ROMA
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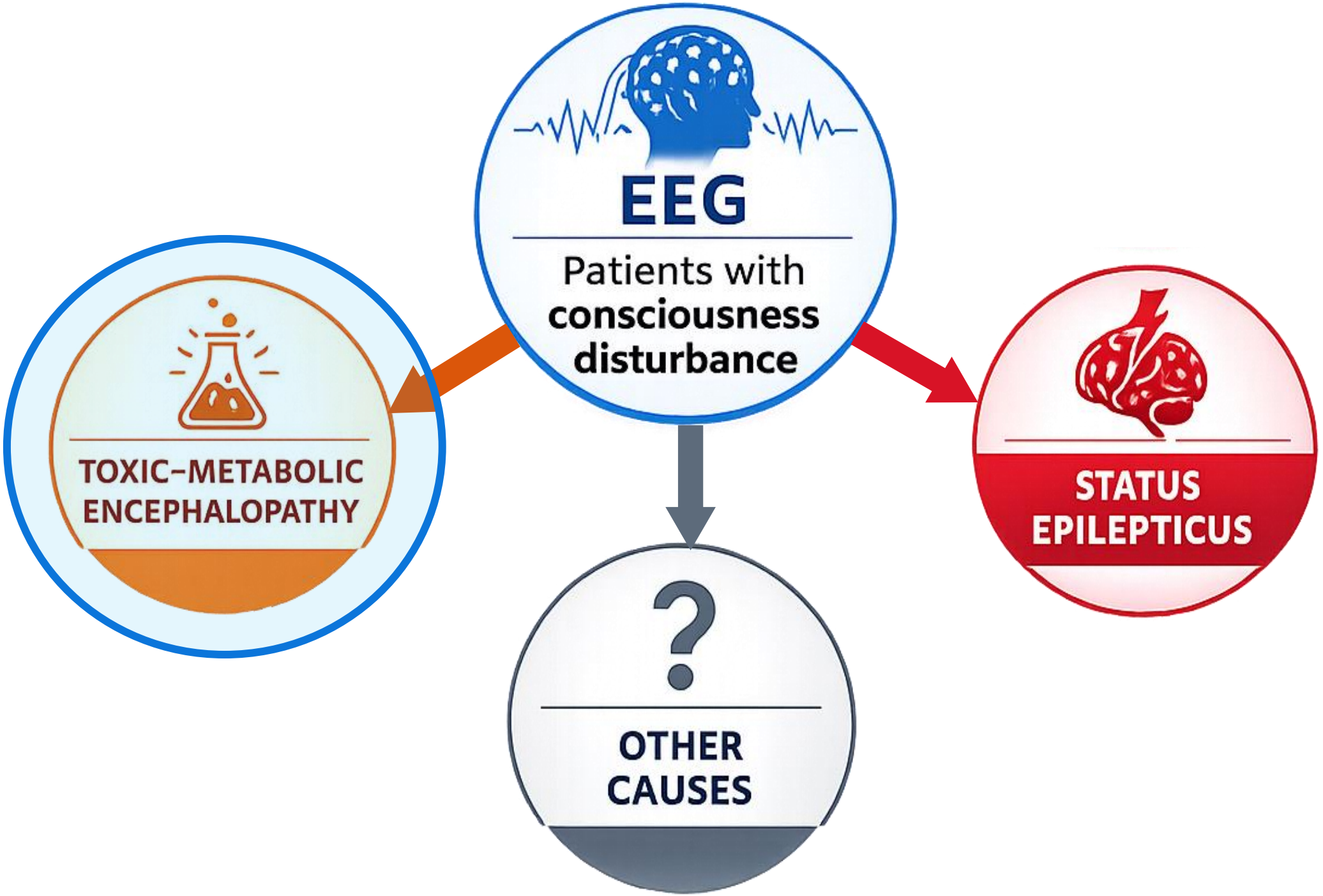
Encefalopatie, stato epilettico e demenze atipiche: il ruolo dell'EEG

Dott. Adolfo Mazzeo
Sapienza Università di Roma

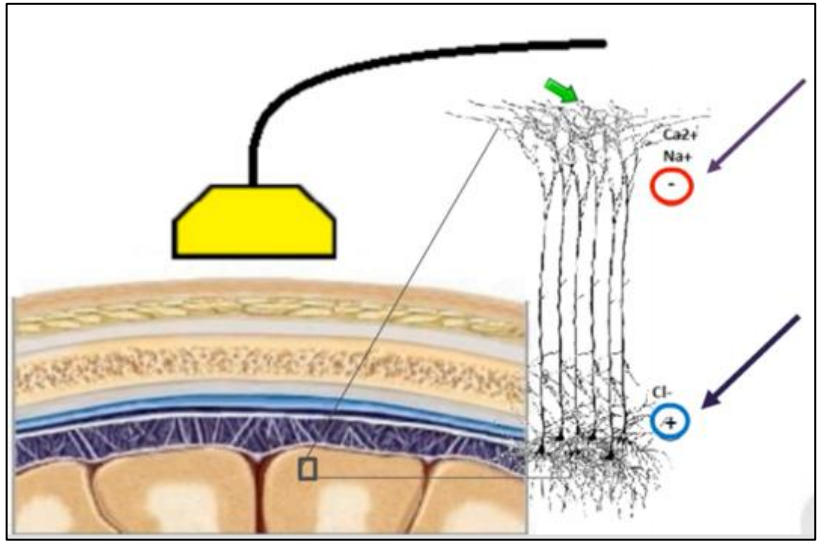
adolfo.mazzeo@uniroma1.it

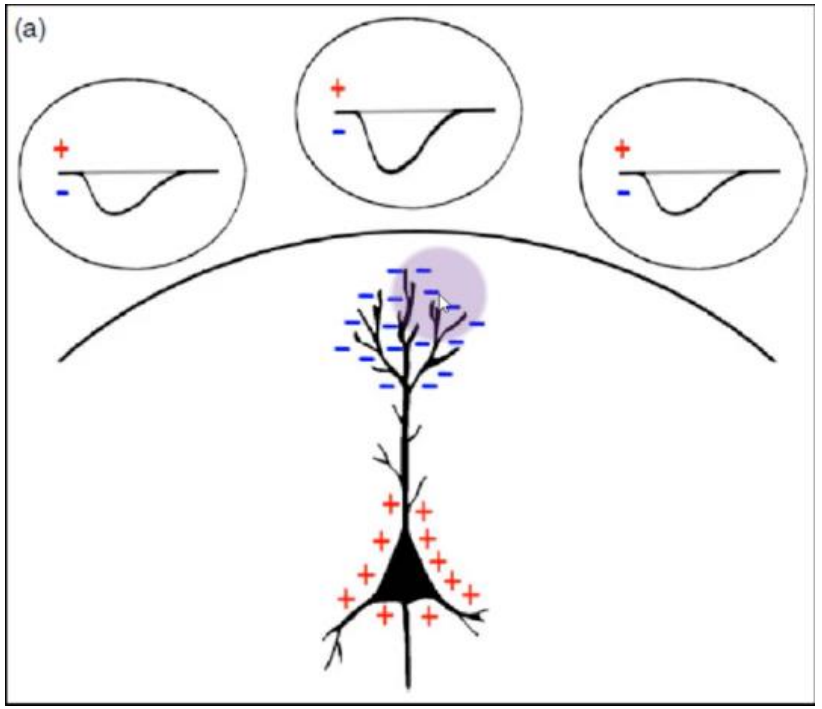

Occhi aperti
Occhi chiusi
FP - F8
F8 - T4
T4 - T6
T6 - O2
FP - F4
F4 - C4
C4 - P4
P4 - O2
FZ - CZ
CZ - PZ
FP1 - F3
F3 - C3
C3 - P3
P3 - O1
FP1 - F7
F7 - T3
T3 - T5
T5 - O1


EEG
Patients with consciousness disturbance
TOXIC–METABOLIC ENCEPHALOPATHY
STATUS EPILEPTICUS
?
OTHER CAUSES

Advatanges of EEG

- High sensitivity
- Low specificity
- High temporal resolution
- (Moderate) spatial resolution
- Accessible, low-cost, non invasive

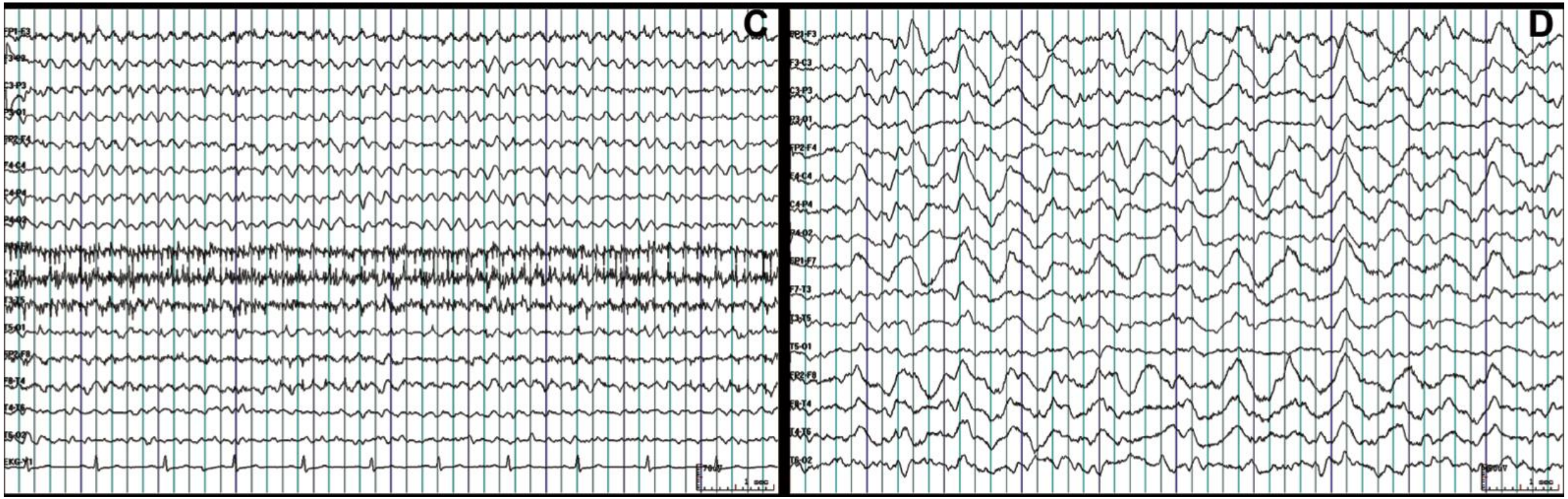




Toxic-metabolic encephalopathy

1. Slowing of the PDR
2. Slowing in wakefulness (Theta > delta)
3. Loss of the PDR, loss of normal sleep transients
4. Loss of reactivity to external stimuli
5. Burst attenuation/suppression
6. Diffuse attenuation/suppression
7. Electrocerebral inactivity

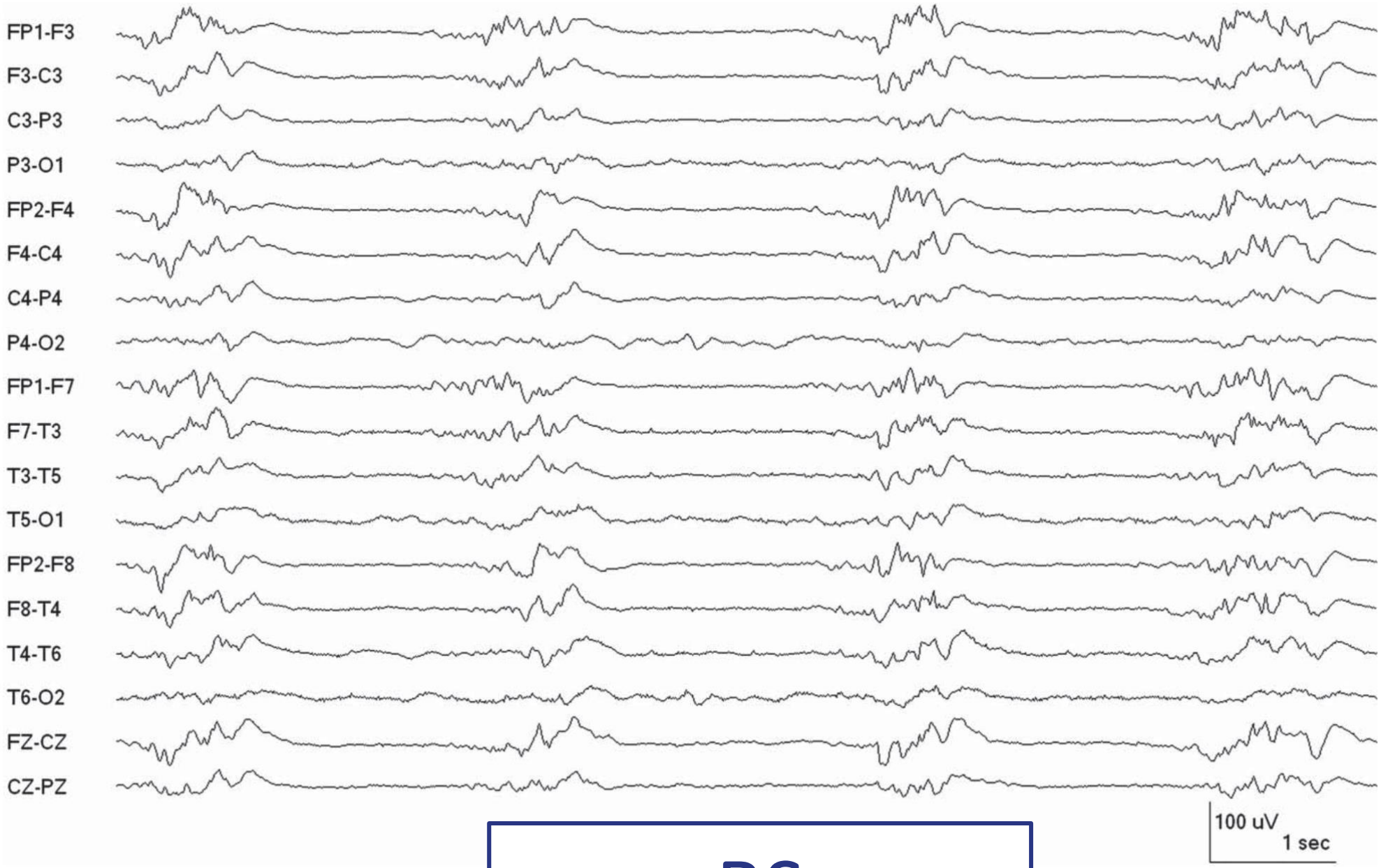
PROGRESSIVE BRAIN DYSFUNCTION



THETA COMA

DELTA COMA

Sutter and Kaplan, 2012



BS

Hirsch and Brenner

ALFA COMA

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Electroencephalographic patterns in coma: When things slow down

December 2012

Lab: Neurointensive Care Study Group

Raoul Christian Sutter · Peter W Kaplan

Coma pattern	Etiologies	EEG background reactivity*	Most frequent outcome
Beta coma			
Intermingled with alpha activity	Intoxications or withdrawal (barbiturates or benzodiazepines), severe hyperthyroidism	+ / -	favorable
Intermingled with delta activity	Brainstem lesions	-	unfavorable
Alpha coma			
More diffusely	Intoxication (barbiturates, benzodiazepines, anesthetic agents, meprobamate, imipramine)	+	favorable
Monomorphic posterior	Brainstem lesions, locked-in syndrome	+ / -	unfavorable
More diffusely	Hypoxic-ischemic encephalopathy	(+) / -	unfavorable
Theta coma	Hypoxic-ischemic encephalopathy, mild to moderate metabolic encephalopathies, severe systemic infections	(+) / -	unfavorable
High-voltage delta coma			
Anterior predominance or focal, unilateral	Metabolic encephalopathies, focal or unilateral white matter lesions	+	favorable
More diffusely	Severe metabolic encephalopathy, severe encephalitis, vasculitis, large white matter lesions, markedly increased intracranial pressure	(+) / -	unfavorable
Spindle coma			
Theta and delta activity with paroxysmal bursts symmetric spindles	Traumatic brain injury, intracerebral hemorrhage, post-ictal states, intoxication	+	favorable
Theta and delta activity with paroxysmal bursts of symmetric spindles	Hypoxic-ischemic encephalopathy, severe traumatic brain injury, large intracerebral hemorrhage	(+) / -	unfavorable
Burst-suppression			
With interruptions	Intoxication (sedative drugs), anesthetic drug use, and hypothermia	+ / (-)	favorable
No interruption	Hypoxic-ischemic encephalopathy, severe intoxication	(+) / - (controversial for outcome)	unfavorable
Low-voltage delta coma			
Theta and delta activity with intrusions of alpha and beta activity	Traumatic brain injury, healthy individuals	+	favorable
Theta and delta activity without intrusions of higher frequency activity	Hypoxic-ischemic encephalopathy, severe traumatic brain injury	(+) / -	unfavorable
Electro-cerebral inactivity			
No spontaneous neuronal activity detectable	Marked hypothermia, severe intoxications (nervous system depressant drugs)	-	favorable
	Hypoxic-ischemic encephalopathy	-	unfavorable

+ = preserved EEG background activity, - = no EEG background activity EEG = electroencephalography, * to external noxious stimulation

How to tackle EEG in encephalopathy?

1. Look for a PDR
2. What is the dominant frequency?

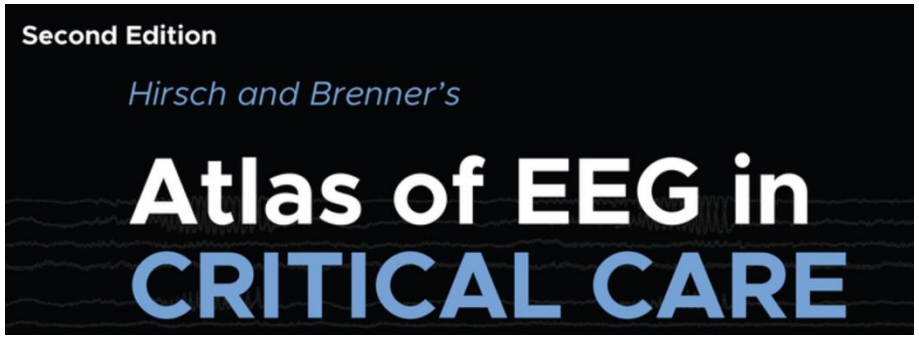
3. Continous?
4. Reactive? – **CYSTE -** call, yell, sternal rub, nasal tickle, passive eye opening

SEVERITY

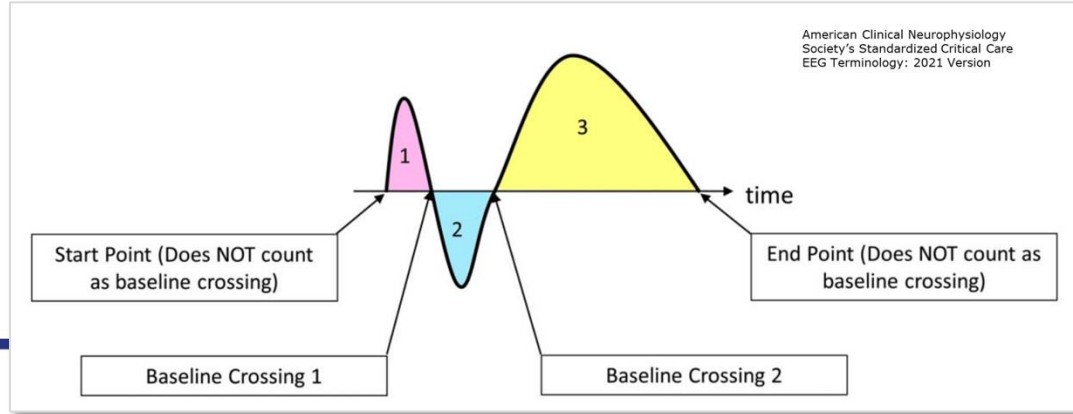
TABLE 3.3 Yale Adult Encephalopathy Scale (2021)

Required fields highlighted in yellow	Normal	Mild	Mild - Moderate	Moderate	Moderate - Severe	Severe	Complete Suppression of Cerebral Rhythms[c]
Predominant frequency,most awake state	Alpha or LVF[a] with minimal theta, no delta	≥7 Hz or LVF[a] with prominent theta or any delta (whether rhythmic or not)	For patients who do not meet all criteria for either Mild or Moderate but fall somewhere in between	3–6 Hz	For patients who do not meet all criteria for either Moderate or Severe but fall somewhere in between, OR with LVF in a non-interactive patient[a]	Any	NA
Posterior dominant rhythm (PDR)	≥8.5 or LVF[a]	Present (any freq) or LVF[a] background		Absent		Absent	NA
Continuity, most awake state	Continuous	Continuous		Continuous or nearly continuous		NR	NA
Reactivity	NR[b]	NR[b]		Reactive or SIRPIDs-only		Unreactive or SIRPIDs-only	NA
State changes	NR[b]	NR[b]		Present		Absent	NA

[a] LVF = low voltage beta (or faster) activity diffusely: In awake patients with meaningful interaction, LVF can be part of a normal background, mild dysfunction or mild-moderate dysfunction based on other features. In patients without meaningful interaction, LVF should be classified as moderate to severe if EEG is reactive, and severe if unreactive and without state changes.
[b] NR: Not required. These features are not part of the definition of that level of dysfunction, but all records will be reactive and have state changes if a long enough recording is obtained.
[c] Complete suppression: No discernible cerebral rhythms whether or not standards for determination of electrocerebral silence are met. NA: Not Applicable




Fp1-F7
F7-T7
T7-P7
P7-O1
Fp2-F8
F8-T8
T8-P8
P8-O2
Fp1-F3
F3-C3
C3-P3
P3-O1
Fp2-F4
F4-C4
C4-P4
P4-O2
Fz-Cz
Cz-Pz
ECGL-ECGR
140 uV
1 sec


American Clinical Neurophysiology Society's Standardized Critical Care EEG Terminology: 2021 Version
1
2
3
time
Start Point (Does NOT count as baseline crossing)
End Point (Does NOT count as baseline crossing)
Baseline Crossing 1
Baseline Crossing 2

Triphasic waves

- 1950 – “hepatic encephalopathy”
- Not different from GPDs? Variant?
- Can cause secondary brain injury?
- A form of NCSE?

Comparison of typical and atypical features of triphasic waves.

	Typical triphasic waves	Atypical triphasic waves
Symmetry	Symmetrical or shifting asymmetry; may be asymmetrical depending on underlying structural abnormalities#	Consistently lateralized or asymmetrical, or focal/ multifocal negativity; maybe asymmetrical depending on underlying structural abnormalities as well as potential epileptogenicity#
Phases and Location	Triphasic or biphasic with prominent frontal/ frontocentral positivity	Triphasic or biphasic but prominent negativity as opposed to positivity
Frequency	$\leq$ 2 Hertz frequency	$>$ 2 Hertz frequency
State/Stimulus Dependence	Stimulus or state dependent, often resolve completely or becoming less frequent in sleep or drowsiness; may also be seen more frequently in sleep or drowsiness and less frequently in wakefulness	Continuous, without state or stimulus dependence
Presence of Absence of Lag	Anterior-posterior or posterior-anterior lag	No lag
Contour	Blunted	Spiky/sharp contour
Benzodiazepine responsiveness	May resolve with benzodiazepine; would not typically demonstrate clinical responsiveness to benzodiazepine	May resolve with benzodiazepine; may or may not demonstrate clinical responsiveness to benzodiazepine
Background	Background activity is present though slow	Slow or attenuated background
Dynamic or Monotonous	When present the waveforms have a similar appearance and are somewhat monotonous	When present the waveforms have dynamic appearance

Review article

Triphasic waves: To treat or not to treat?

Brin E. Freund [a,*], Khalil S. Husari [b], José L. Fernández-Torre [c,d,e], Philippe Gélisse [f], Peter W. Kaplan [b]

Etiologies of Triphasic Waves.

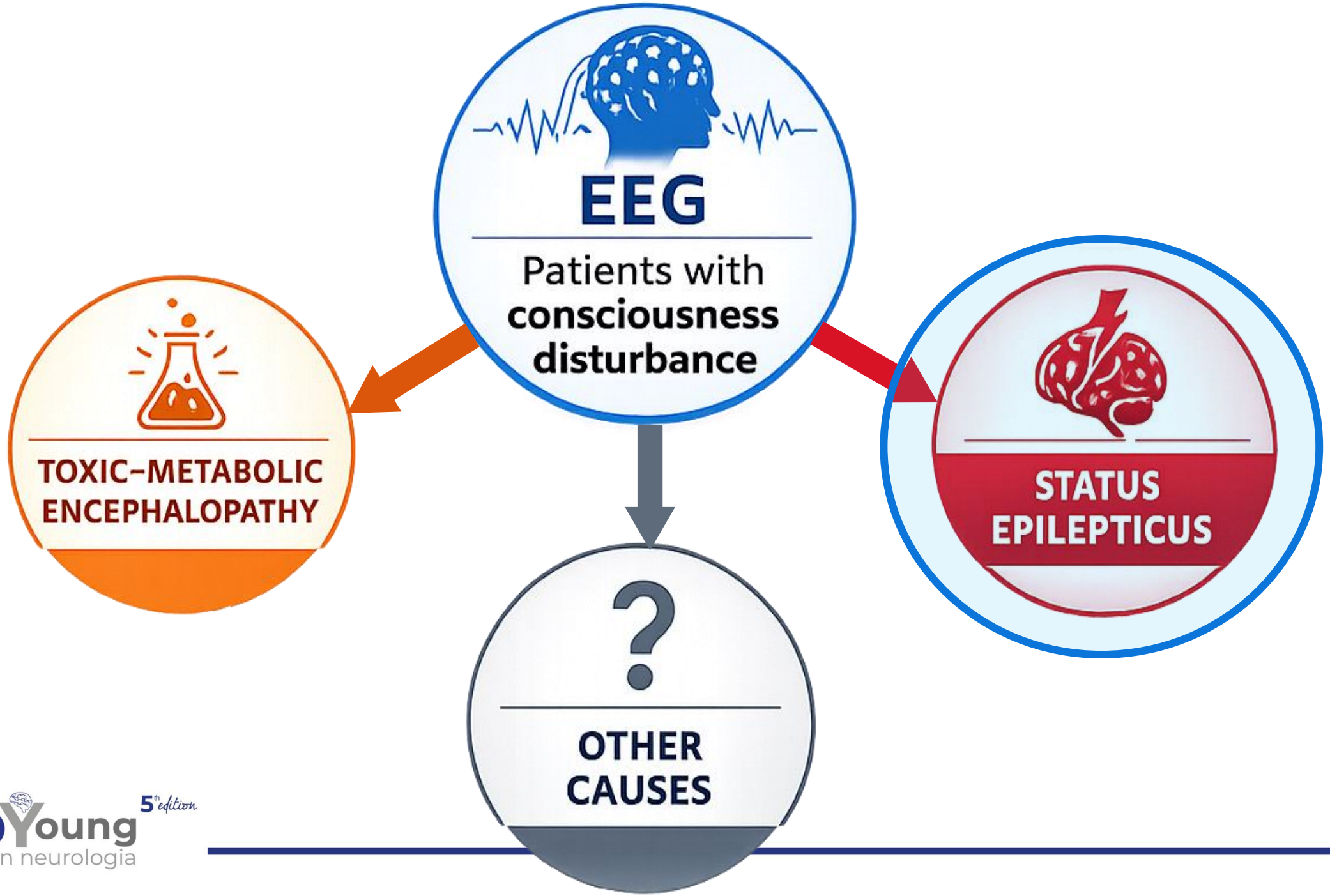
Clinical
Hepatic failure/Hyperammonemia
Renal dysfunction
Hypothermia
Systemic or CNS infections
Intracranial hypertension
Angelman syndrome
Rett syndrome
Hashimoto encephalopathy/Thyroid disorders
Medications (levodopa, metrizamide, pentobarbital, naproxen, gadolinium, levetiracetam, baclofen, cefepime, ifosfamide, lithium, cefoperazone, pregabalin, ceftriaxone, aztreonam)
Alzheimer disease/Dementia
Hypoparathyroidism and other endocrinologic disorders
Radiographic
White matter lesions
*Brainstem or diencephalic lesions
*Acute ischemic stroke (cortical and subcortical)
*Acute intracranial hemorrhage (including all types)
*Hydrocephalus
*Traumatic Brain Injury
*Hypertensive emergency/posterior reversible leukoencephalopathy

*Confounded by presence of cerebral atrophy and/or white matter lesions.

Triphasic Waves

- Frequency ≤ 2 Hz → Typical: Evaluate for other risk factors for ictogenesis (i.e. 2HELPS2B score)
  Atypical: Empiric ASM/benzodiazepine trial; Continuous EEG monitoring
- Frequency 2-2.5 Hz → Empiric ASM/benzodiazepine trial
  Continuous EEG monitoring
- Frequency >2.5 Hz → Diagnostic of nonconvulsive status epilepticus

* In the absence of any subtle clinical manifestation of nonconvulsive status epilepticus/seizures


EEG
Patients with consciousness disturbance
TOXIC–METABOLIC ENCEPHALOPATHY
STATUS EPILEPTICUS
?
OTHER CAUSES

Status epilepticus

- High sensitivity
- High specificity
- High temporal resolution
- High spatial resolution

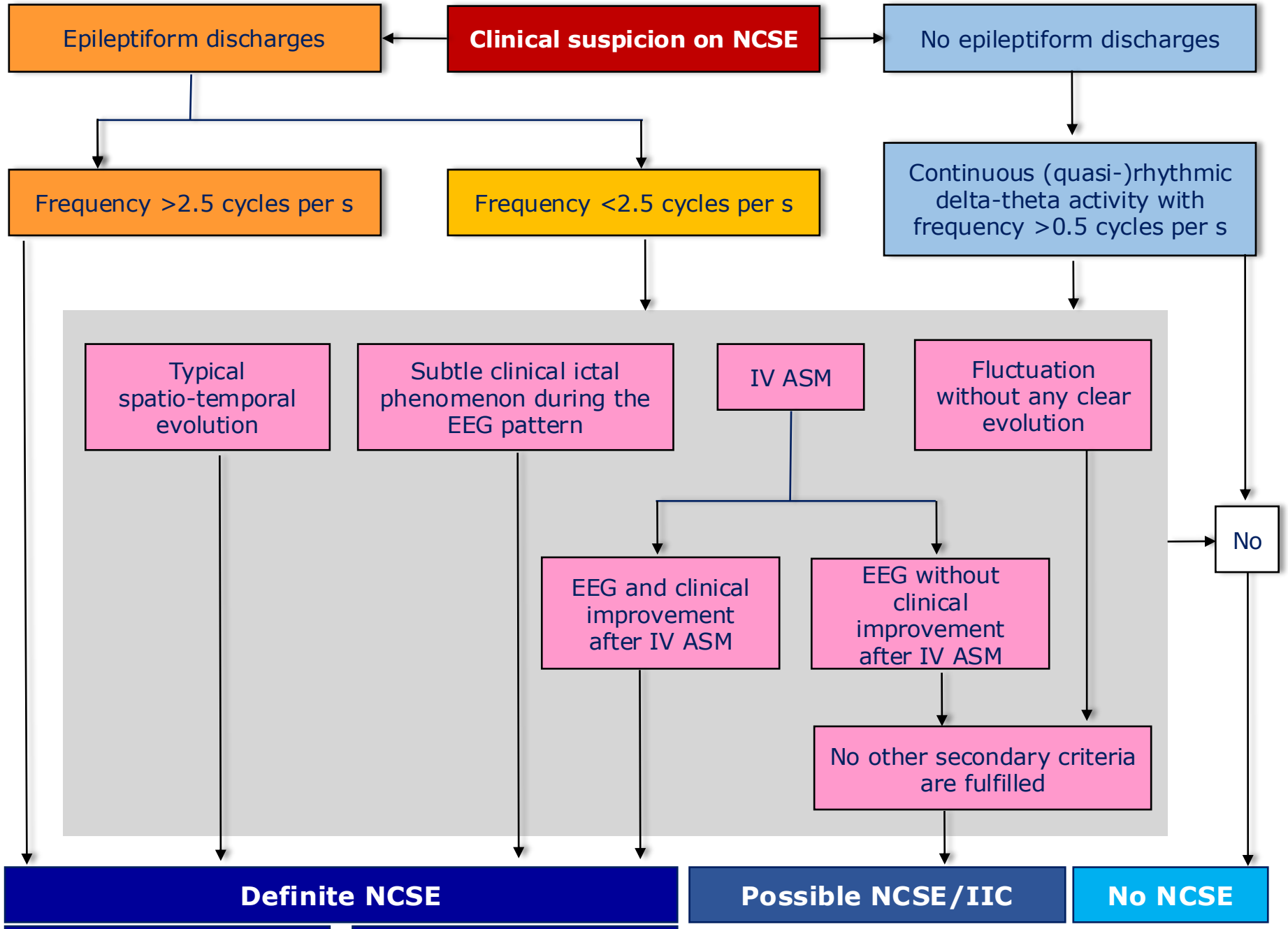
EEG IS THE GOLD STANDARD FOR SE DIAGNOSIS

When to suspect it?

- Convulsive SE → motor signs
- NCSE
  - Consciousness alteration (quantitative or qualitative)
  - After/in between a clear seizure, no return to baseline status
  - Subtle motor signs – twitching, nystagmus/ocular deviation, hippus, pyloerection
  - A history of epilepsy, recent withdrawal of antiseizure medications, *acute brain injury, or metabolic disturbances*

Epilepsia

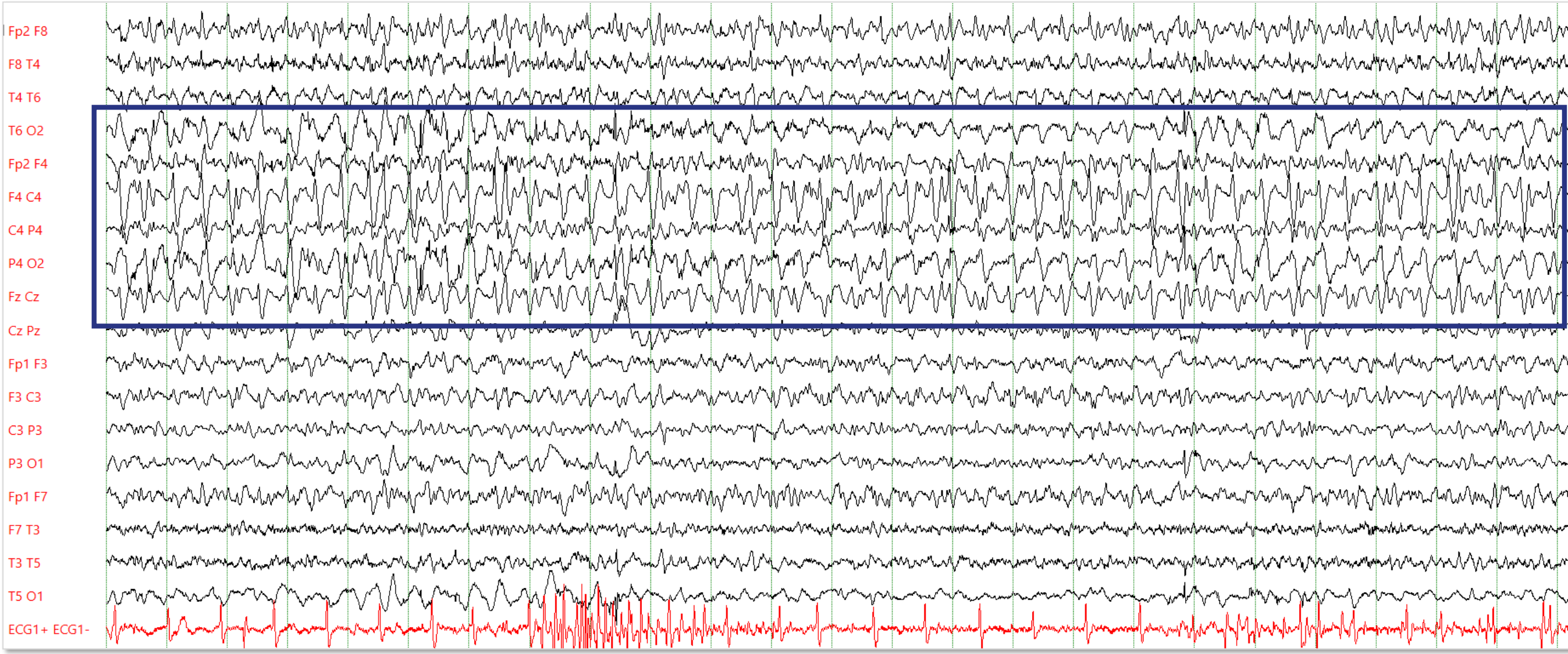
Salzburg criteria for nonconvulsive status epilepticus: Details matter



>10’ or >20% of one hour

Adapted from Leitinger M et al., Epilepsia 2019

GRAY MATTERS
Letter



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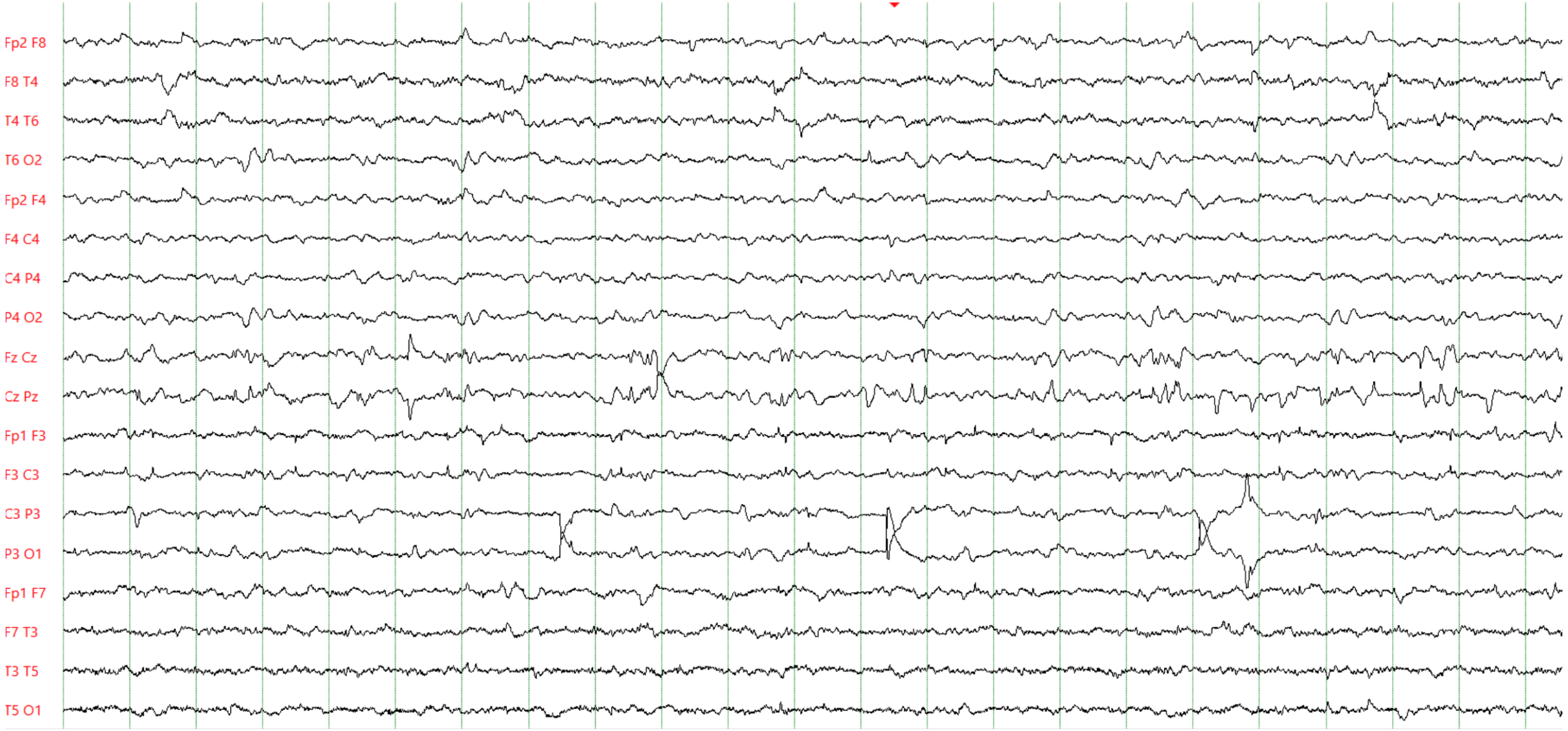
> 2.5 Hz

DEFINITE SE



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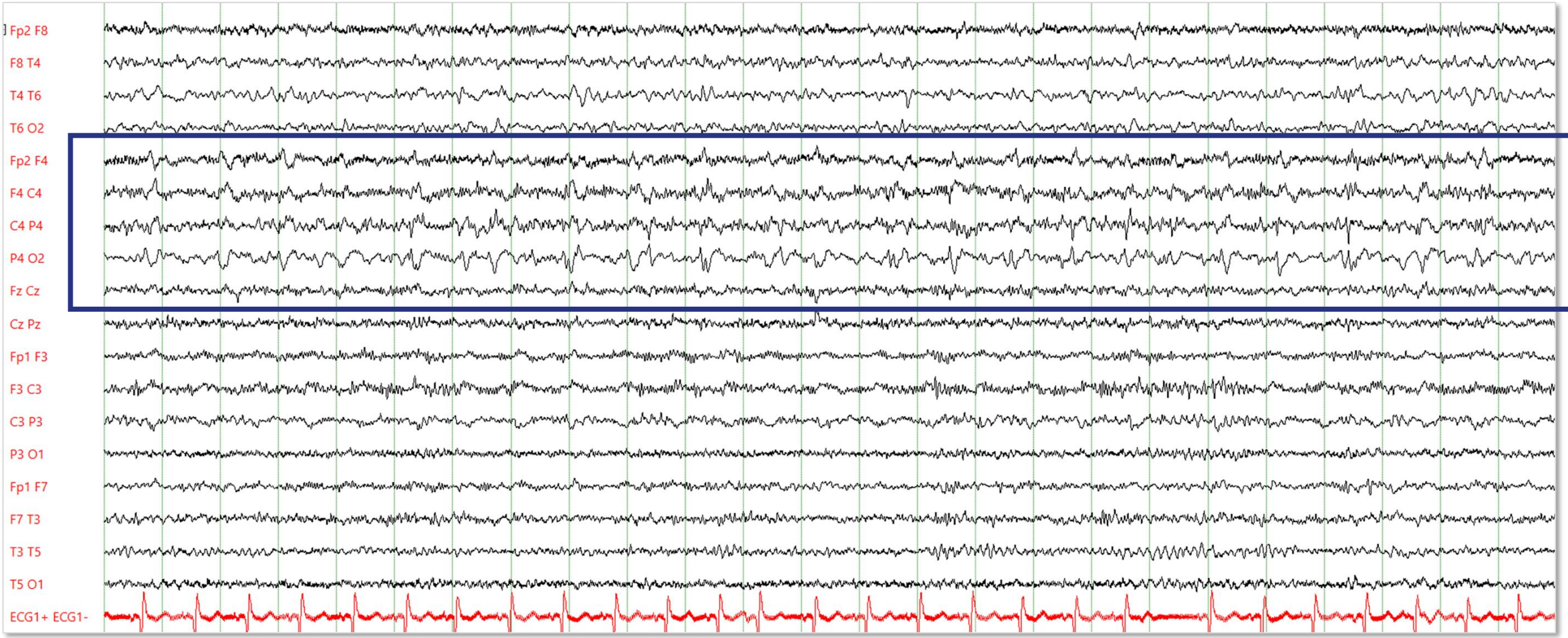
DEFINITE SE



IIC – POSSIBLE NCSE

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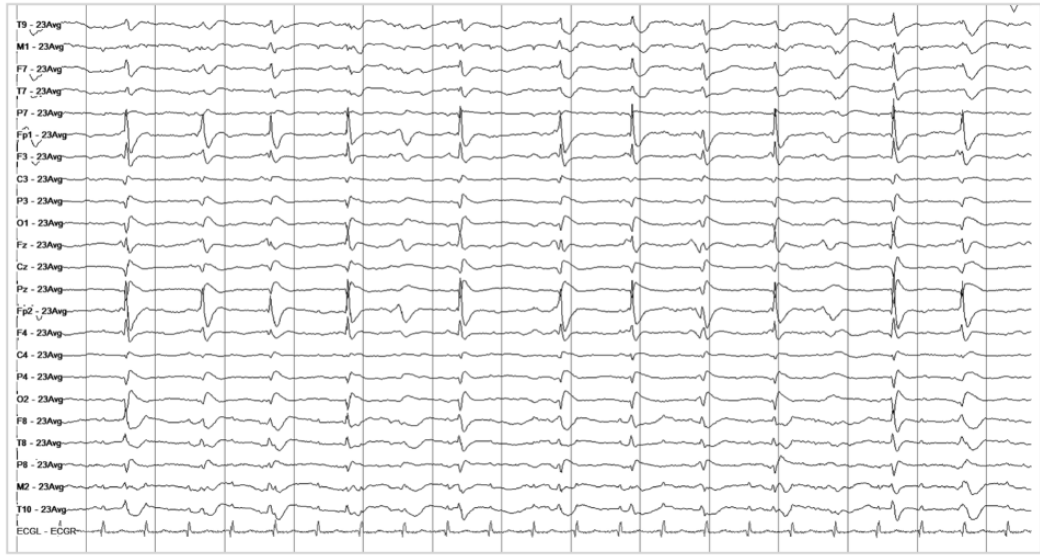
Lateralized Periodic Discharges (LPDs)

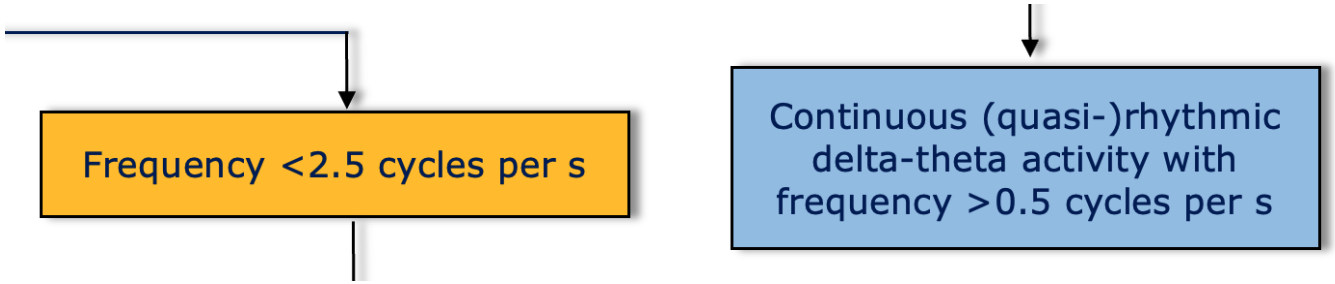
Generalized Periodic Discharges (GPDs)



Lateralized Rhythmic Delta Activity (LRDA)

Generalized Rhythmic Delta Activity (GRDA)

What is IIC?



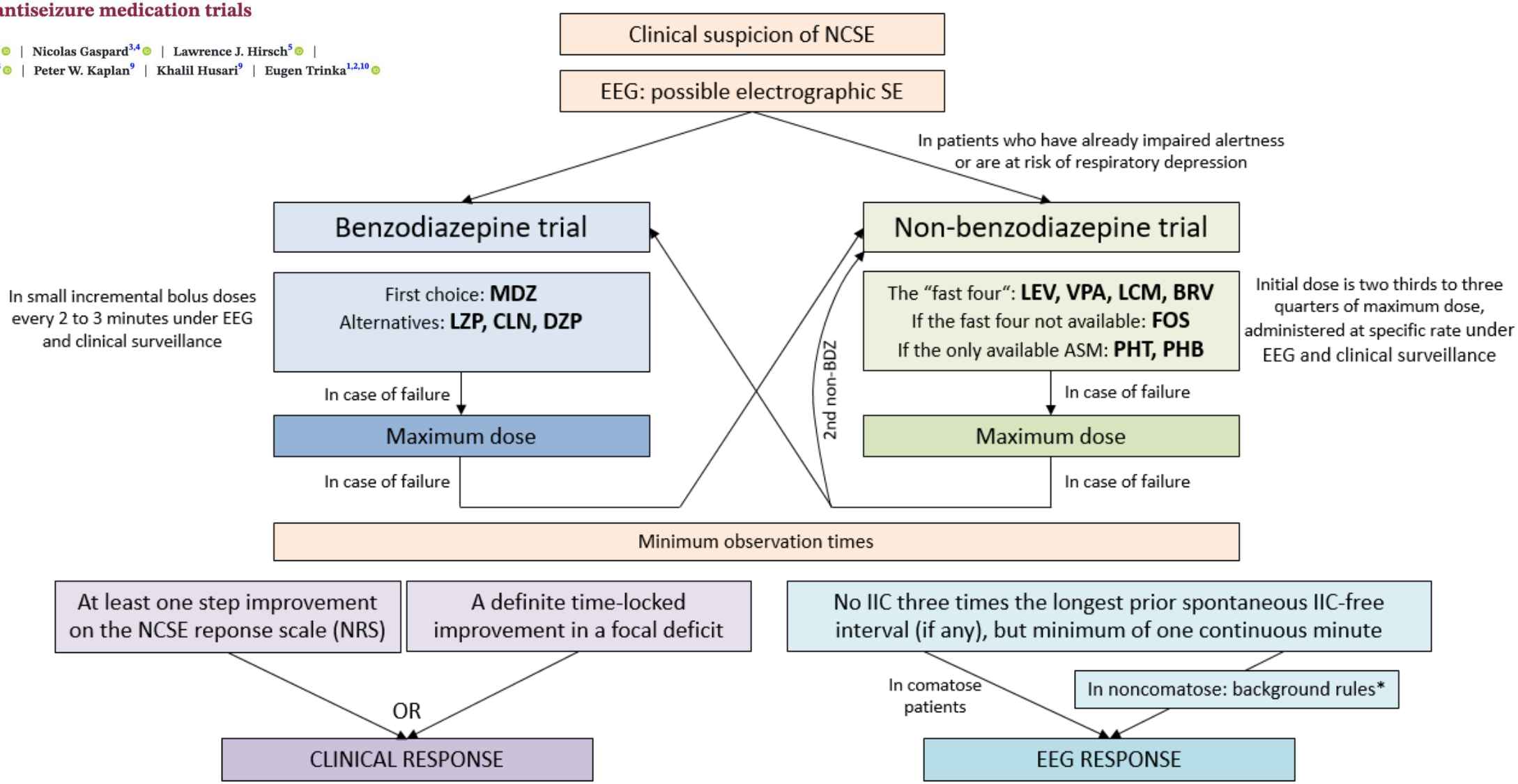
- Purely electrographic phenomenon
- Borderland or a “boundary syndrome” of SE, with no clear distinction between ictal and interictal EEG activity
- Deciding if ictal or interictal is, however, **critical**

Adapted from Bosque-Varela P et al., Epilepsia 2026

Diagnosing nonconvulsive status epilepticus: Defining electroencephalographic and clinical response to diagnostic intravenous antiseizure medication trials

Markus Leitinger[1,2] | Nicolas Gaspard[3,4] | Lawrence J. Hirsch[5] | Sándor Beniczky[6,7,8] | Peter W. Kaplan[9] | Khalil Husari[9] | Eugen Trinka[1,2,10]

RESPONSE TO DIAGNOSTIC IV ASM TRIAL



*Background rules: If baseline EEG background frequency is known: return to baseline OR appearance of previously absent normal features. In patients with no acute neurological or systemic condition that can affect the background activity: normal background OR appearance of previously absent normal features. In all other cases: the criterion cannot be applied

BDZ

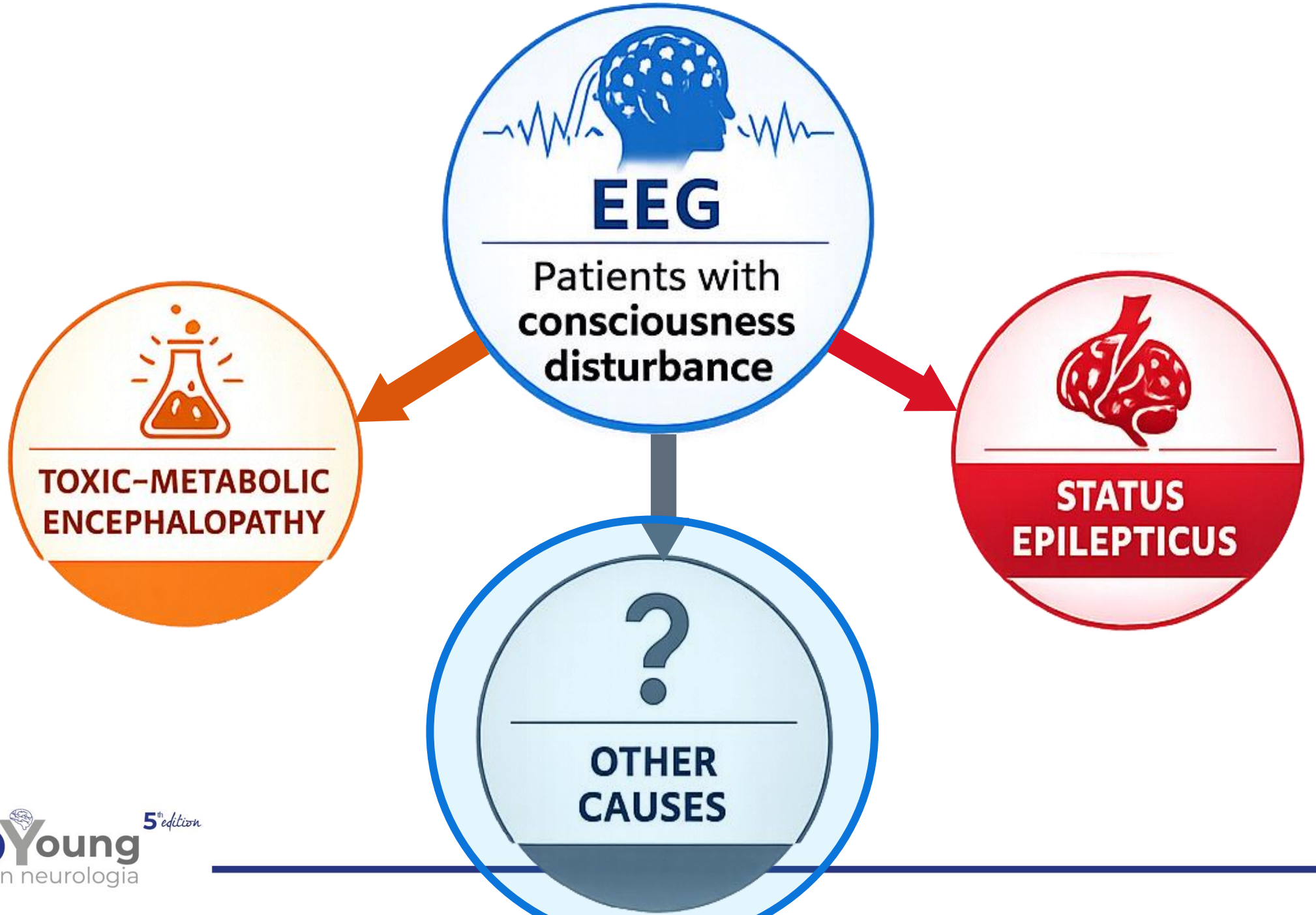
	Midazolam	Lorazepam	Clonazepam	Diazepam
Starting dose	1 mg	0.5 mg	0.25 mg	4 mg
if there is no EEG-response give incremental doses (same as starting doses) every 2-3 minutes of (under EEG- and clinical monitoring)				
up to a maximum dose of (whichever is lower)				
Maximum dose, weight based	0.2 mg/ kg	0.1 mg/ kg	0.05 mg/ kg	0.25 mg/kg
Maximum dose, absolute	5 mg	2.5mg	1.25 mg	20 mg

Non-BDZ

	Levetiracetam	Valproate	Lacosamide	Brivaracetam	Fosphenytoin*	Phenobarbital**
Choice	1	1	1	1	2	3
Starting dose	40 mg/kg	30 mg/kg	6 mg/ kg	4 mg/kg	15 mg PE/ kg	10 mg/kg
Administration time	5 minutes	5 minutes	10 minutes	5 minutes	10-15 minutes (max 150 mg/min)	15+ minutes
With additional boluses up to a maximum dose of (whichever is lower)						
Maximum total loading dose, weight based	60 mg/kg	40 mg/kg	8 mg/ kg	6 mg/ kg	20 mg PE/ kg	20 mg/kg
Maximum total loading dose, absolute	4500 mg	3000 mg	600 mg	450 mg	1500 mg PE	1500
Special measures			ECG monitoring		ECG monitoring	

*phenytoin: where fosphenytoin is not available, at a maximum rate of 50 mg/min;
**in countries where only phenobarbital is available
PE phenytoin sodium equivalent;

LEVEL 10: **normal**
LEVEL 9: **speaks or writes words** with clear sense, oriented to person, year and city or region, but behaviour or performance different than normal
LEVEL 8: **speaks or writes words** with clear sense, but disoriented to person, year and city or region
LEVEL 7: **speaks or writes words or syllables,** incomprehensible or confused
LEVEL 6: **Follows commands** (verbally or by demonstration) (e.g. open/close eyes, raise arms, say "1,2,3")
LEVEL 5: **directed gaze to** examiner (or responsive vertical eye movement in locked-in syndrome), does not follow any commands (neither verbal nor by demonstration.)
LEVEL 4: **opens eyes** spontaneously or to verbal stimulus or to light touch, (no directed gaze), does not follow any commands
LEVEL 3: **opens eyes** to (strong) tactile stimulus (R or L shoulder, nose), does not follow commands (verbal NOR by demonstration)
LEVEL 2: **localizes** to/ **wards off** painful stimuli
LEVEL 1: **no purposeful response to painful stimuli**

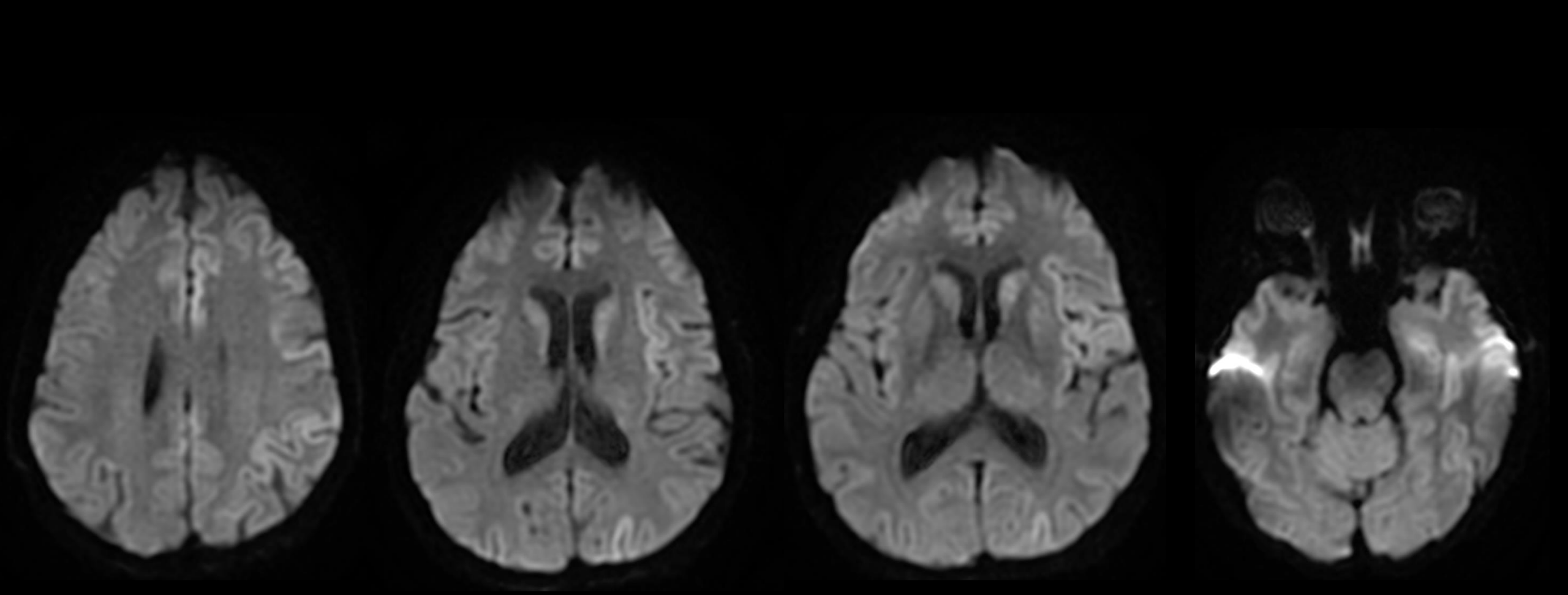

EEG
Patients with consciousness disturbance
TOXIC–METABOLIC ENCEPHALOPATHY
STATUS EPILEPTICUS
?
OTHER CAUSES

Case #1

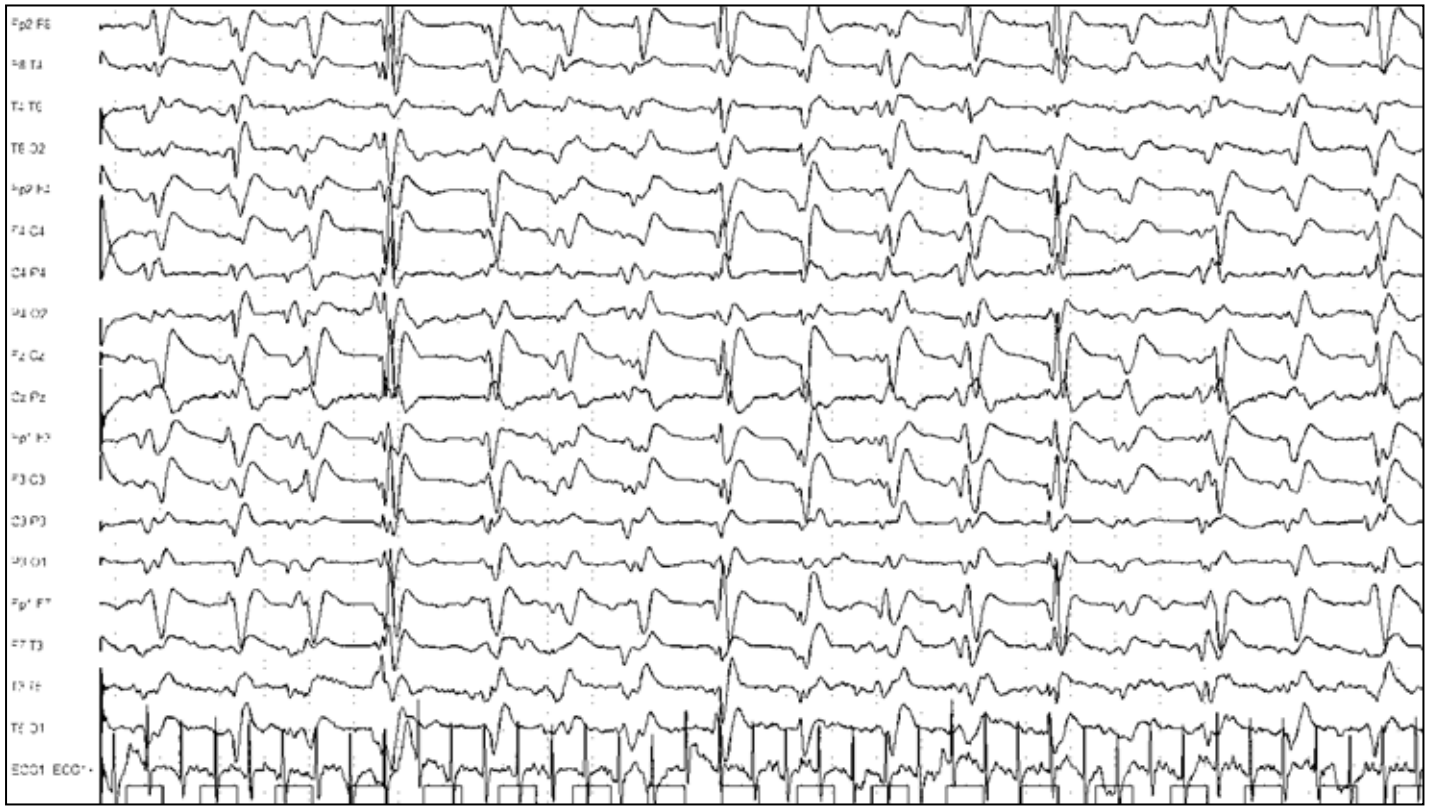
- E.H., 57 yo, female
- Past medical hx: nihil
- Presenting complaint: subjective cognitive impairment (phone!) three months prior > progressive
- Physical examination: upper limb dystonia, dysarthria, aphasia nominum

Adapted from Bosque-Varela P et al., Epilepsia 2026


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



CJD – E200K

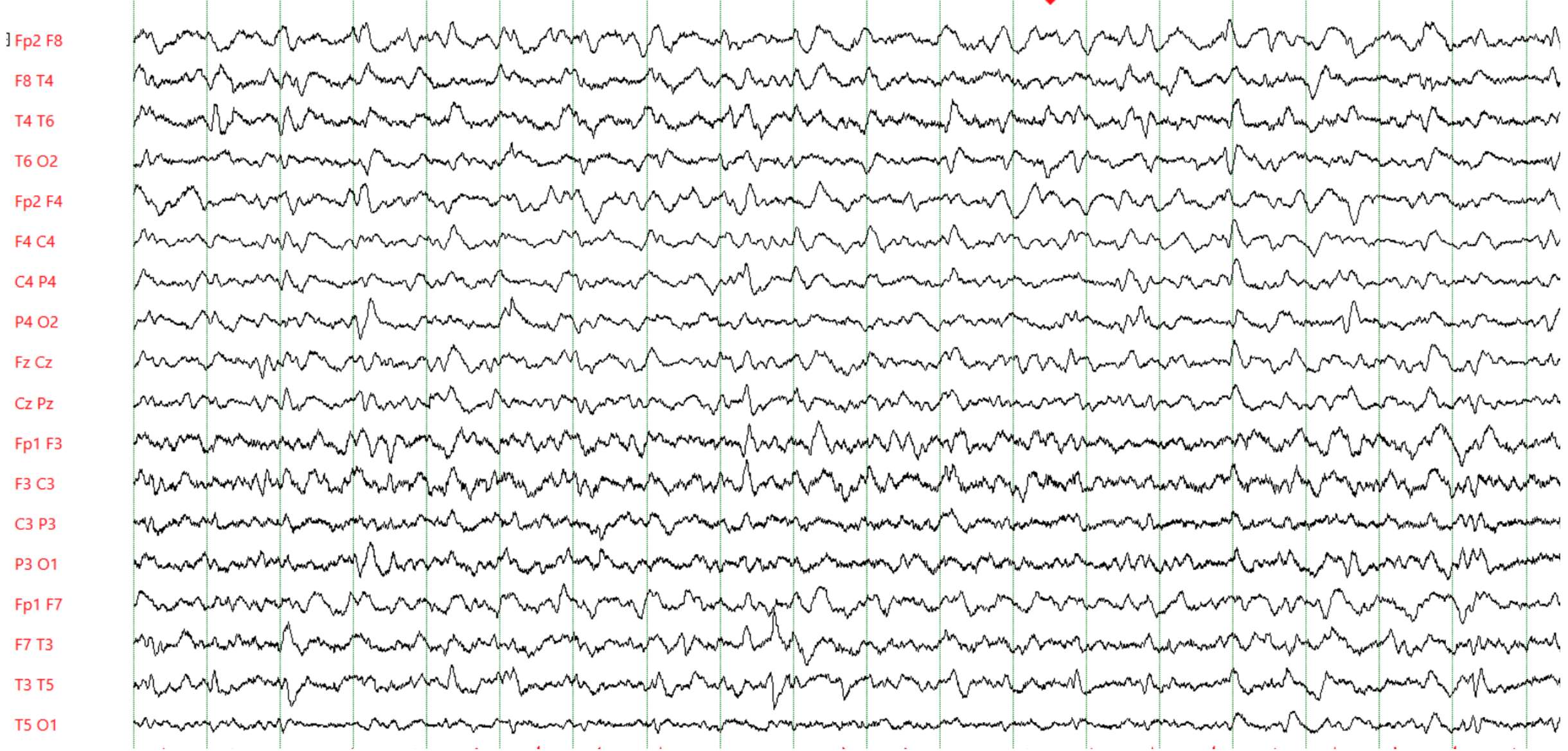


Periodic Sharp Wave Complexes (PSWC)

DIAGNOSTIC CRITERIA OF PSWC TYPICAL FOR **sCJD**

- Biphasic or triphasic
- Periodic, the majority with a duration of 100-600 ms and an intercomplex interval of 500-2000 ms
- Generalized and lateralized complexes accepted
- Frontal-precentral

Steinhoff et al, Arch Neurol 1996



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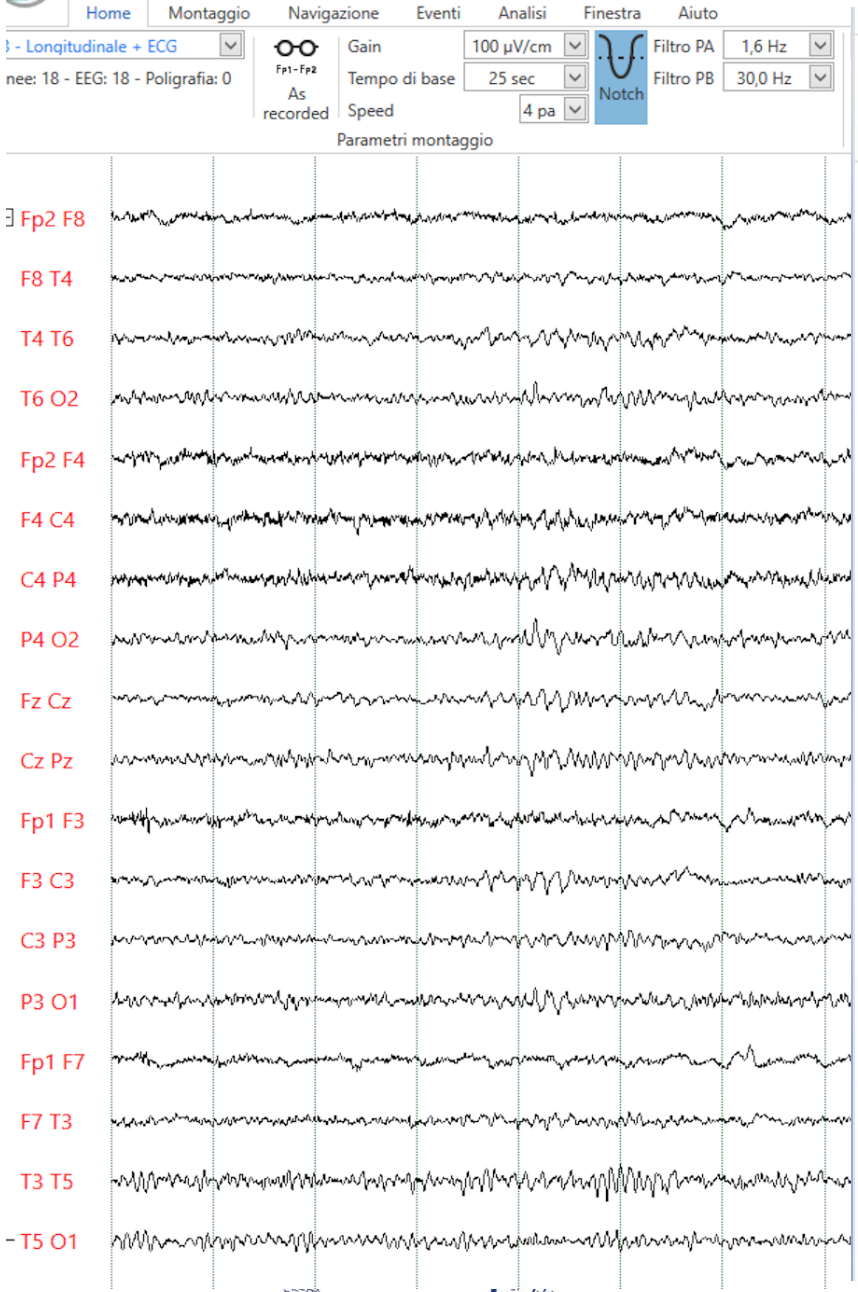
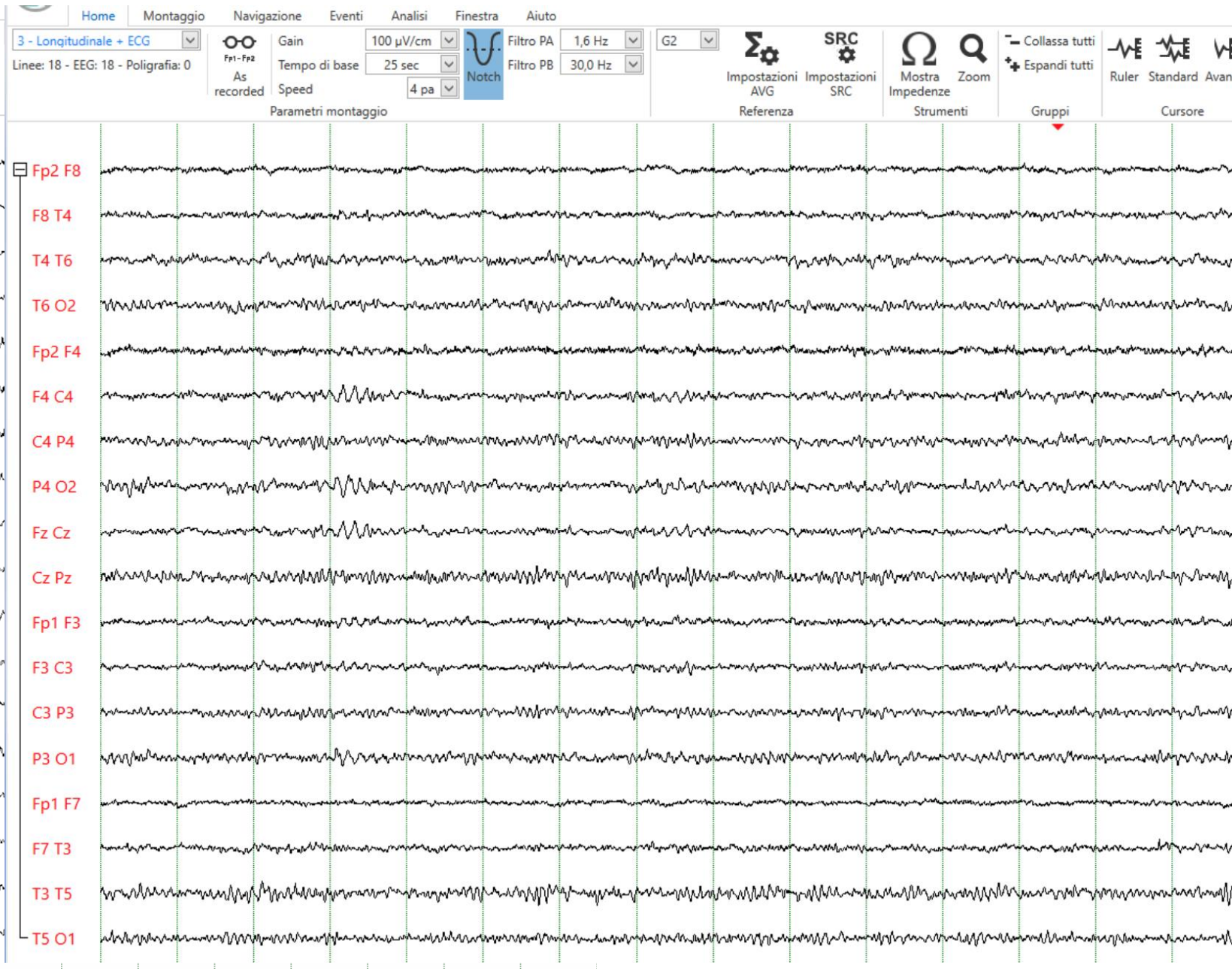
Another pt with prion disease

Case #2

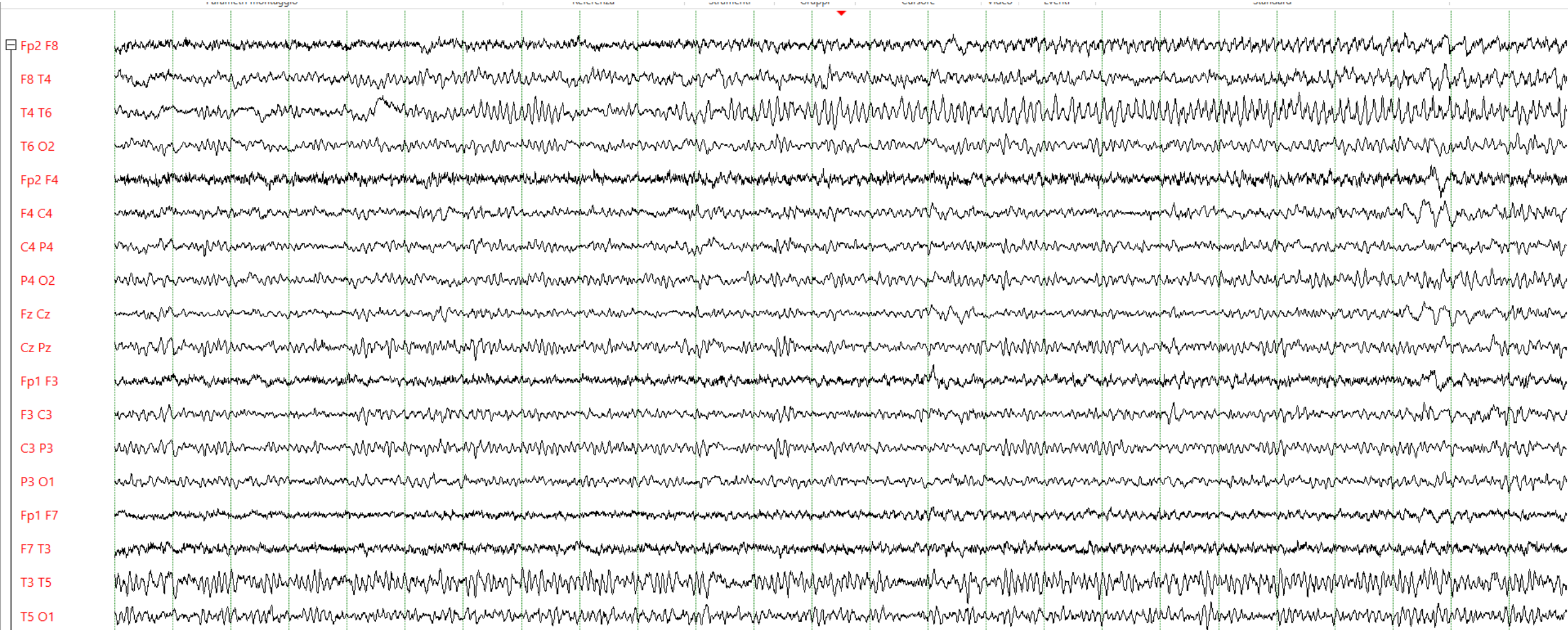
- A.B., 25 yo, female
- Past medical hx: anxiety, CMT type 1
- Presenting complaint: episodes of difficulty in ''moving the tongue" + worsening of anxiety, slowness

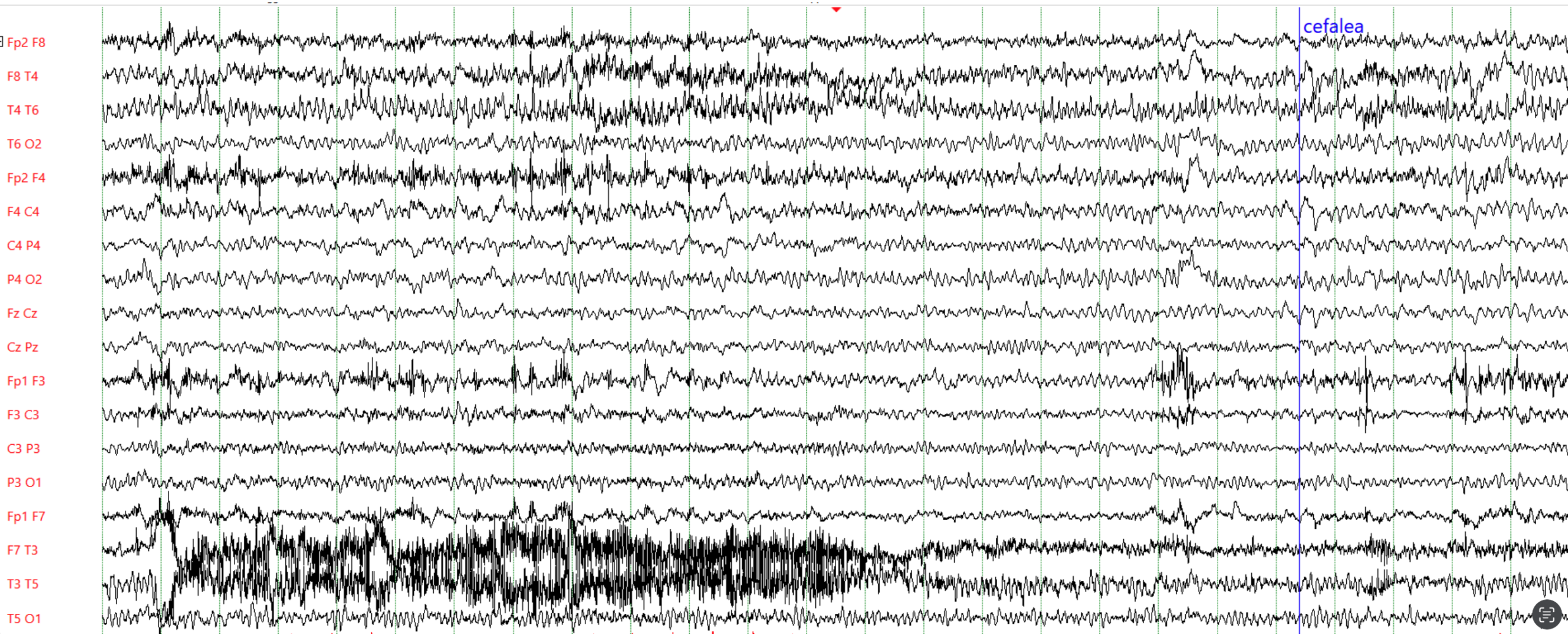

Home
Montaggio
Navigazione
Eventi
Analisi
Finestra
Aiuto
- Longitudinale + ECG
nee: 18 - EEG: 18 - Poligrafia: 0
Fp1-Fp2
As recorded
Gain
100 µV/cm
Tempo di base
25 sec
Speed
4 pa
Notch
Filtro PA
1,6 Hz
Filtro PB
30,0 Hz
Parametri montaggio
Fp2 F8
F8 T4
T4 T6
T6 O2
Fp2 F4
F4 C4
C4 P4
P4 O2
Fz Cz
Cz Pz
Fp1 F3
F3 C3
C3 P3
P3 O1
Fp1 F7
F7 T3
T3 T5
T5 O1

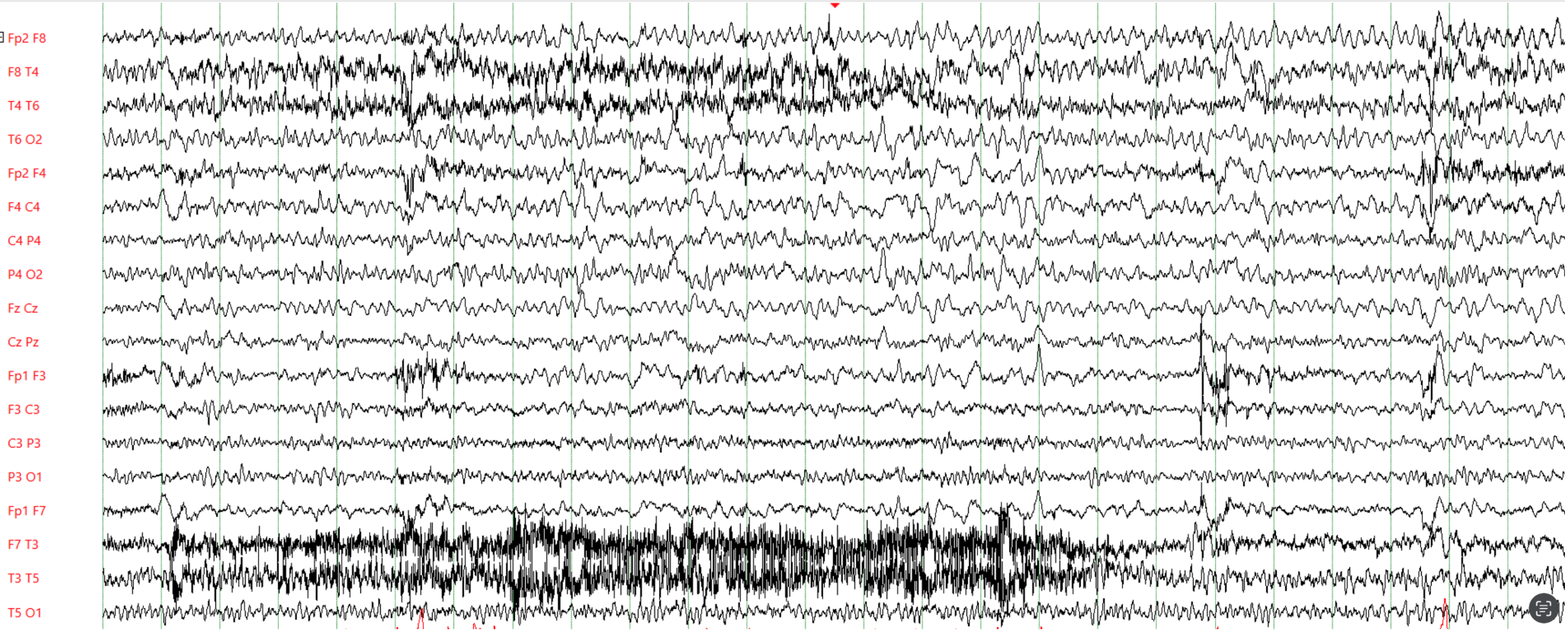

Home
Montaggio
Navigazione
Eventi
Analisi
Finestra
Aiuto
3 - Longitudinale + ECG
Linee: 18 - EEG: 18 - Poligrafia: 0
Fp1-Fp2
As recorded
Gain
100 µV/cm
Tempo di base
25 sec
Speed
4 pa
Notch
Filtro PA
1,6 Hz
Filtro PB
30,0 Hz
Parametri montaggio
G2
Impostazioni AVG
Impostazioni SRC
Referenza
Mostra Impedenze
Zoom
Strumenti
Collassa tutti
Espandi tutti
Gruppi
Ruler
Standard
Cursore
Fp2 F8
F8 T4
T4 T6
T6 O2
Fp2 F4
F4 C4
C4 P4
P4 O2
Fz Cz
Cz Pz
Fp1 F3
F3 C3
C3 P3
P3 O1
Fp1 F7
F7 T3
T3 T5
T5 O1

Il primo avvenuto sabato sera (18 novembre): sono uscita per negozi, (sottolineo che ultimamente non avviene spesso che esca di casa e che a ripensarci ora mi è difficile realizzare che siano passati solo pochi giorni, mi sembra un'eternità, cosi come mi sembra molto lontano il giorno in cui ci siamo visti) mi sono subito resa conto che era disturbante la musica che c'era e le corsie dei supermercati ma ho cercato di rimanere più calma possibile ricordando che posso pian piano tornare alla normalità. Uscendo dall'edificio ho avuto un déjà-vu... Salita in macchina la testa si è affollata di pensieri ed ho provato una sensazione particolare che si è ripetuta poi negli episodi successivi che proverò in qualche modo a descriverle e che si sono inoltre ripetuti in tutta la giornata di sabato. Dopo pochi secondi dalla salita in macchina ho avuto come una sensazione di svenimento, sentendo le gambe e le mani come addormentate accompagnata da nausea. Il tutto avrà avuto la durata di 3-4 minuti, successivamente sono andata a prendere una mia amica che non vedevo da circa 3 settimane ed ho come avuto la difficoltà nel riconoscerle il viso.... dopo qualche giorno... ho avvertito anche lì lo stesso disagio della sera prima nel supermercato, cercando sempre di razionalizzarlo e tranquillizzarmi, ma dopo avvertita la stessa sensazione descritta sopra ho avuto lo stesso sentore di svenimento e disorientamento, della stessa durata e a seguire puntuale il mal di testa, molto più forte, alle tempie e agli occhi, tanto da non riuscire a tenerli aperti e a costringerci a rientrare a casa, ho provato sollievo 2-3 ore dopo.... Un ulteriore episodio si è presentato ieri pomeriggio, sapevo che stava per succedere perché avevo riconosciuto il disorientamento, come di déjà-vu, ma diverso e più complesso da spiegare, a seguire sempre lo stesso mal di testa, ero a casa con un amico che non vedevo da anni e anche lì ho avuto difficoltà a focalizzarne il volto e seguire i discorsi che facevamo....

One week later

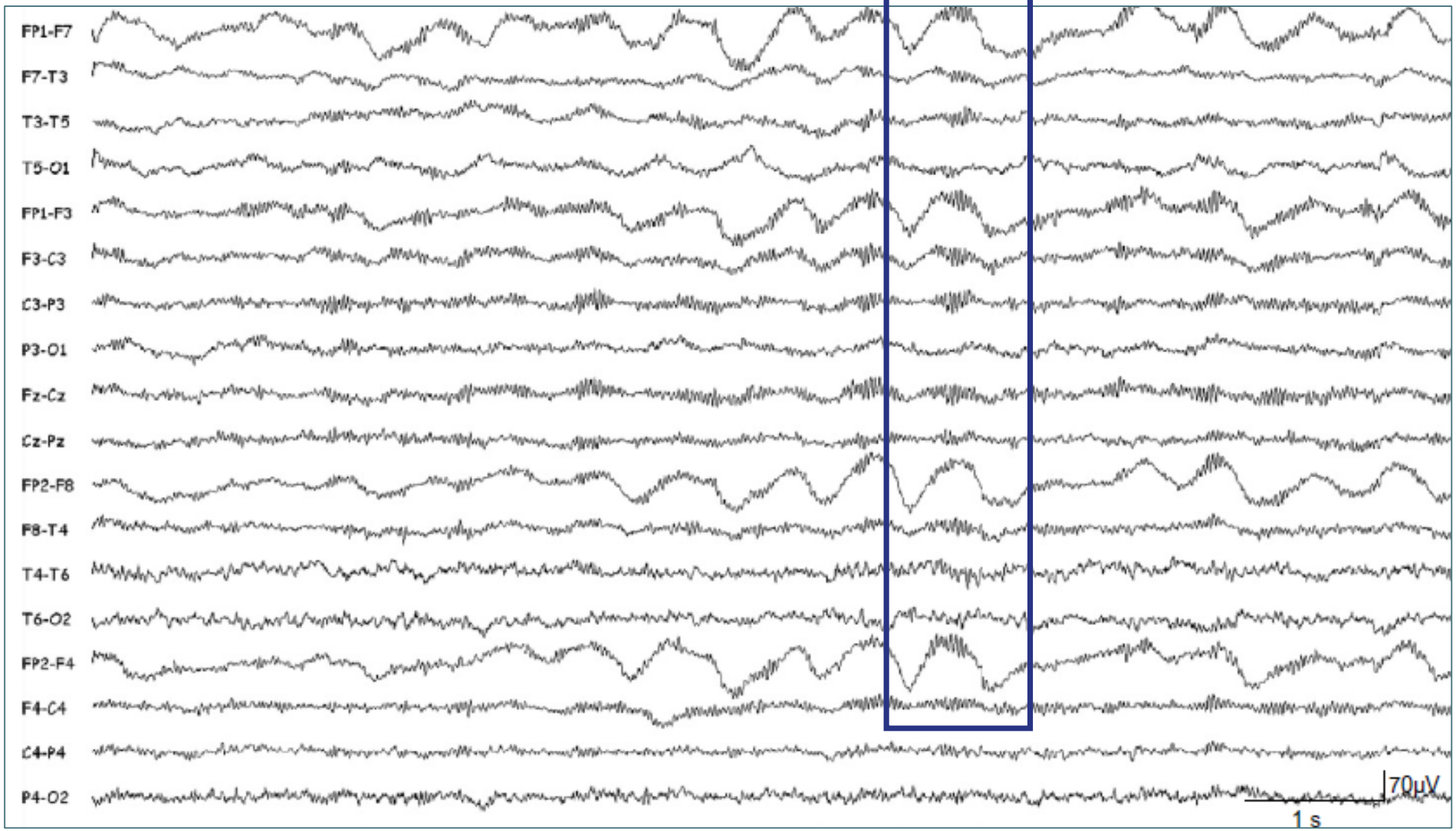

Fp2 F8
F8 T4
T4 T6
T6 O2
Fp2 F4
F4 C4
C4 P4
P4 O2
Fz Cz
Cz Pz
Fp1 F3
F3 C3
C3 P3
P3 O1
Fp1 F7
F7 T3
T3 T5
T5 O1


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


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

- Normal MRI
- Increased proteins, IgG in the CSF

Anti-NMDA+



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DELTA BRUSH

AE: clues

CLINICAL RED FLAGS

- Subacute onset
- High frequency (weekly/daily)
- Possible status epilepticus at onset
- Faciobrachial dystonic seizures (LGI1)
- Pilomotor seizures
- Multi-modal auras

EEG RED FLAGS

- Subclinical seizures
- Sensitivity to HV
- Possible peculiar triggers
- Bilateral independent seizures
- Ictal shift
- Possible multiple ictal patterns

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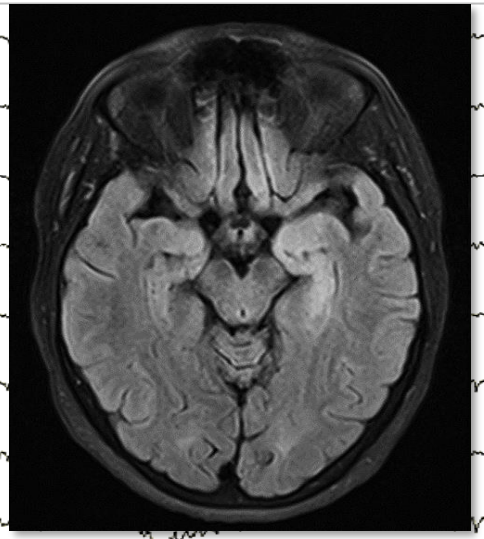
Steriade et al, Epilepsia 2016; Steriade et al, E&B 2018; Morano et al, Seizure 2020; McGinty et al, JNNP 2021; Wennberg et al, Epilepsia 2024

262 SEIZURES/24H
126 CLINICAL (PILOMOTOR)
136 SUBCLINICAL



Fp2 T4
T4 O2
Fp2 C4
C4 O2
Fp1 C3
C3 O1
Fp1 T3
T3 O1
ECG1+ ECG1-

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SUBCLINICAL, STORMY ONSET

ANNALS of Clinical and Translational Neurology
Open Access
AMERICAN NEUROLOGICAL ASSOCIATION
Check for updates

BRIEF COMMUNICATION

Hyperventilation-induced focal seizures in adults: think autoimmune encephalitis

Richard A. Wennberg

Table 1. Routine EEG recordings with hyperventilation-induced focal seizures.

Patient (Age, y/Sex)	1 (61/M)	1 (61/M)	2 (35/F)	3 (66/M)	4 (49/M)	5 (71/M)	6 (43/M)
HV-induced seizures	2 L T subclinical	1 L → R T subclinical	1 R T clinical	1 R → L T subclinical	1 R T subclinical	1 L → R T clinical	1 L T subclinical
Spontaneous seizures	1 R T subclinical	0	0	2 L T subclinical	0	0	0
Interictal epileptiform discharges	None	None	None	None	None	None	None
Other EEG abnormalities	None	None	None	B T int theta/delta	None	None	None
Time from illness onset to EEG	1 week	2 weeks	18 months	4 years	10 months	1 month	9 months
Diagnosis before EEG	No	No	AE[a]	AE[b]	AE[a]	No	AE[a]
Diagnosis after EEG	AE[a]		AE[a]	AE[b]	AE[a]	AE[c]	AE[a]
Hyponatremia at illness onset	No		Yes[d]	Yes[e]	Yes[e]	No	Yes[e]
Anti-seizure medication at time of EEG	None	None	LVT 1500bid, CLB 10qhs	LVT 1500bid	LVT 500bid	None	LVT 1250bid
Immunotherapy before EEG	No	No	Yes[f,g,h,i,j]	Yes[f,g,h,k]	Yes[f,g,h]	No	Yes[f,g,h]
Immunotherapy at time of EEG	No	No	No	No	No	No	Yes[h]
Immunotherapy after EEG	Yes[h]		Yes[g]	Yes[f,g,k]	Yes[f,g,j]	Yes[f,h]	Yes[h,j]

- ✓ HV is a standard activating procedure in EEG
- ✓ The proportion of focal epilepsy patients with HV-triggered seizures is 0.46%

Holmes et al, Epilepsia 2004; Wennberg, Ann Clin Trans Neurol 2023

Seizure: European Journal of Epilepsy 69 (2019) 204–206

Contents lists available at ScienceDirect

Seizure: European Journal of Epilepsy

journal homepage: www.elsevier.com/locate/seizure

Clinical letter

Olfactory stimulus-induced temporal lobe seizures in limbic encephalitis

Federica Avorio, Alessandra Morano, Martina Fanella, Jinane Fattouch, Mariarita Albini, Luca M. Basili, Emanuele Cerulli Irelli, Giacomo Fisco, Mario Manfredi, Anna T. Giallonardo, Carlo Di Bonaventura*

Clinical commentary

Epileptic Disord 2021; 23 (5): 1-6

Epileptic Disorders

Musicogenic epilepsy in paraneoplastic limbic encephalitis: a video-EEG case report

Alessandra Morano[1], Biagio Orlando[1], Martina Fanella[1,2], Emanuele Cerulli Irelli[1], Claudio Colonnese[3], Pierpaolo Quarato[4], Anna Teresa Giallonardo[1], Carlo Di Bonaventura[1]

Accepted: 29 June 2017

DOI: 10.1111/ane.12799

CLINICAL COMMENTARY

WILEY Acta Neurologica Scandinavica

Musicogenic reflex seizures in epilepsy with glutamic acid decarbocylase antibodies

M. Falip[1] | L. Rodriguez-Bel[2] | S. Castañer[3] | J. Miro[1] | S. Jaraba[1] | J. Mora[4] | J. Bas[5] | M. Carreño[6]

Received: 5 December 2020 | Revised: 5 March 2021 | Accepted: 10 March 2021

DOI: 10.1111/epi.16888

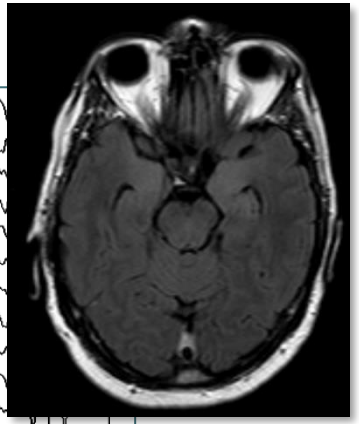
BRIEF COMMUNICATION

Epilepsia

Musicogenic epilepsy: Expanding the spectrum of glutamic acid decarboxylase 65 neurological autoimmunity

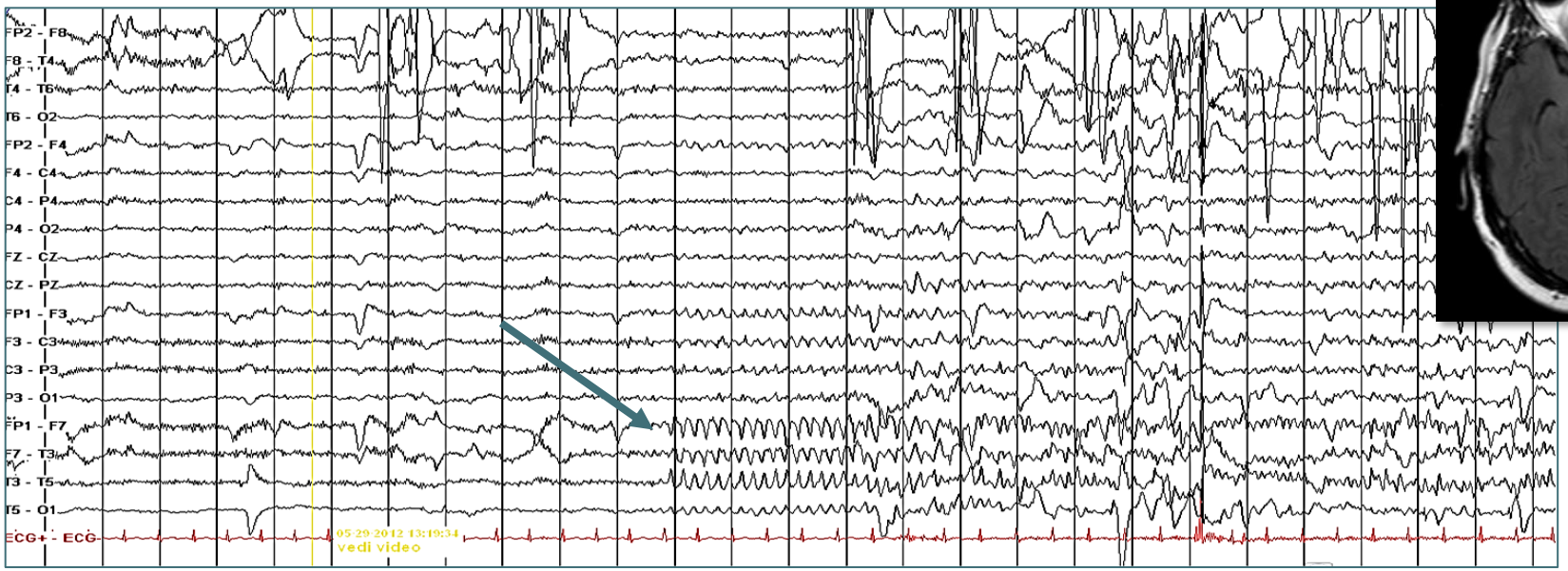
Kelsey M. Smith[1] | Nicholas L. Zalewski[2] | Adrian Budhram[1,3] | Jeffrey W. Britton[1] | Elson So[1] | Gregory D. Cascino[1] | Anthony L. Ritaccio[4] | Andrew McKeon[1,5] | Sean J. Pittock[1,5] | Divyanshu Dubey[1,5]

BILATERAL INDEPENDENT



LEFT mTL SEIZURE

Epigastric sensation, aphasia, amnesia



RIGHT mTL SEIZURE

No signs/symptoms

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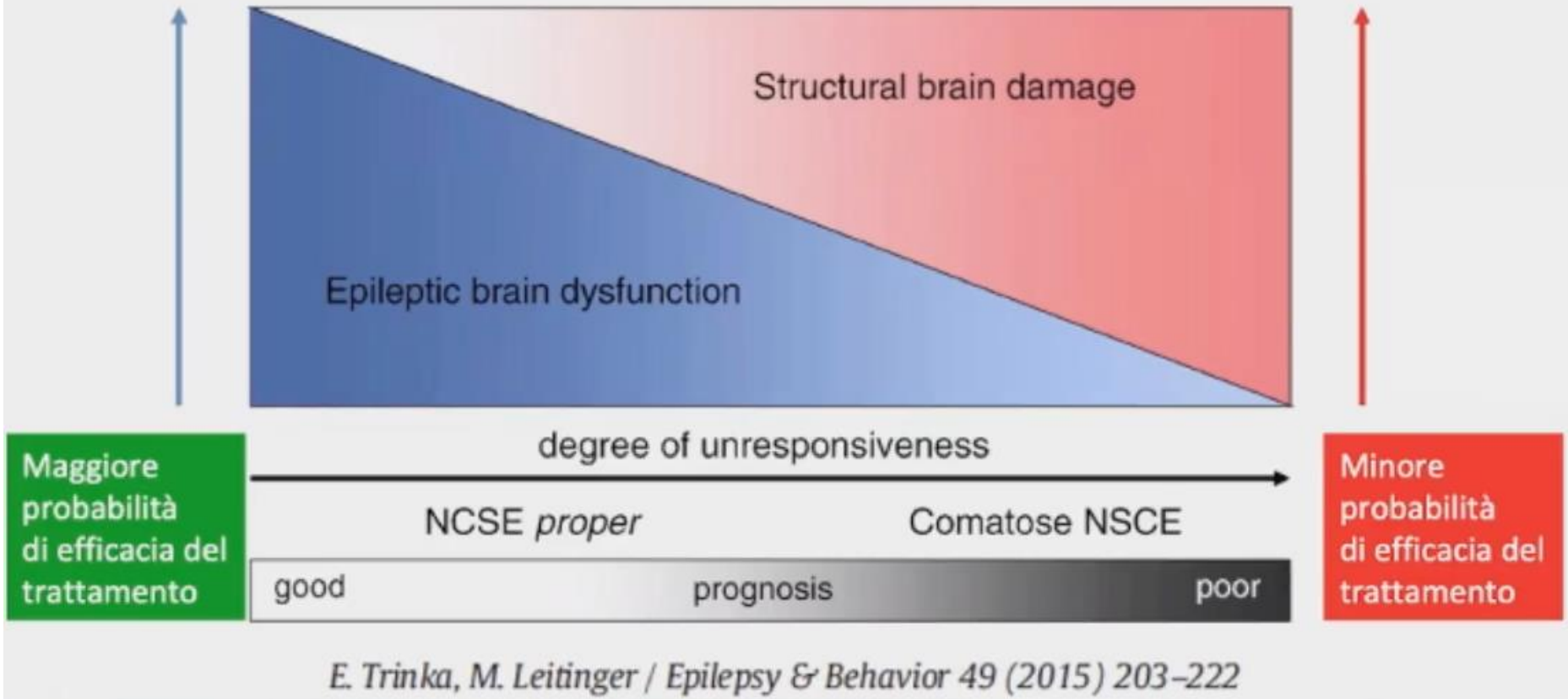
ICTAL SHIFTING

SENC come causa del disturbo di coscienza

SENC come conseguenza del danno cerebrale strutturale

AS in IGE Late AS de novo Atypical AS	Focal SE with impaired consciousness, Aura continua, Status aphasicus	Acute symptomatic focal SE +/- EPC Subtle SE	→ Coma with GPD Coma with LPD



E. Trinka, M. Leitinger / Epilepsy & Behavior 49 (2015) 203–222

To sum up

- Sensitive, but not specific
- High suspicion of NCSE
- Do pharmacological trials, *always*!
- Remember the *'unicorns'*