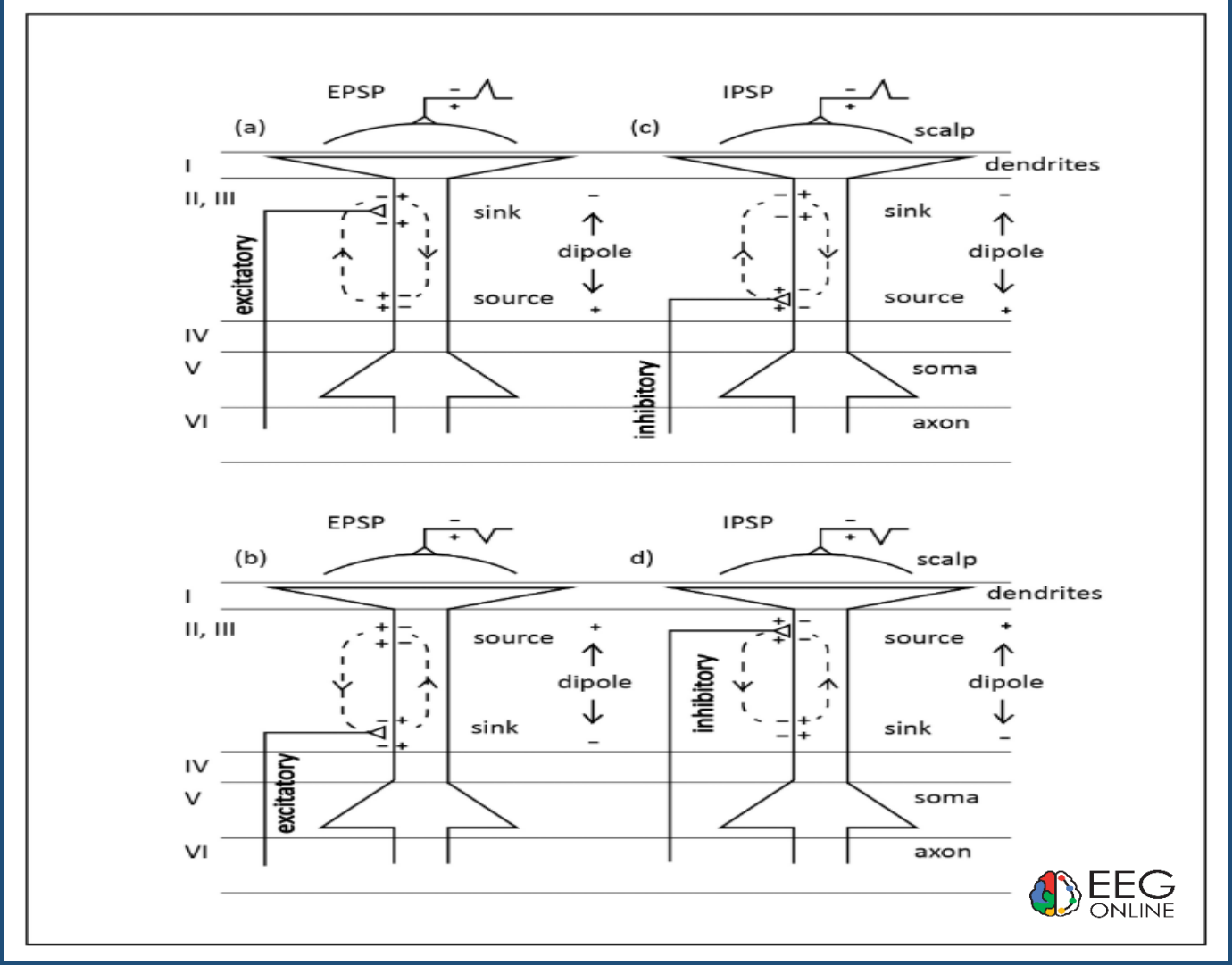
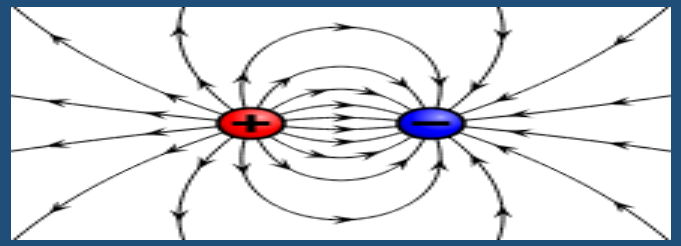
Summation of the voltages (EPSPs and IPSPs) is aided by the fact that the axons and dendrites of neurones are arranged in parallel in the cortex, referred to as dendritic palisades

Dipoles



It is mostly the EPSPs and IPSPs of the **large pyramidal neurones** with cell bodies in the cortical layers IV and V that generate the EEG

The positivity and negativity created can be viewed as a **dipole**, with current flow occurring between the poles.

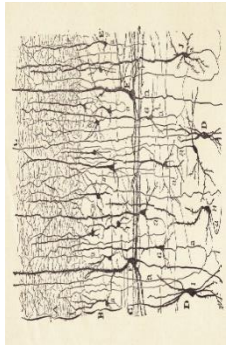


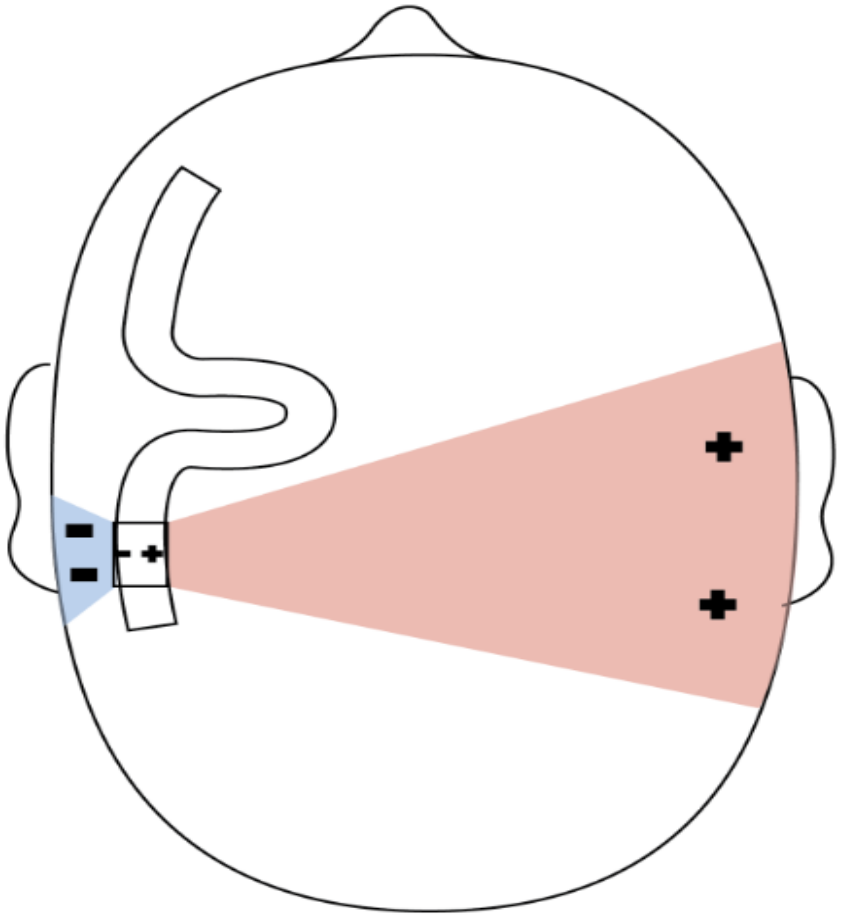
Orientation of the Cortical Layer & Cancellation Effect

Orientation of the cortical layer generating the electrical potentials is important

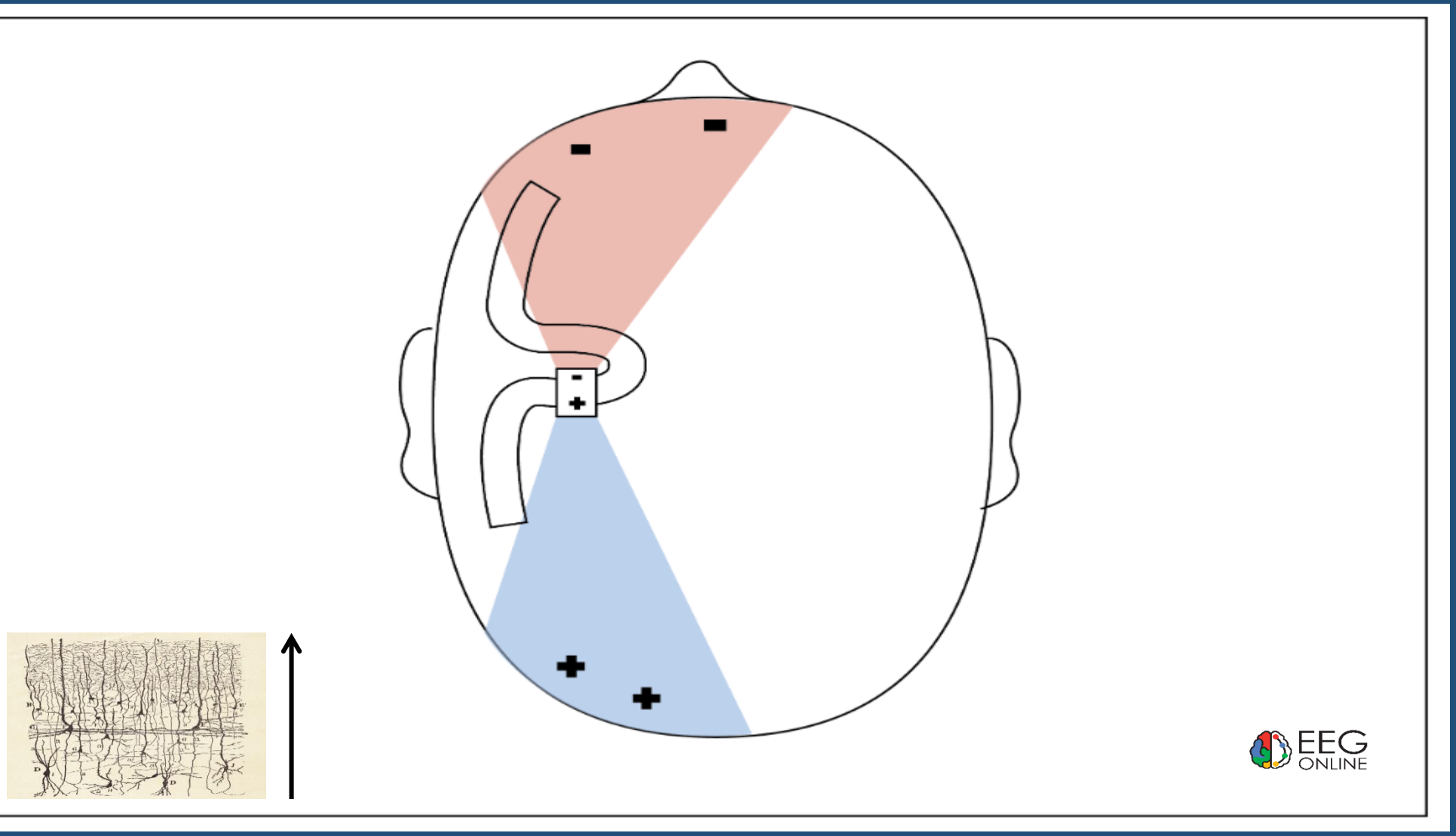
For example, the summation of electrical potentials generated by dendritic palisades in one region of cortex may **“cancel out”** potentials generated in another if these regions are in an opposite orientation.

Radial Dipole

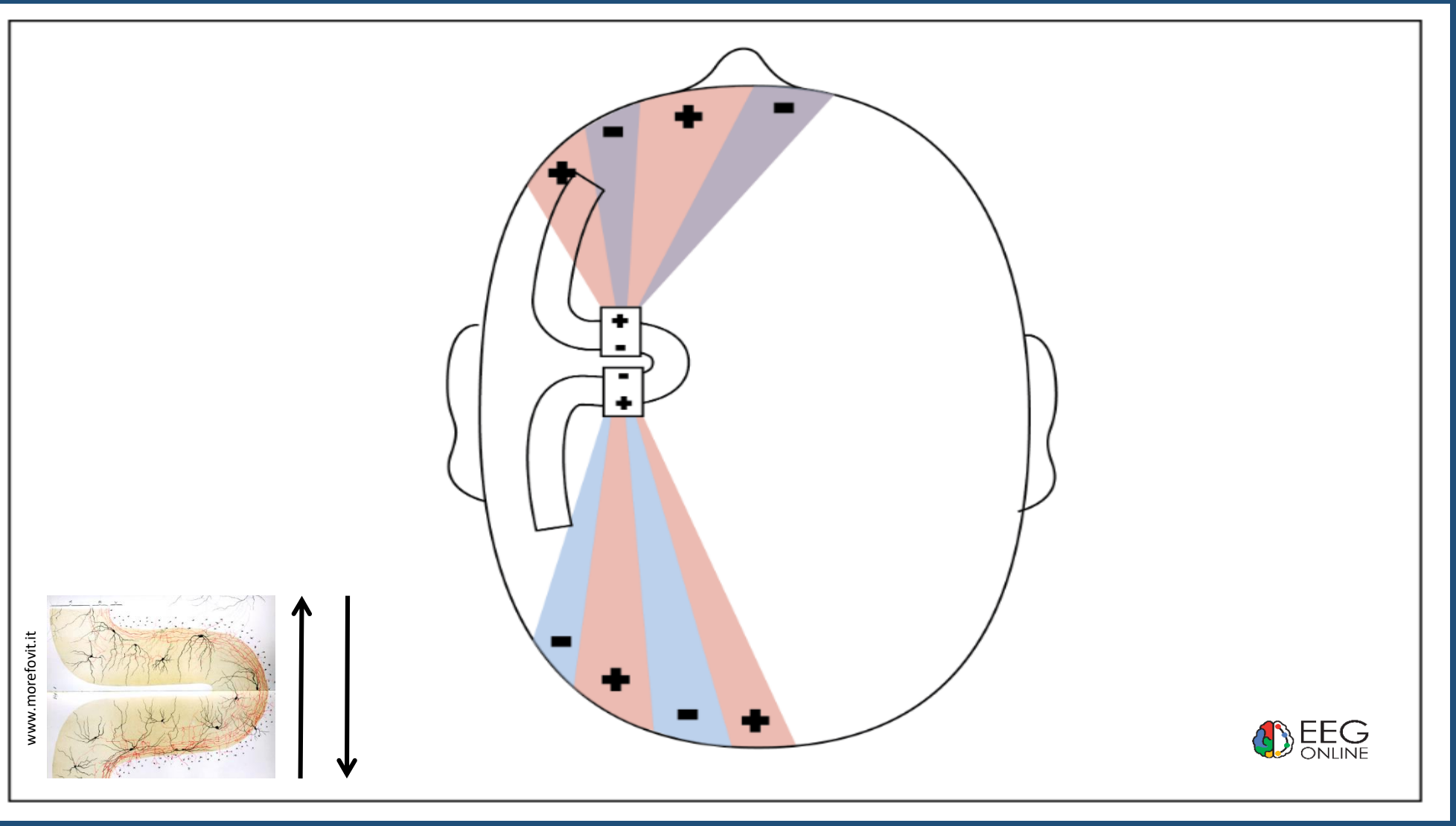




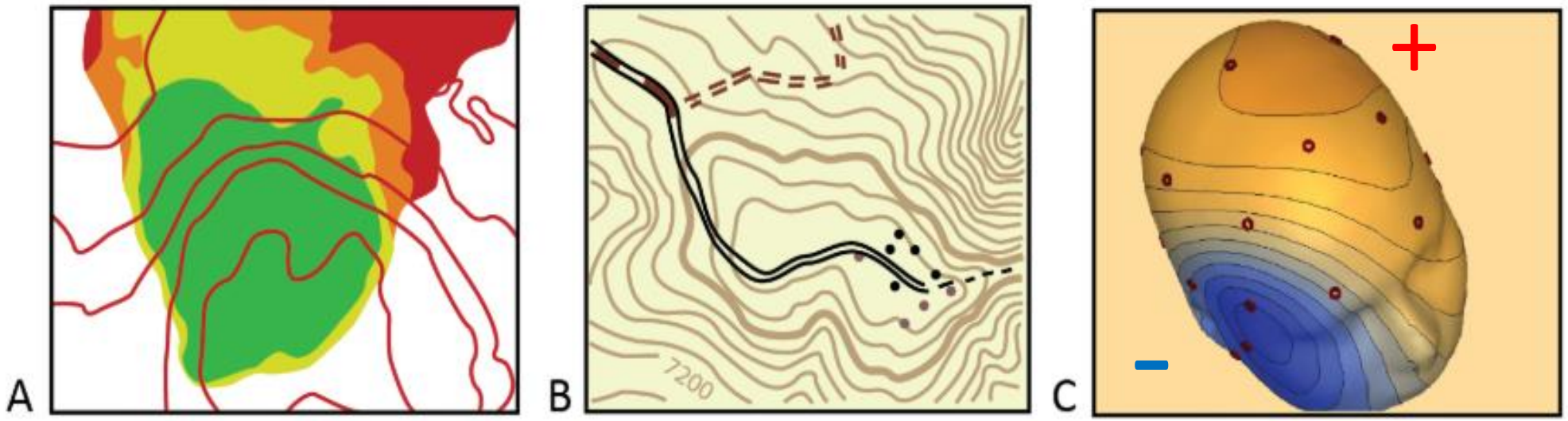
Tangential Dipole



Opposing Tangential Dipoles

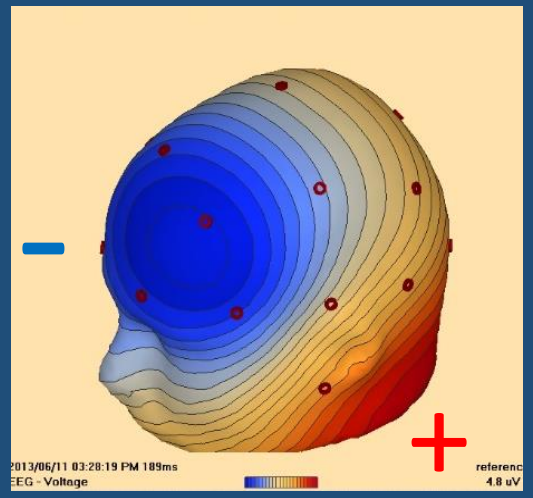


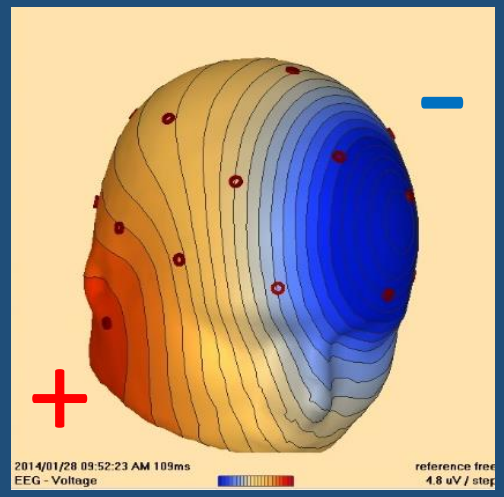
Electrical/Voltage Fields Represented as Contour Maps

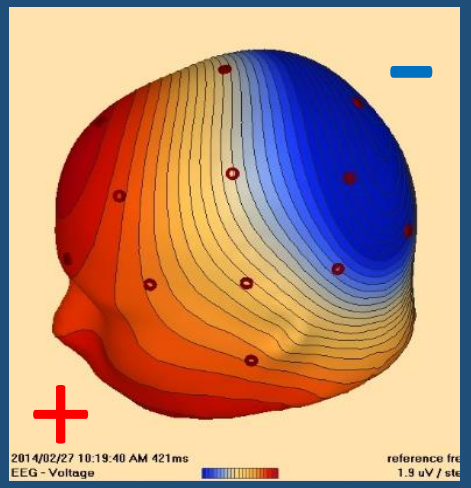


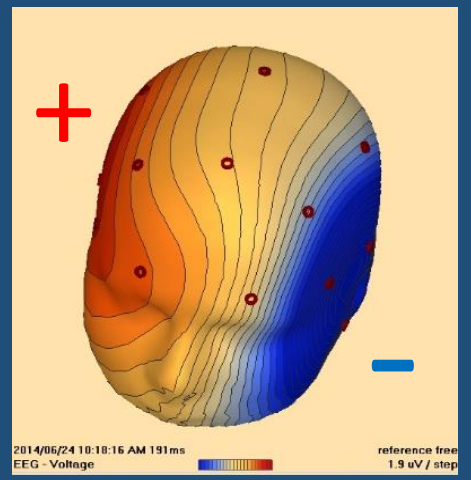
Contours representing pressure on a weather chart (A) and representing altitude on a topographical map (B). In (C), contours are used in a similar way to represent an electrical field on the surface of the brain.

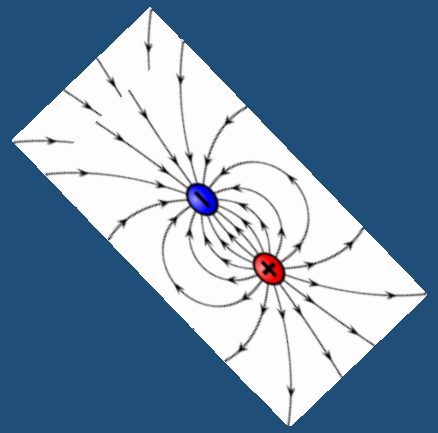
Electrical/Voltage Fields Represented as Contour Maps

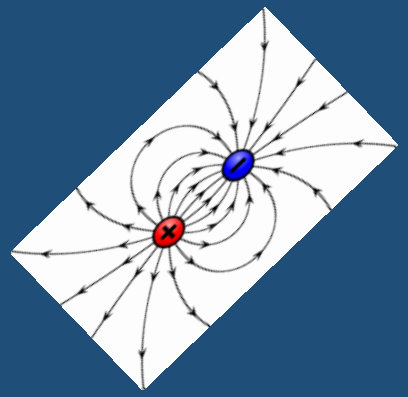


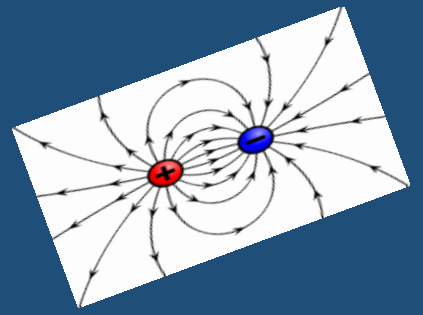


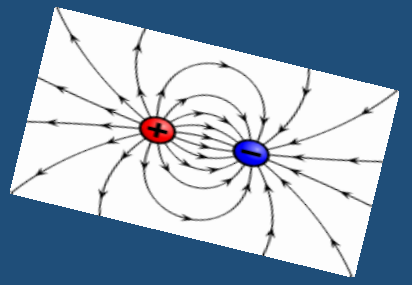




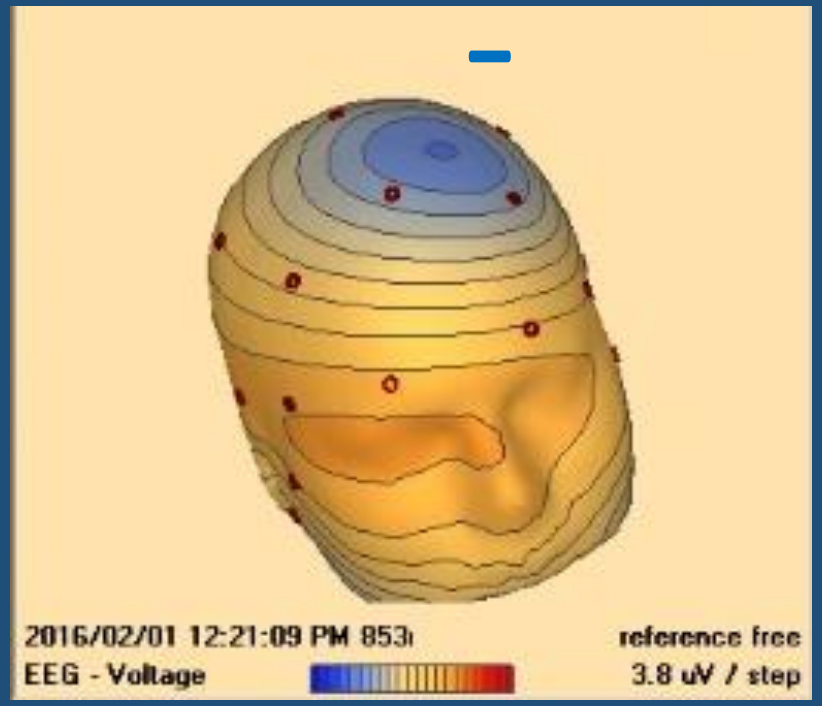


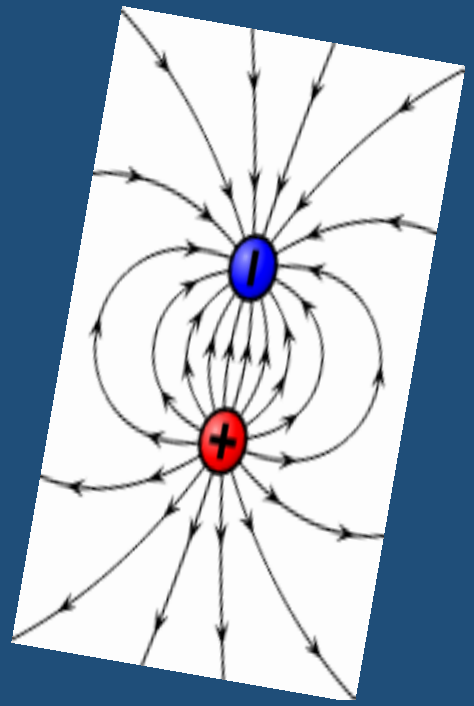






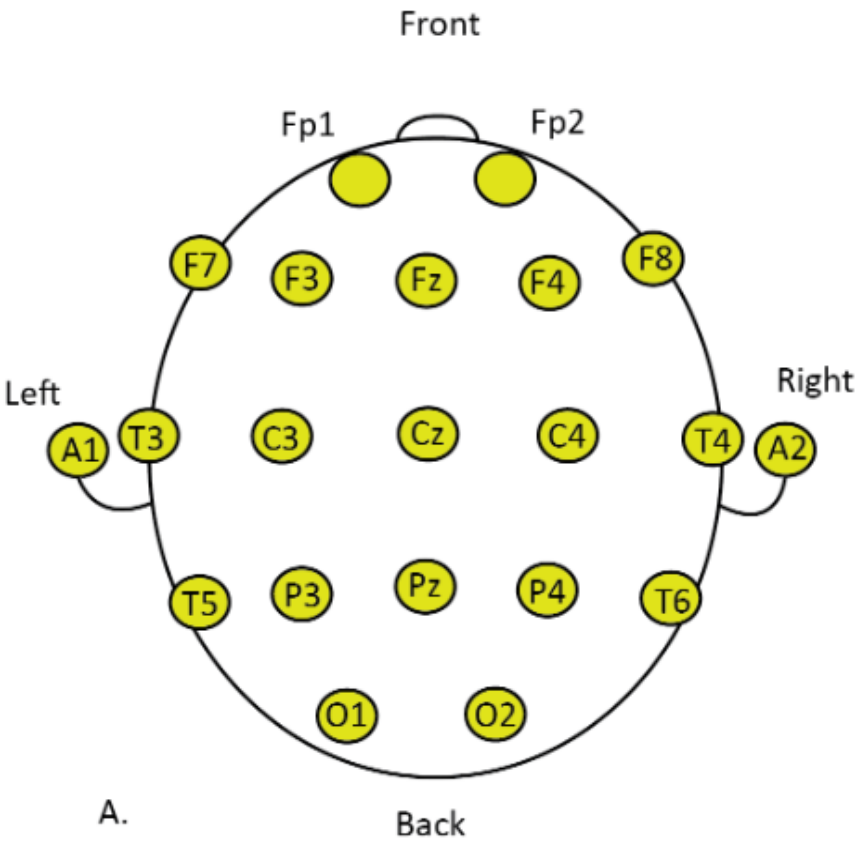
Electrical/Voltage Fields Represented as Contour Maps



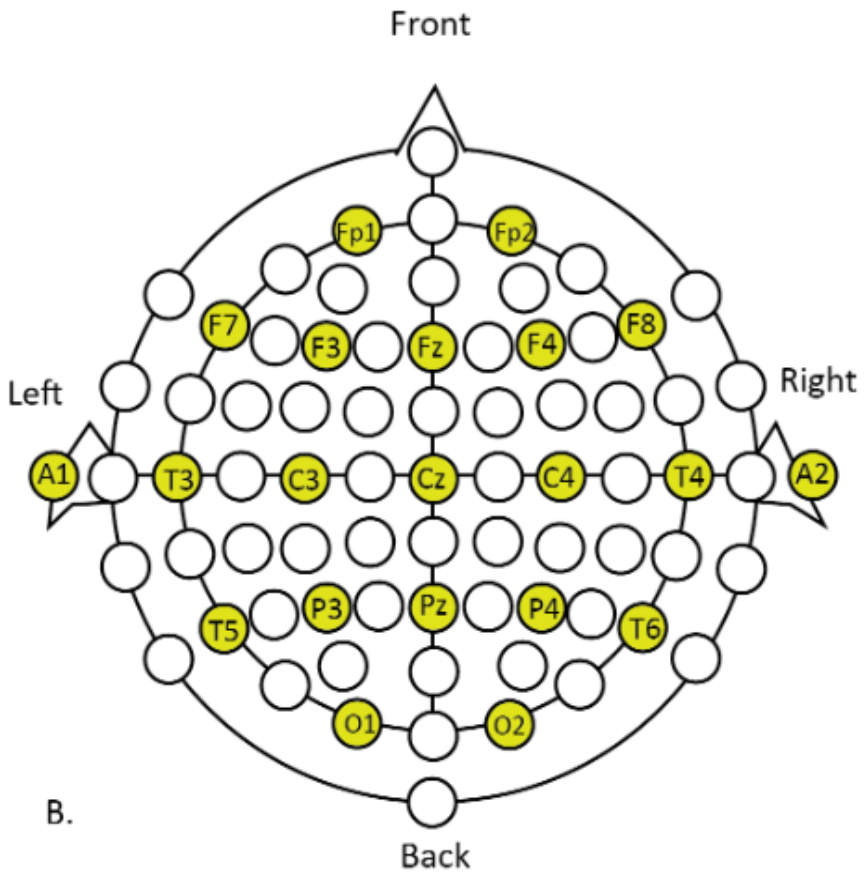


A dipole with its negative pole on the cortical surface may have its positive pole hidden deep within the brain itself and this may not be detectable by scalp EEG

Electrode Placement: 10-20 / 10-10 System

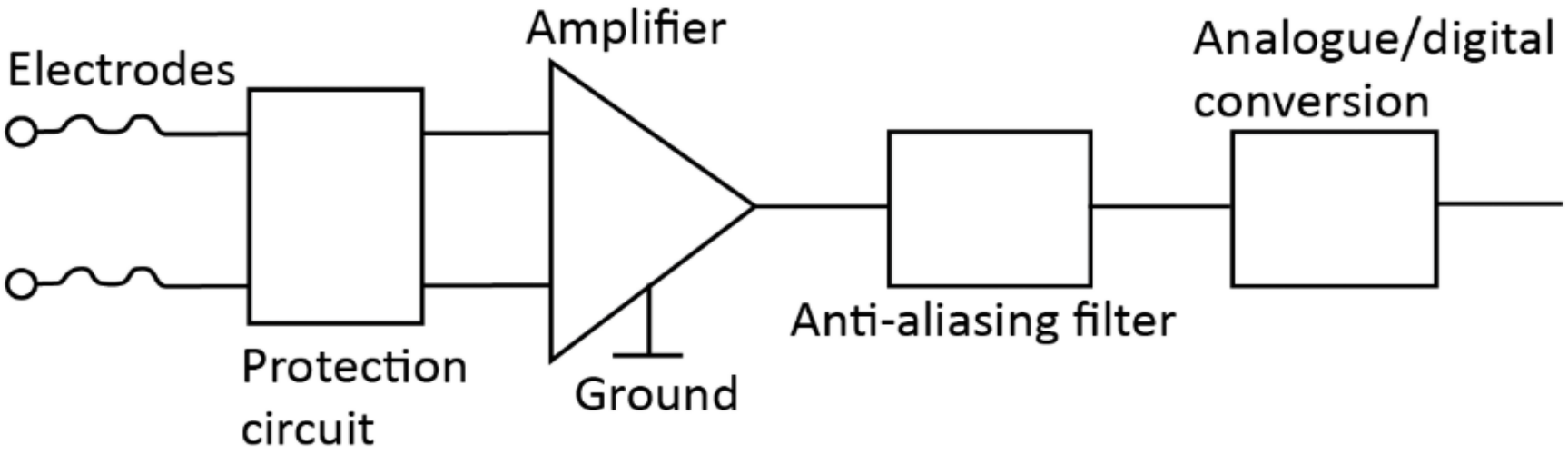


10-20 System



10-10 System

Signal Processing



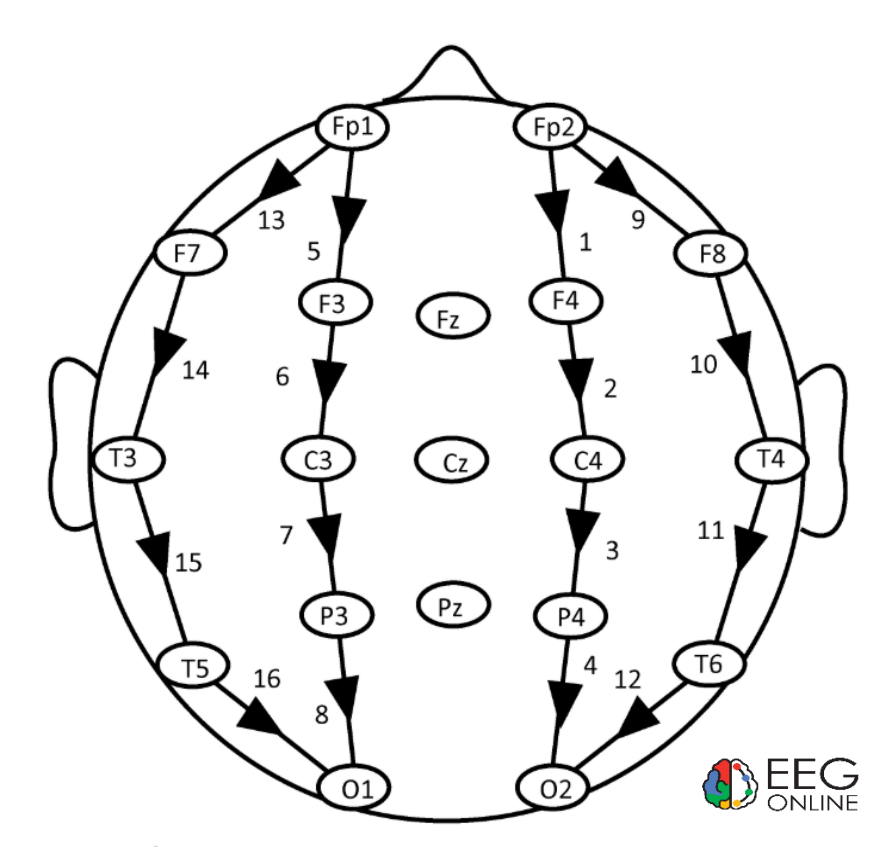
A simplified diagrammatic representation of the signal modification which occurs in an EEG machine.

Montages

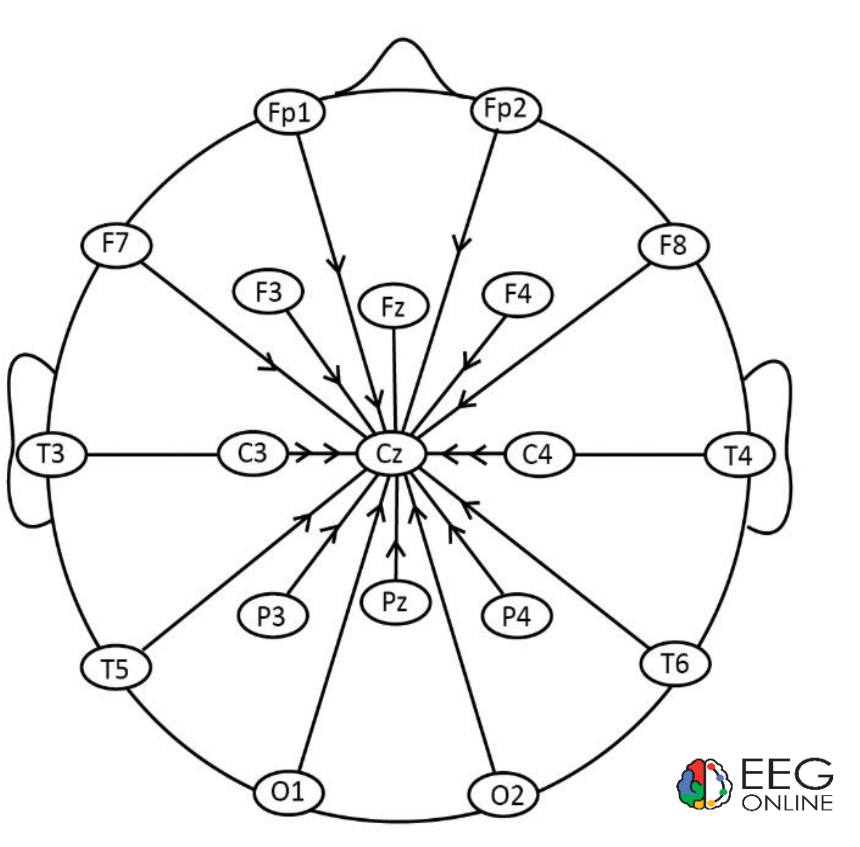
There are numerous ways one can connect scalp electrodes on the head and these different arrangements will result in different advantages and disadvantages. Each of these arrangements constitutes a **“montage”**.

For instance, there are certain montages, which are particularly useful when analysing temporal lobe abnormalities and others which are better at identifying, respectively, frontal, paracentral or occipital abnormalities.

Montages

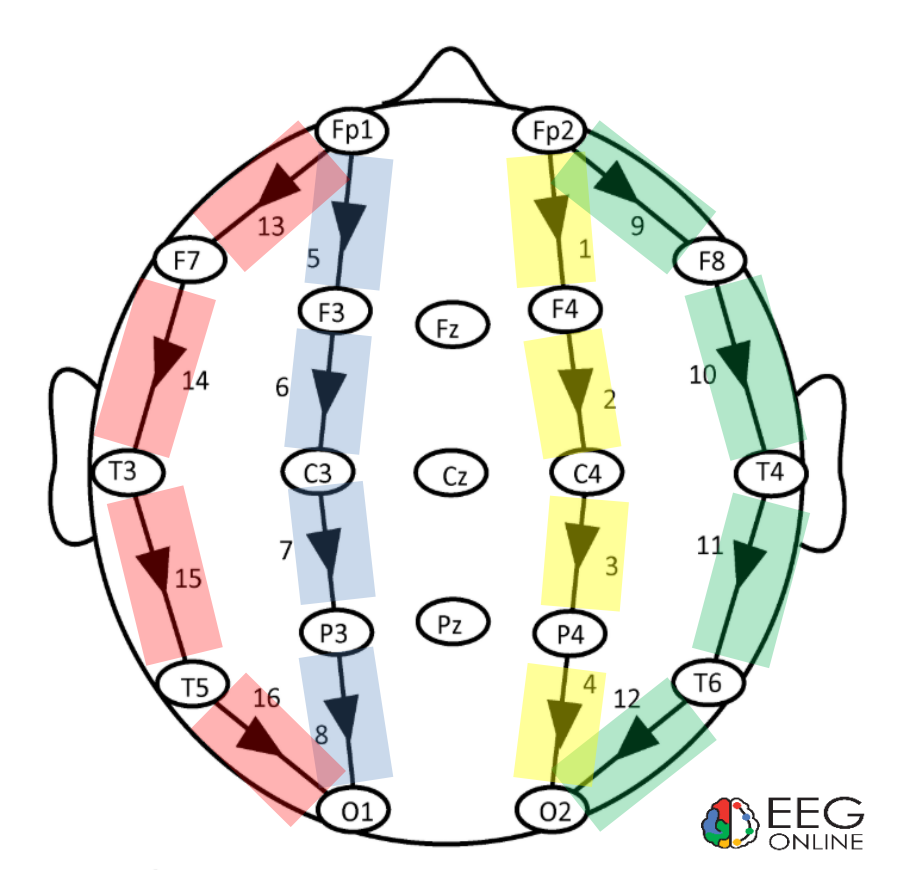


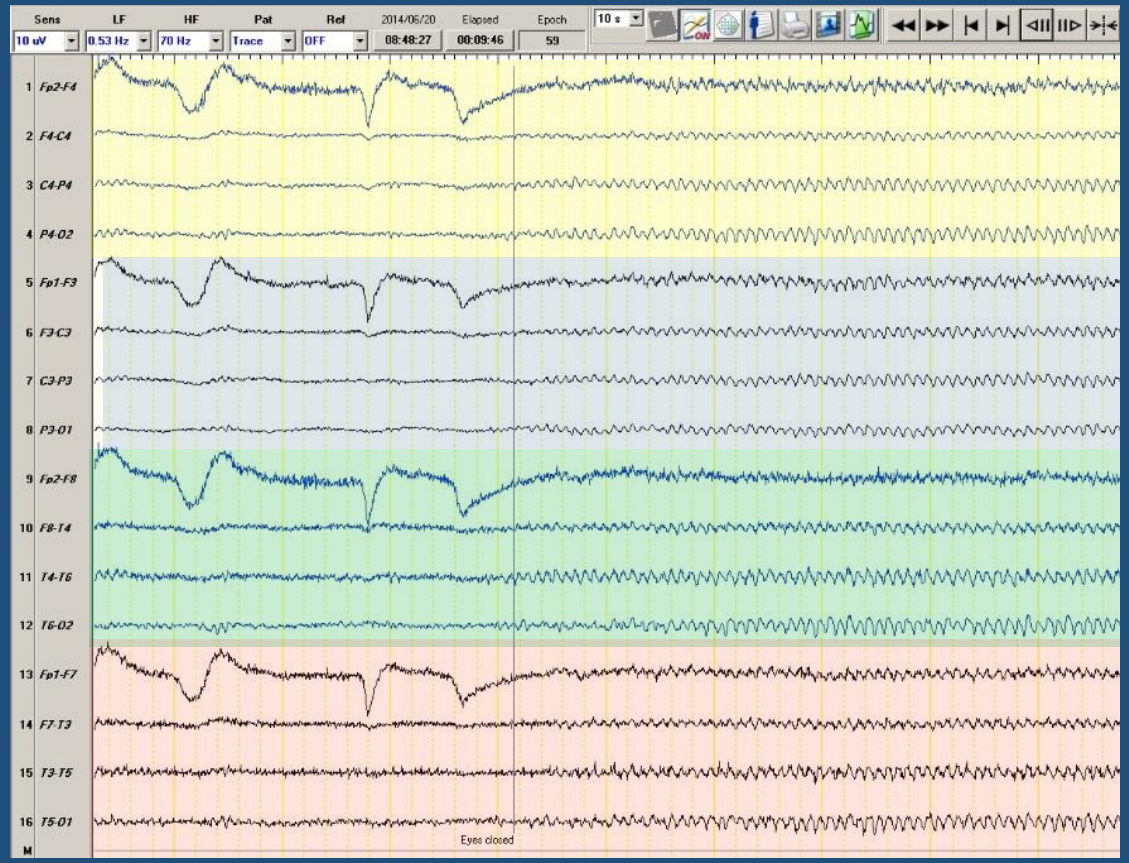
Longitudinal Bipolar Montage



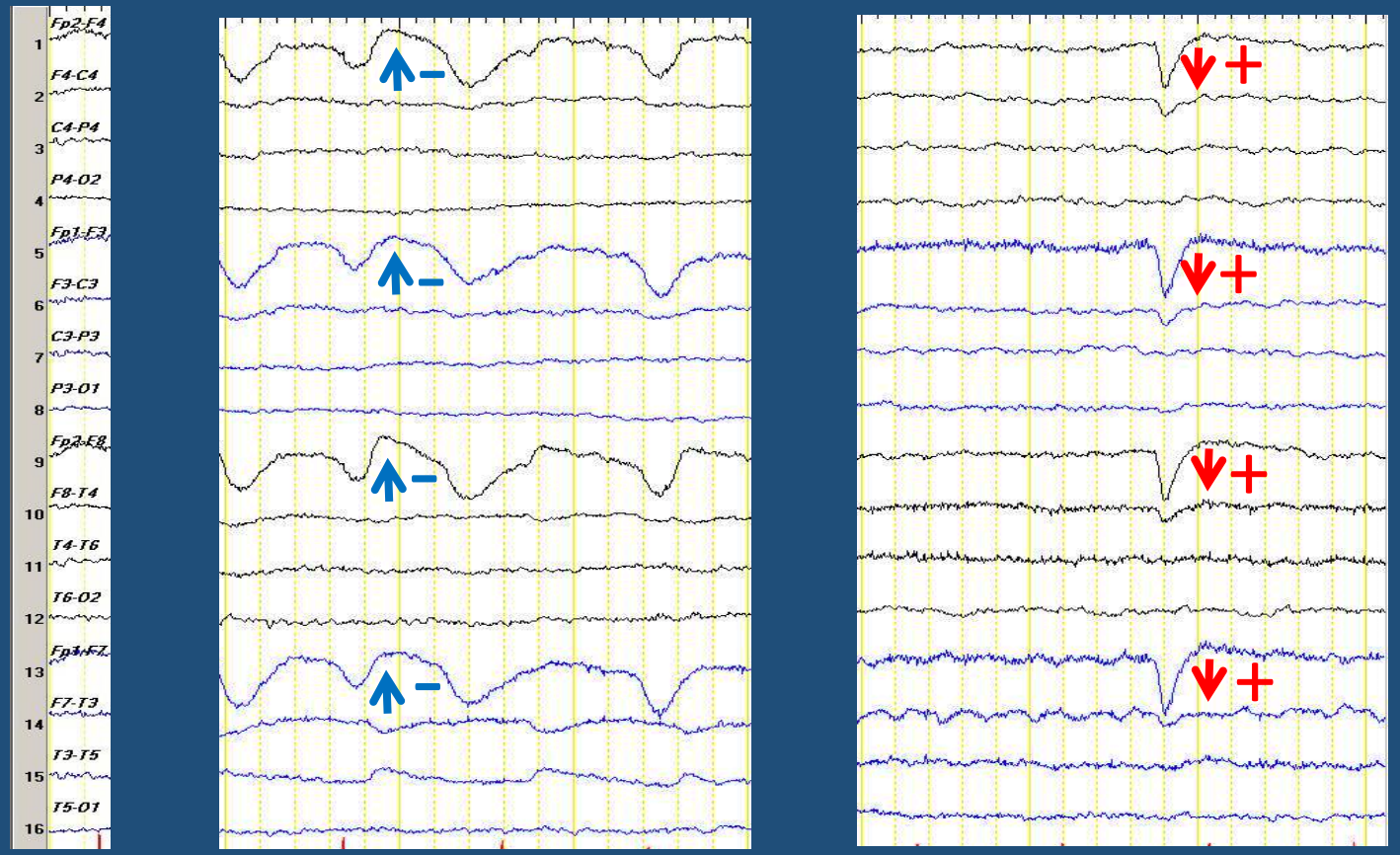
Cz-Common Reference Montage

In this presentation:
Only the Longitudinal Bipolar Montage will be used





Polarity Convention



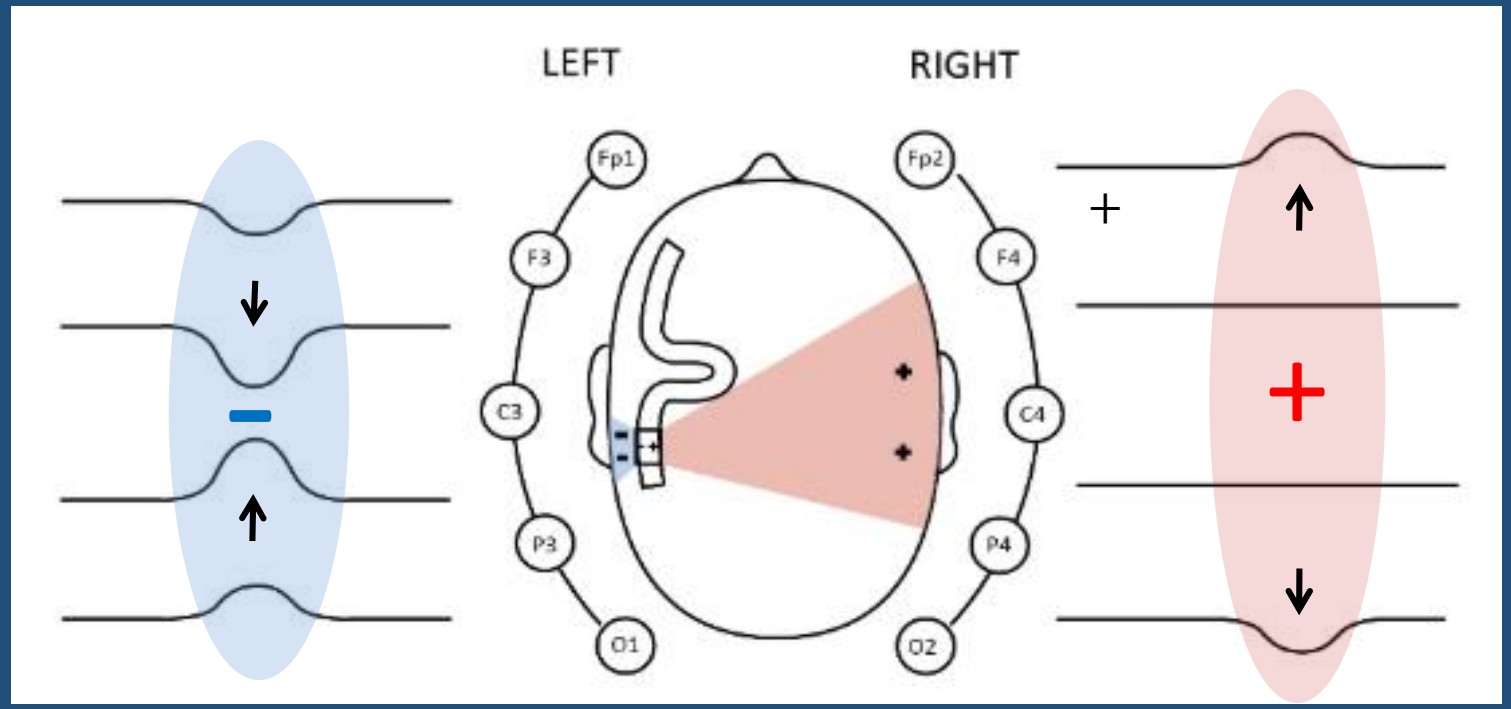
Eye movement Artefact A

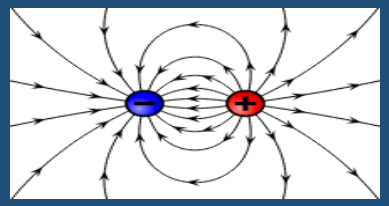
Eye movement Artefact B

By convention:

- upward deflections are negative
- downward deflections are positive

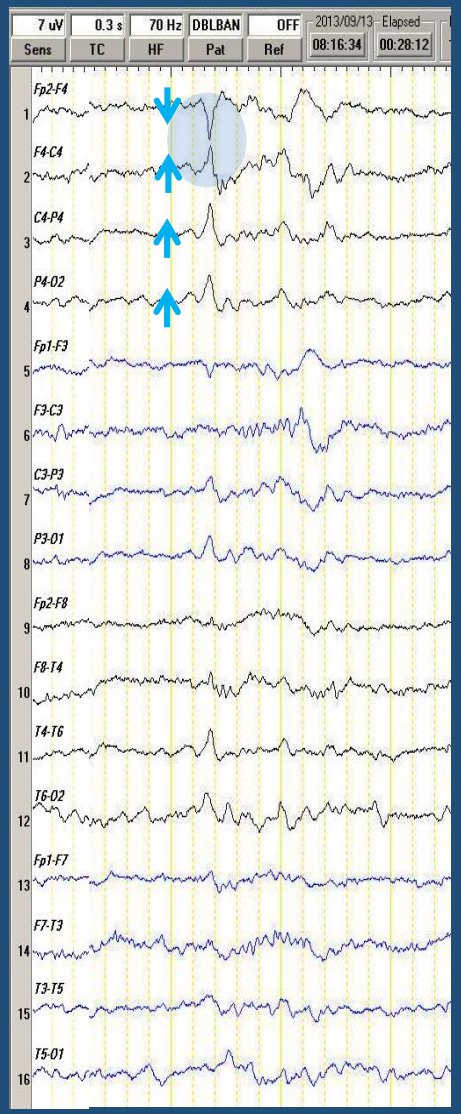
Longitudinal Bipolar Montage: **Radial Dipole**

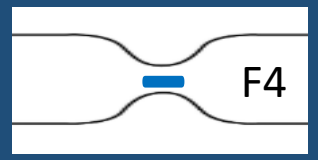




Note how the negative pole of the dipole on this montage results in deflections which move together while the more widely distributed positive pole results in deflections which move apart.

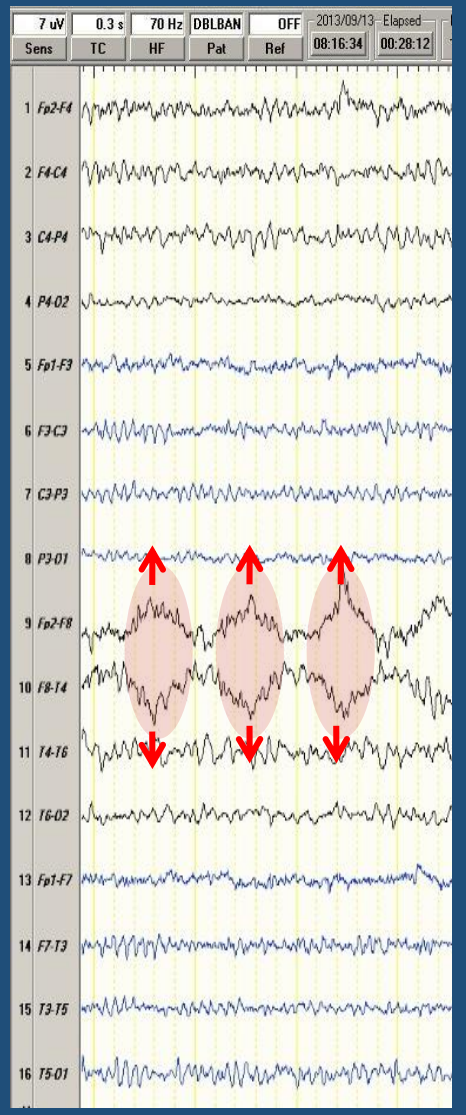
Foci of Negative and Positive Charge on the Bipolar Montage

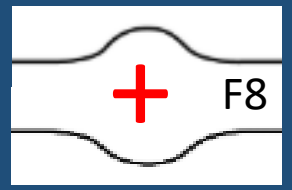




A

Asymmetrical V-Wave





B

F8 Electrode Artefact

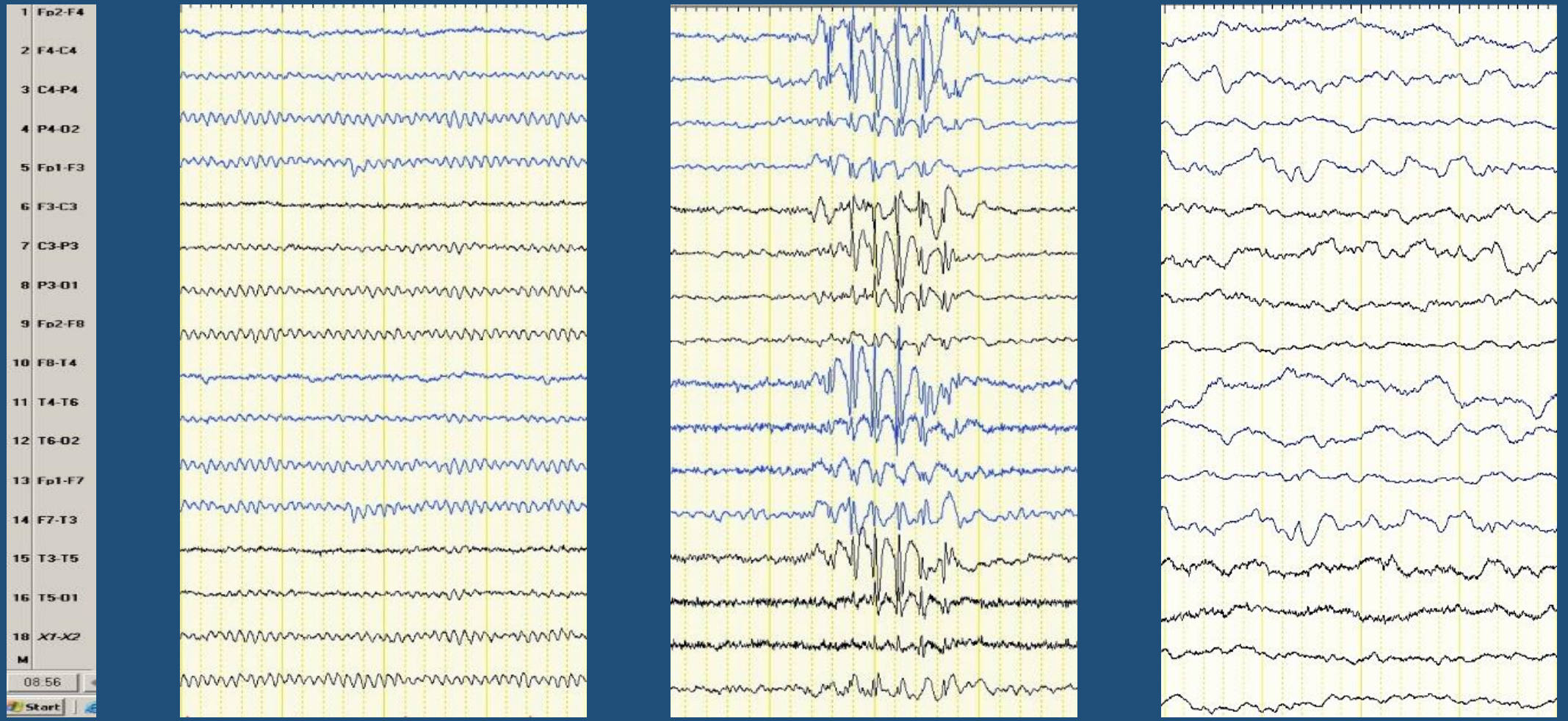
Most clinically relevant waveforms have a predominantly **negative polarity** at the surface of the brain and, consequently, most montages are designed to localize these regions of high negativity.

Waveform Pattern Recognition and Interpretation

In order to successfully analyse and interpret the EEG, the reader must adopt an **unbiased and rigorously systematic approach**.

It is best to analyse an EEG recording "BLIND"

1. Take note of the subjects age and state of vigilance
2. Take note of settings (sensitivity, paper speed and filtering)
3. Identify any artefacts
4. Beware of bias
5. Identify and assess the predominant background rhythm (posterior dominant rhythm)
6. Identify and assess any additional rhythms
7. Identify and assess any other waveforms of interest
8. Correlate your EEG findings with the reason for referral and clinical features



Normal

Epileptiform

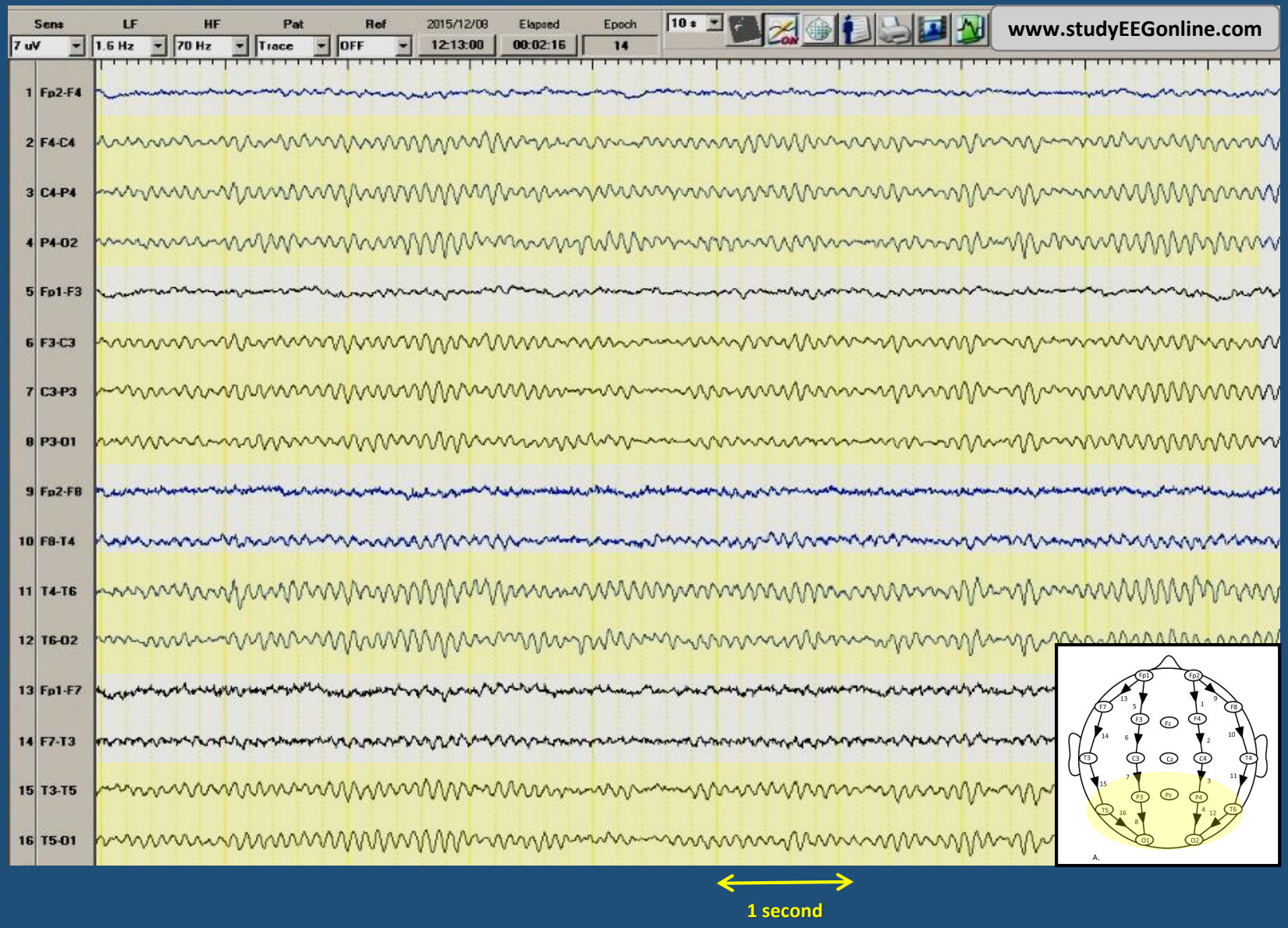
Slowing

The EEG of Normal Wakefulness

Background / Posterior Dominant Rhythm (PDR)

*Background rhythms of normal wakeful adult subjects are assessed in the **posterior regions of the head** and represent the summation of different cortically-generated rhythms of varying frequencies. These background rhythms are referred to as the **Posterior Dominant Rhythm** (PDR) and typically run at a frequency of between **8Hz and 14 Hz** which is referred to as the **Alpha Range***

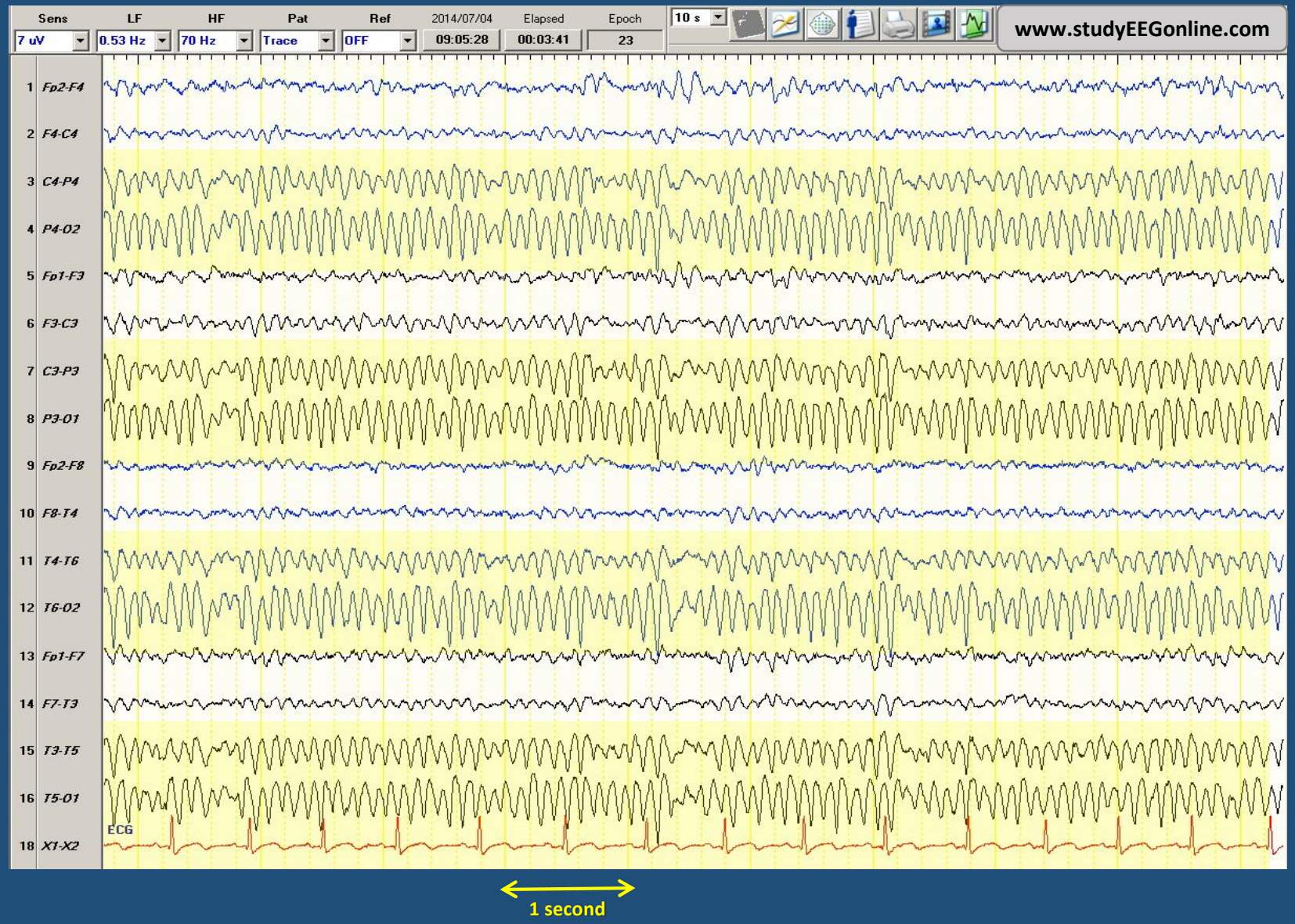
Normal Wakefulness: **Alpha Posterior Dominant Rhythm (PDR)**



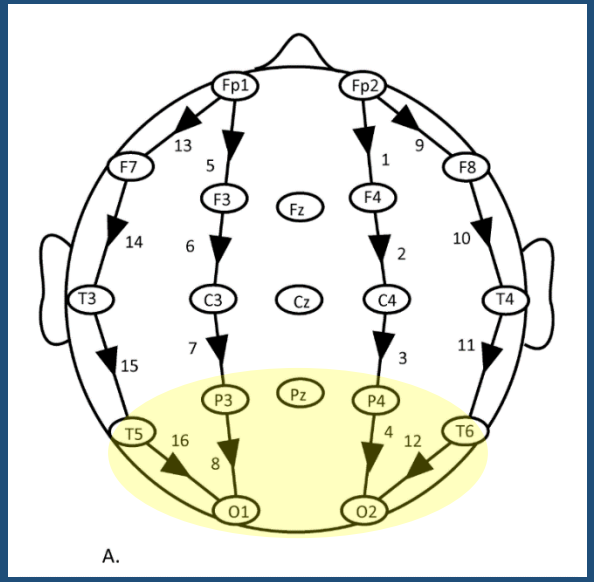
This is an example of a **Normal Posterior Dominant Rhythm**.

Note that it is **symmetrical, rhythmical, sinsusoidal**, largely confined to the posterior channels and runs in the **alpha range (i.e. 8-14 Hz)**

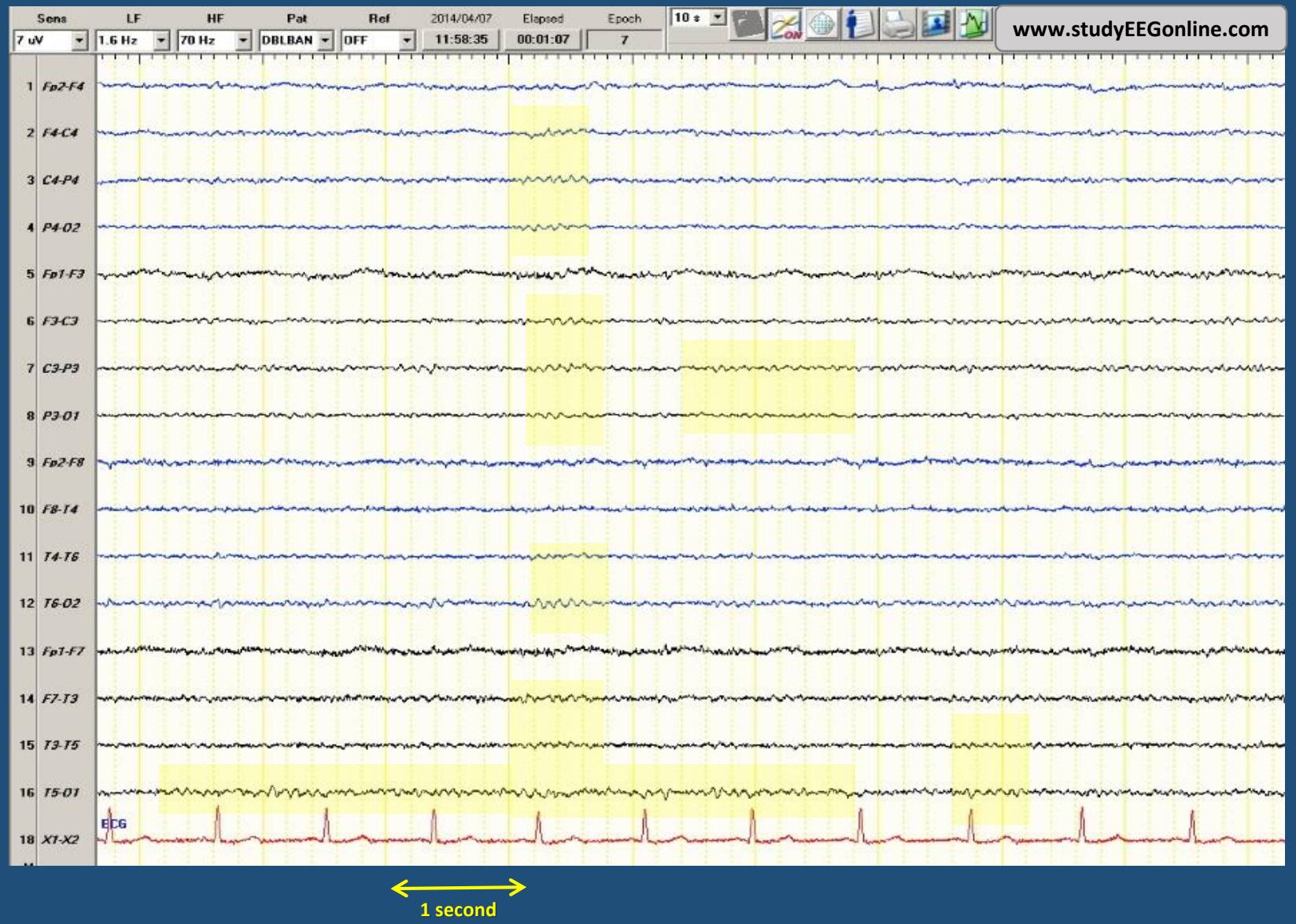
Normal Wakefulness: **Normal Alpha PDR (High Amplitude)**



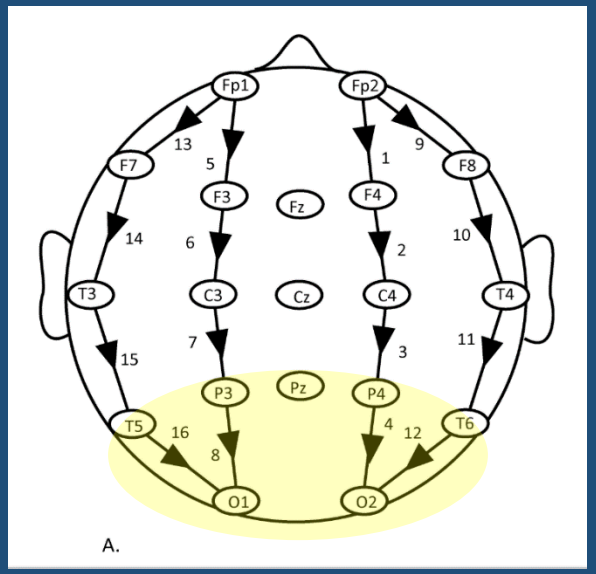
Normal, high amplitude 9-10 Hz alpha PDR



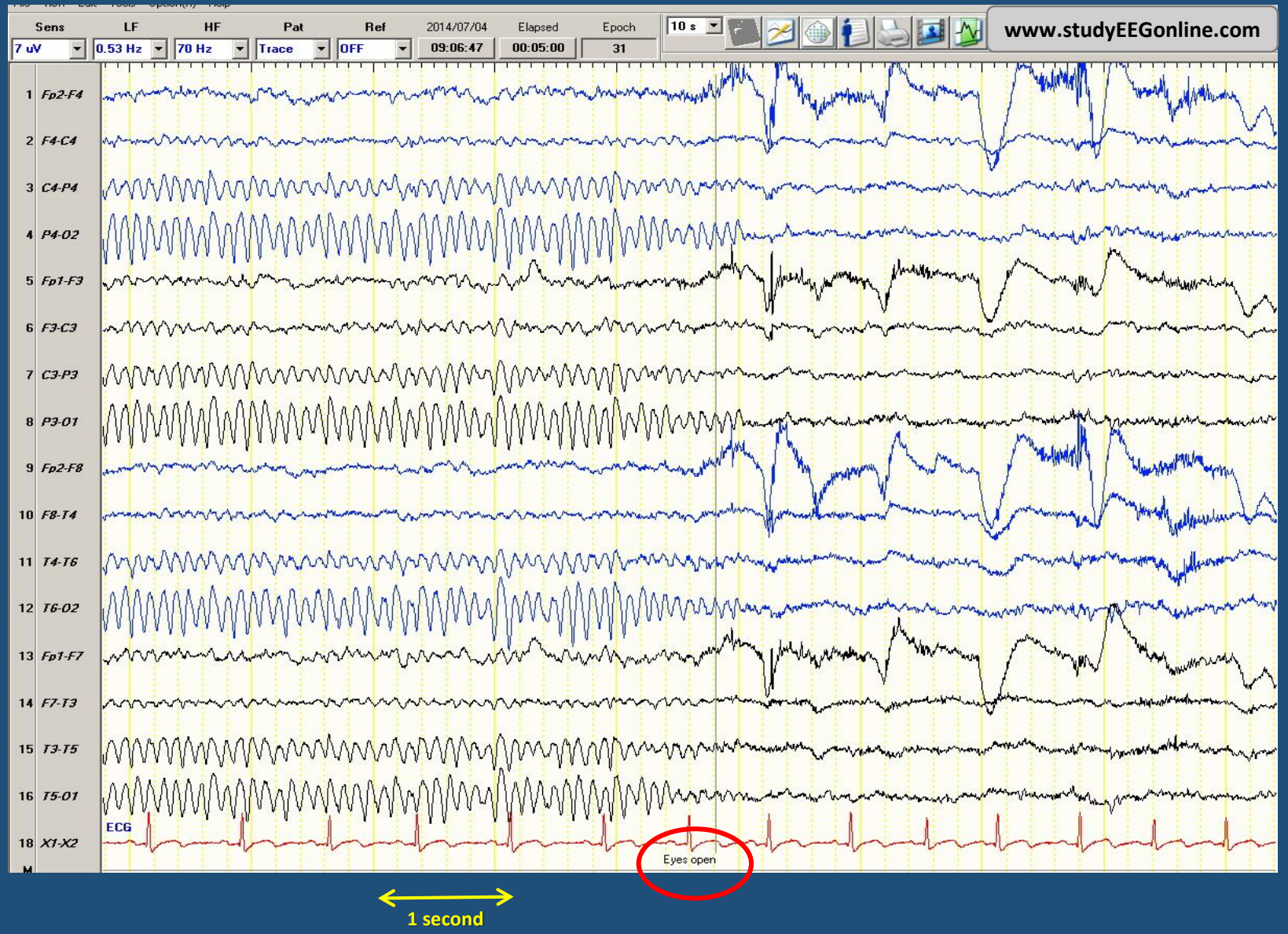
Normal Wakefulness: **Normal Alpha PDR (Low Amplitude)**



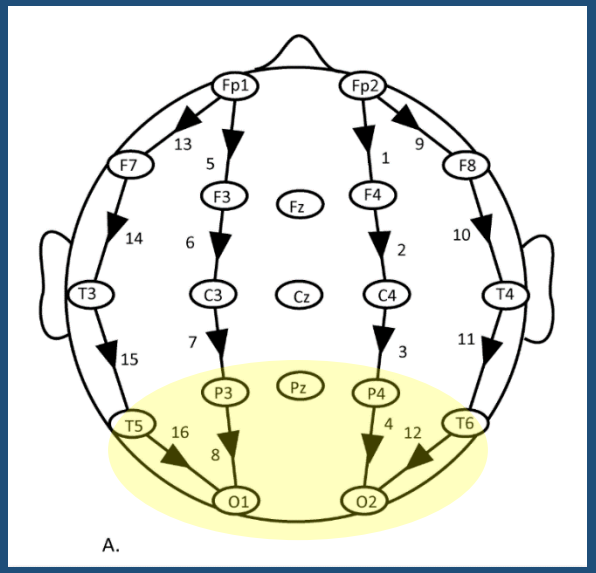
Normal scanty, low amplitude 10-11 Hz alpha PDR



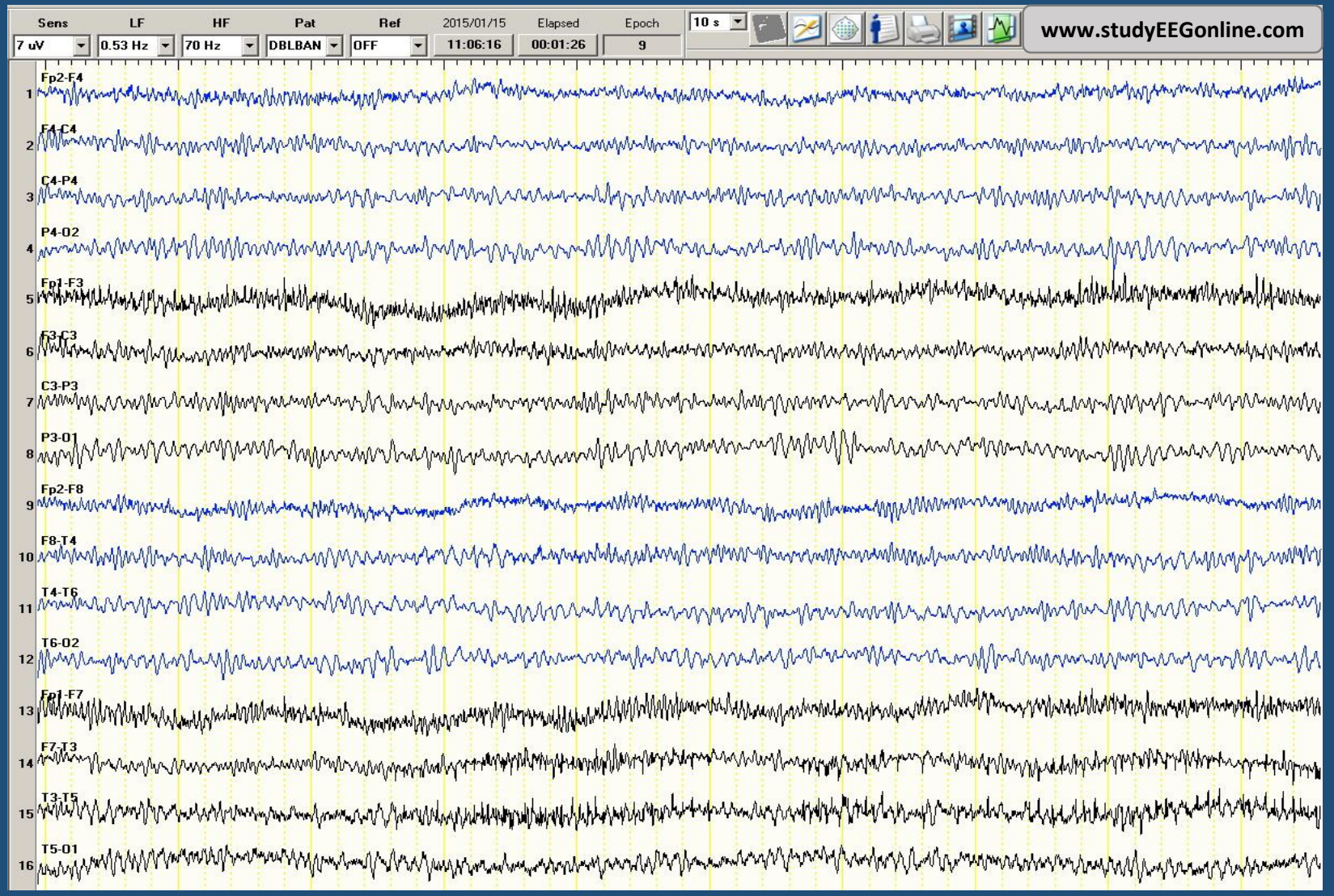
Normal Wakefulness: **Reactive Alpha PDR**



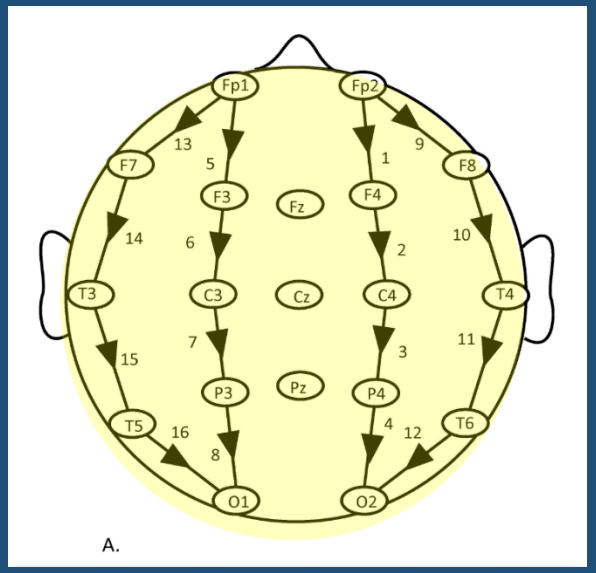
Normal reactive 9-10Hz alpha PDR, which attenuates on eye opening. Note eye blink artefacts in the second half of this epoch



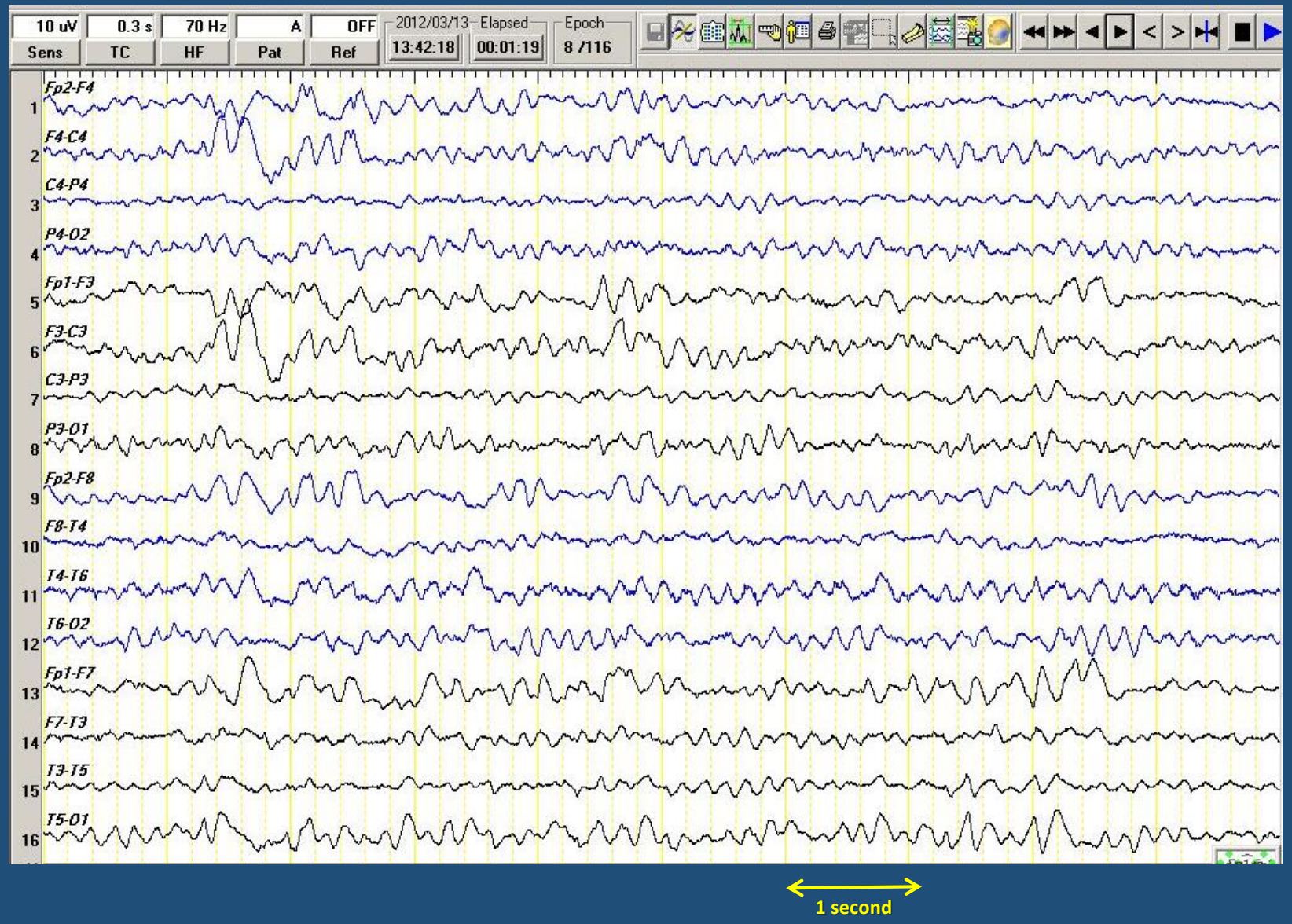
Normal Wakefulness: **Beta PDR**



Normal beta PDR running faster than 14 Hz.



Slowing of the PDR: Normal Drowsiness vs. Abnormal



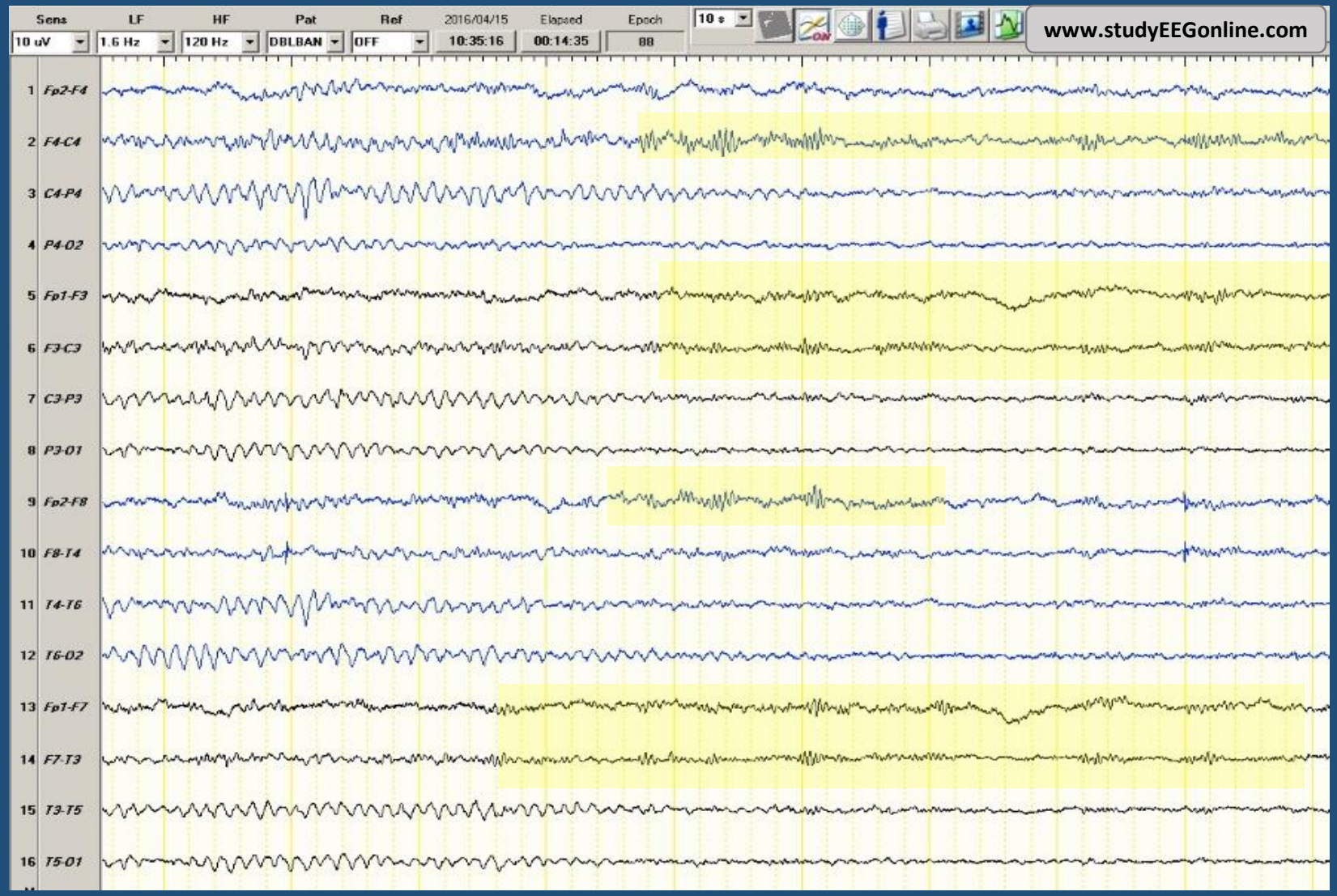
A PDR running slower than 8 Hz (i.e. in the theta or delta range) may be due to normal drowsiness or it may be pathological.

It is therefore very important to recognise the EEG features of drowsiness

Normal Drowsiness / Somnolence

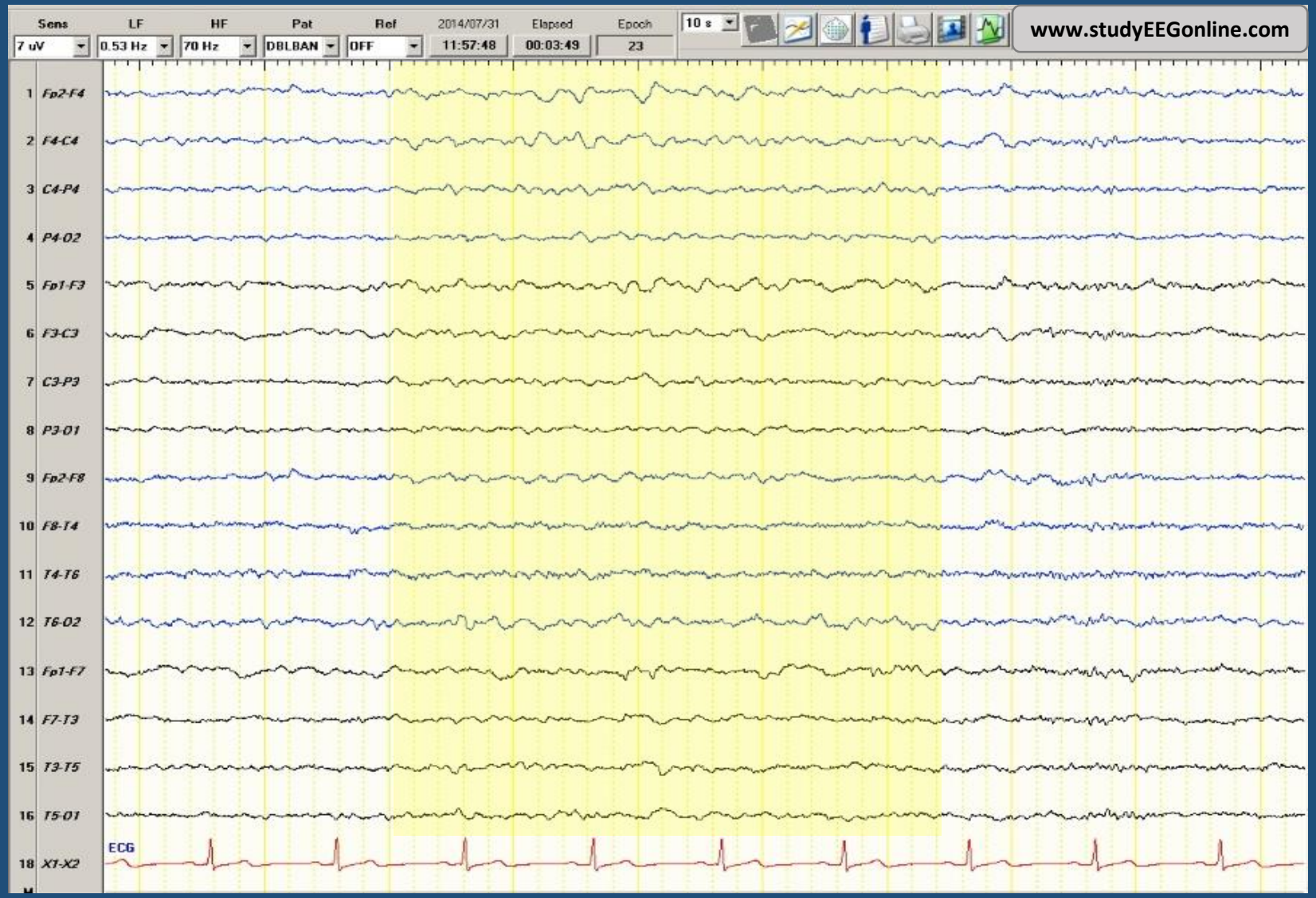
With the onset of drowsiness and subsequent progression to somnolence in normal adults, a number of characteristic changes occur sequentially in the electroencephalogram. These include alterations in, and eventual loss of, the alpha rhythm, the emergence of generalised slow waves, and the appearance of specific drowsiness- or sleep-associated electrographic waveforms such as sleep spindles, V-waves and K-complexes. Many of these waveforms may be mistaken for abnormalities.

Drowsiness: **Drop-Out of Alpha**

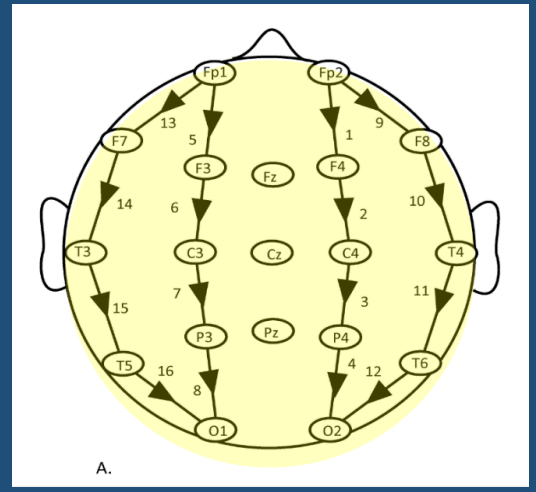


Typically, as a subject becomes drowsy, there is dropout of the alpha PDR which is replaced by generalised beta frequencies.

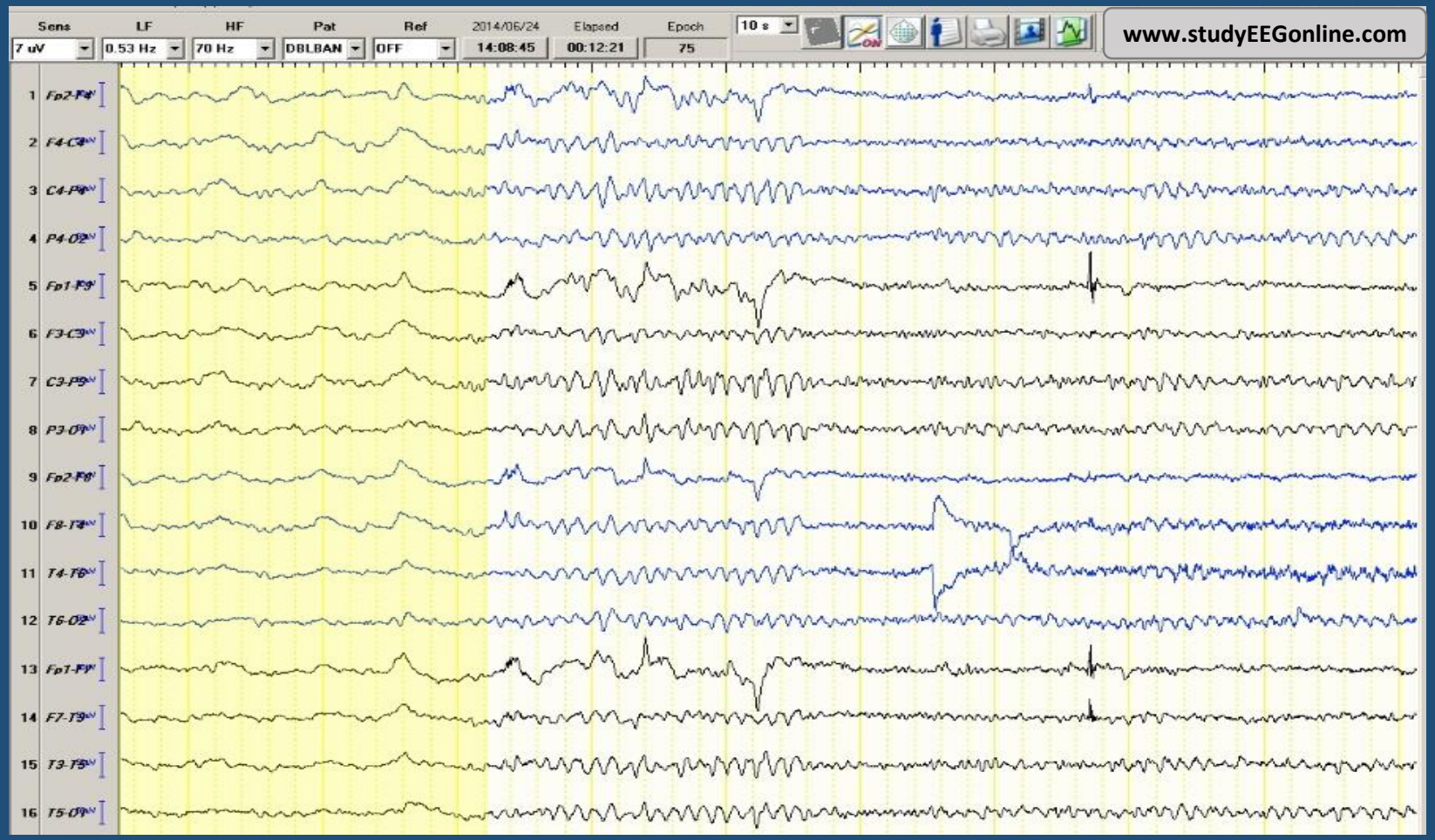
Drowsiness: **Theta**



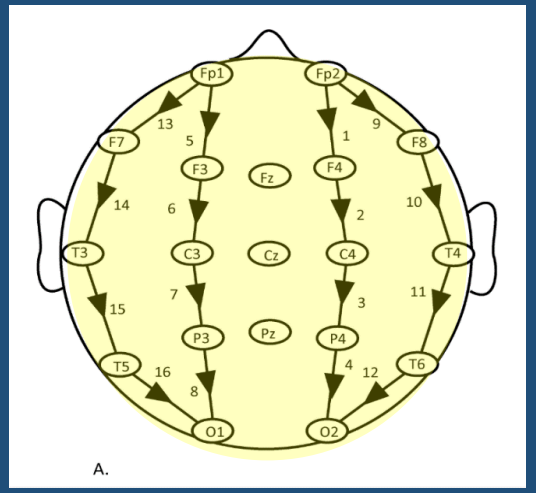
This is followed by emergence of generalised slowing in the theta range (5-7 Hz)



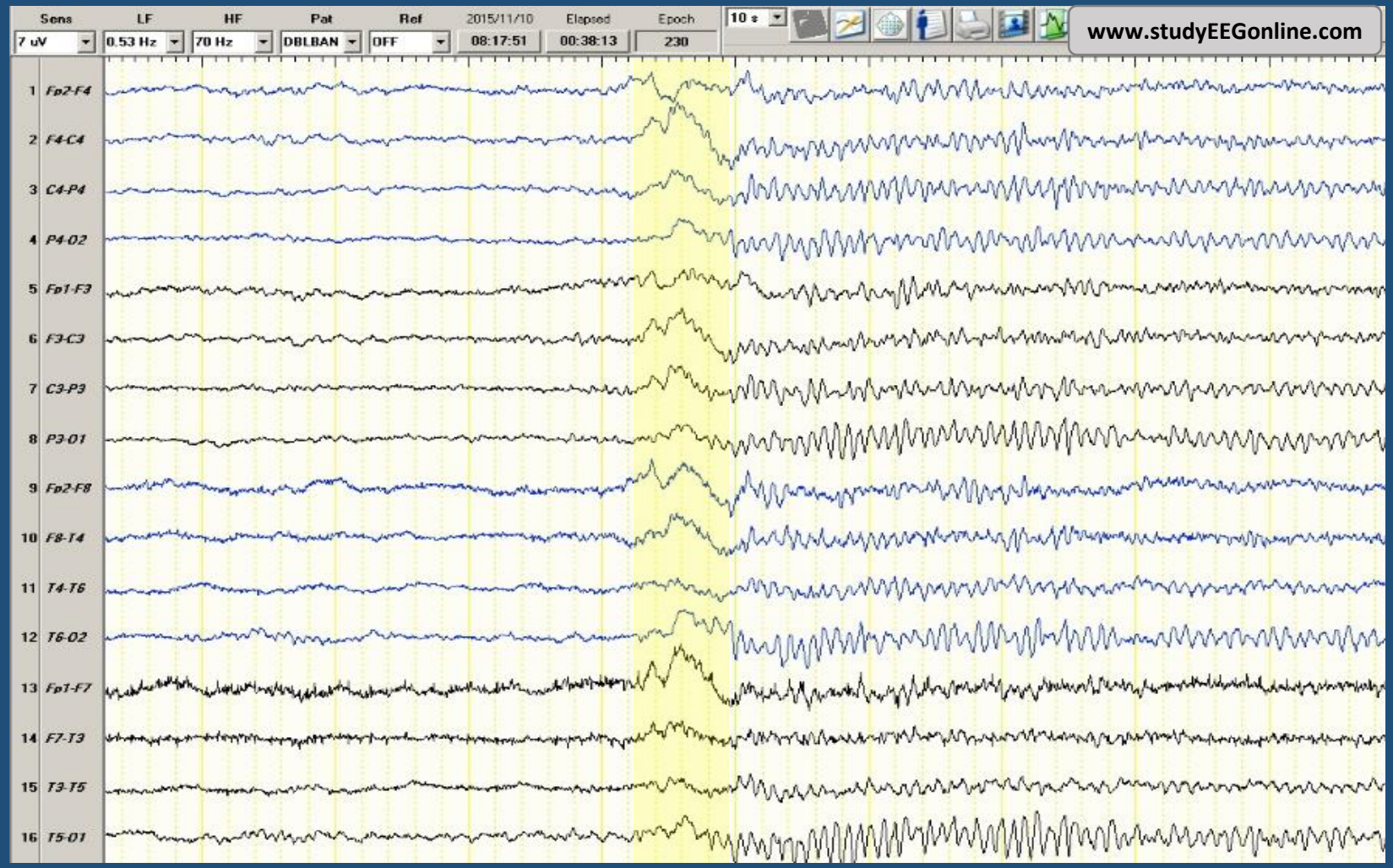
Drowsiness: **Delta and Arousal**



The theta, in turn, is followed by slowing in the delta range (i.e. < 5Hz) consistent with slow wave sleep.

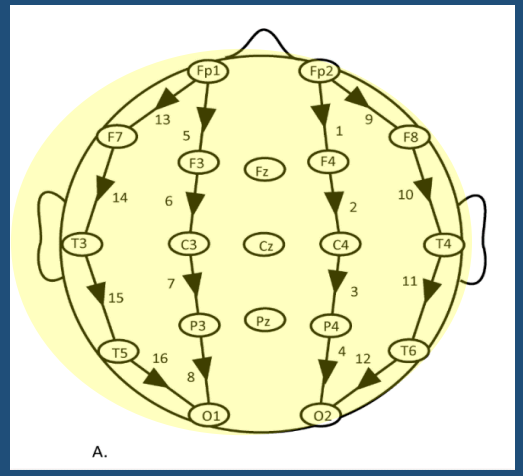


Arousal

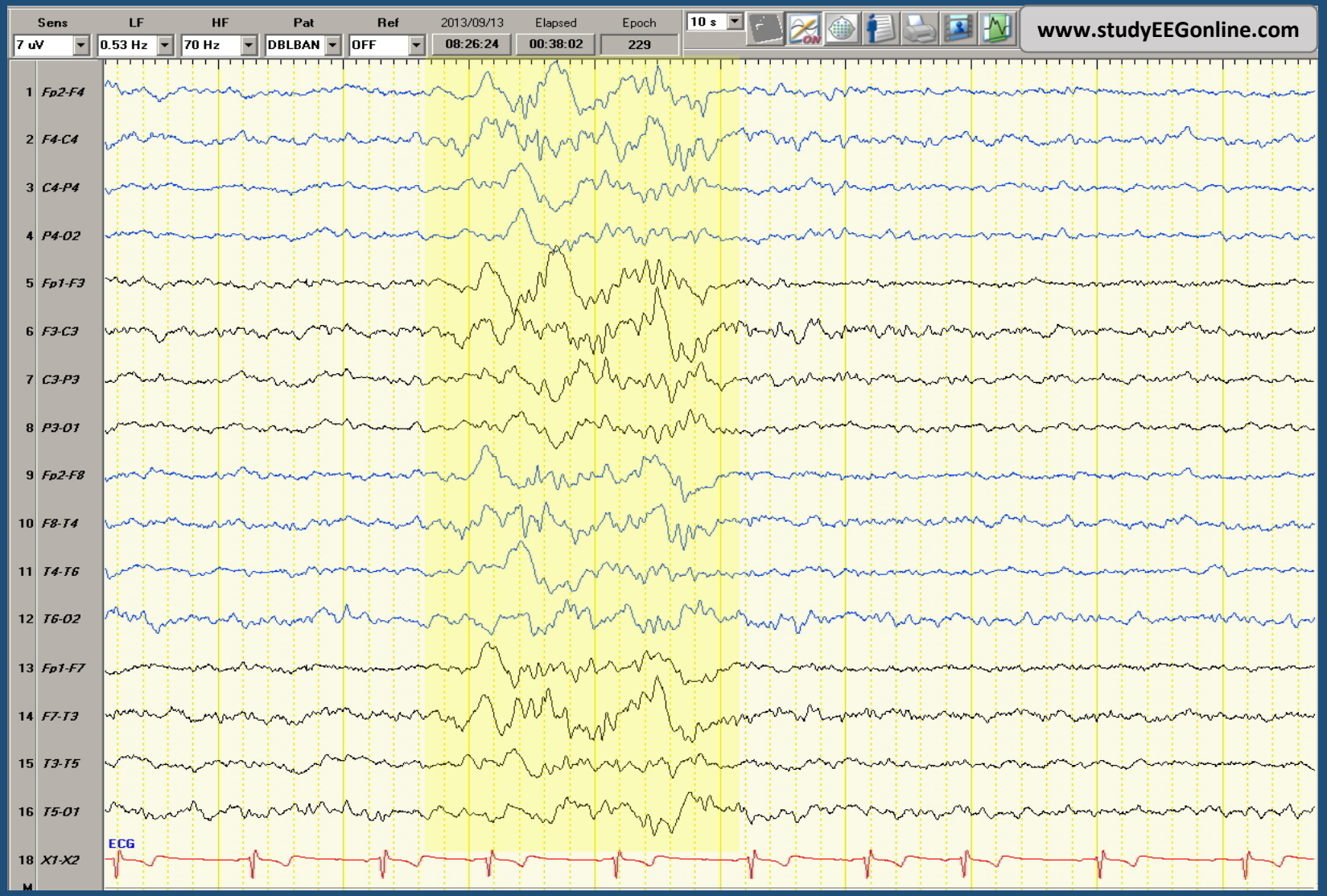


Somnolence → Arousal

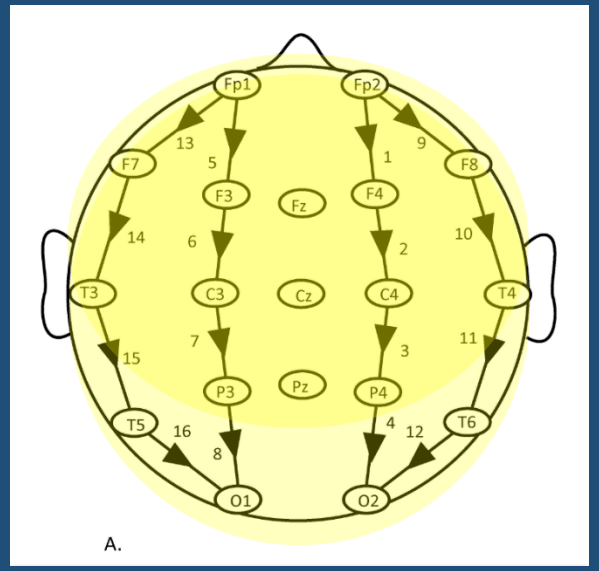
Occasionally, arousals may be preceded by waveforms associated with physiological sleep which may be erroneously regarded as pathological.



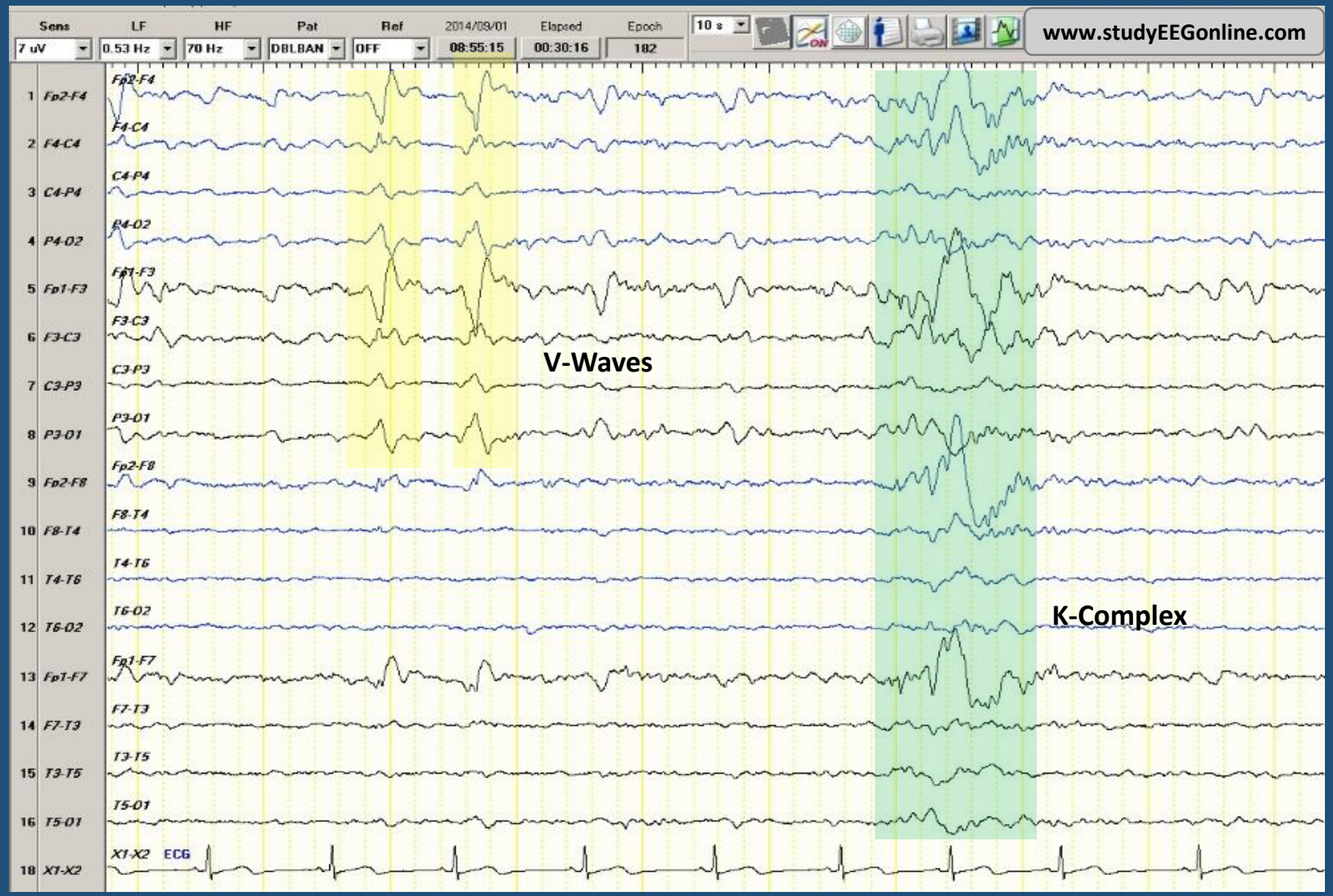
Drowsiness & Somnolence: **K-Complexes**



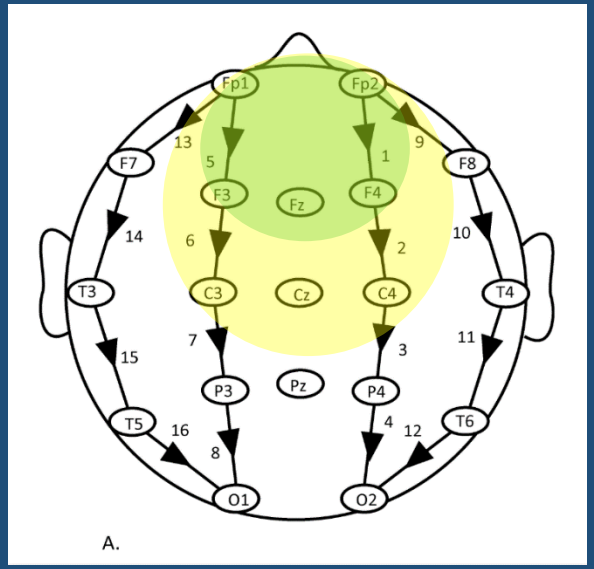
K-complexes which occur during somnolence may be dramatic and mistaken as pathological



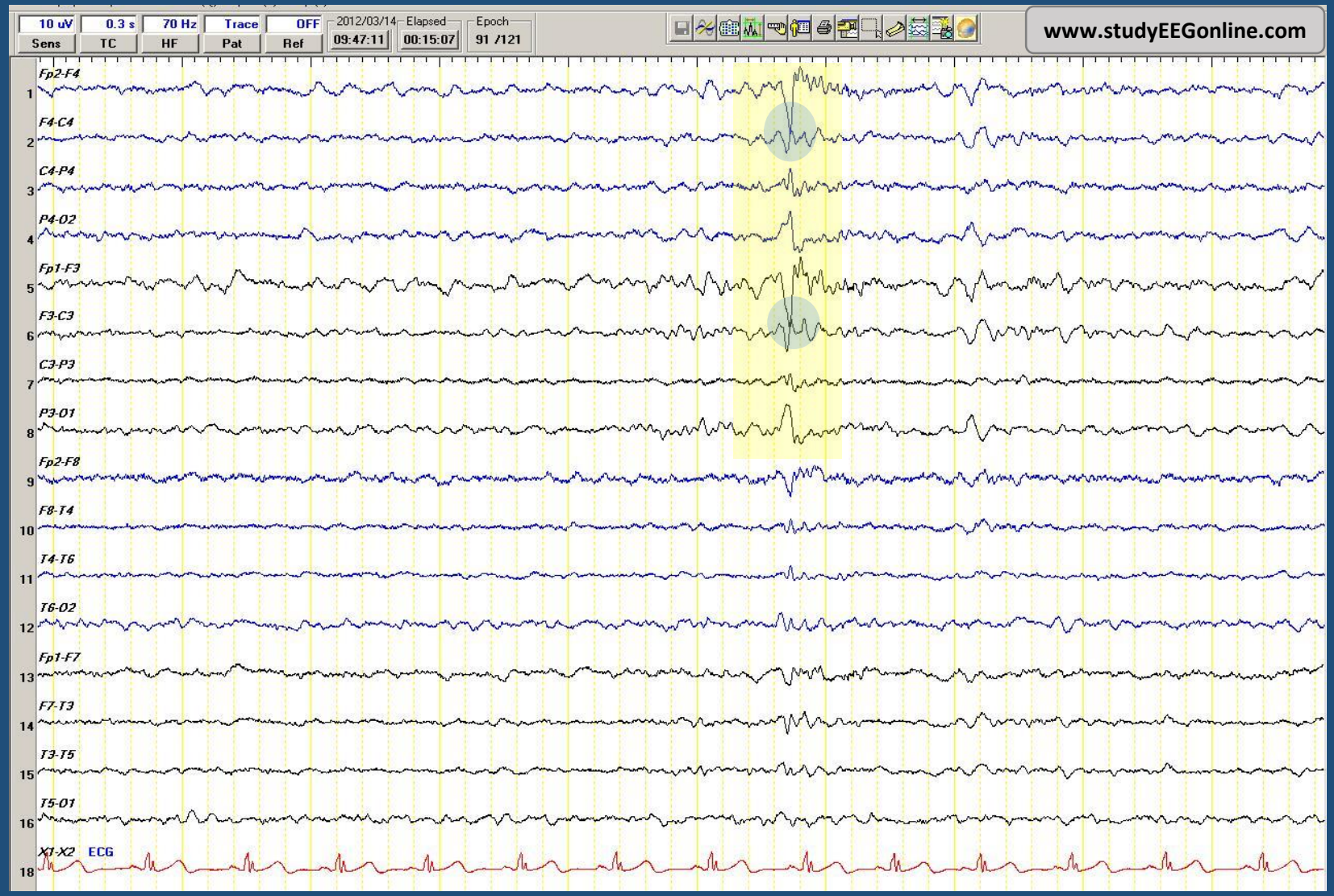
Drowsiness & Somnolence: **Vertex (V)-Waves**



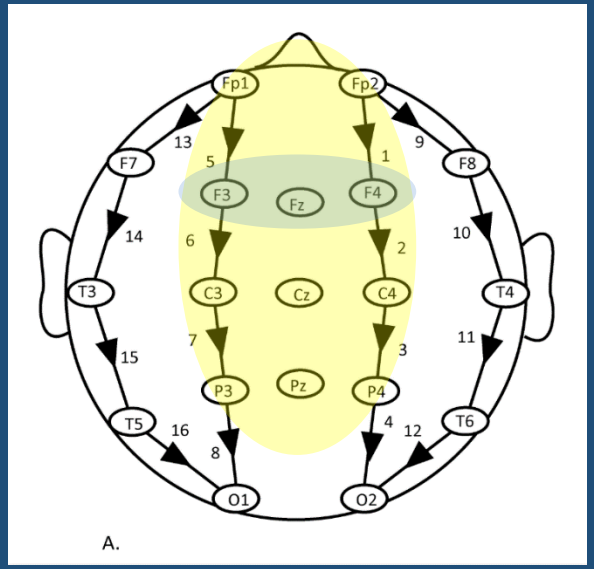
V-waves often have a sharply-contoured morphology which closely mimic epileptiform discharges



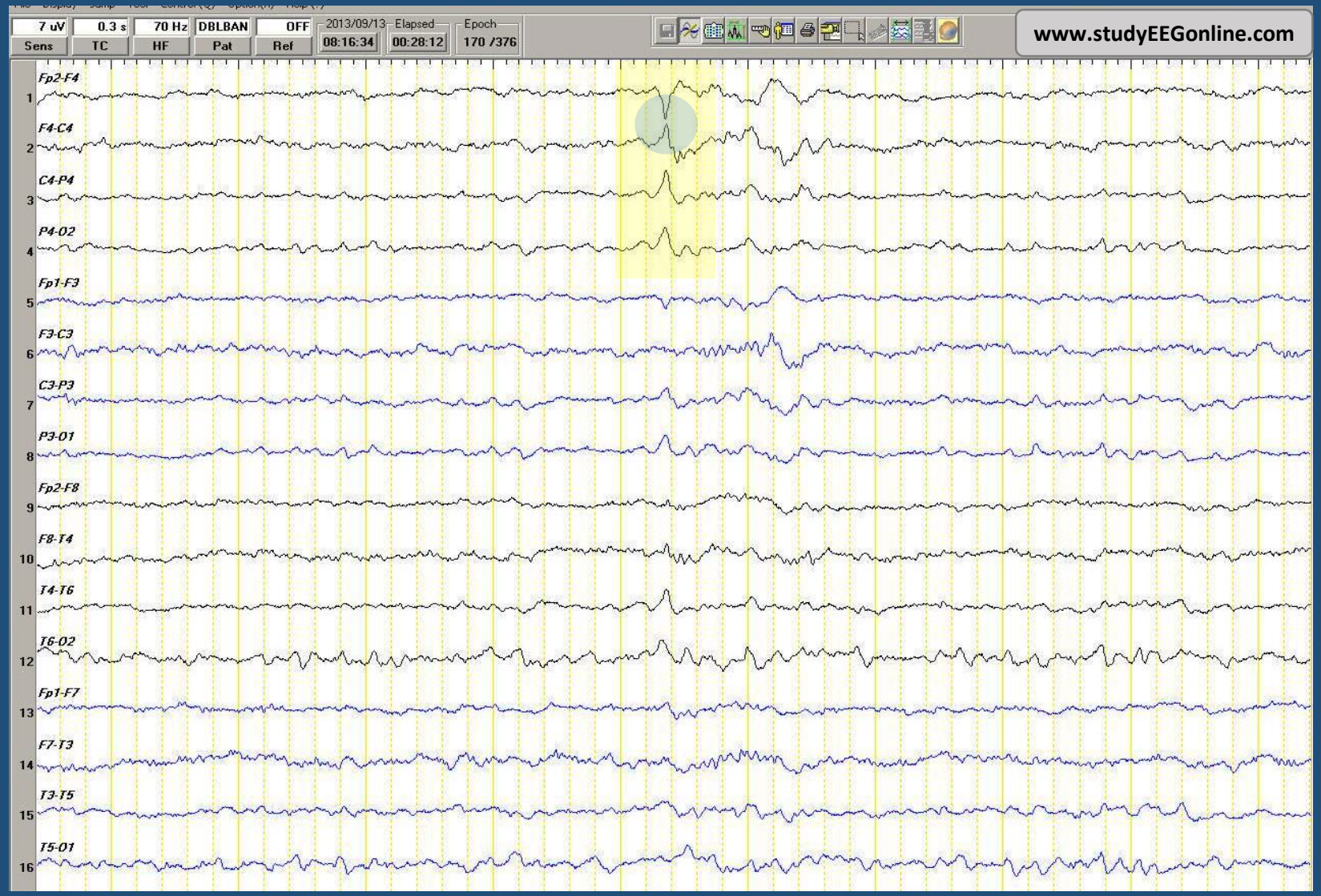
Drowsiness & Somnolence: **Symmetrical Frontal (F)-wave**



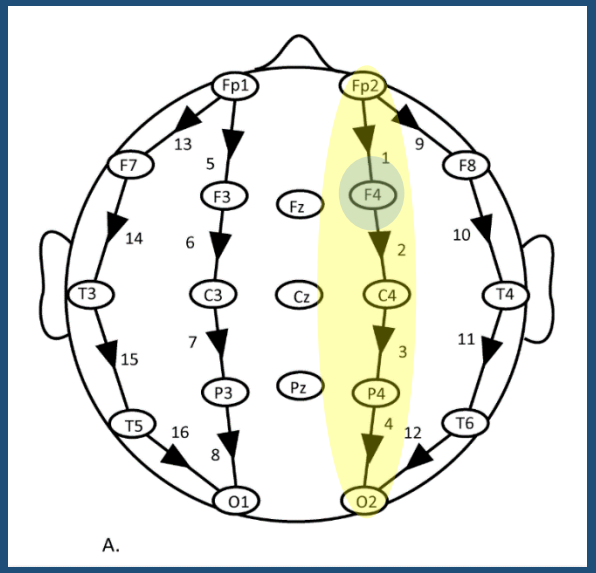
When “V”-waves phase-reverse in the frontal channels they are often referred to as F-waves



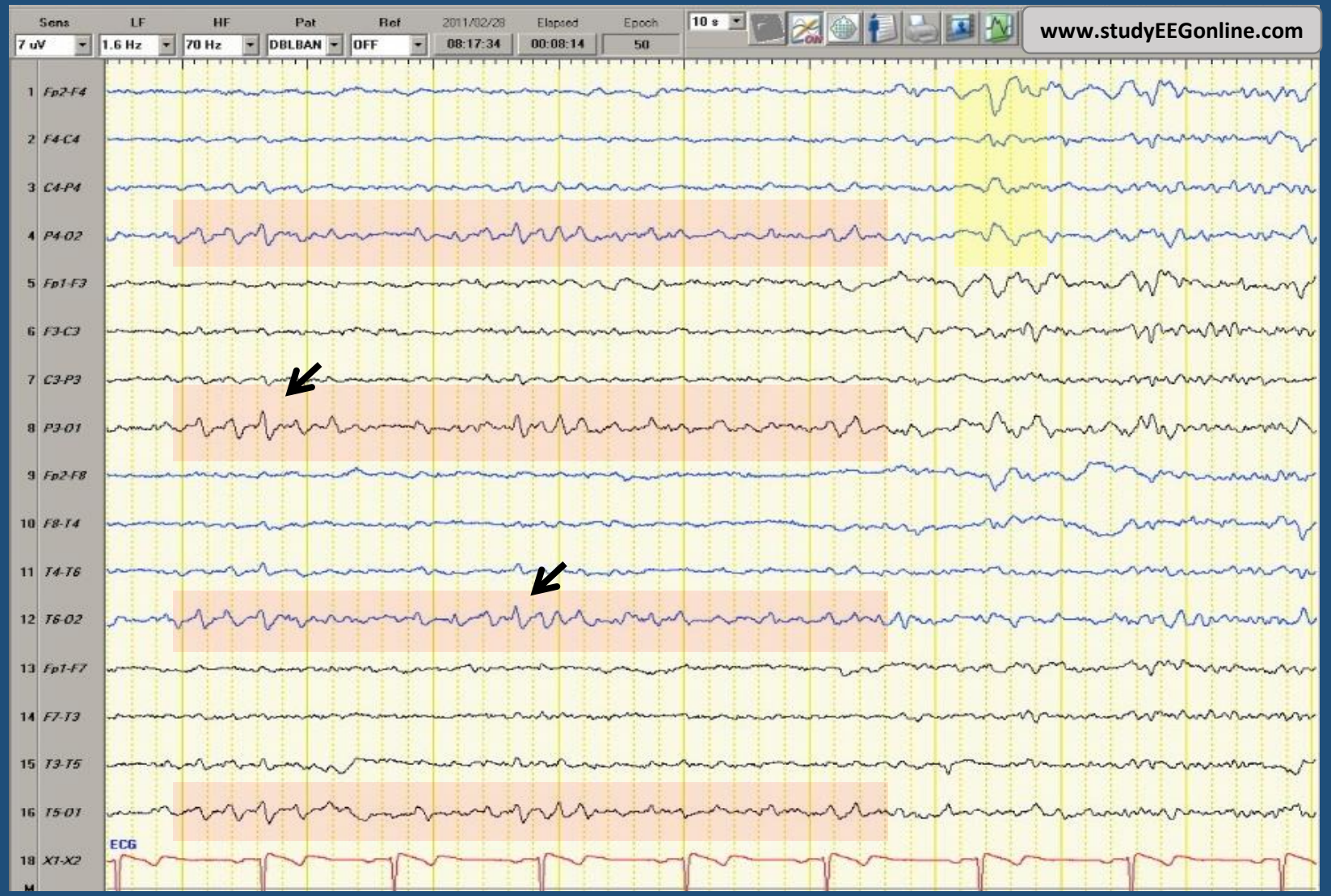
Drowsiness & Somnolence: **Asymmetrical F-wave**



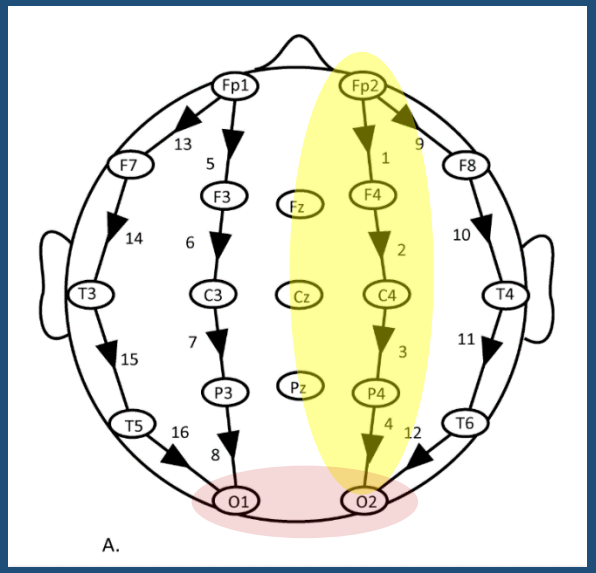
Importantly, V- and F-waves may occasionally be asymmetrical



Drowsiness & Somnolence: **POSTS**



Posterior Occipital Sharp Transients of Sleep (POSTS) may be mistaken as evidence of epilepsy



The take home message is not to mistake normal physiological waveforms of drowsiness and sleep as epileptiform or pathological slowing.

The Abnormal EEG

The Abnormal EEG

Epileptiform Waveforms:

Sharp Waves
Spikes / Polyspikes
Spike/Poly-spike/Sharp & Slow Wave Complexes

Non-Epileptiform Waveforms:

Abnormal Slowing

Epileptiform Waveforms

The EEG is probably most commonly used to assist in diagnosing and characterising epilepsy. It may be especially helpful where conditions such as syncope, panic attack, hyperventilation, and TIAs, etc. present with clinical symptoms mimicking those of a seizure

Epileptiform Discharges

Three features characterise epileptiform discharges:

a) They should be **sharply contoured**

b) They should be "**superimposed upon**" and "**disrupt**" the **background rhythms**

c) They should have **credible electrical fields**

A diagnosis of epilepsy is primarily based on clinical presentation

The presence of iterictal epileptiform discharges strongly support a clinical diagnosis of epilepsy

BUT

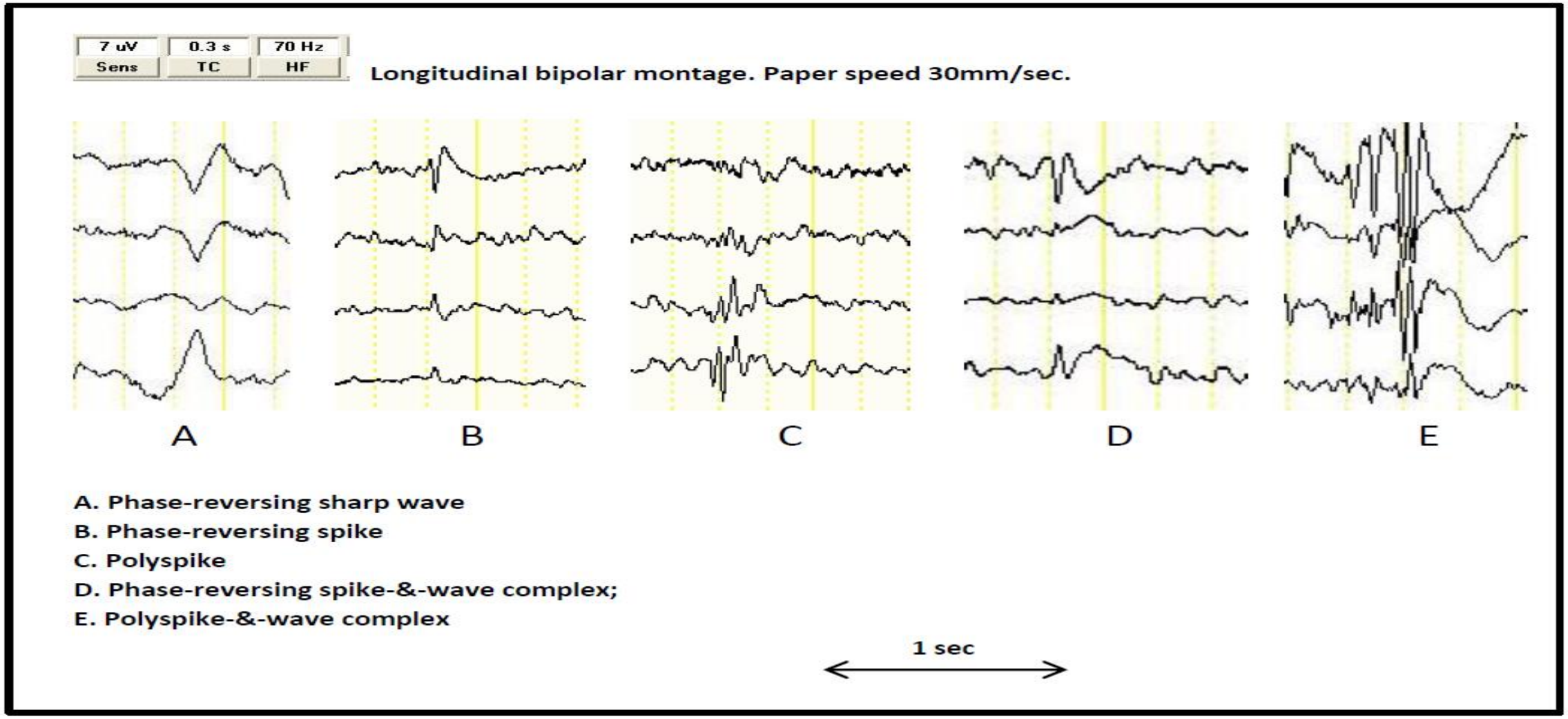
The absence of epileptiform discharges does not exclude a clinical diagnosis of epilepsy

Epileptiform Discharges: Focal vs Generalised

Another useful role of EEG is to determine whether interictal epileptiform discharges are **focal** or **generalised**

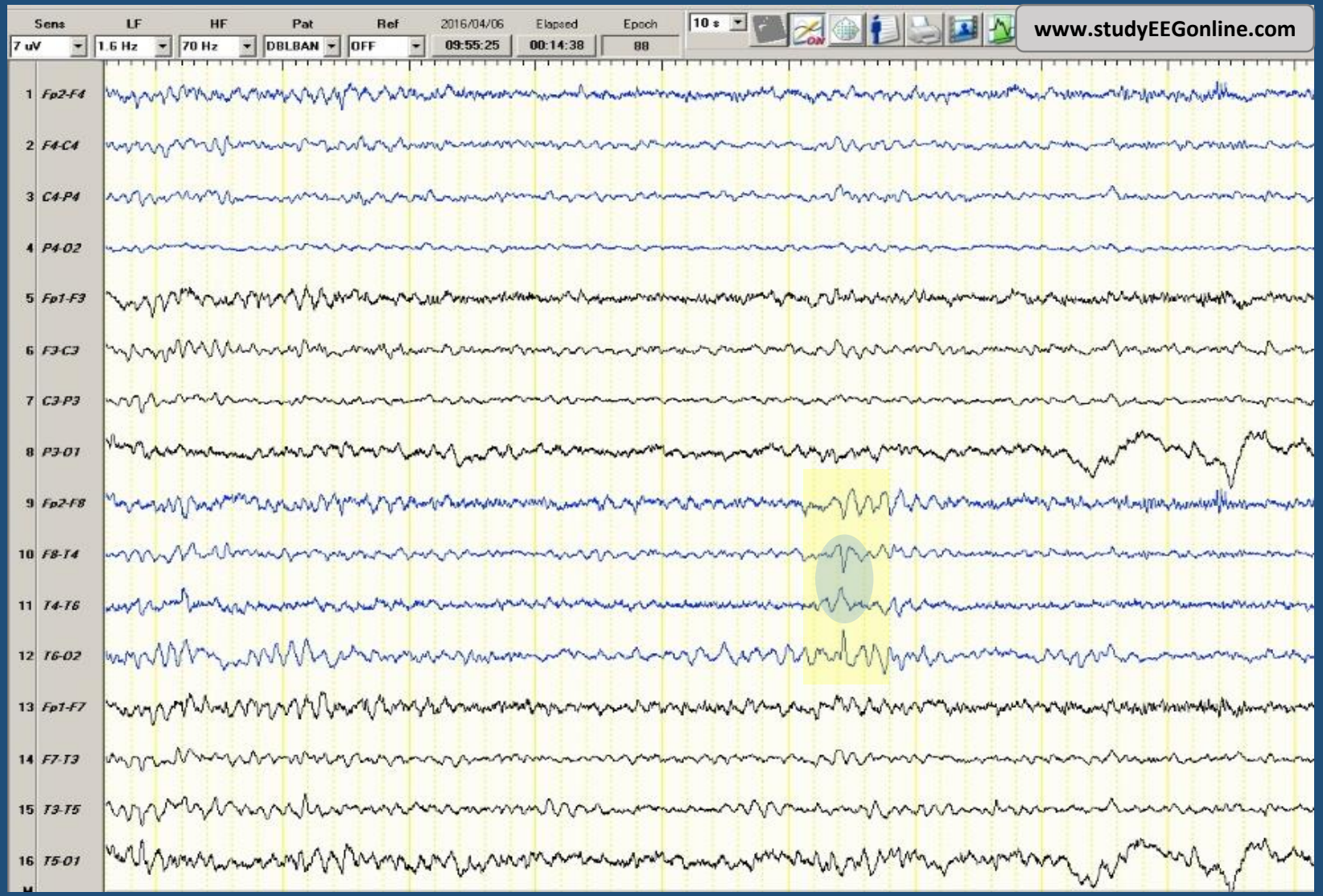
This has important implications for both the **aetiology** and **management** of a patient with epilepsy

Epileptiform Dischages (longitudinal bipolar montage)

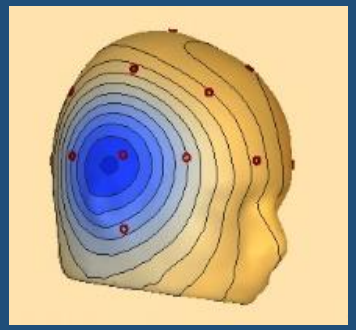


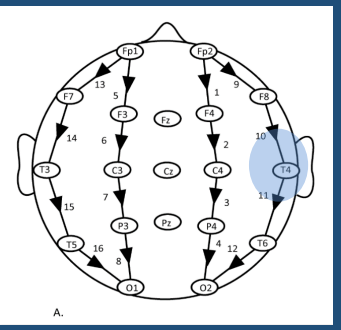
Focal Epileptiform Discharges

Focal Spike-&-Slow Wave

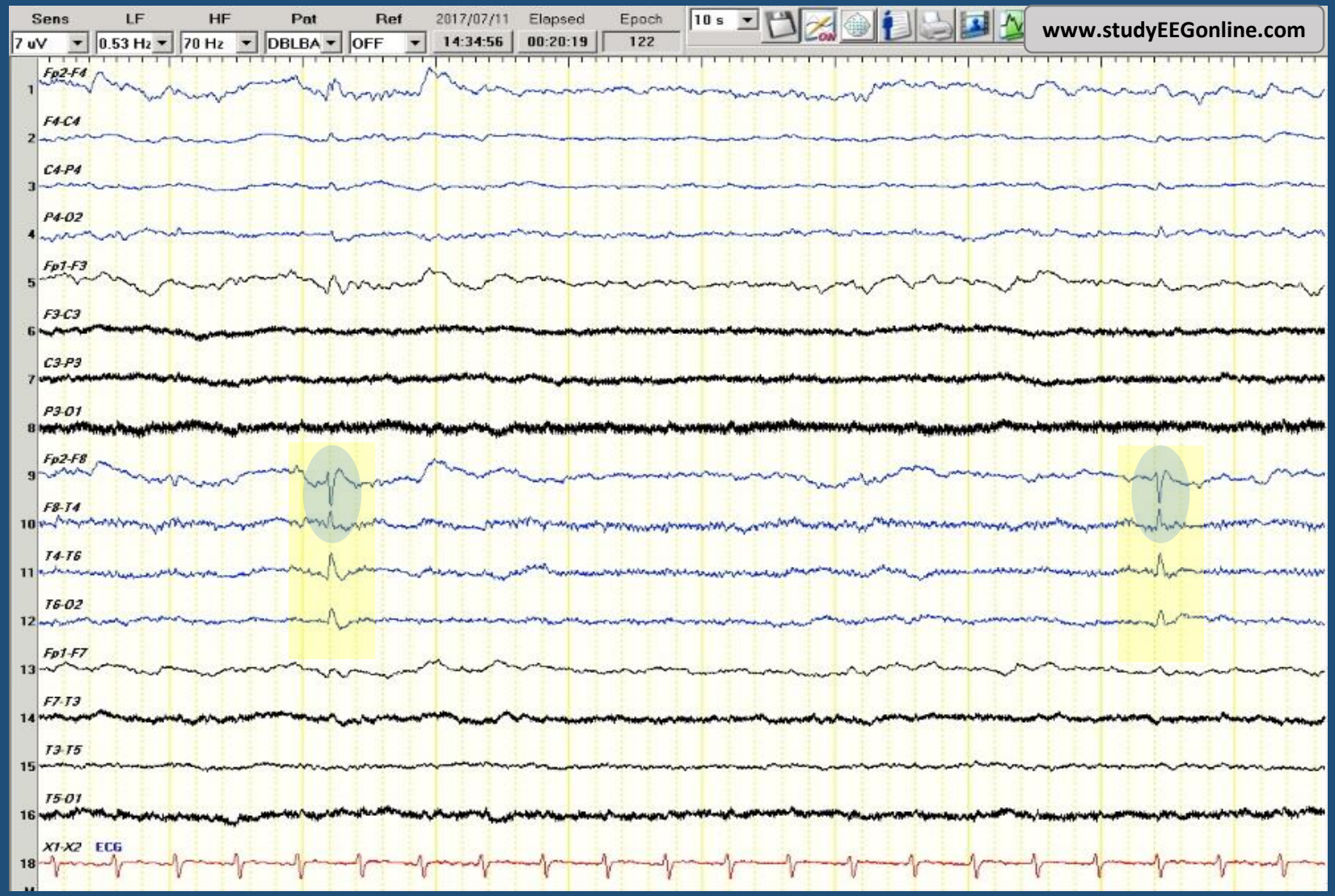


A focal spike is present, which phase-reverses at T4 in the right mid-temporal lobe

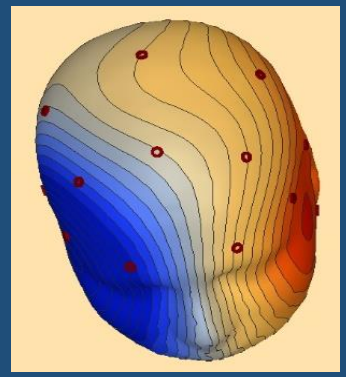


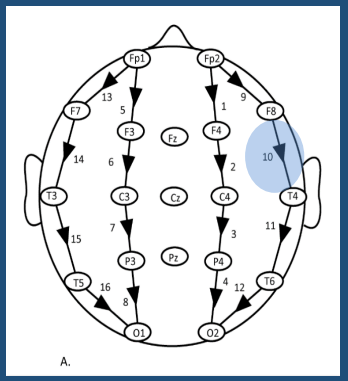


Focal Spikes

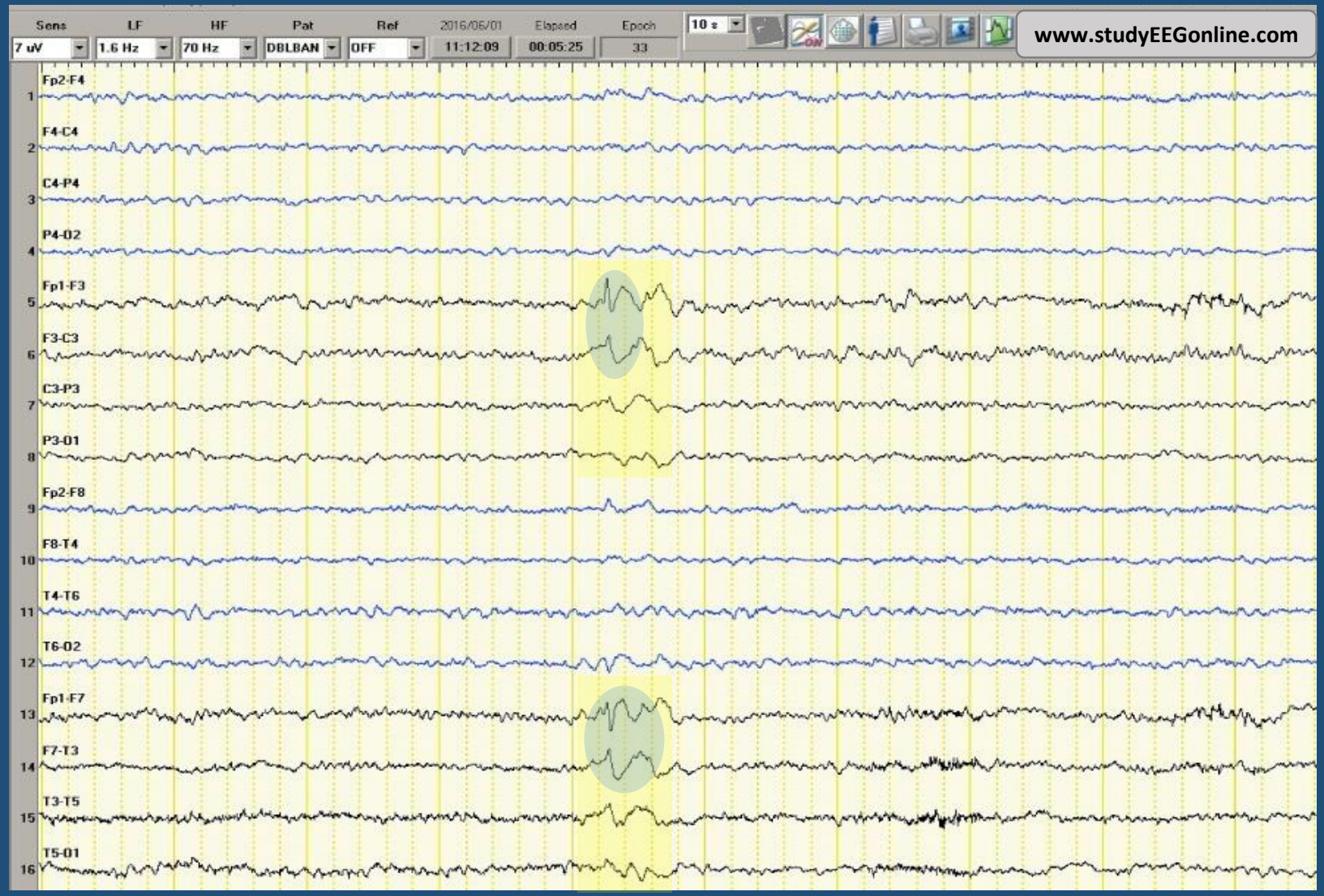


Two focal spikes are present, which phase reverse at F8 in the right anterior temporal lobe

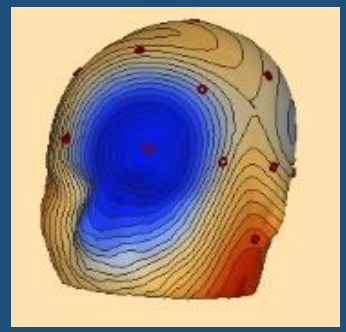


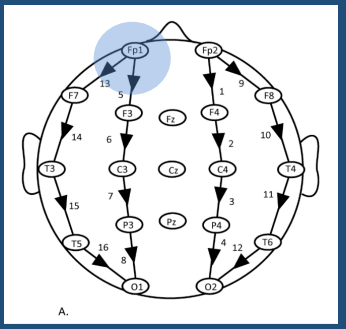


Focal Spike-&-Slow Wave

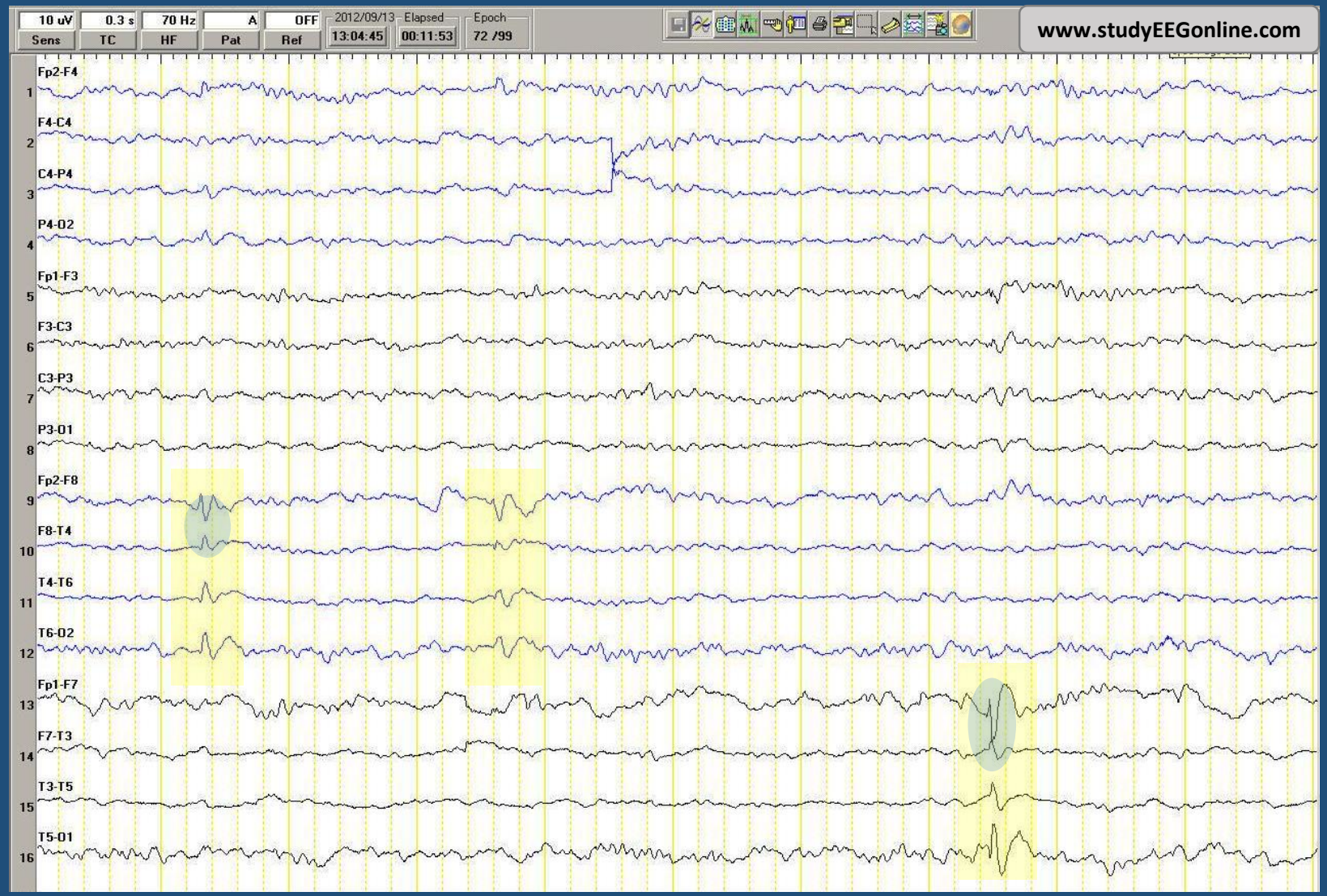


A focal spike-&-slow wave discharge is present, which is maximal at Fp1 and F7 in the left frontal and left anterior temporal region.





Independent Multi-Focal Epileptiform Discharges

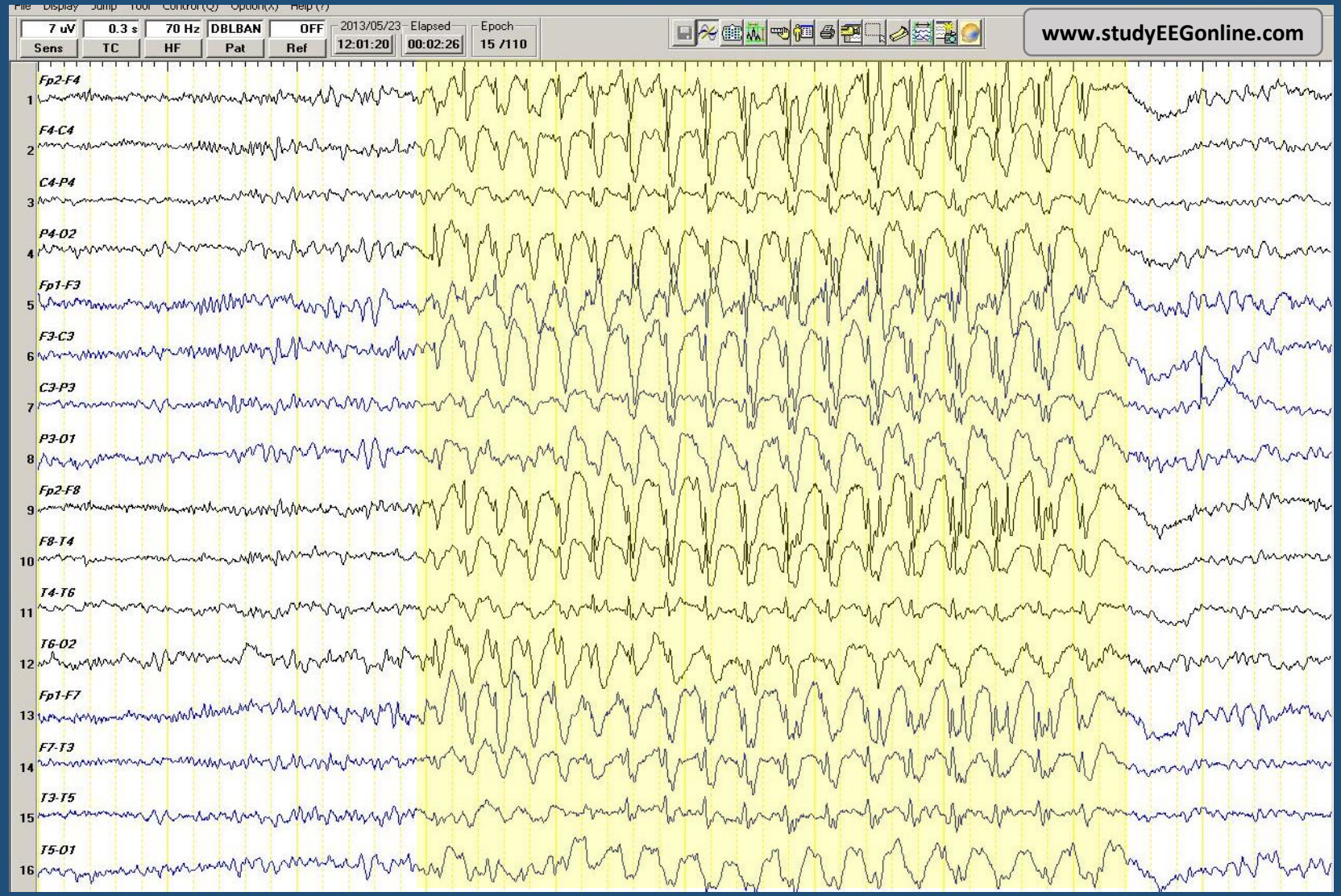


Independent multi-focal epileptiform discharges are seen in both temporal lobes.

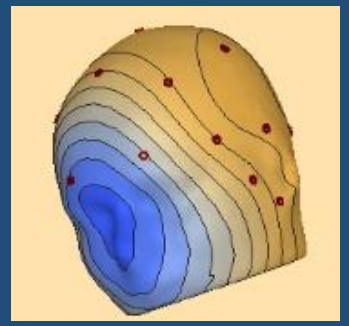
Generalised Epileptiform Discharges

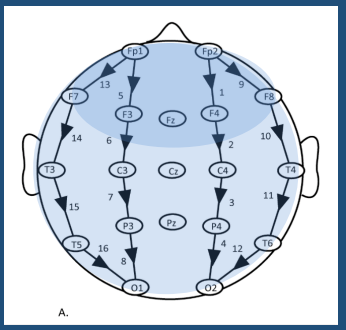
Generalised Epileptiform Discharges usually
imply an inherited generalised form of epilepsy

Generalised (“Typical”) 3 Hz Spike-&-Slow Wave

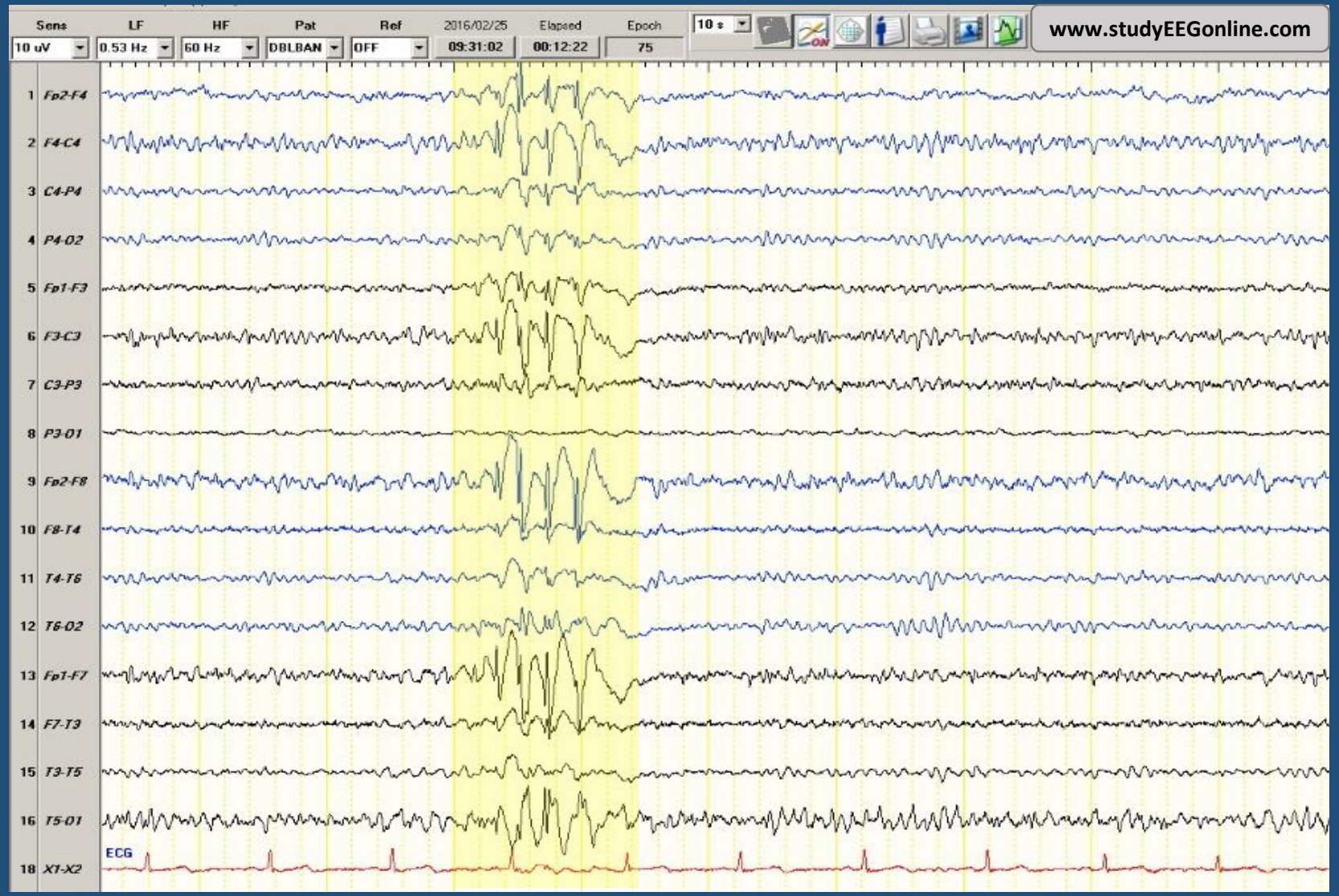


Generalised, but frontally-predominant, rhythmic, 3Hz spike-&-slow wave discharge characteristic of Childhood Absence Epilepsy

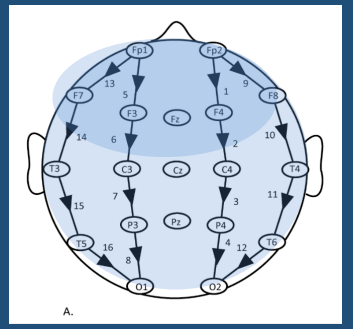




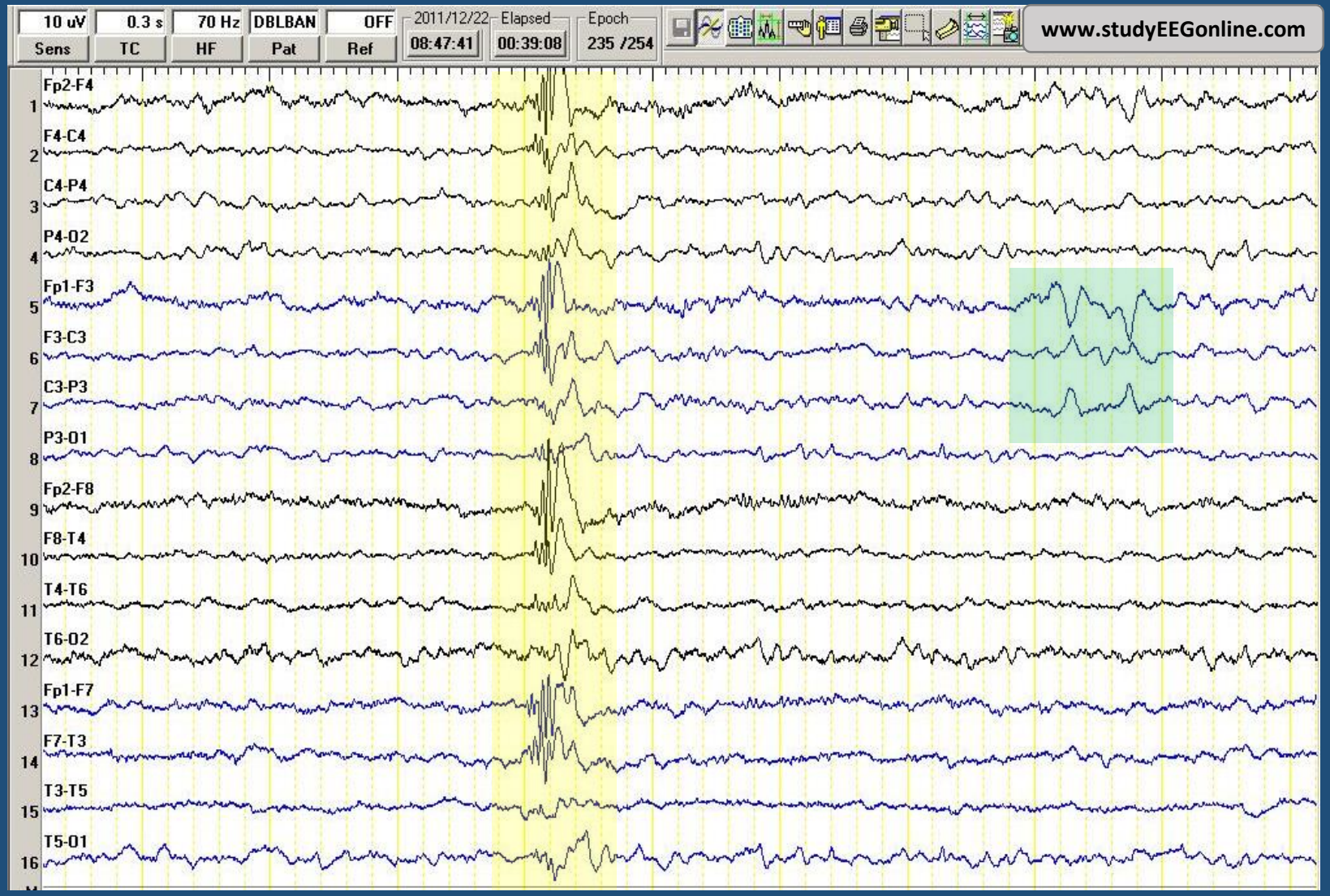
Generalised (“Atypical”) 4-5 Hz Spike & Slow Wave



Generalised, but frontally-predominant, atypical, 4-5 Hz spike-&-slow wave discharge characteristic Juvenile Myoclonic Epilepsy



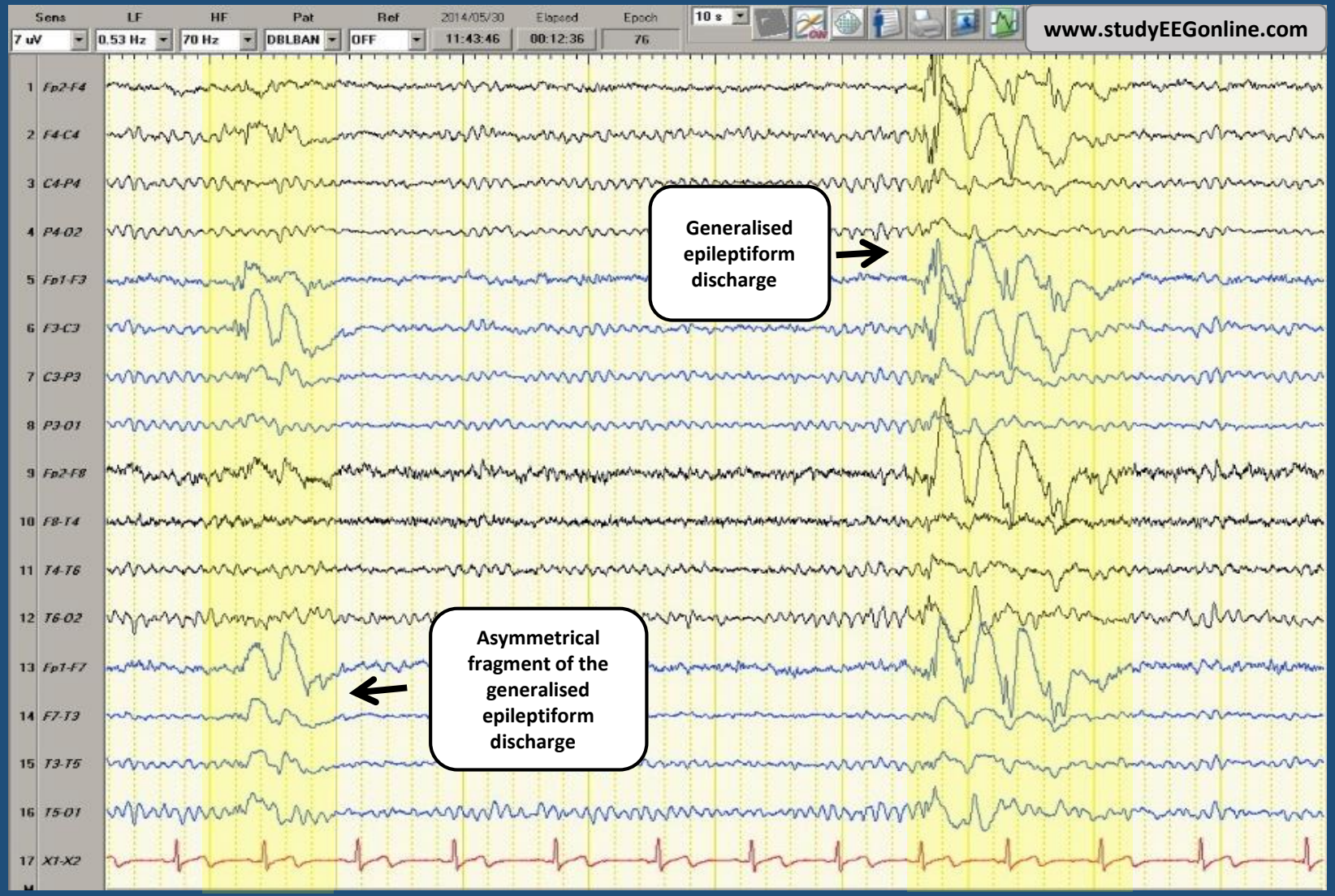
Generalised Polyspike-&-Slow Wave



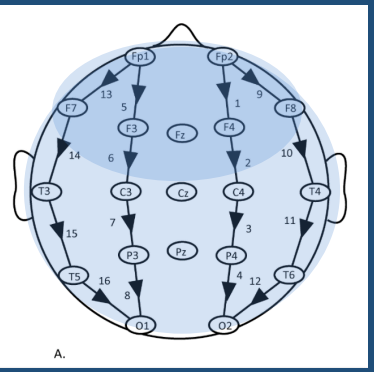
Generalised, frontally-predominant, polyspike-&-slow wave complex consistent with an Inherited Generalised Form of Epilepsy

Note the asymmetrical V-waves resembling sharp waves (highlighted in green)

Generalised Spike & Slow Wave: With Fragment



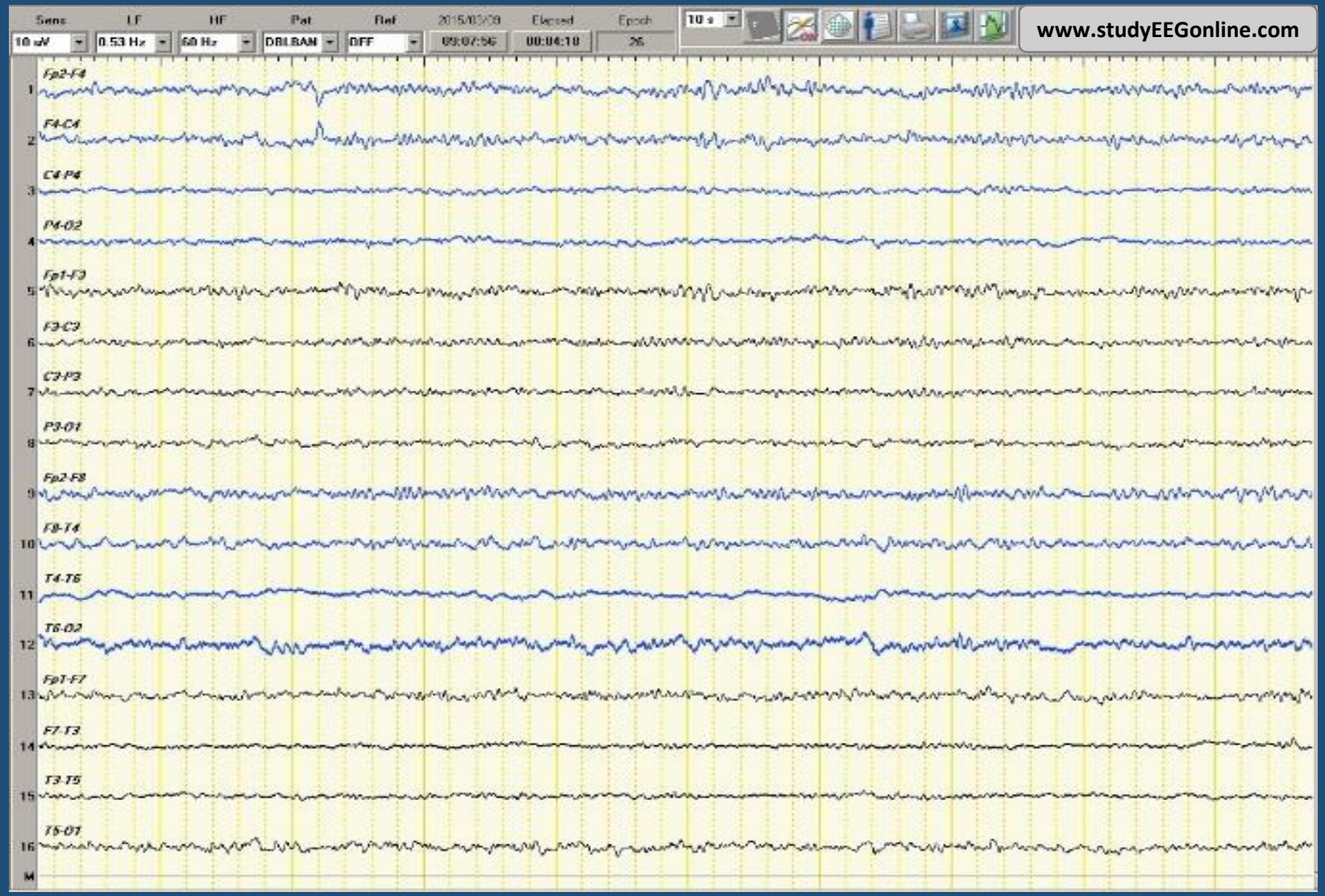
Occasionally fragments of generalised epileptiform discharges may be seen, which may be confused with focal discharges.



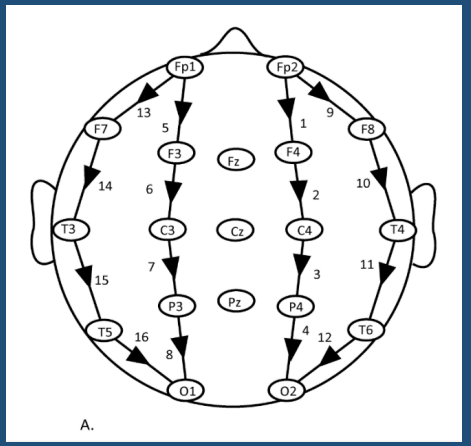
Electrographic Seizures

Generalised Seizure

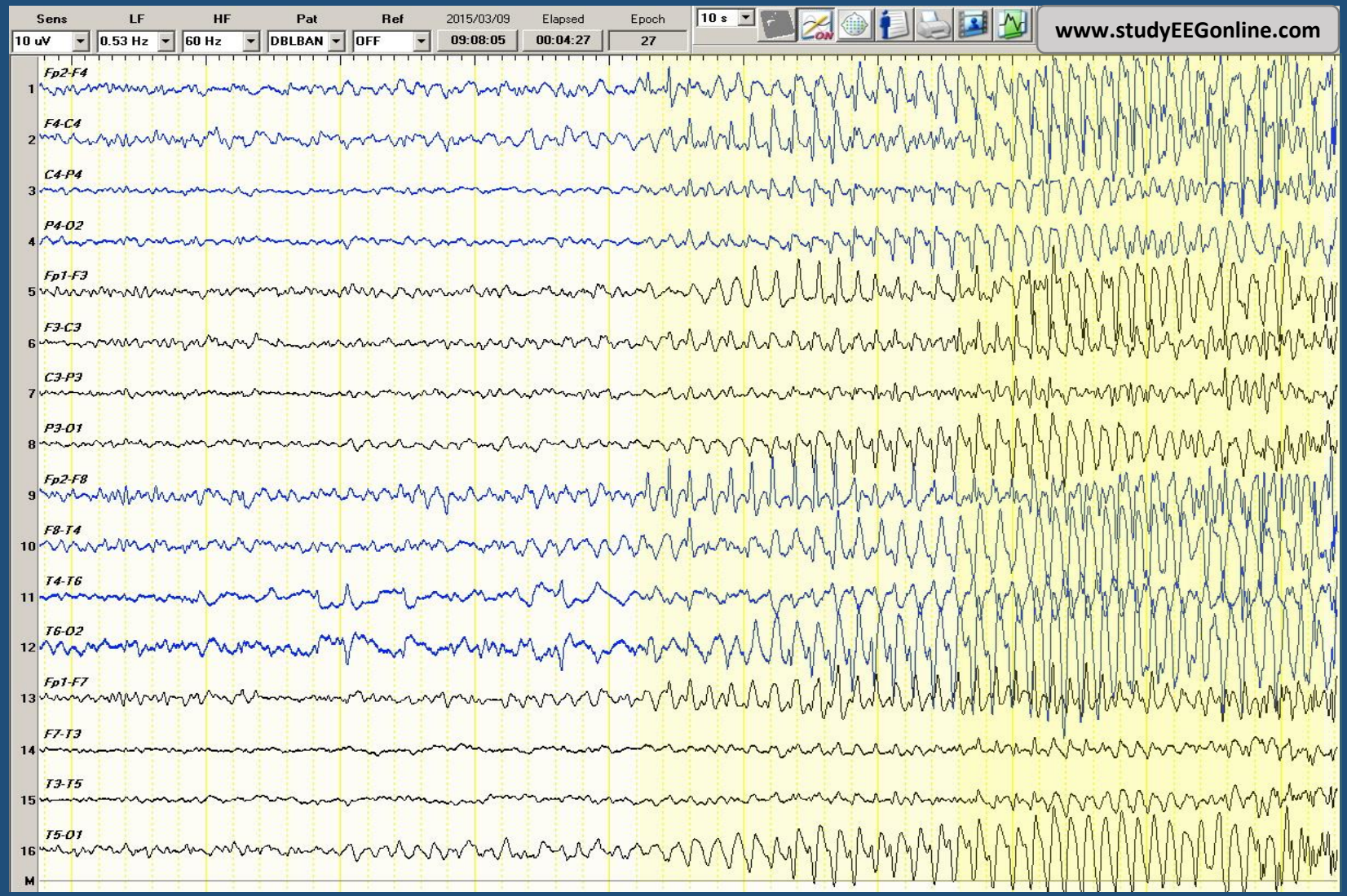
Generalised Seizure: **Background**



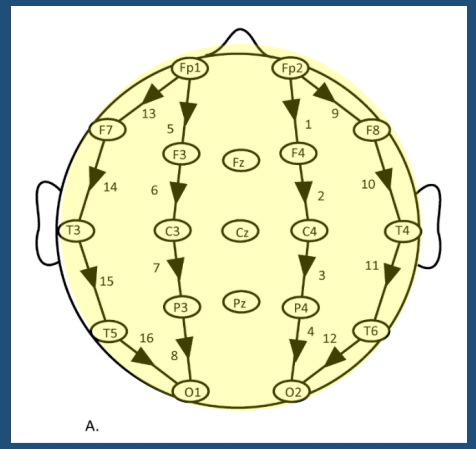
Note the
normal background



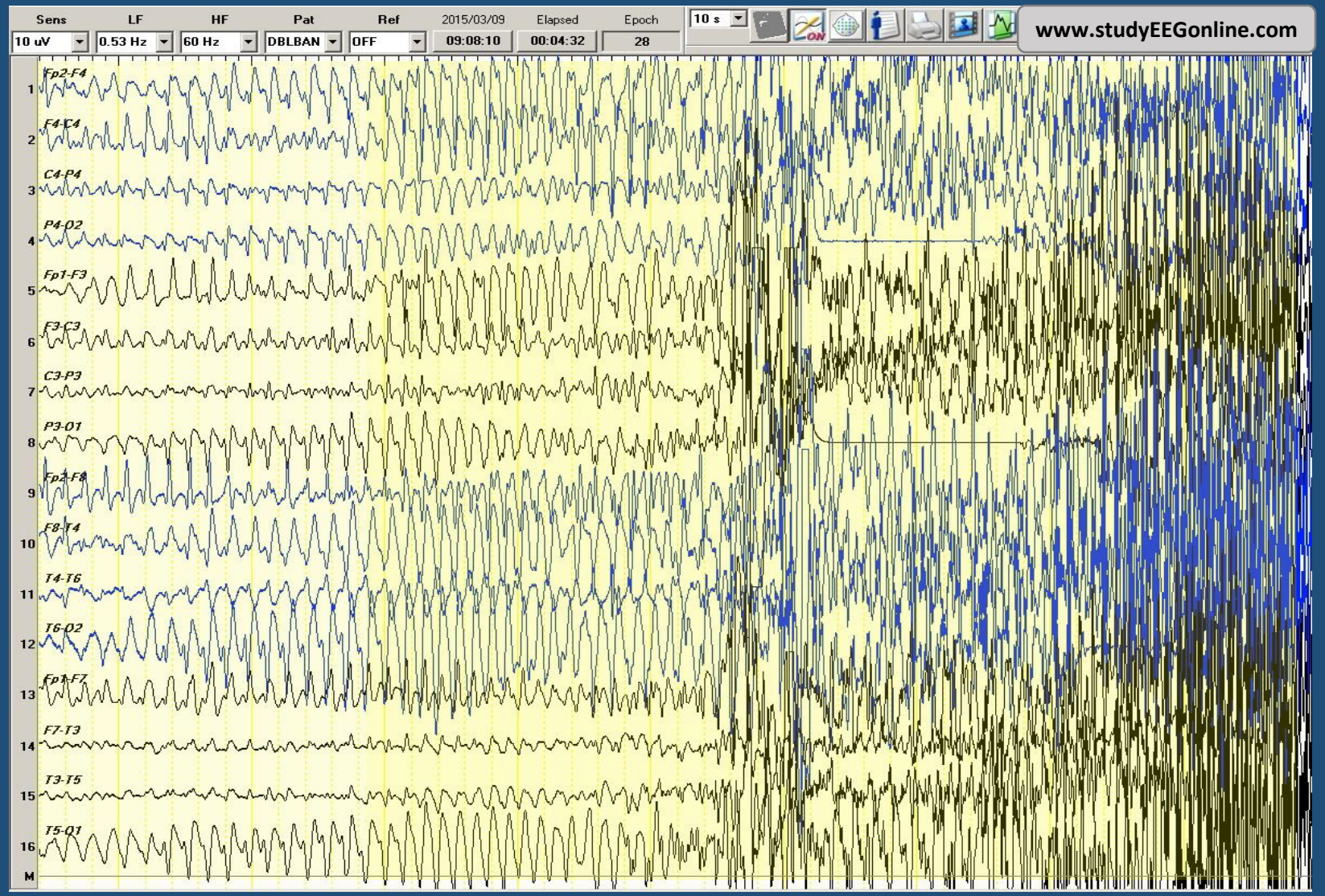
Generalised Seizure: Onset



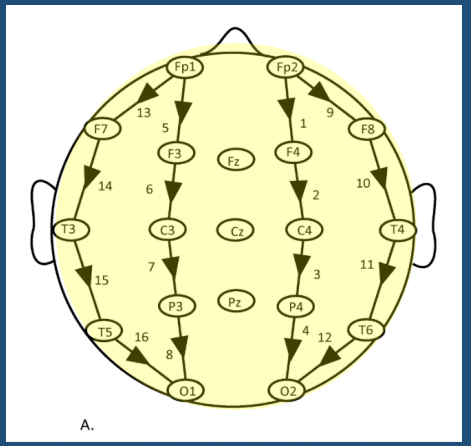
Simultaneous onset in all channels of a relatively high frequency, sharply contoured beta rhythm



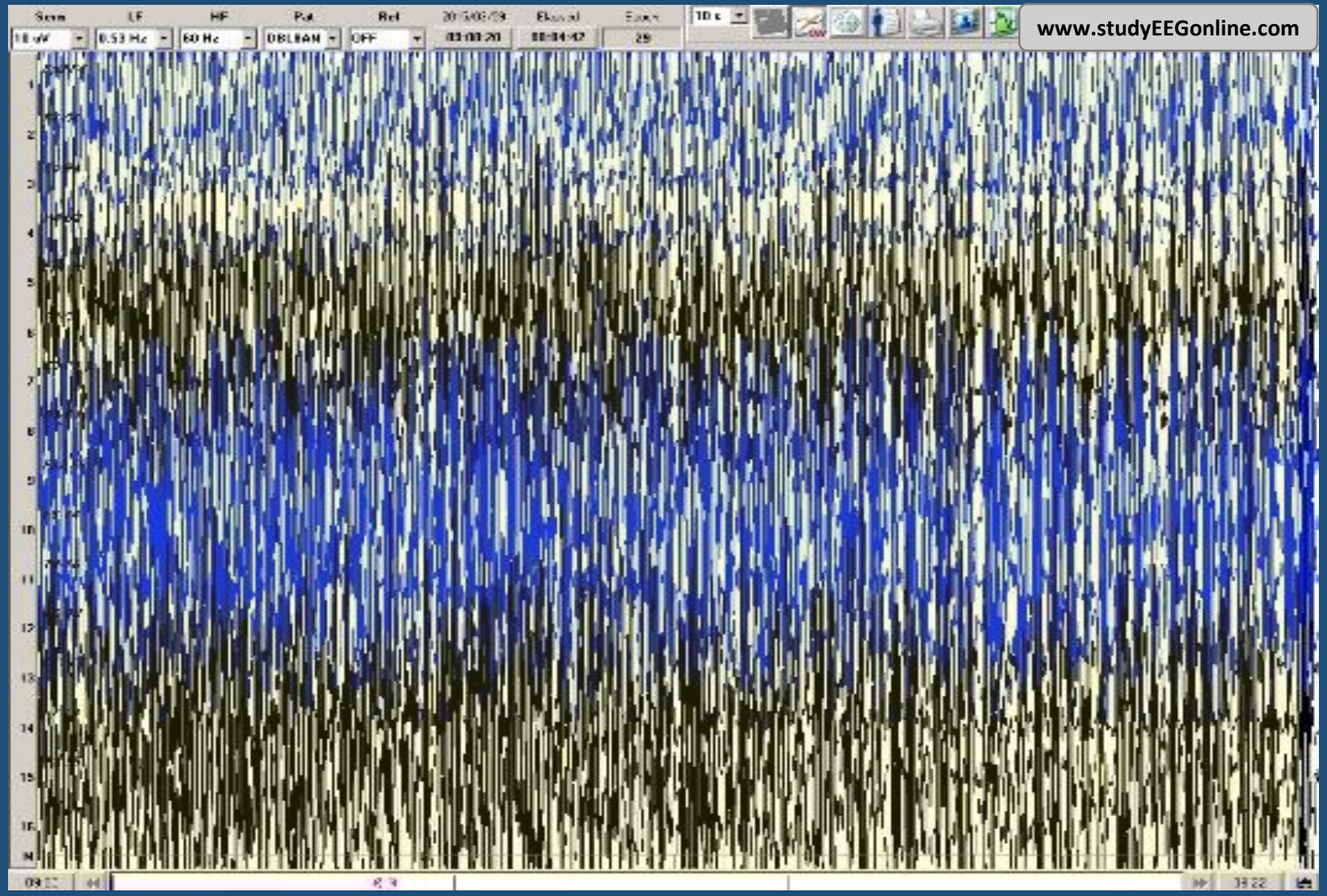
Generalised Seizure: Evolution



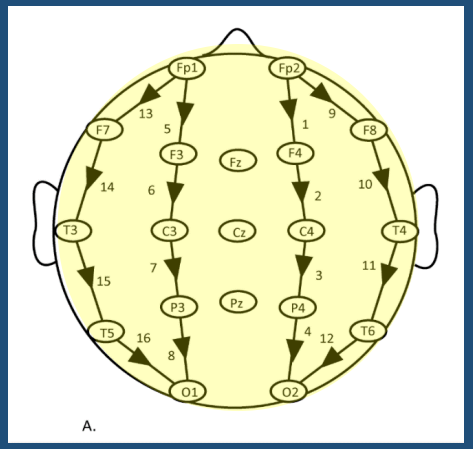
Gradual evolution (reducing frequency and increasing amplitude)



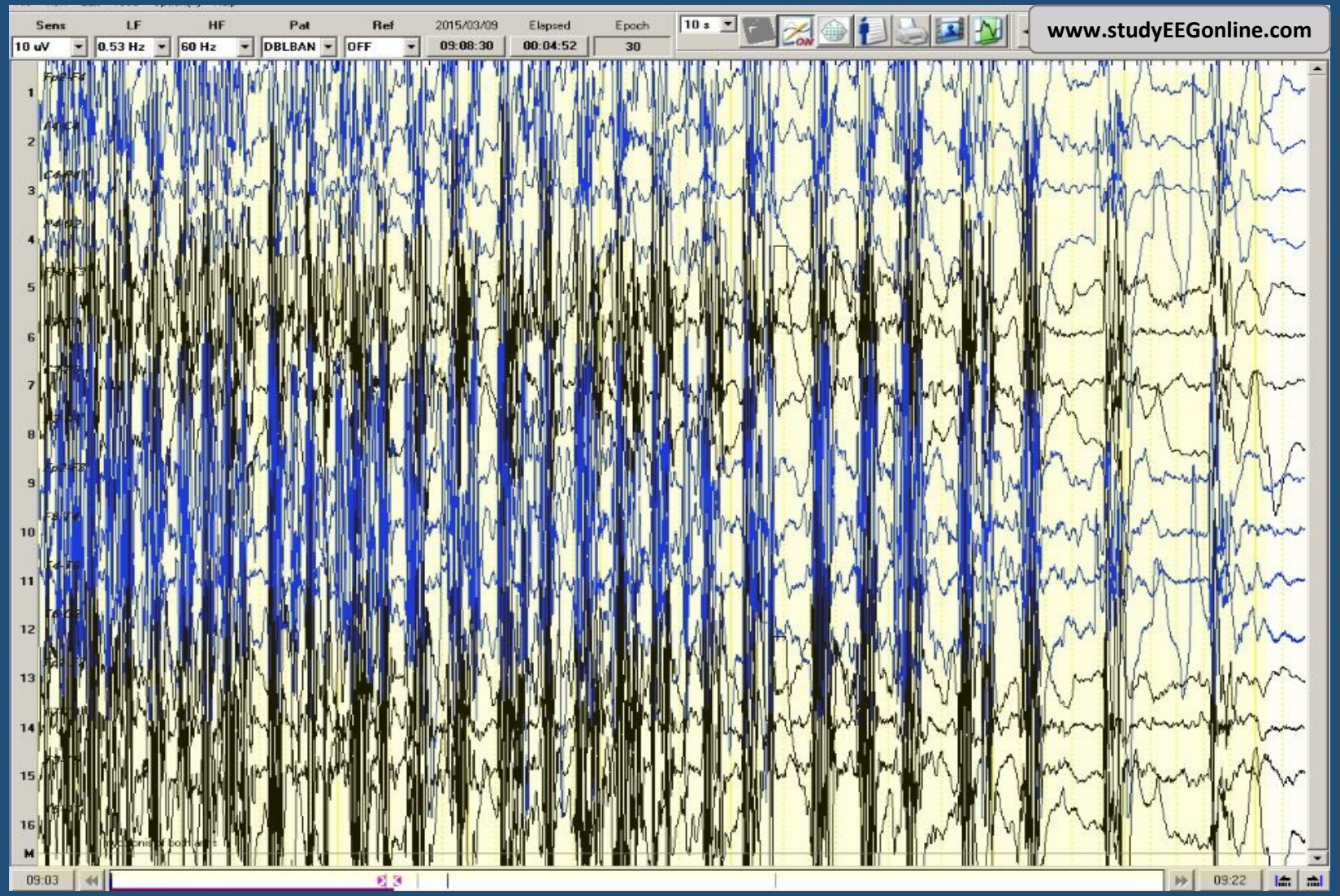
Generalised Seizure: **Evolution**



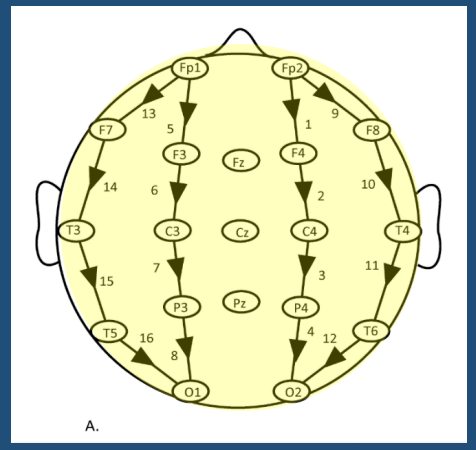
Muscle (EMG) artefact



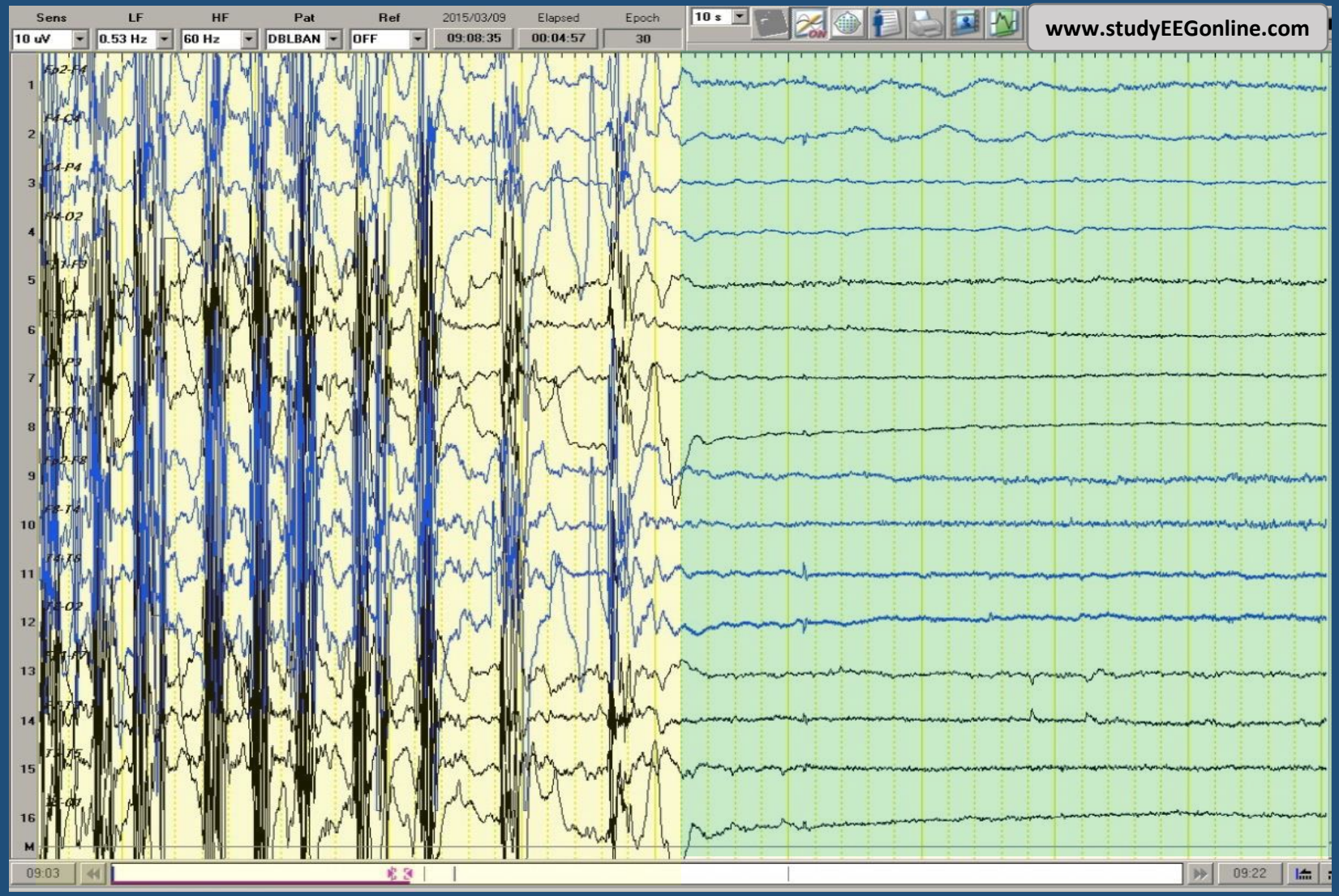
Generalised Seizure: **Evolution**



Continued evolution (reducing frequency) of discharges/EMG artefact



Generalised Seizure: **Offset & Post-ictal Suppression**

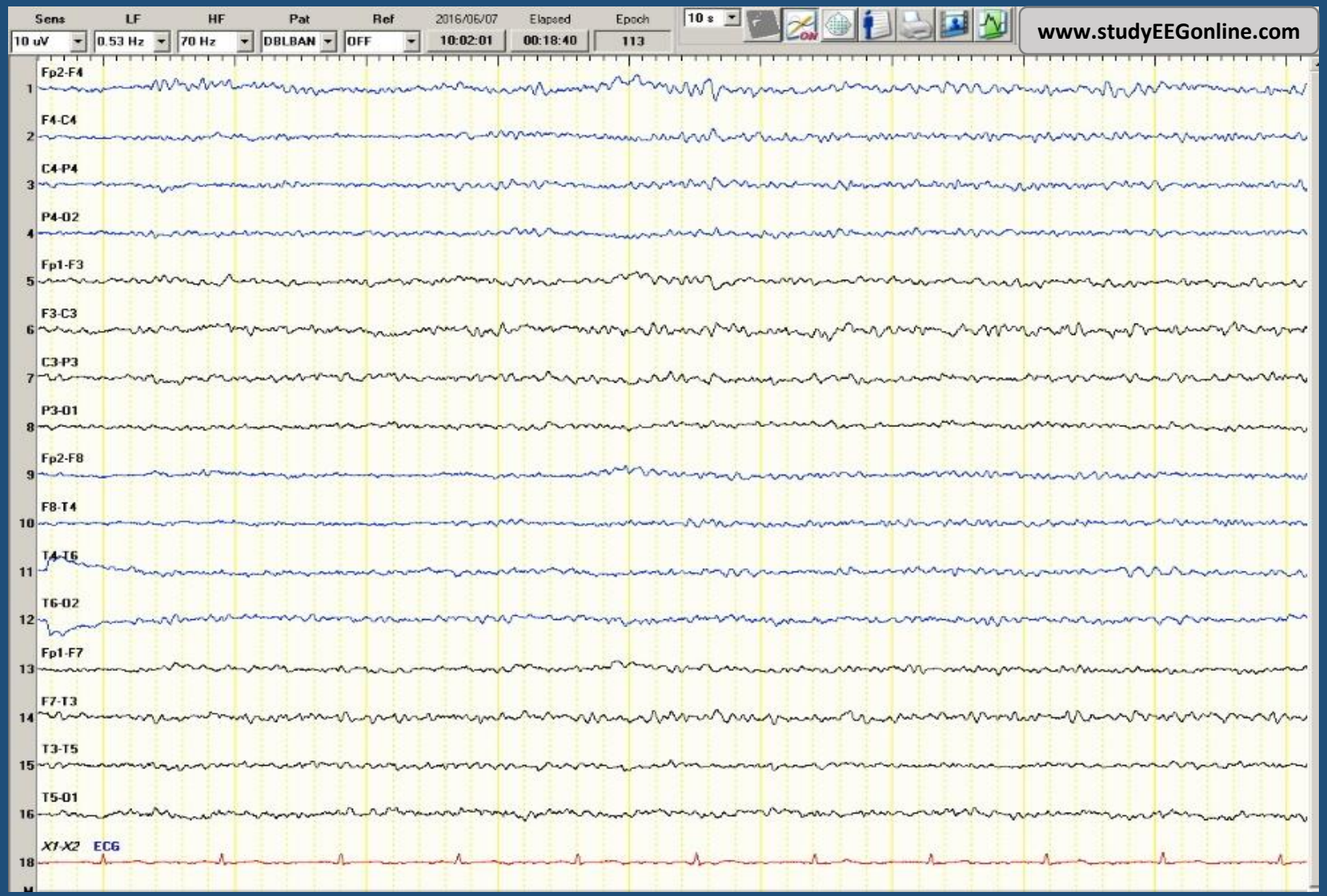


Continued evolution (reducing frequency) of discharges/EMG artefact and abrupt offset

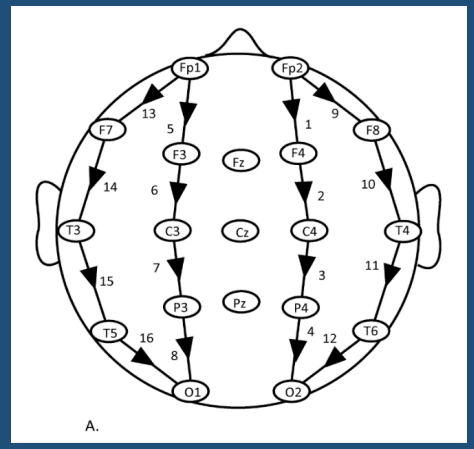
Note post-ictal suppression

Focal Seizure with Altered Awareness

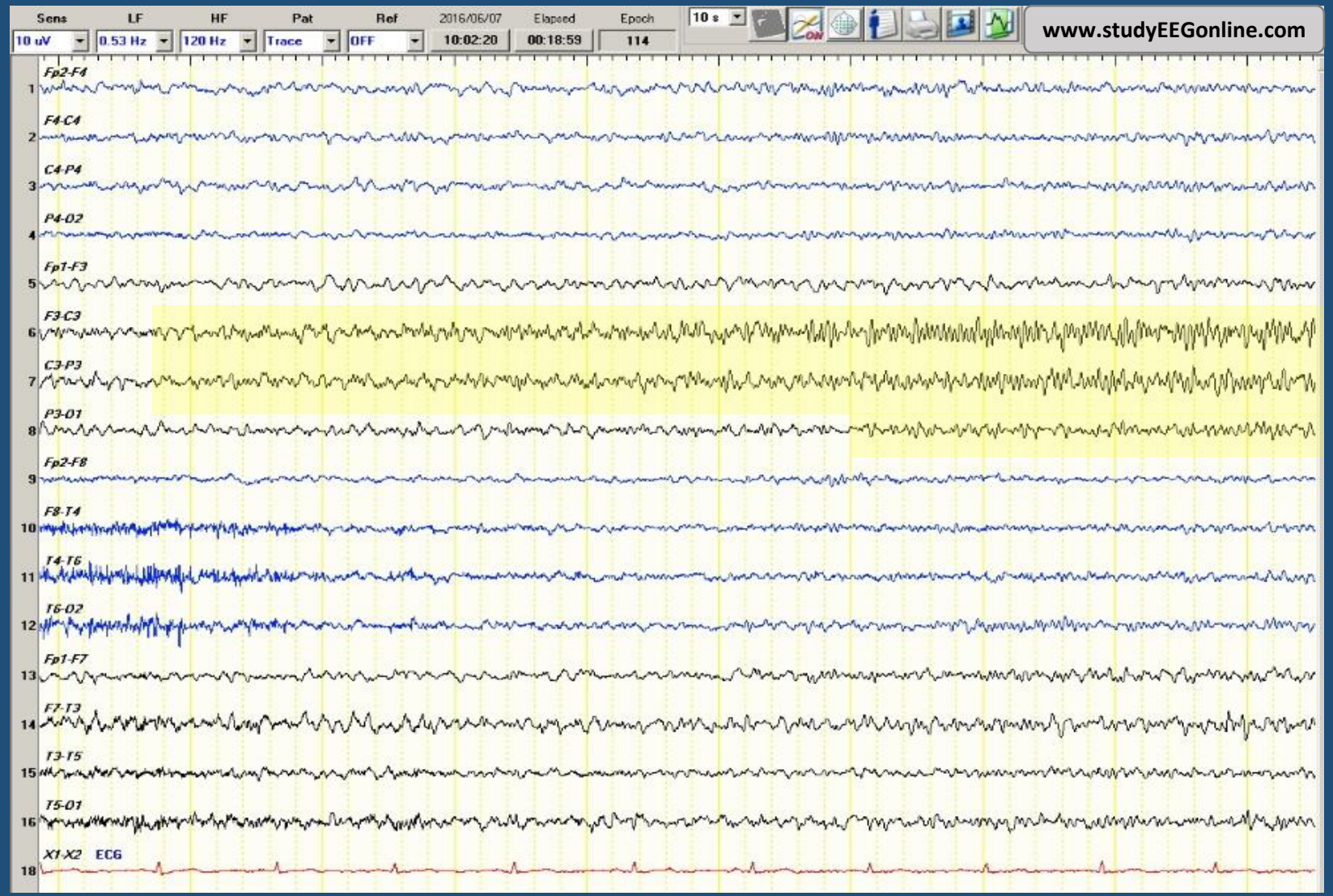
Focal Seizure with Altered Awareness: **Background**



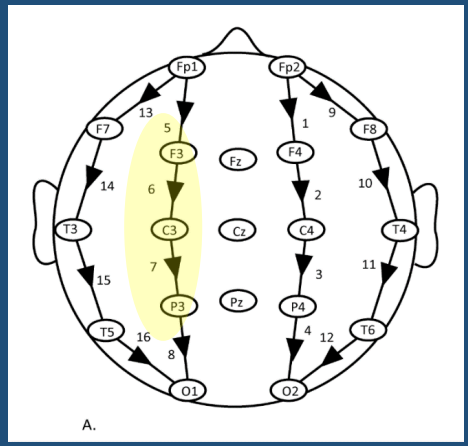
Note the
normal background



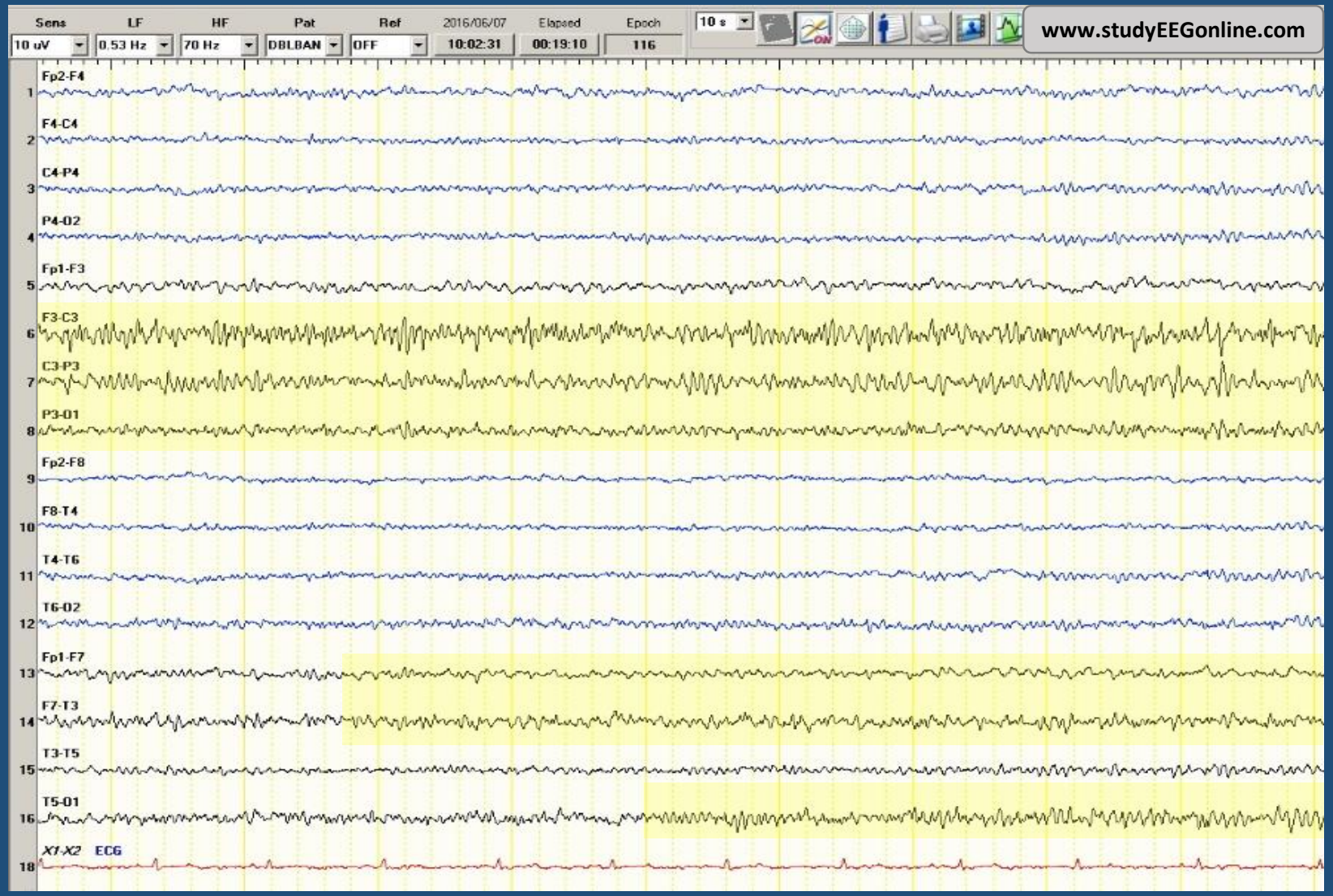
Focal Seizure with Altered Awareness: **Onset**



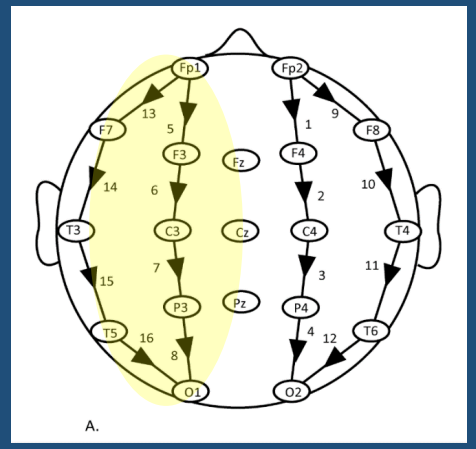
Gradual emergence of fast beta rhythm over F3, C3 and P3



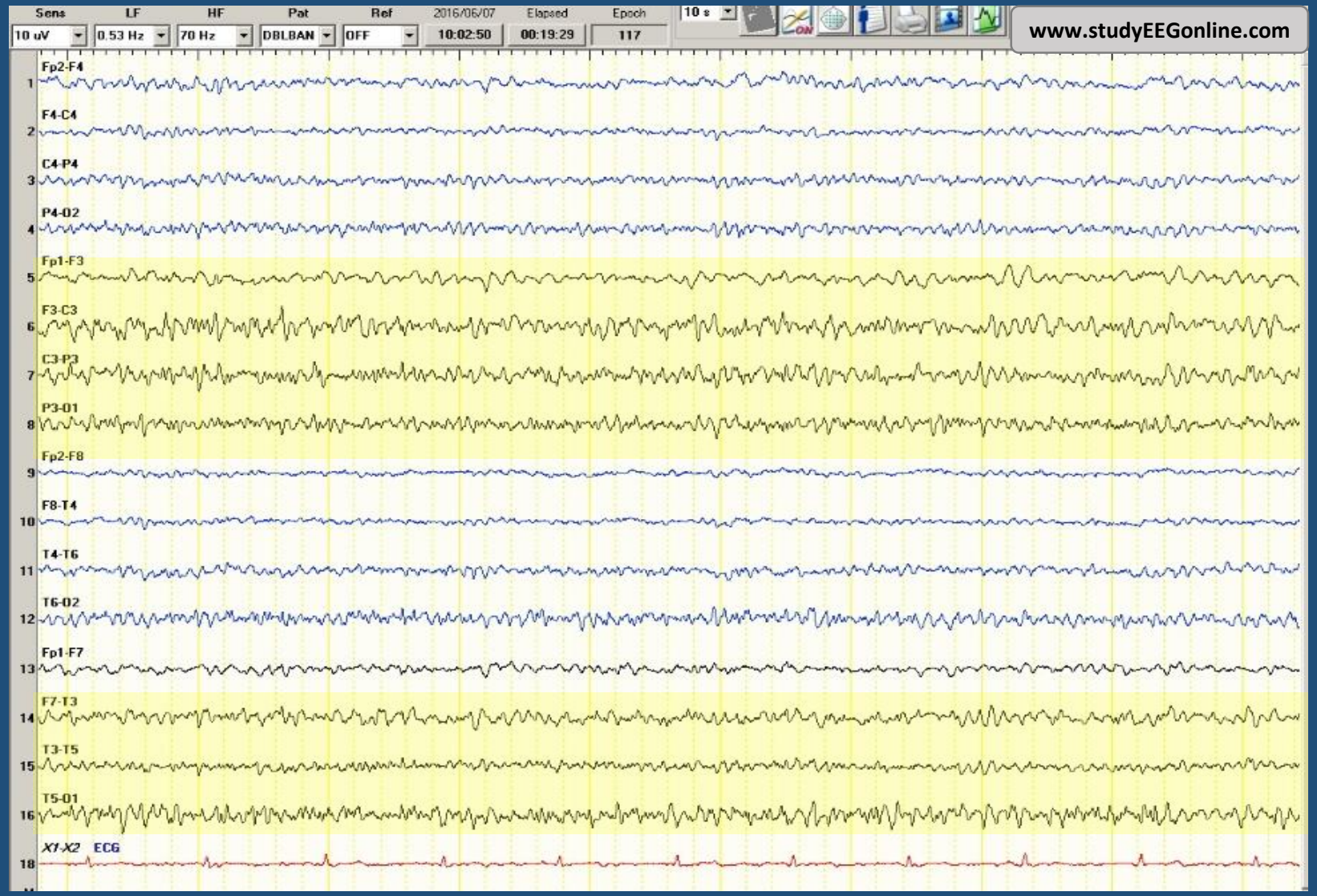
Focal Seizure with Altered Awareness: **Evolution**



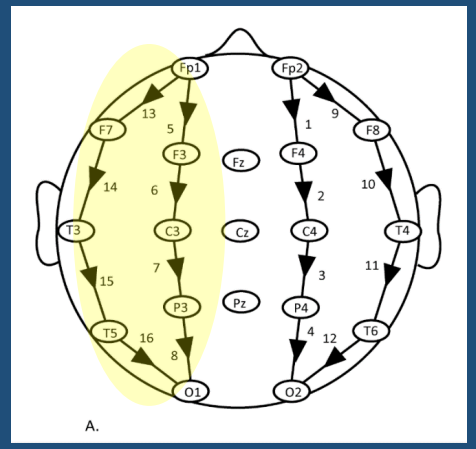
Gradual evolution (reducing frequency and increasing amplitude) and involvement of temporal channels



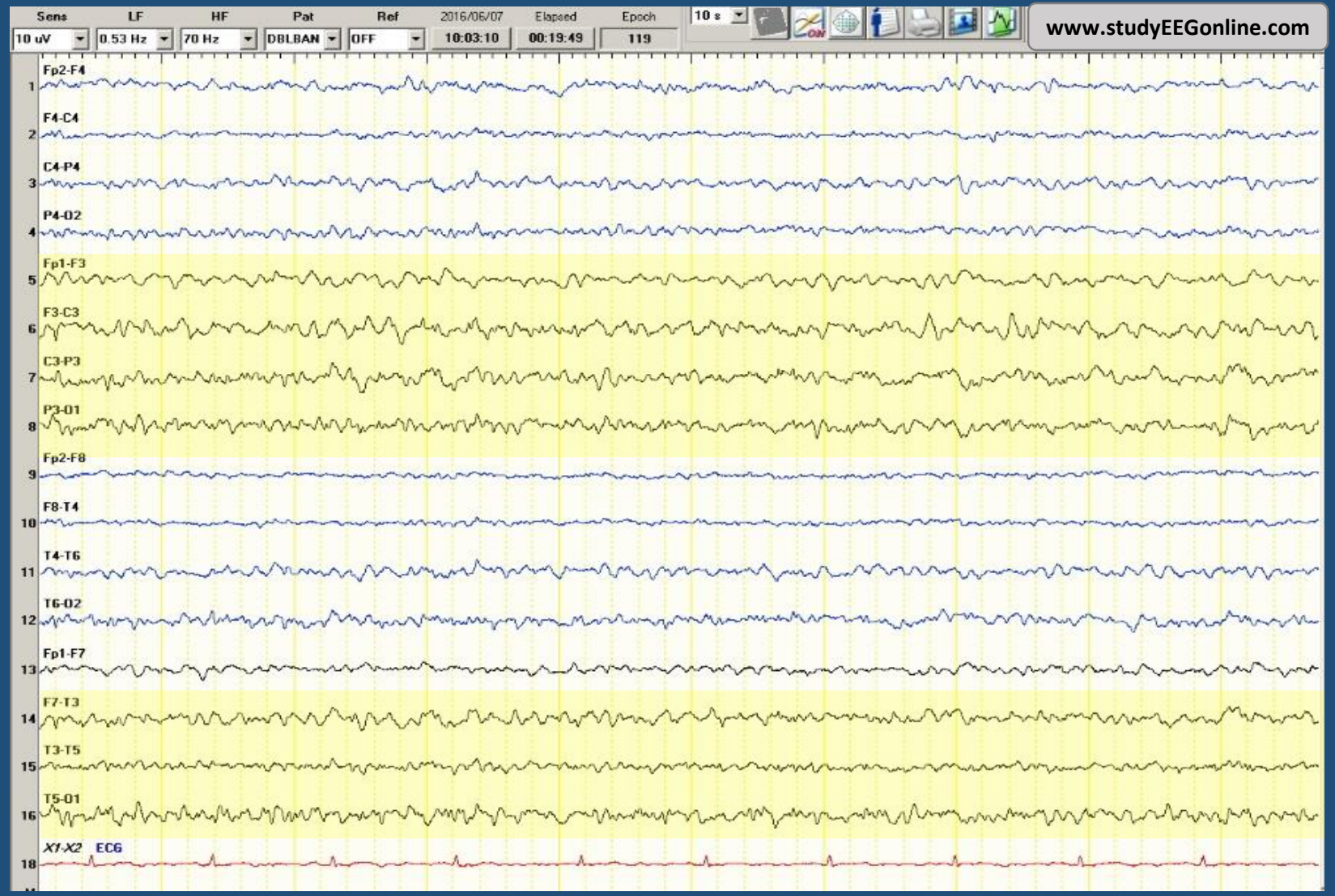
Focal Seizure with Altered Awareness: **Evolution**



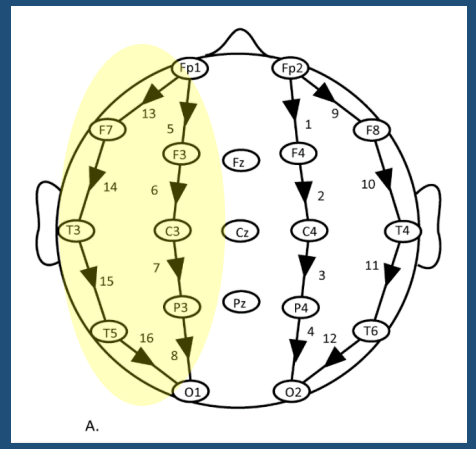
Continuing evolution (reducing frequency and increasing amplitude)



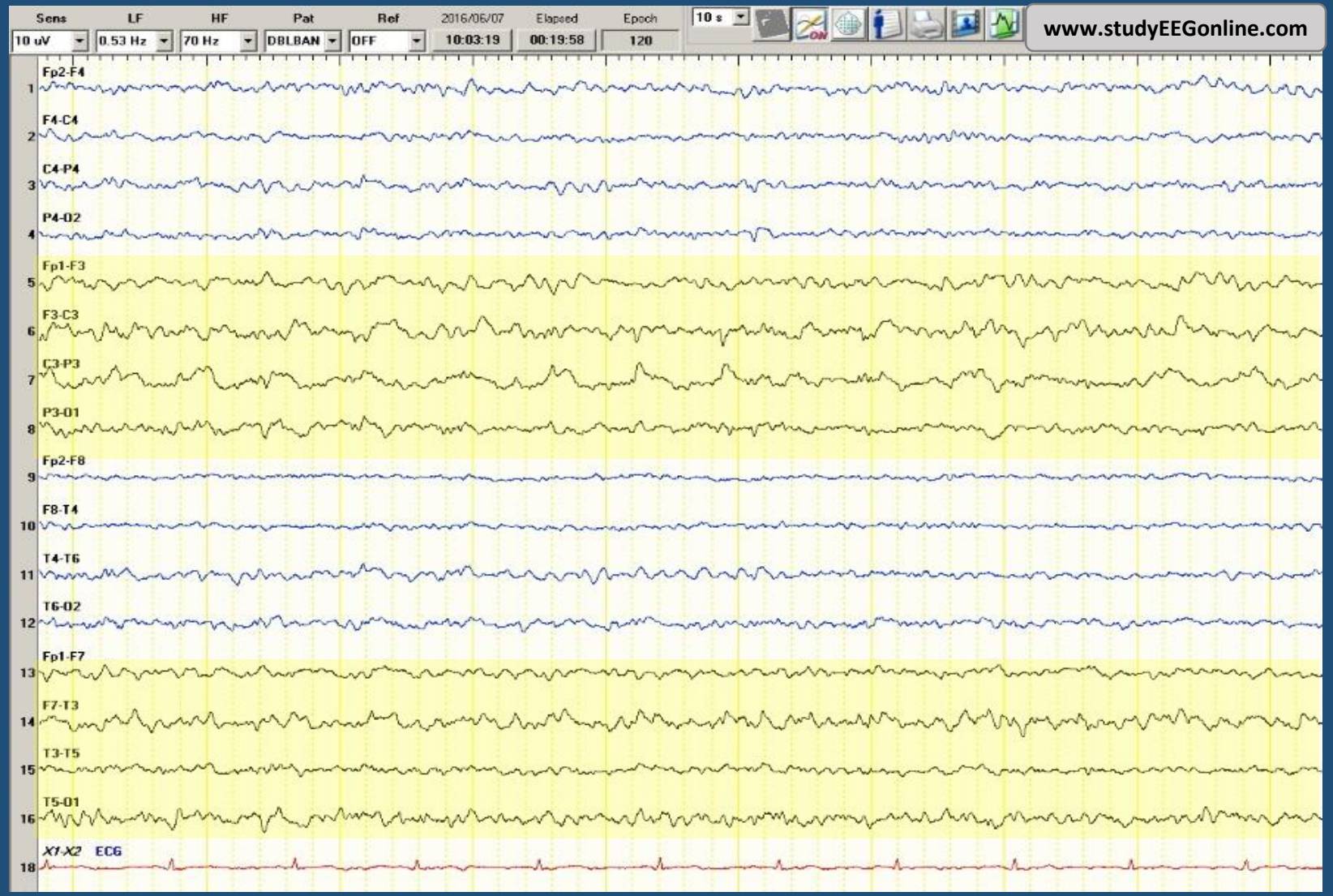
Focal Seizure with Altered Awareness: **Evolution**



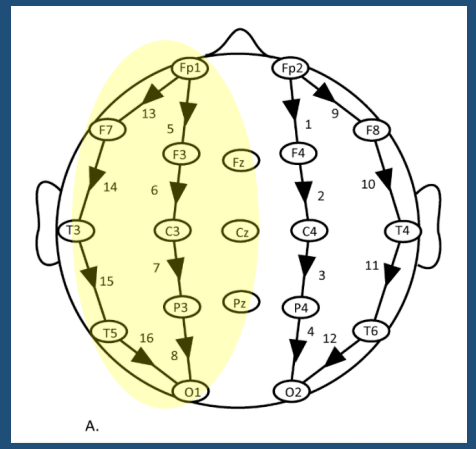
Continuing evolution (reducing frequency and increasing amplitude)



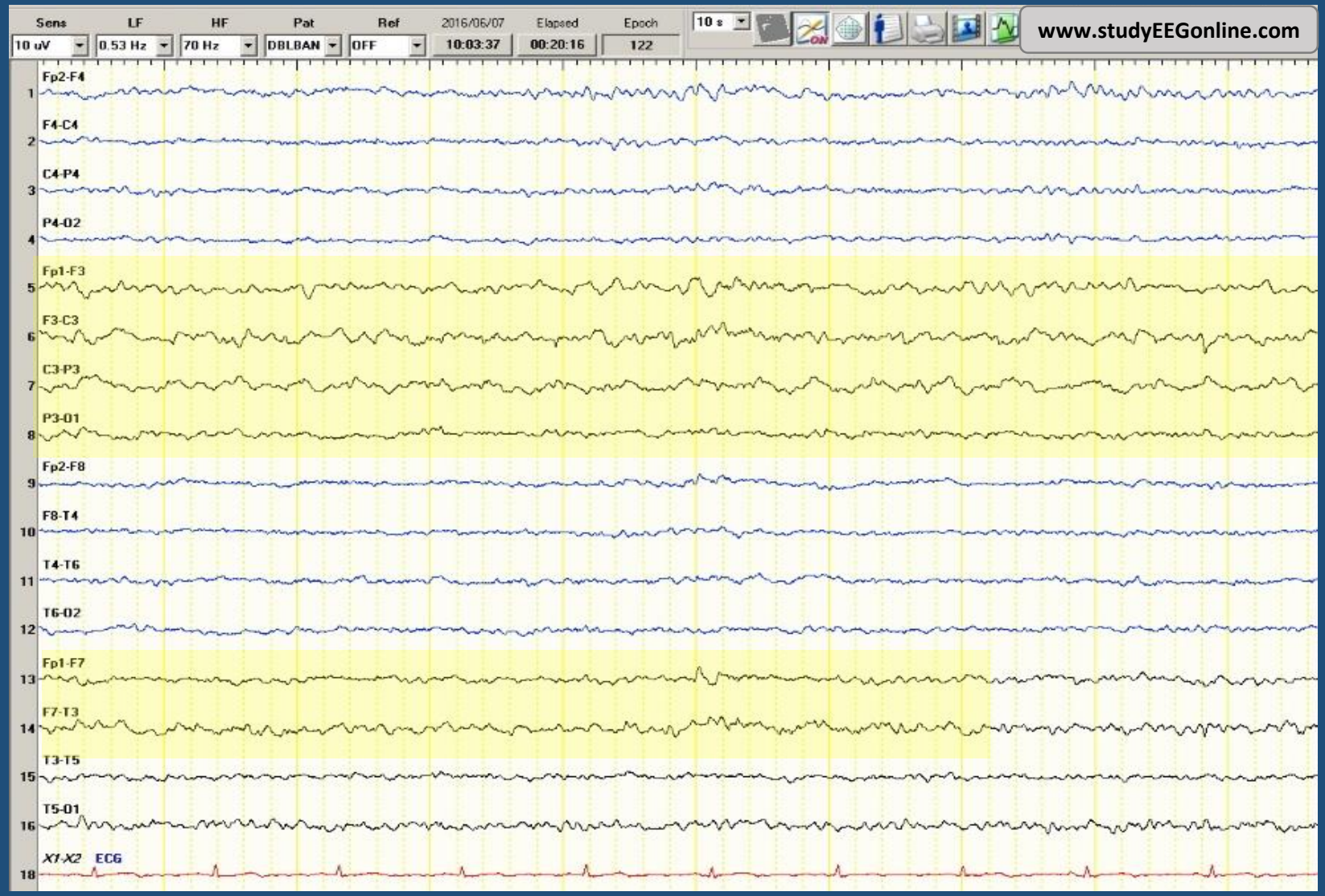
Focal Seizure with Altered Awareness: **Evolution**



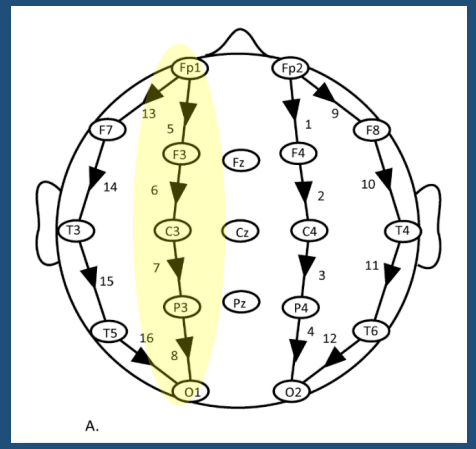
Continuing evolution (reducing frequency and increasing amplitude)



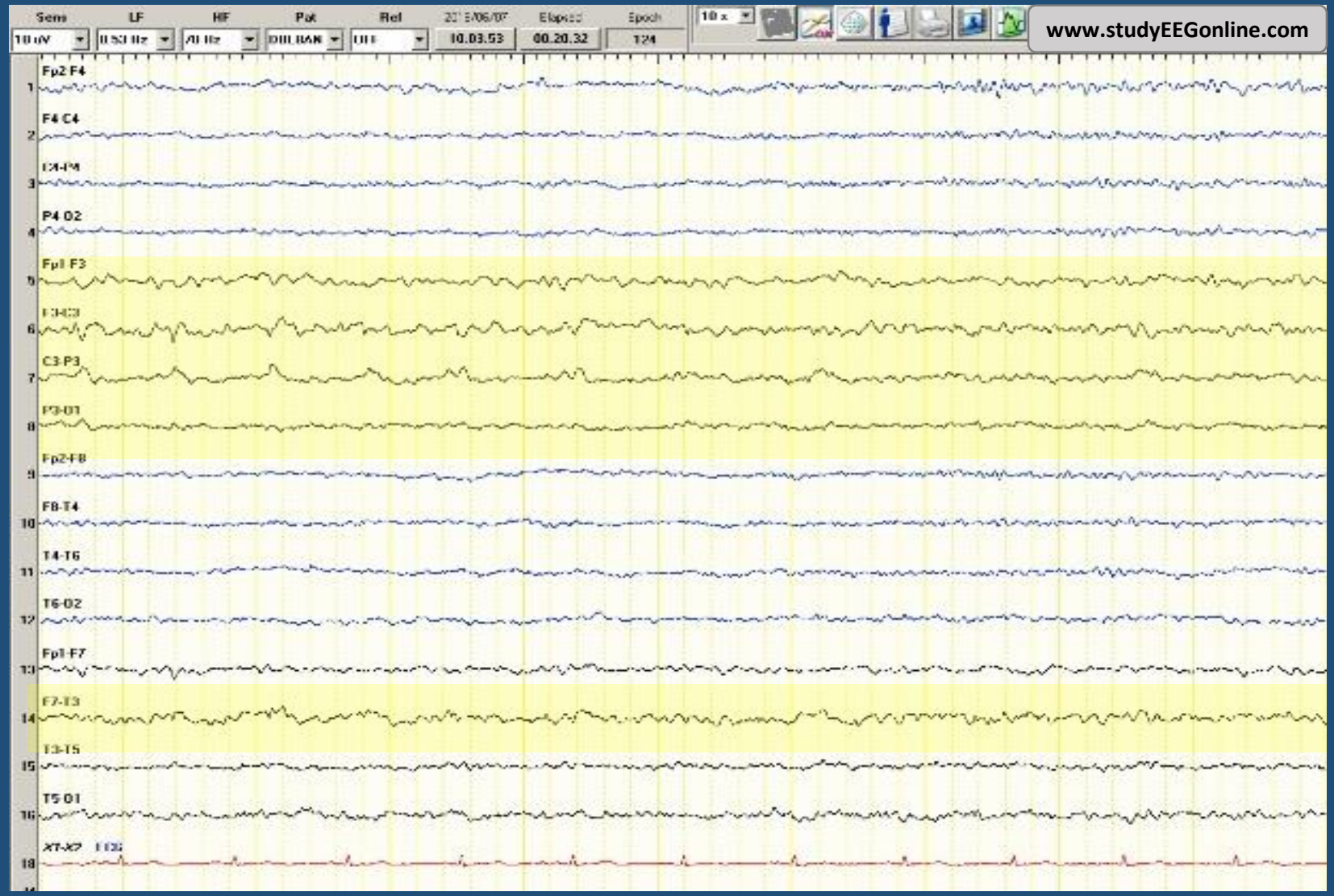
Focal Seizure with Altered Awareness: **Evolution**



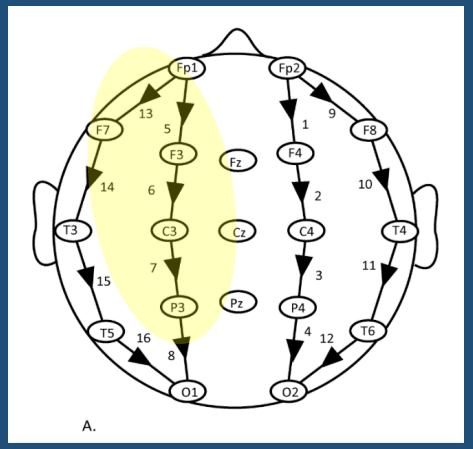
Continuing evolution (reducing frequency and increasing amplitude)



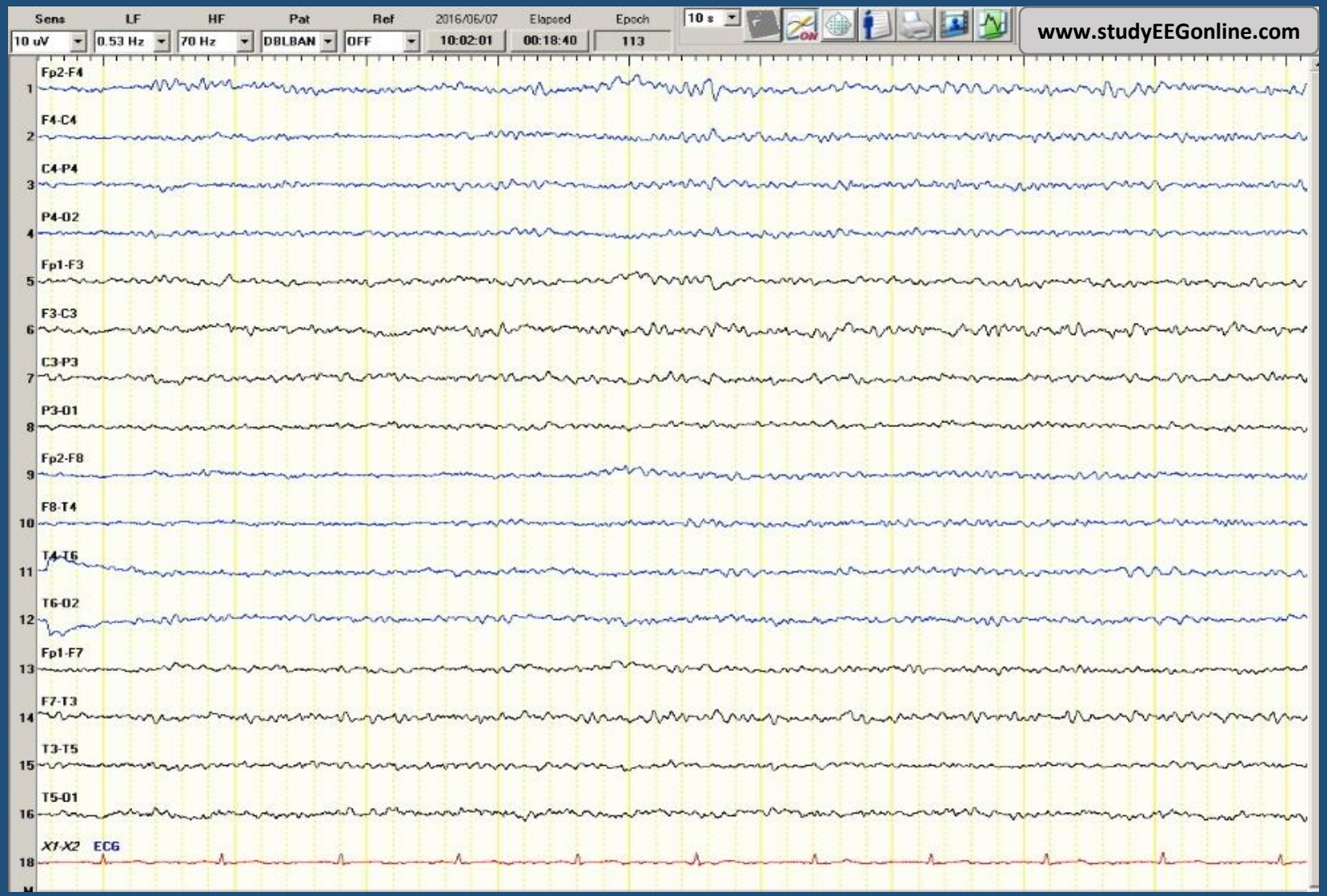
Focal Seizure with Altered Awareness: **Offset**



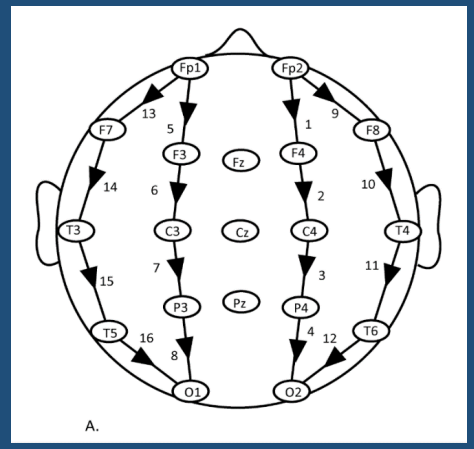
Continuing evolution (reducing frequency and increasing amplitude)



Focal Seizure with Altered Awareness: **Post-Ictal**

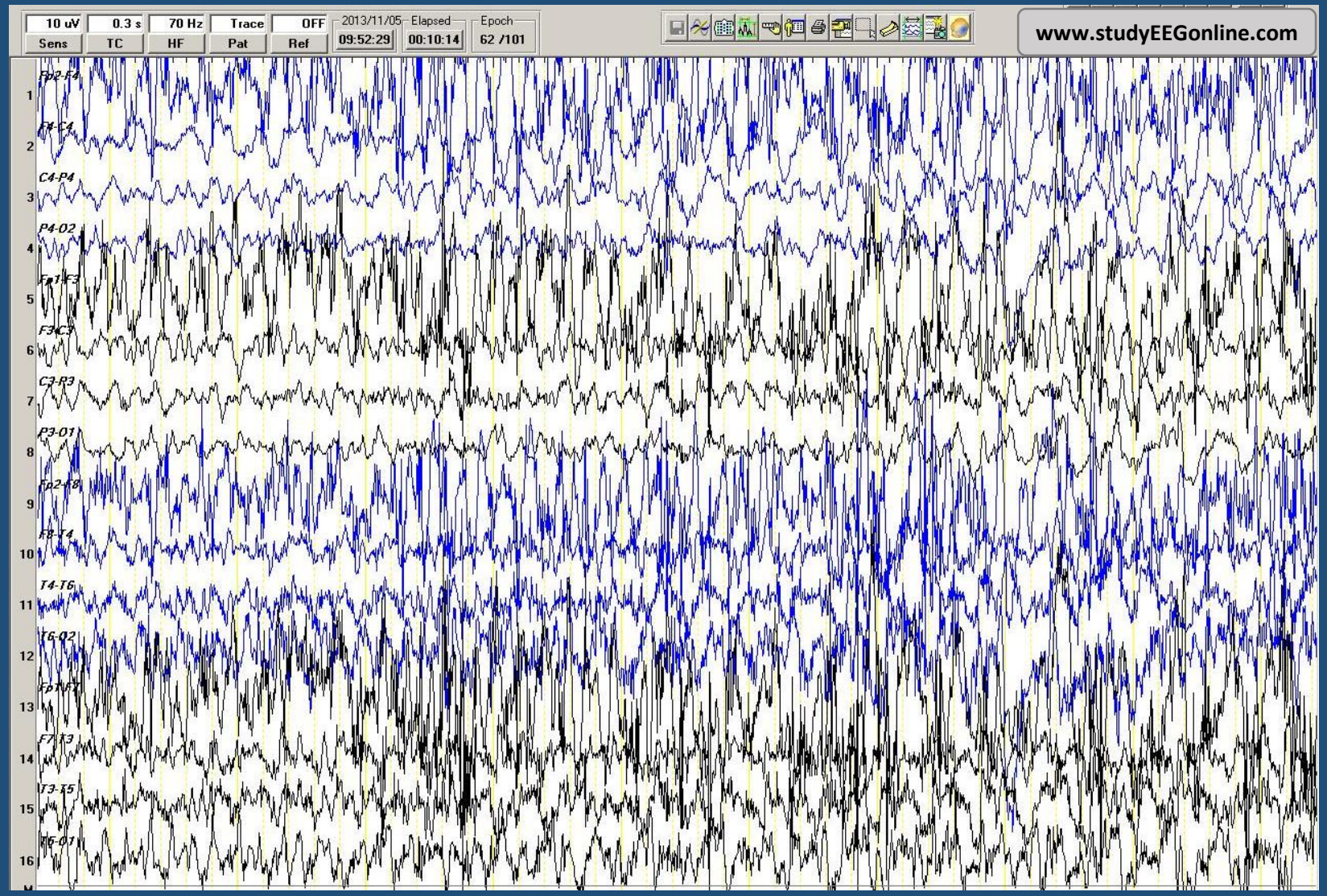


Back to base-line

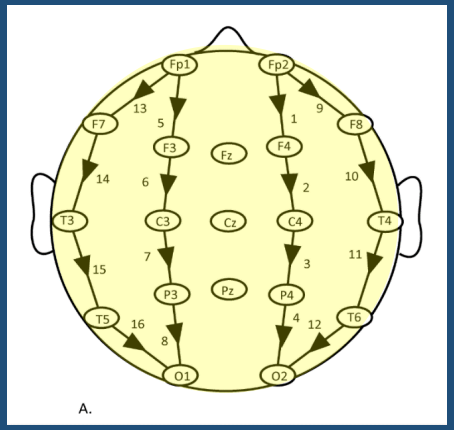


Status Epilepticus

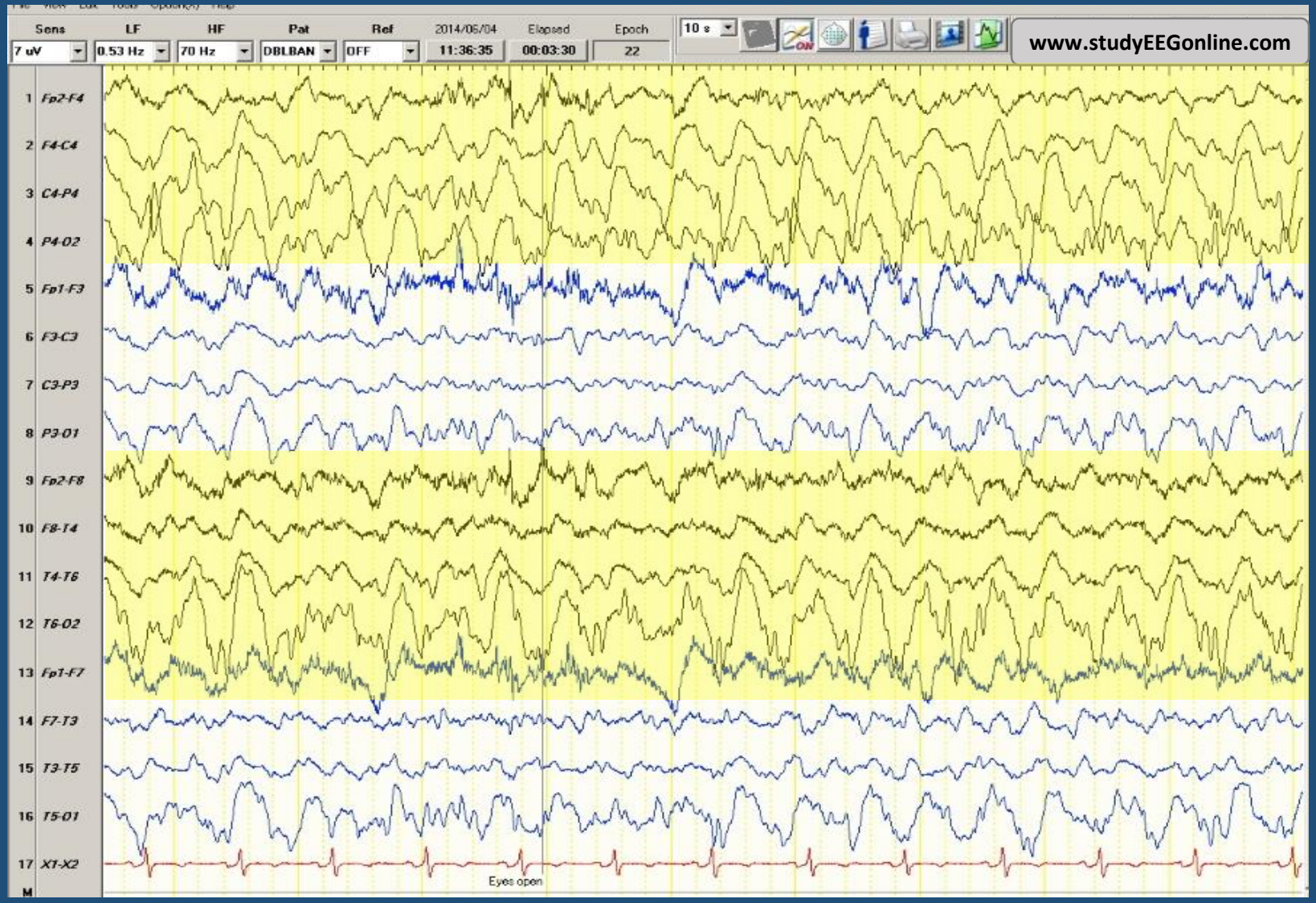
Generalised Status Epilepticus



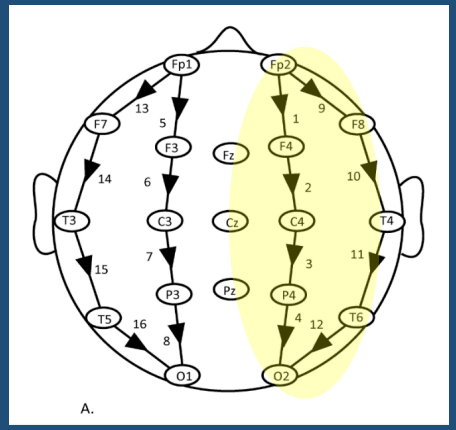
Continuous generalised epileptiform activity



Focal Status Epilepticus: Right Hemisphere

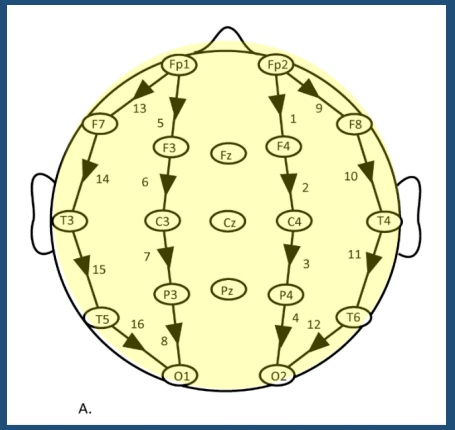


Continuous focal epileptiform activity



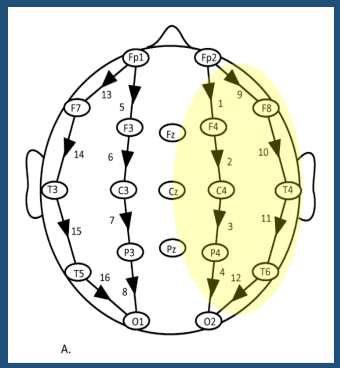
PEDS (Periodic Epileptiform Discharges)

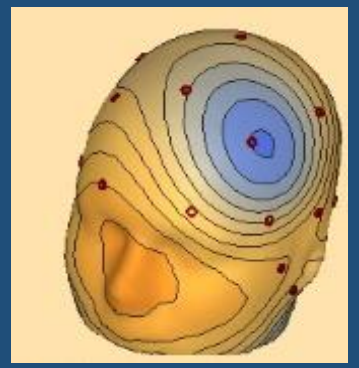
Continuous generalised periodic epileptiform discharges (PEDS)



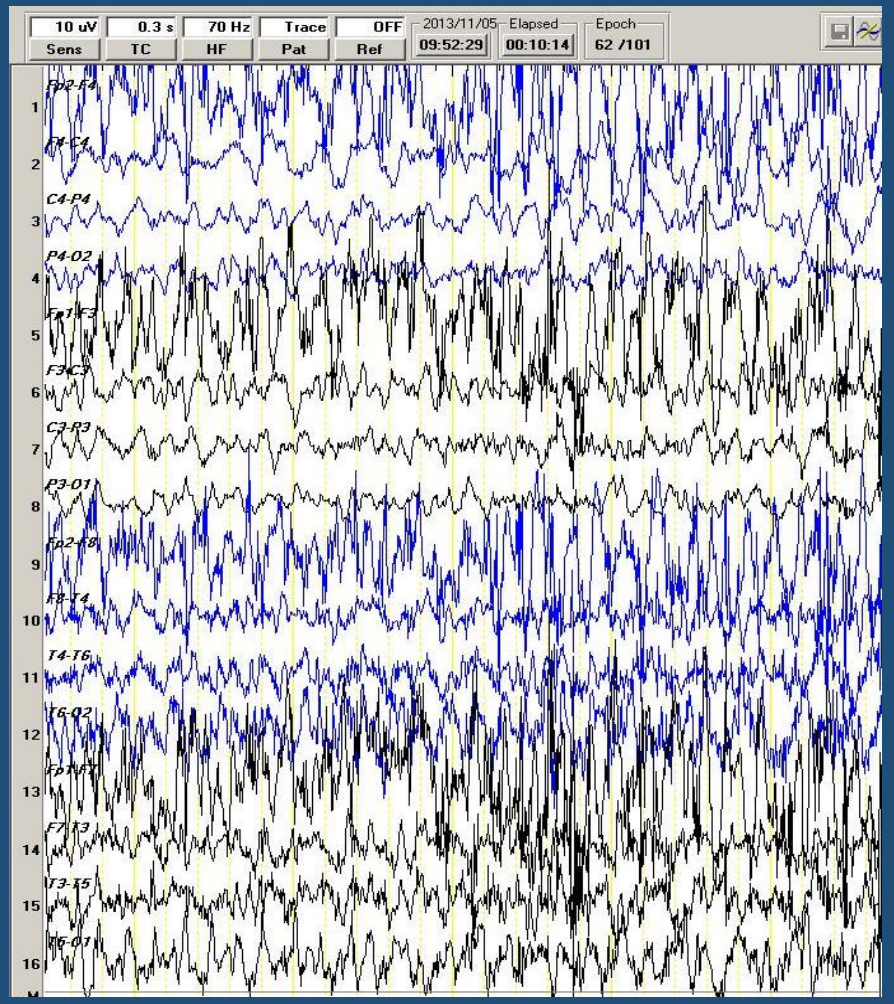
PLEDS (Periodic Lateralised Epileptiform Discharges)

Continuous lateralised periodic epileptiform discharges (PLEDS)

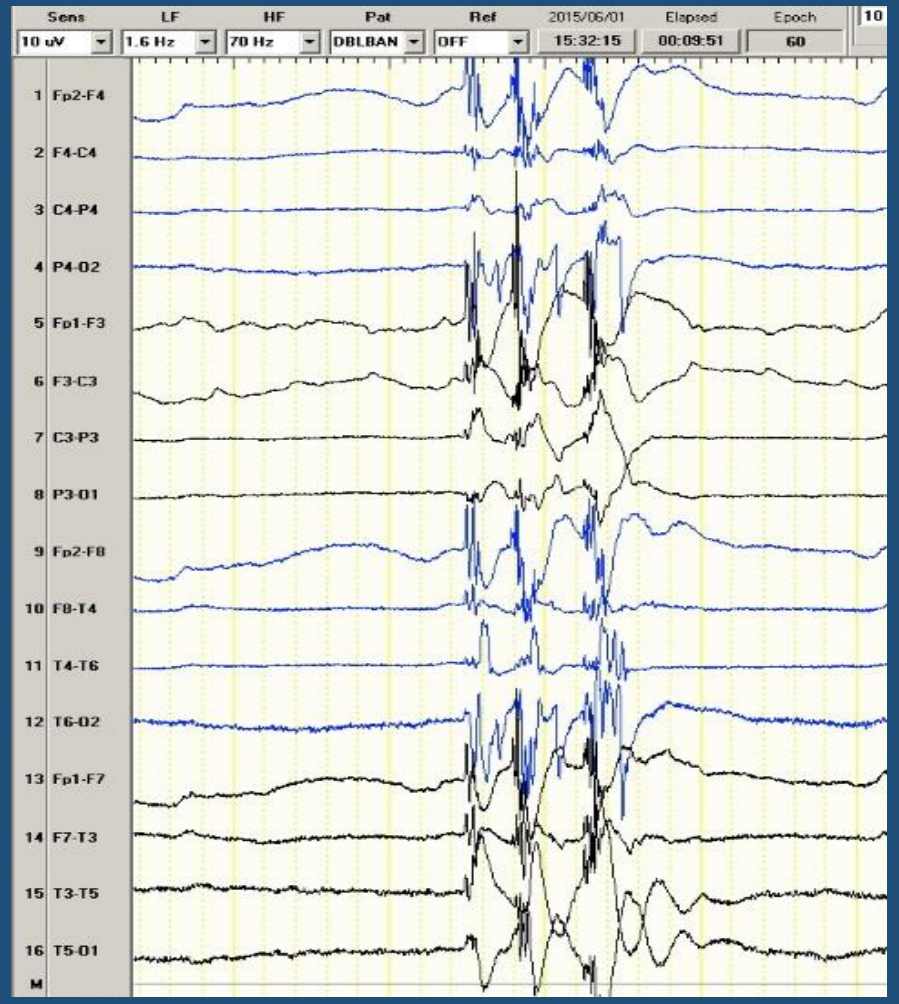




Burst Suppression



General Anaesthesia



Status Epilepticus

Burst Suppression

Non-Epileptiform EEG Abnormalities

Unreactive Slowing

Most non-epileptiform abnormalities of the EEG take the form of **slowing**; in other words transient waves or rhythms with frequencies lower than 8Hz and in the **theta** and **delta** ranges.

However, it is crucial to keep in mind that **not all slowing is pathological**. For instance, normal drowsiness and sleep are associated with physiological slowing of the EEG

Unreactive Slowing

As a rule of thumb:

- **Generalised slowing** is **non-specific** and indicates **encephalopathy** of many possible causes

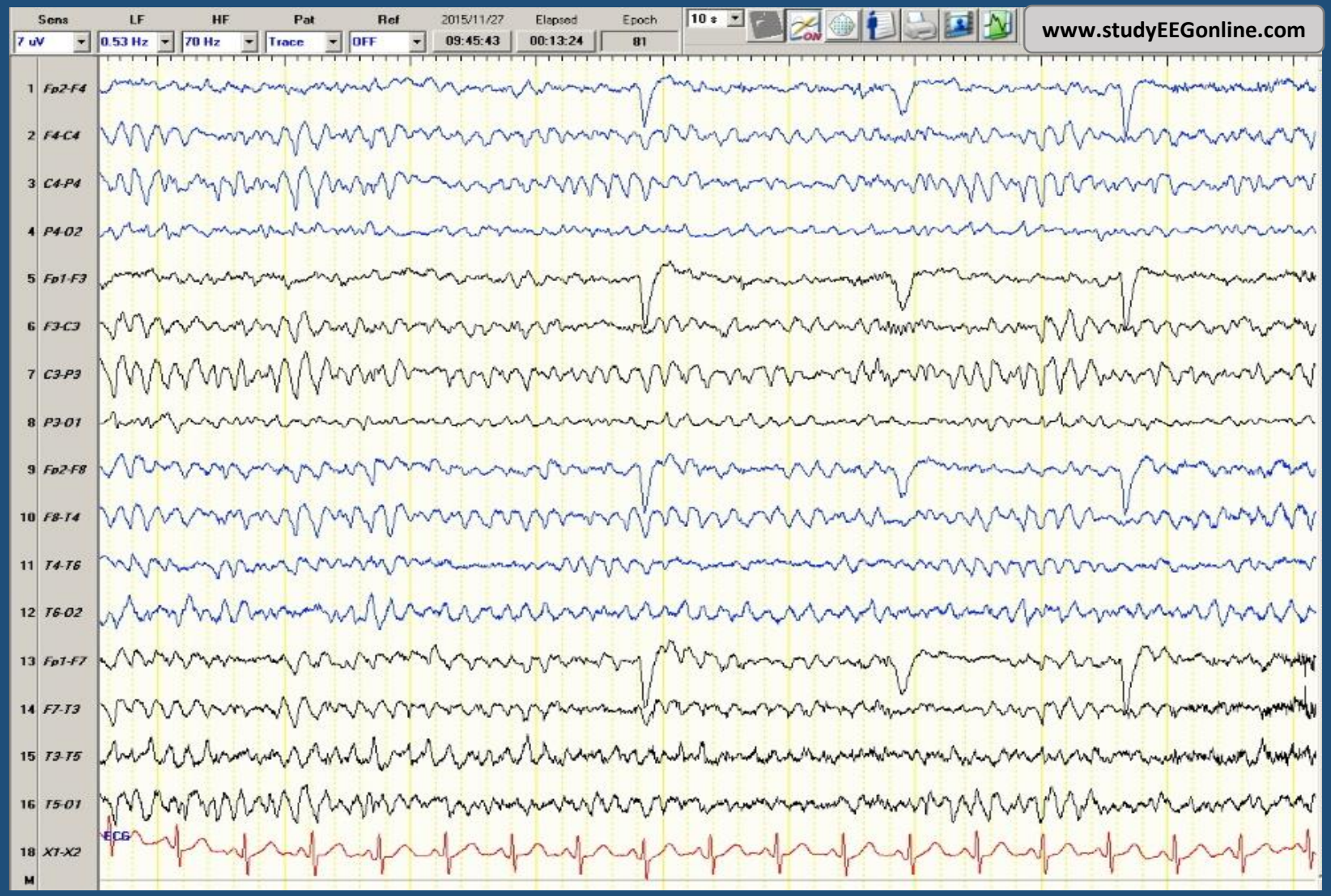
- **Focal slowing** suggests **focal cerebral dysfunction**

  (e.g. stroke, tumour, focal encephalitis)

- The **slower the rhythm, the more pathological it is**

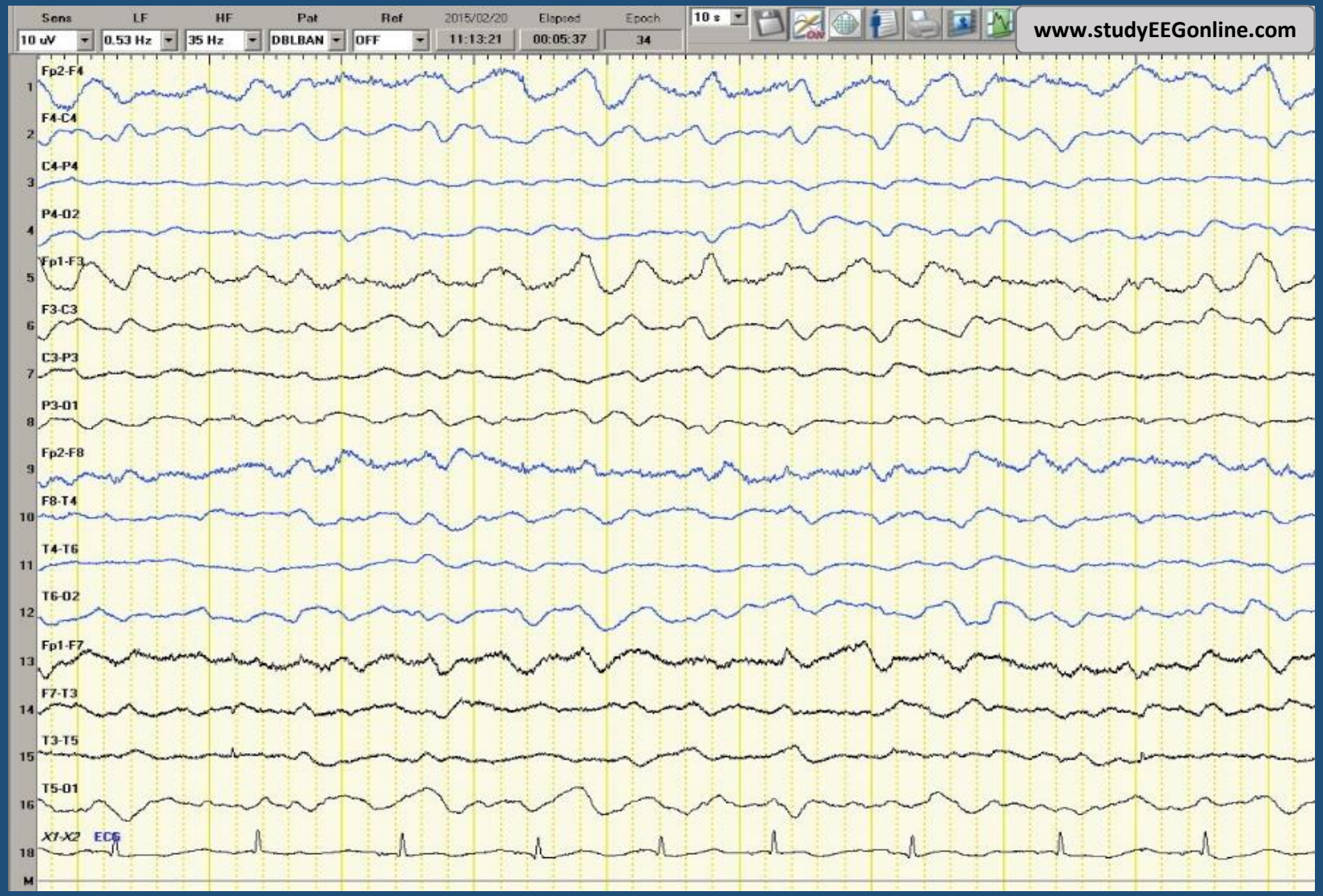
  (i.e. delta is more pathological than theta)

Generalised Slowing: **Theta**



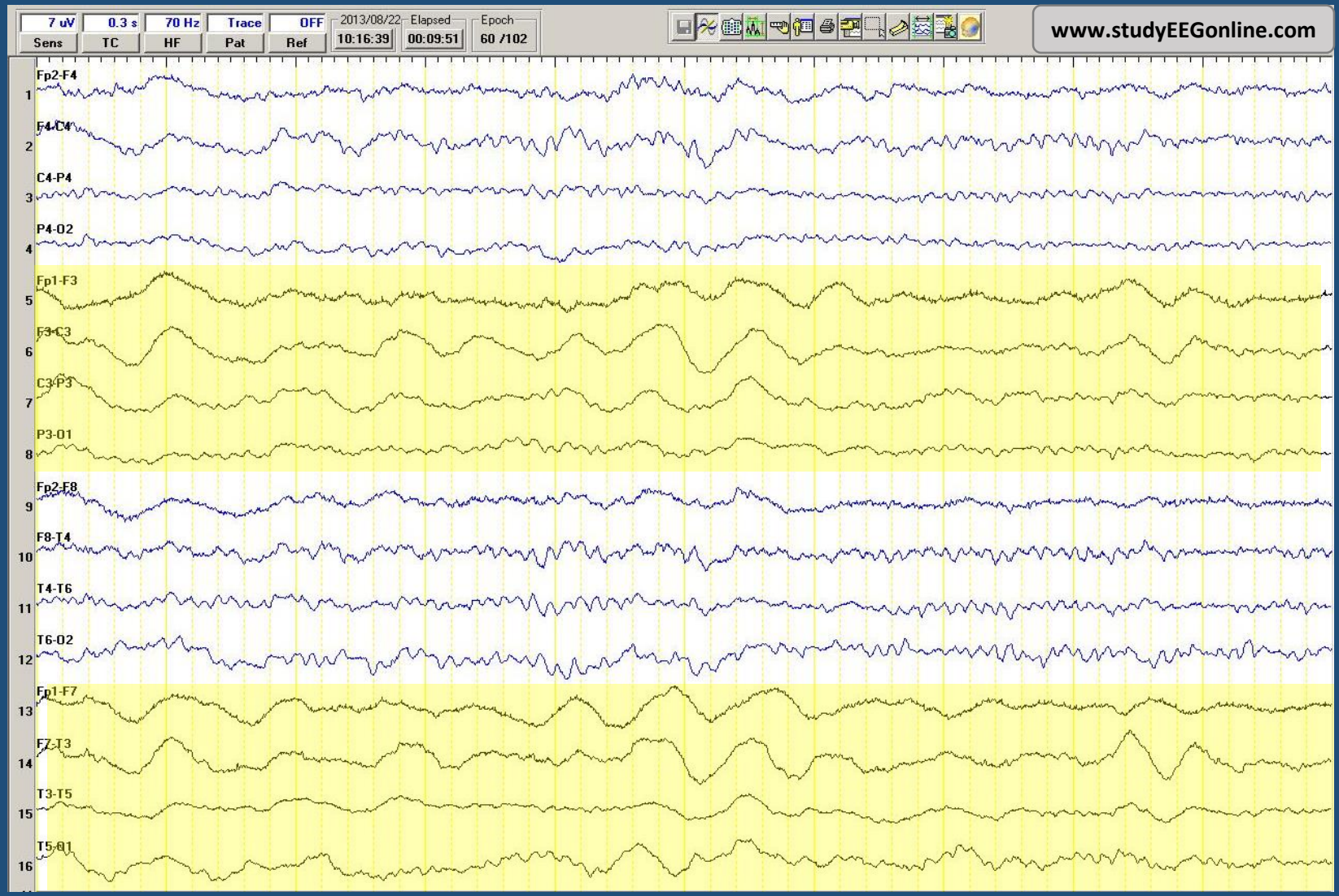
In this epoch the background (PDR) consists of generalised 6 Hz theta which is consistent with mild encephalopathy, unless the patient is drowsy.

Generalised Slowing: **Delta**



In this epoch the background (PDR) consists of generalised 2-3 Hz delta which is consistent with moderate to severe encephalopathy, unless the patient is in slow wave sleep.

Focal Slowing: Left Hemisphere, Delta



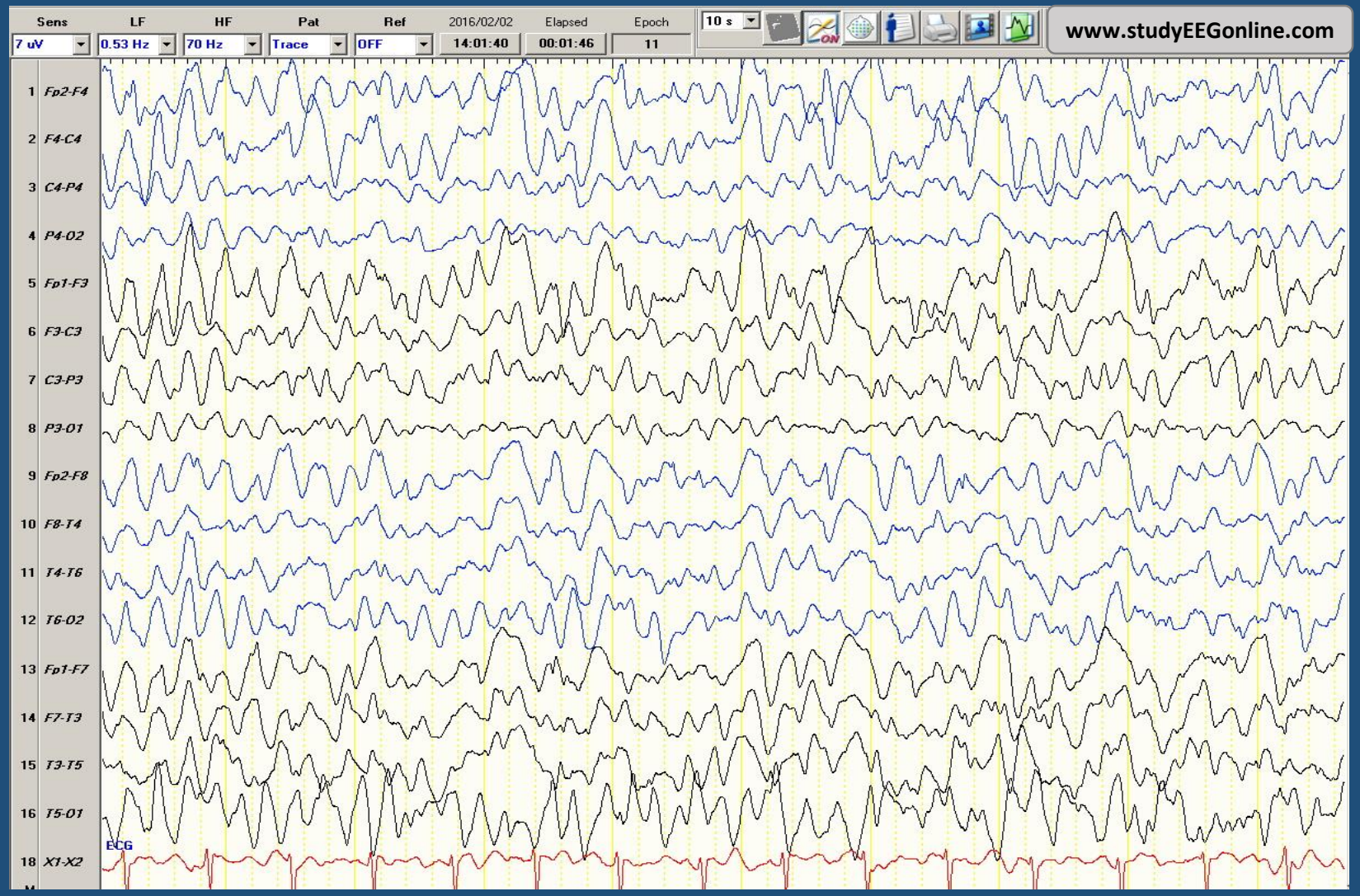
Here focal 2-3 Hz delta affects all channels over the left side of the head. This suggests an underlying structural intracranial abnormality in the left hemisphere.

Triphasic Waves

Triphasic waves
are commonly associated with metabolic derangements and especially
hepatic encephalopathy

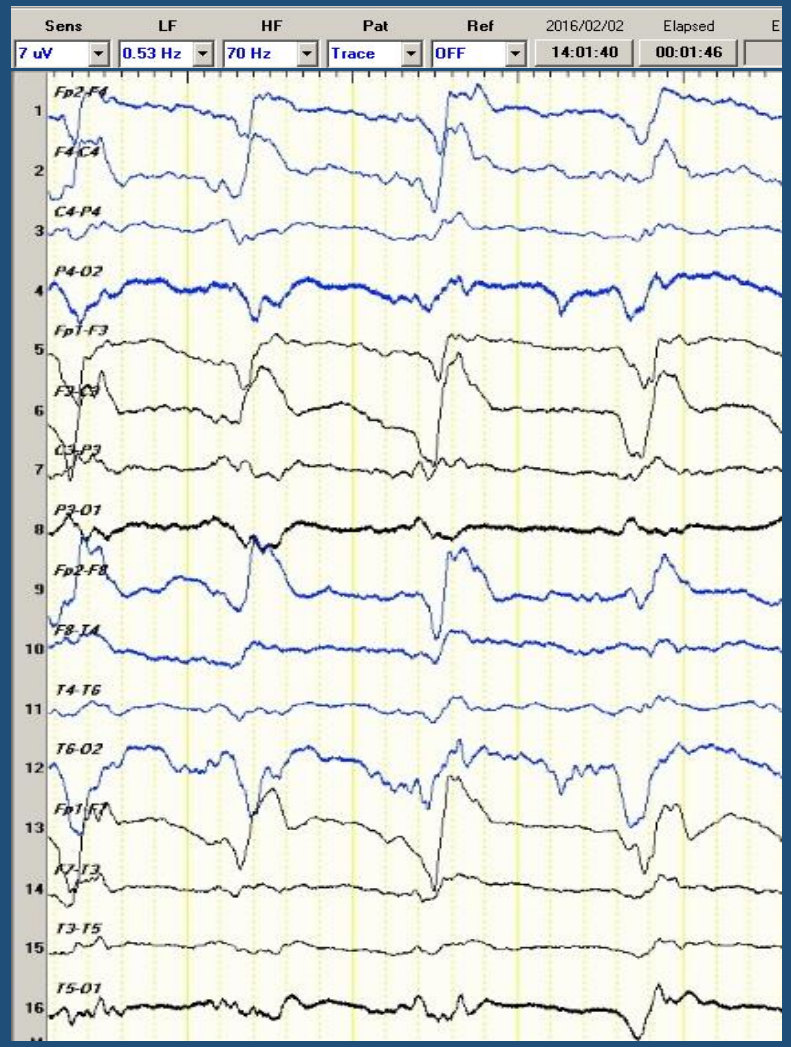
Depression of Consciousness & Coma

Persistent, Unreactive, Polymorphic Delta

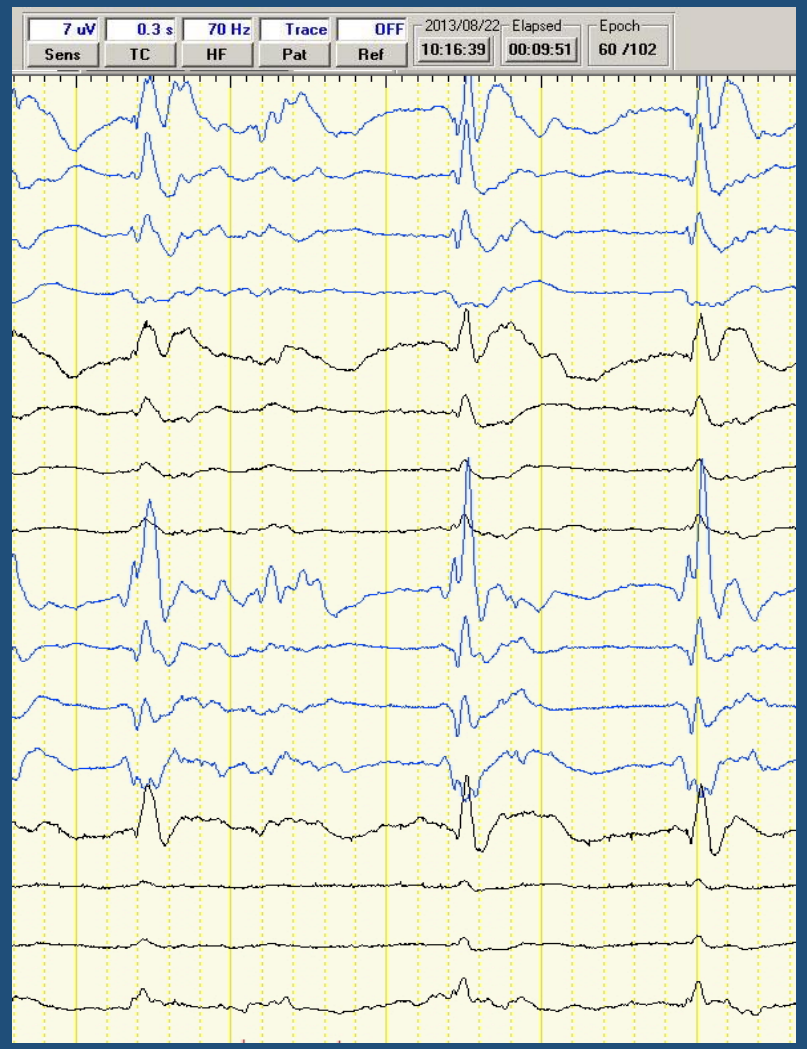


Persistent, generalised, unreactive, polymorphic delta activity is non-specific but implies severe diffuse encephalopathy

PEDS (Periodic Epileptiform Discharges)



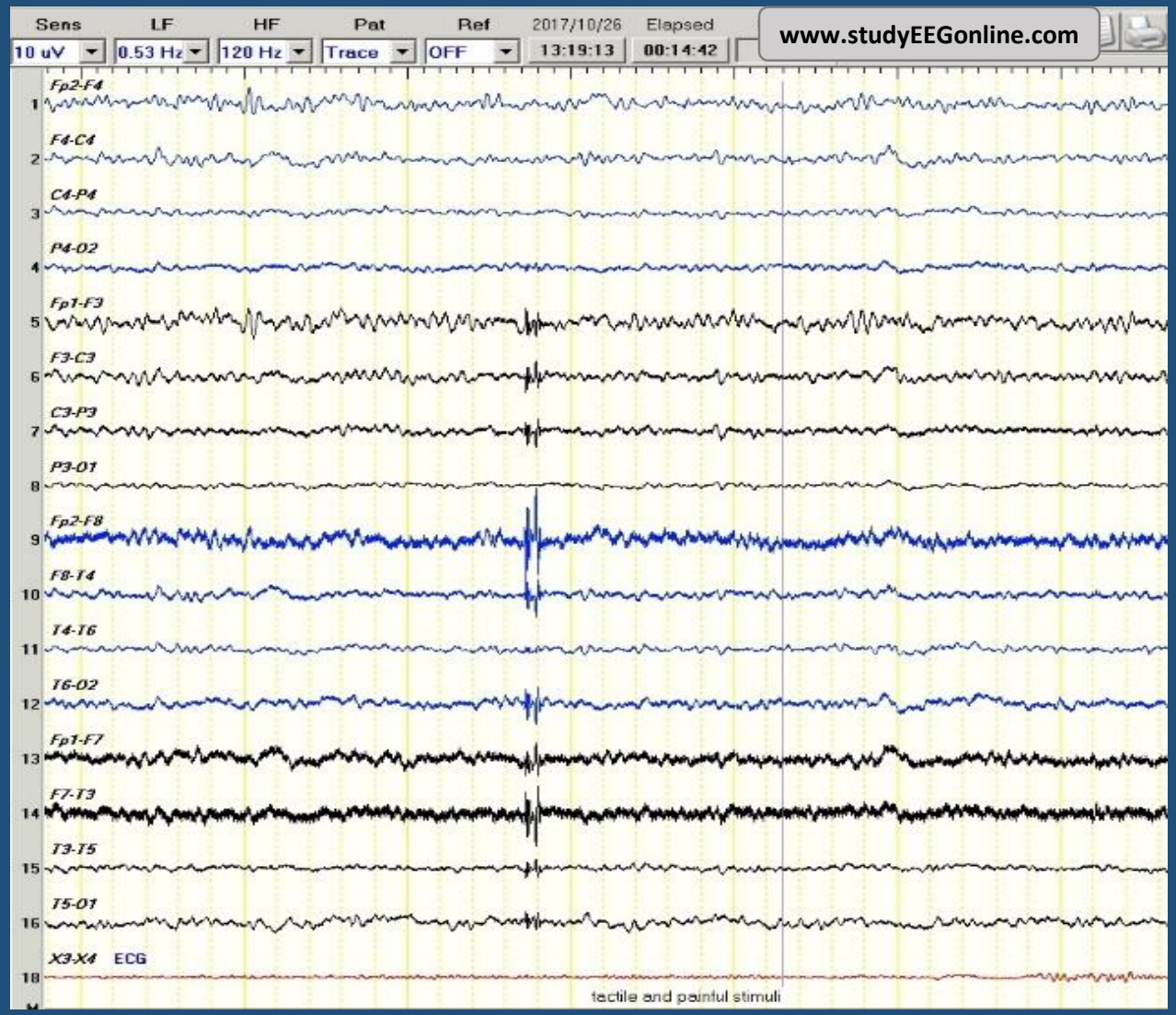
PEDS



PLEDS

PEDS / PLEDS
imply
subclinical status
epilepticus

Alpha Coma

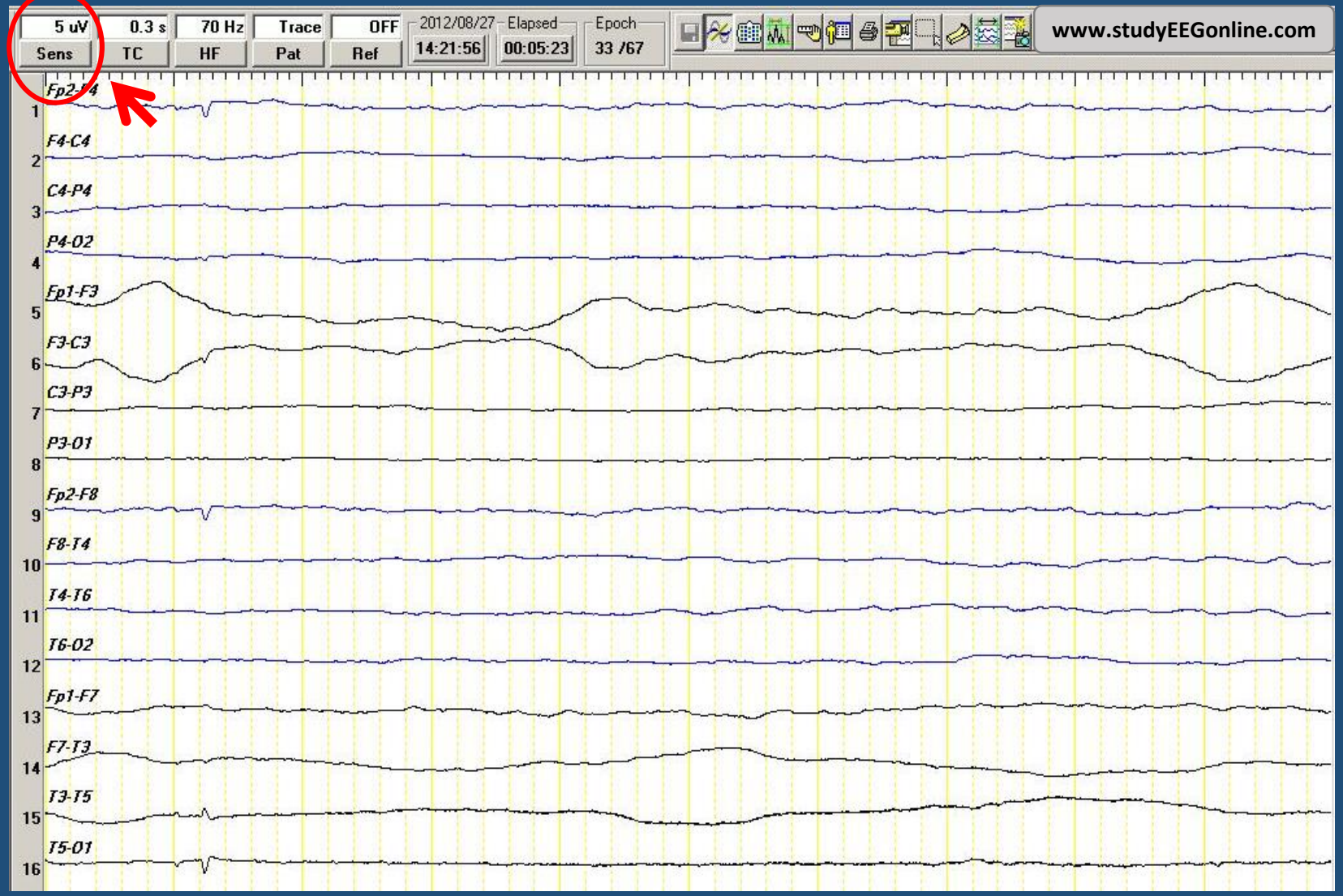


Unreactive to Painful Stimulus

Occasionally the EEGs of patients in severe coma may show generalised unreactive alpha frequencies.

This is typically associated with a poor prognosis

Suppression & Electro-cerebral Silence



Suppression implies reduced cerebral activity while **electro-cerebral silence** indicates that no detectable electrical activity is present

It is important to keep in mind that **suppression and electro-cerebral silence are not necessarily pathological**, and may be reversibly induced in normal people by anaesthetic drugs, hypothermia, and some toxic states

What we *have* covered

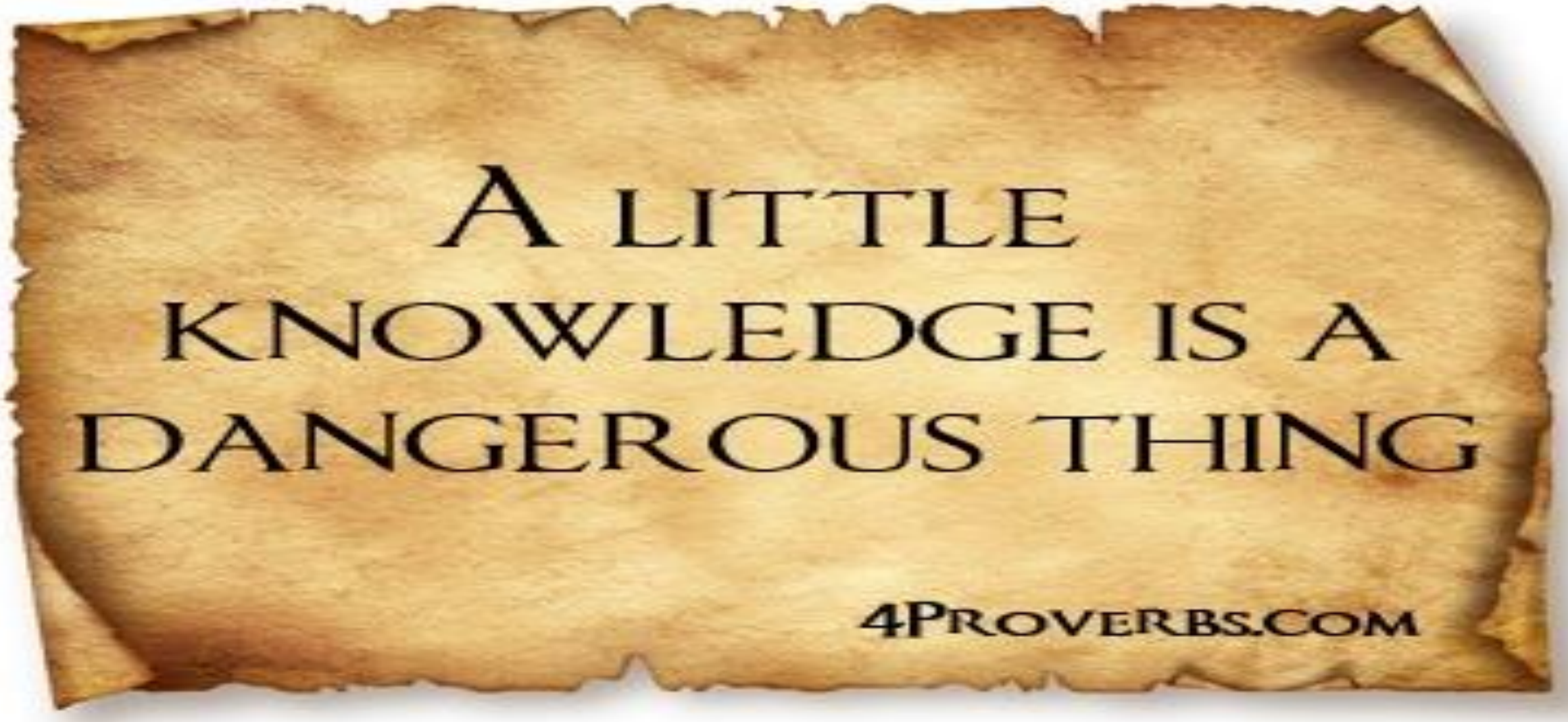
The Basics of:

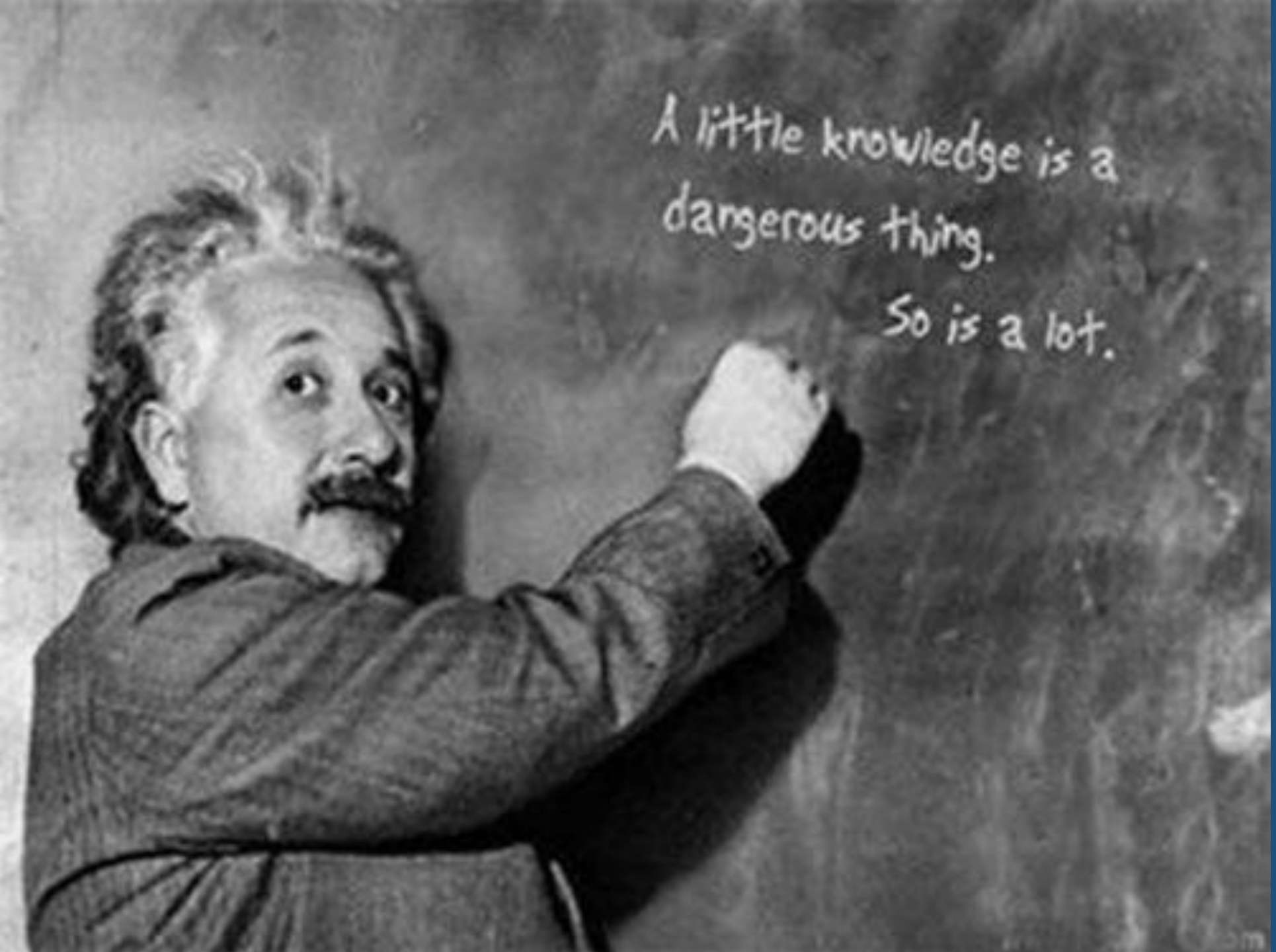
- Generation of Electrical Discharges in the Brain
- Electrical Fields & Dipoles
- Electrode Placement (10-20 and 10-10 System)
- Montages
- Normal EEG in Wakefulness and Drowsiness
- Epileptiform Waveforms
- Non-Epileptiform Waveforms.

What we *have not* covered

Many normal variants and artefacts closely resemble pathological waveforms and which can result in the wrong diagnosis and serious harm

With respect to reading EEGs…




A little knowledge is a
dangerous thing.
So is a lot.