

EEG e Stato Epilettico in area critica

Stefano Meletti



4° CORSO RESIDENZIALE

EEG e POTENZIALI EVOCATI

22 – 27 NOVEMBRE 2021






Argomenti

- Epidemiologia
- Semeiologia
- EEG
- Neuroimmagini

1 Epidemiologia



Epidemiology of status epilepticus in adults: A population-based study on incidence, causes, and outcomes

**Markus Leitinger^{1,2} | Eugen Trinka^{1,2,3}  | Giada Giovannini⁴  | Georg Zimmermann^{1,2,5} |
Cristina Florea^{1,2} | Alexandra Rohracher^{1,2} | Gudrun Kalss^{1,2} | Caroline Neuray^{1,2} |
Rudolf Kreidenhuber^{1,2} | Julia Höfler^{1,2} | Giorgi Kuchukhidze^{1,2} | Claudia Granbichler¹ |
Judith Dobesberger^{1,2} | Helmut F. Novak^{1,2} | Georg Pilz^{1,2} | Stefano Meletti^{4,6}  | Uwe Siebert^{3,7,8}**

Incidenza del NCSE

l'incidenza di NCSE e' intorno ai **12 casi per 100.000 abitanti anno** (forme non convulsive pure, escludendo le forme post-anossiche).

Il 34% di tutti gli SE era rappresentato da NCSE

Il NCSE puo' rappresentare anche l'evoluzione da una precedente forma motoria

Il trend negli anni mostra un **aumento dei NCSE** (miglioramento delle tecniche diagnostiche eg. cEEG in ICU)

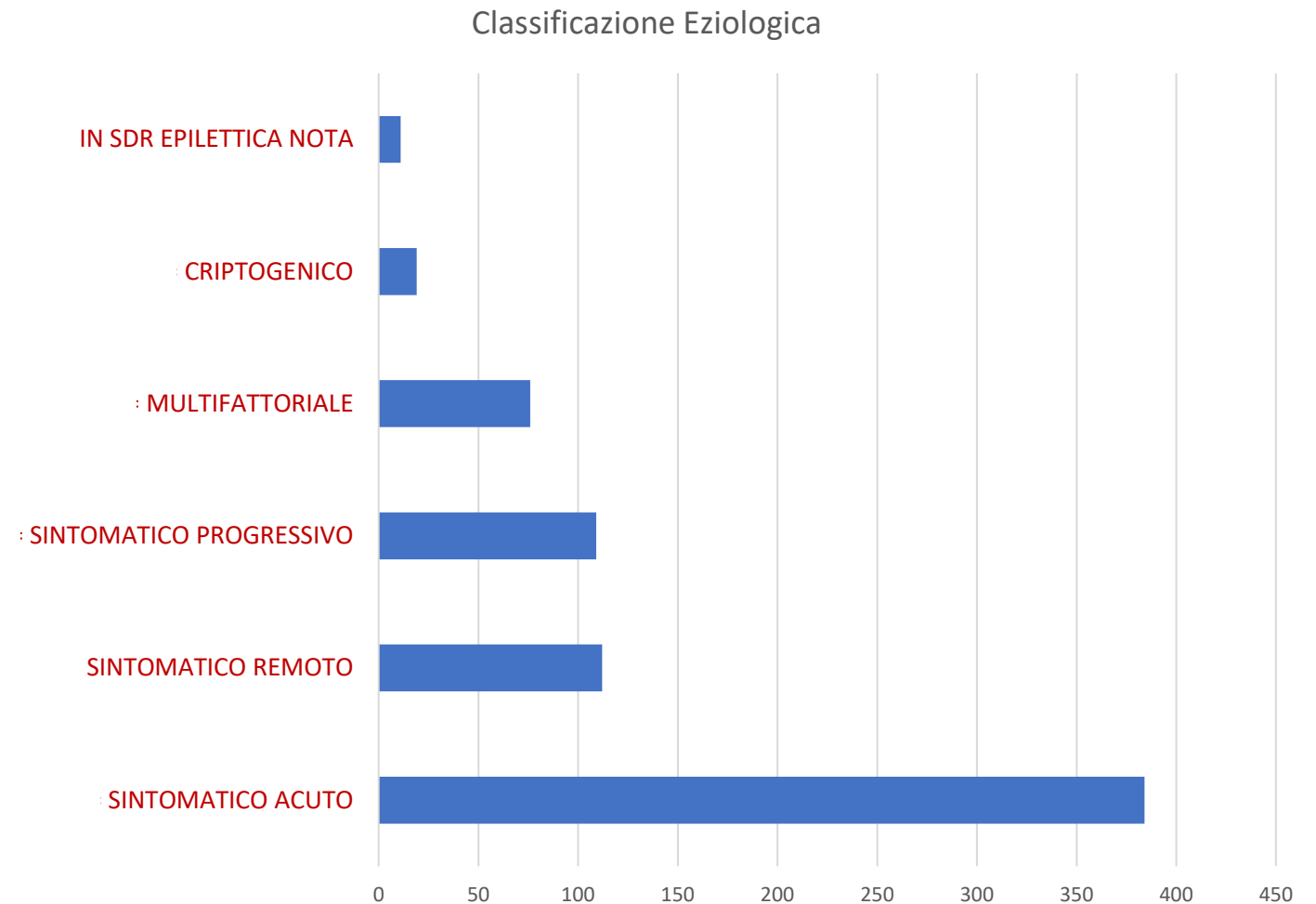
La **mortalita'** del NCSE e' risultata pari al **28% (14% nei NCSE non in coma; 41% nei NCSE in coma)**

Modena – SE registry 2014-2021

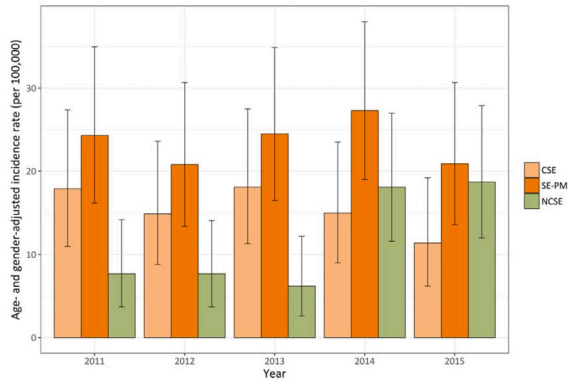
- **711** SE cases
- Mean age 70 yrs



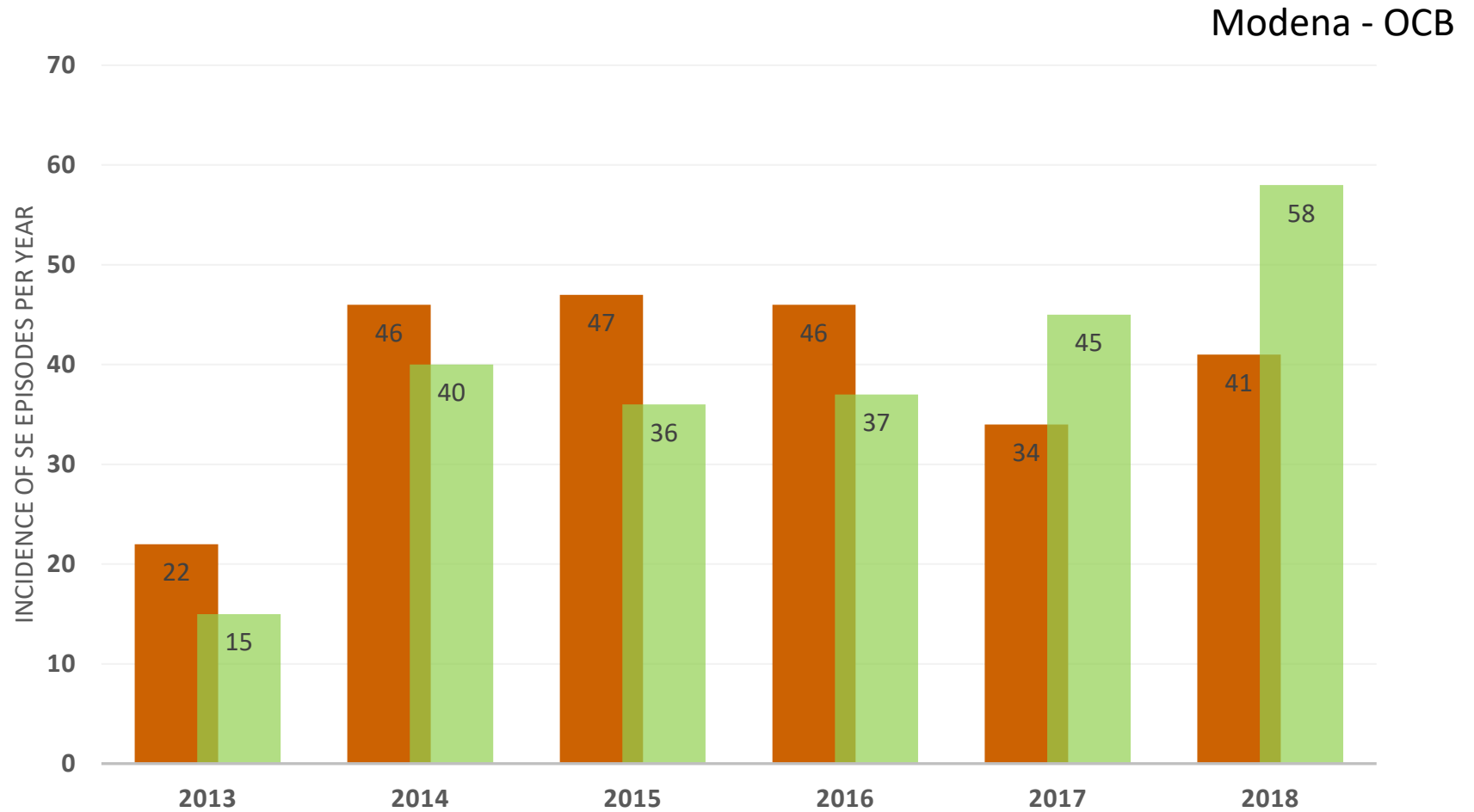
Giada Giovannini



NCSE nel tempo Salzburg / Modena



Leitinger, Giovannini et al., 2019

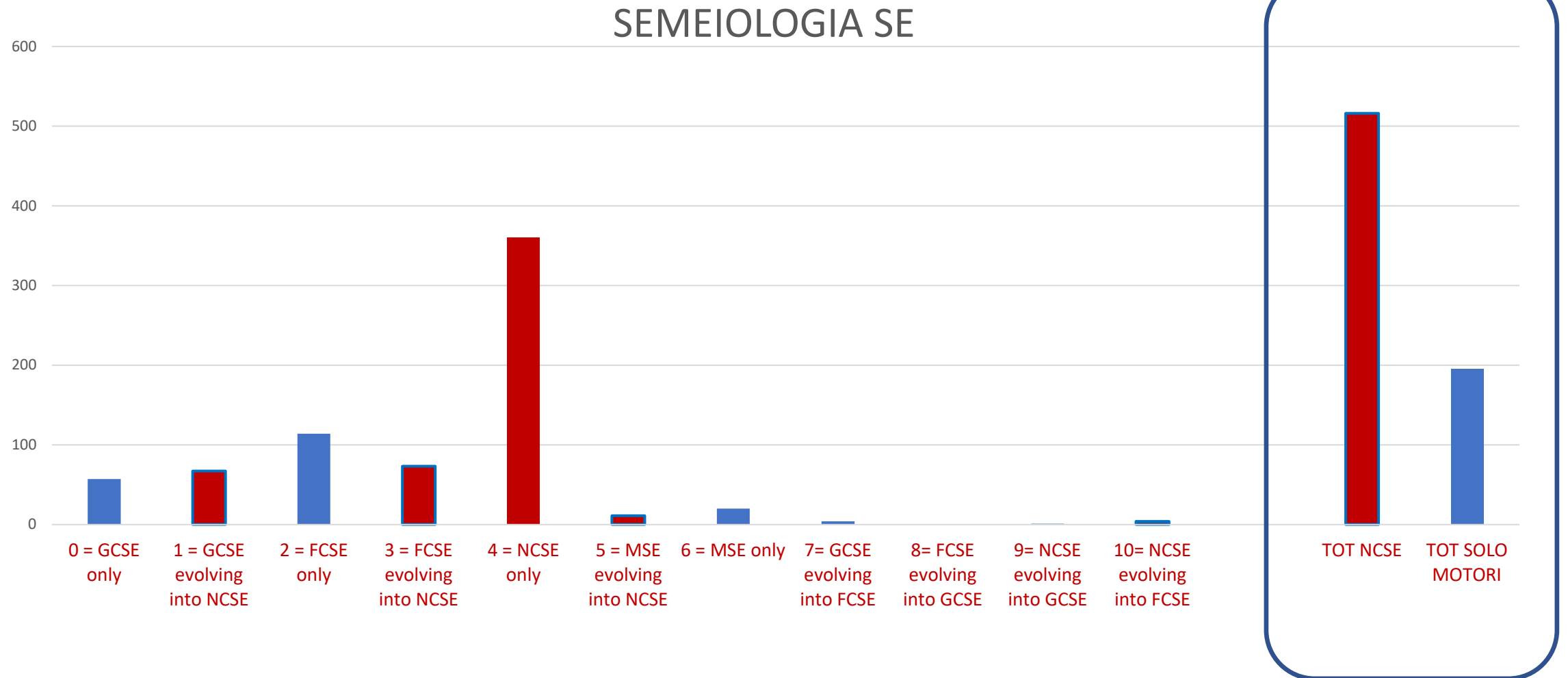


↑ Registro SE

■ MSE ■ NCSE

↑ cEEG monitoring available

Modena SE registry ^{711 casi}



Semeiologia e mortalità

	Level of consciousness	N	Case fatality, % (95% CI)	Age, median (range)
(A) Convulsive semiology at the end of SE, or as the only semiology	NA	57	0.0 (0.0-7.6)	63 (20-91)

	Level of consciousness	N	Case fatality, % (95% CI)	Age, median (range)
(A) Convulsive semiology at the end of SE, or as the only semiology	NA	57	0.0 (0.0-7.6)	63 (20-91)
(B) Convulsive semiology at the beginning of SE; focal motor-, tonic-, myoclonic-, and nonconvulsive semiology in a semiologic sequence; focal motor SE, tonic SE, myoclonic SE	Fully awake and awake with reduced cognition	36	2.8 (0.0-15.4)*	62 (25-87)
	Somnolence, stupor, coma	52	26.9 (16.7-40.4)*	69 (23-97)

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(C) Nonconvulsive SE	Fully awake, and awake with reduced cognition	37	13.5 (5.4-28.5)	71 (20-94)
	Somnolence, stupor, coma	39	41.0 (27.1-56.6)	78 (25-99)
(B and C)	Fully awake, and awake with reduced cognition	73	8.2 (3.5-17.1)*	67 (20-94)
	Somnolence, stupor, coma	91	33 (24.1-43.2)*	72 (23-99)

Clusters nello SE



Simona Lattanzi

Semiology of SE with prominent motor phenomena

Etiology of SE was defined as symptomatic (acute, remote, progressive) or unknown.

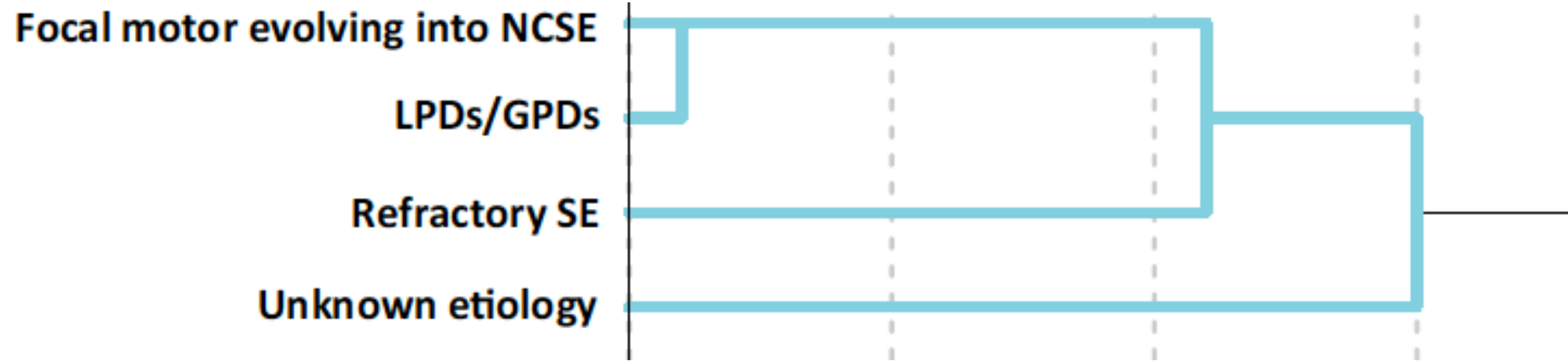
Electroencephalogram (EEG) recordings were searched for lateralized periodic discharges (LPDs), generalized sharply and/or triphasic periodic potentials (GPDs), and spontaneous burst suppression (BS).

Treatment response, SE was classified into responsive, refractory and super-refractory.

Average linkage hierarchical cluster analysis was performed with Pearson's correlation as a similarity measure.

	SE episodes (n = 240) n (%)
Etiology	
Acute symptomatic	139 (57.9)
Progressive symptomatic	56 (23.3)
Remote symptomatic	39 (16.3)
Unknown	6 (2.5)
Semiological dynamics	
Convulsive only	42 (17.5)
Focal motor only	82 (34.2)
Myoclonic only	3 (1.3)
Convulsive evolving into NCSE	50 (20.8)
Focal motor evolving into NCSE	61 (25.4)
Myoclonic evolving into NCSE	2 (0.8)
Electroencephalographic pattern	
LPDs/GPDs	76 (31.7)
Spontaneous BS	2 (0.8)
No LPDs/GPDs/BS	162 (67.5)
Response to treatment	
Responsive	181 (75.4)
Refractory	35 (14.6)
Super-refractory	24 (10.0)

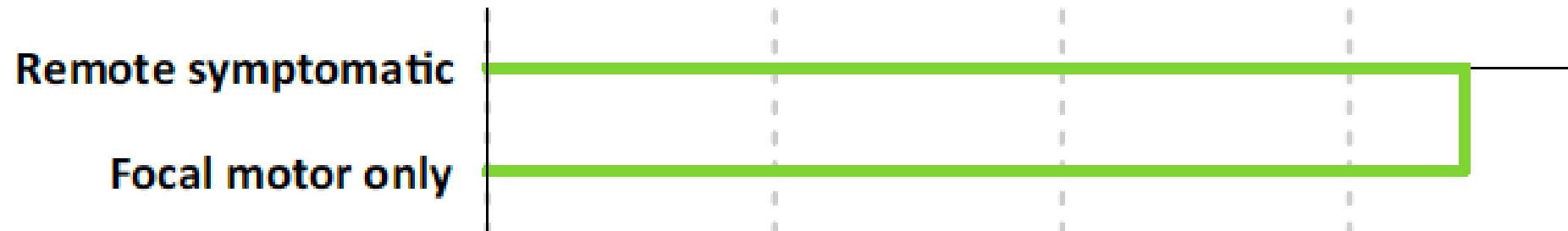
Cluster #1



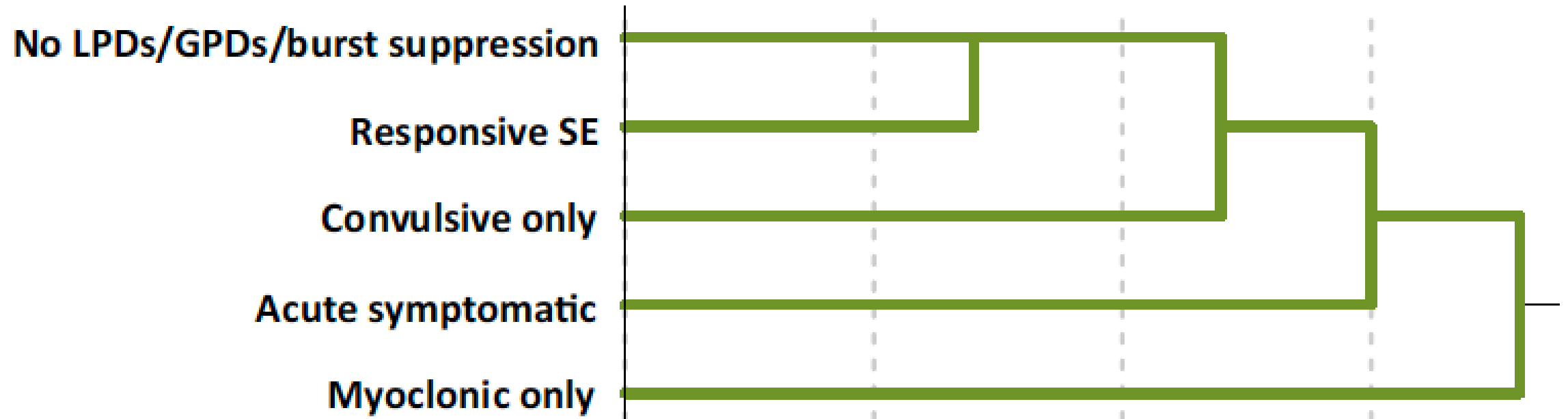
Cluster #3



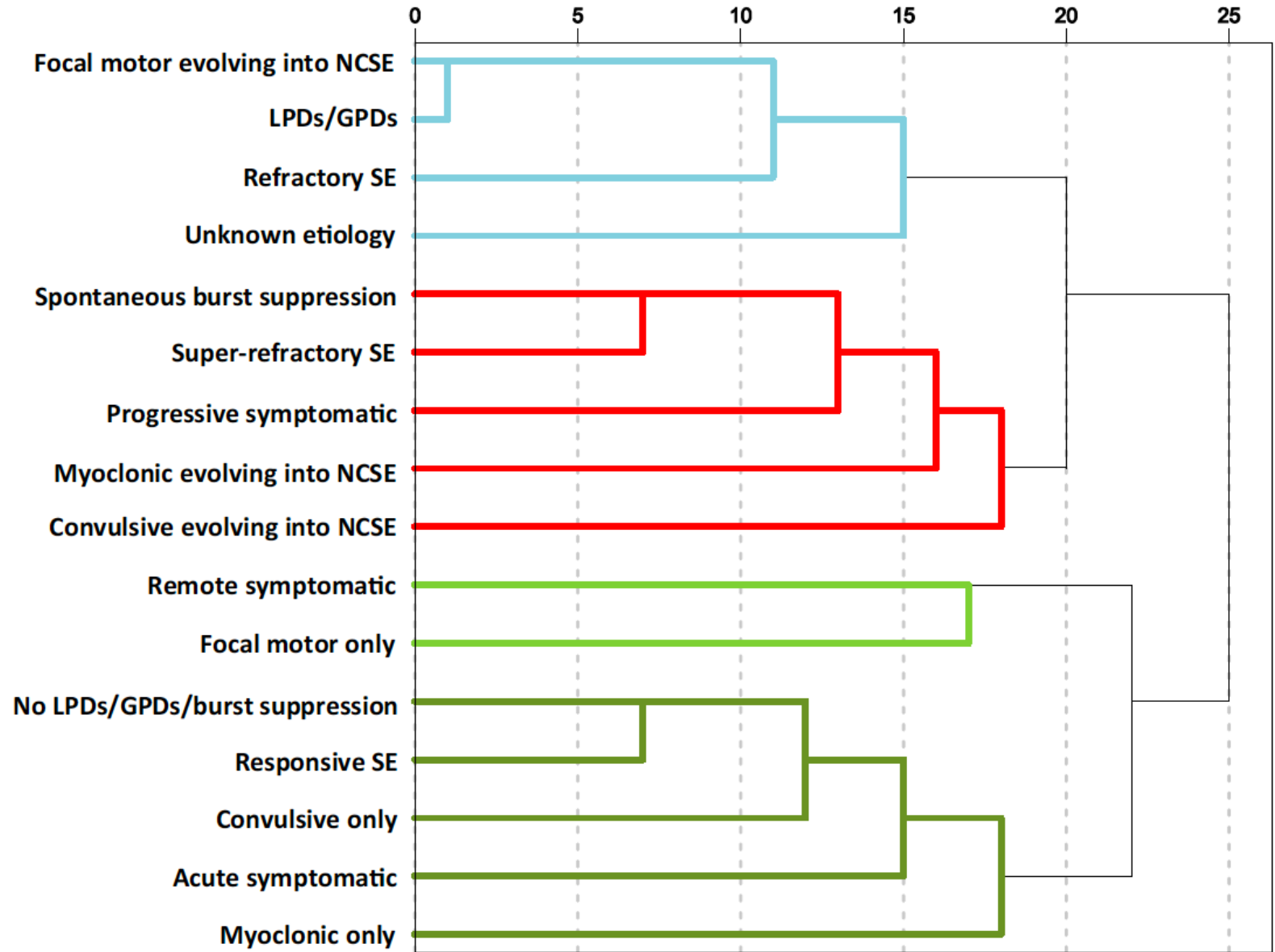
Cluster #3



Cluster #4



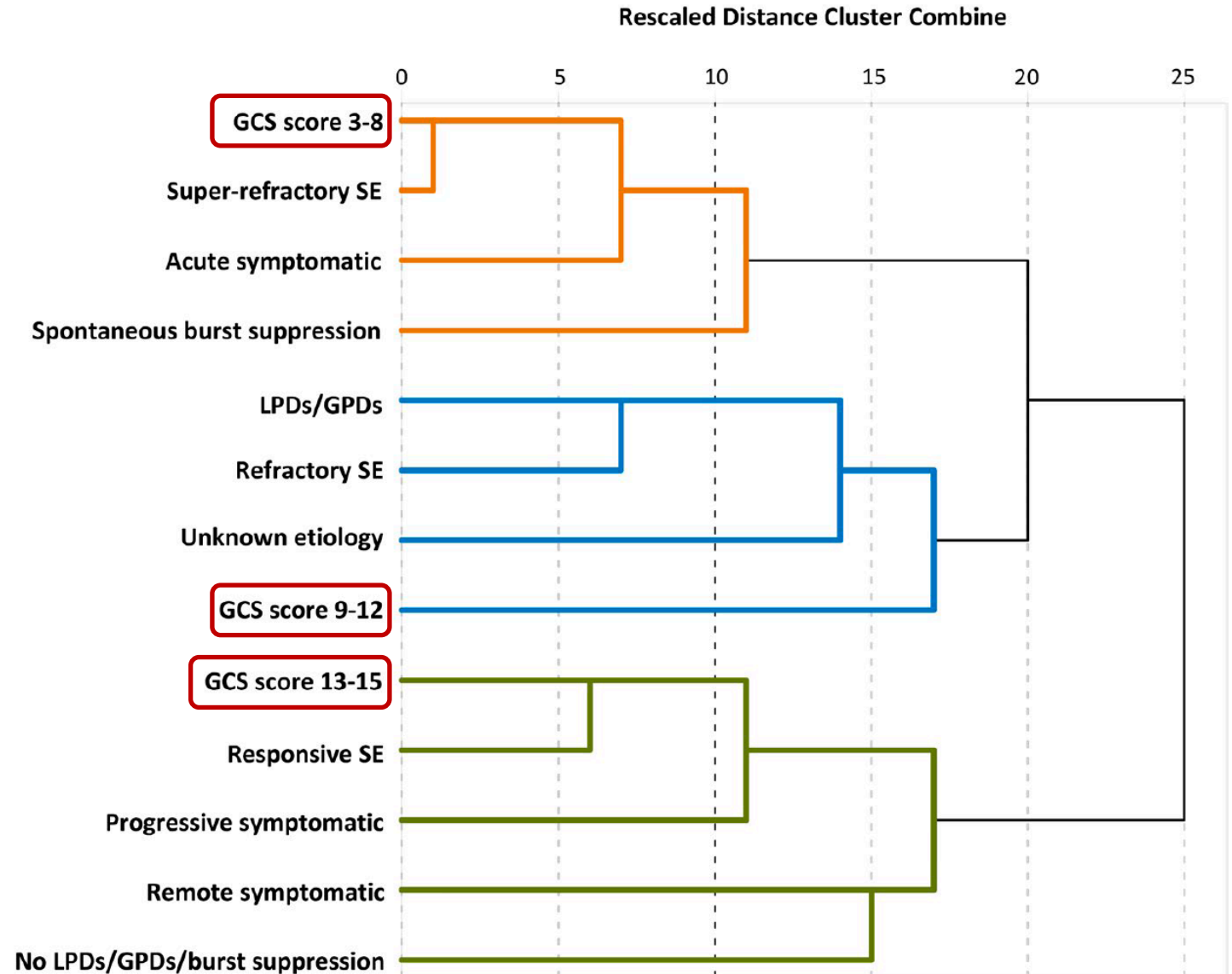
Rescaled Distance Cluster Combine



Clinical phenotypes within nonconvulsive status epilepticus

Simona Lattanzi¹ | Giada Giovannini^{2,3} | Francesco Brigo^{4,5} | Nicolò Orlandi^{2,6} | Eugen Trinka^{7,8,9} | Stefano Meletti^{2,6}

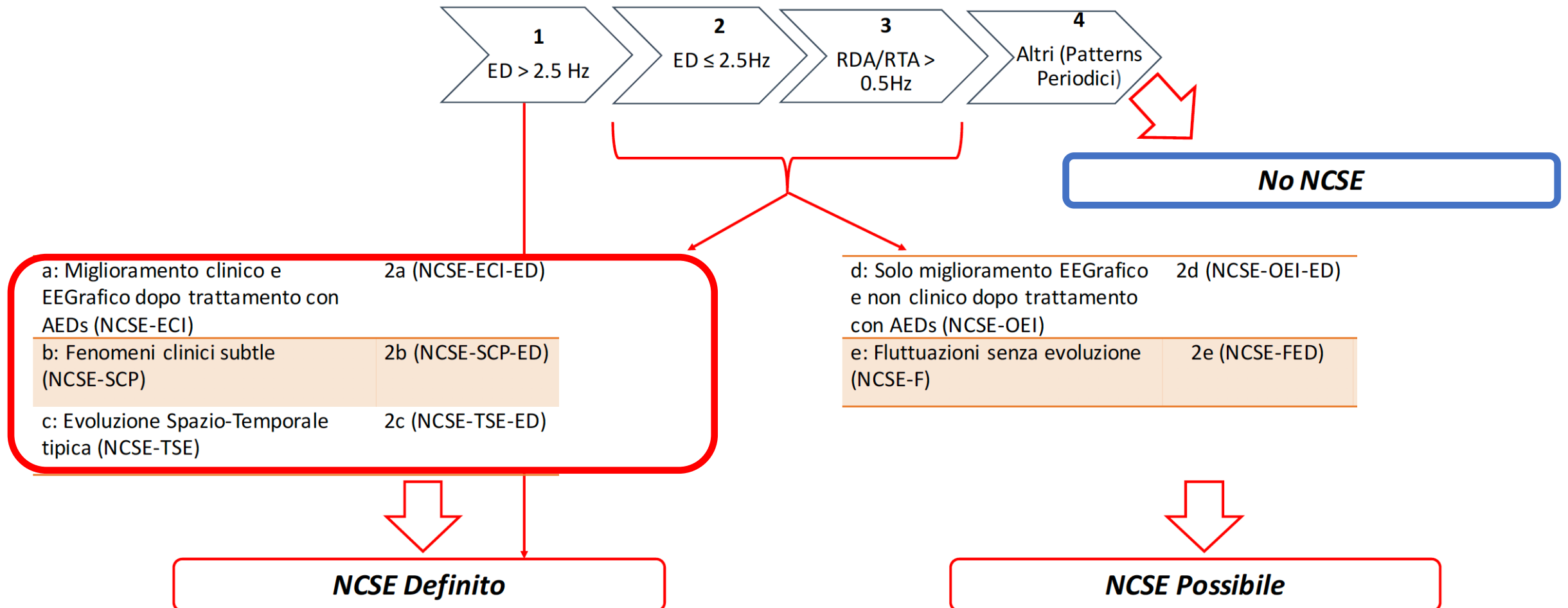
Characteristic	Status epilepticus episodes, <i>n</i> = 229
Etiology	
Acute symptomatic	149 (65.1)
Remote symptomatic	39 (17.0)
Progressive symptomatic	33 (14.4)
Unknown	8 (3.5)
Level of consciousness impairment	
GCS score 3–8	61 (26.6)
GCS score 9–12	69 (30.1)
GCS score 13–15	99 (43.2)
Electroencephalographic pattern	
LPDs/GPDs	61 (26.6)
Spontaneous burst suppression	2 (.9)
No LPDs/GPDs/burst suppression	166 (72.5)
Response to treatment	
Responsive	182 (79.5)
Refractory	26 (11.3)
Superrefractory	21 (9.2)



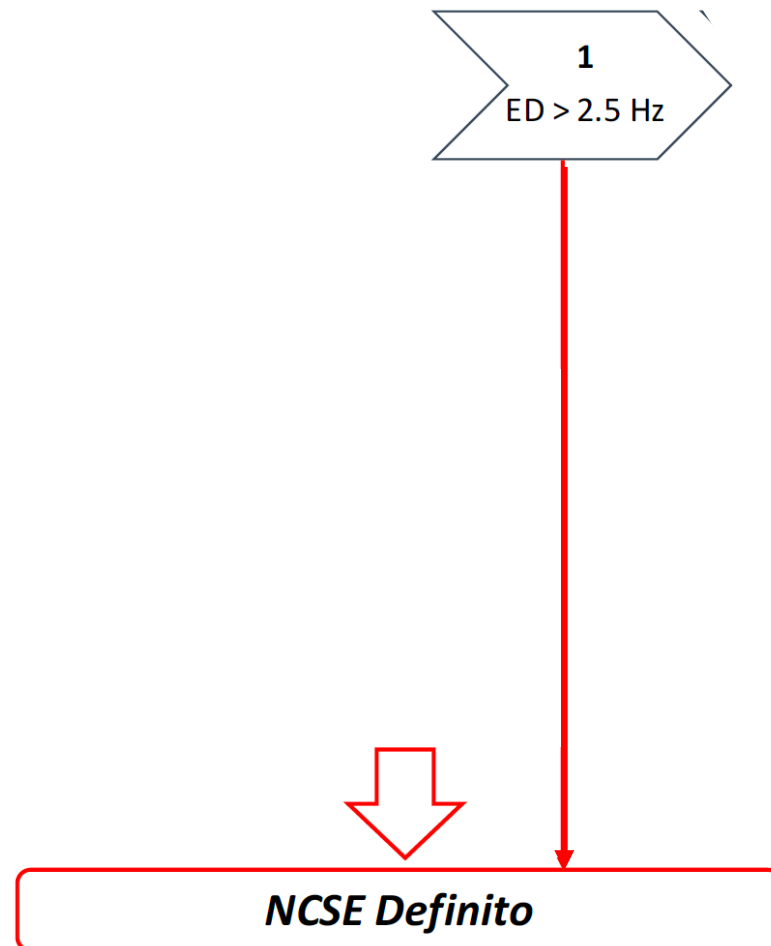
3 EEG



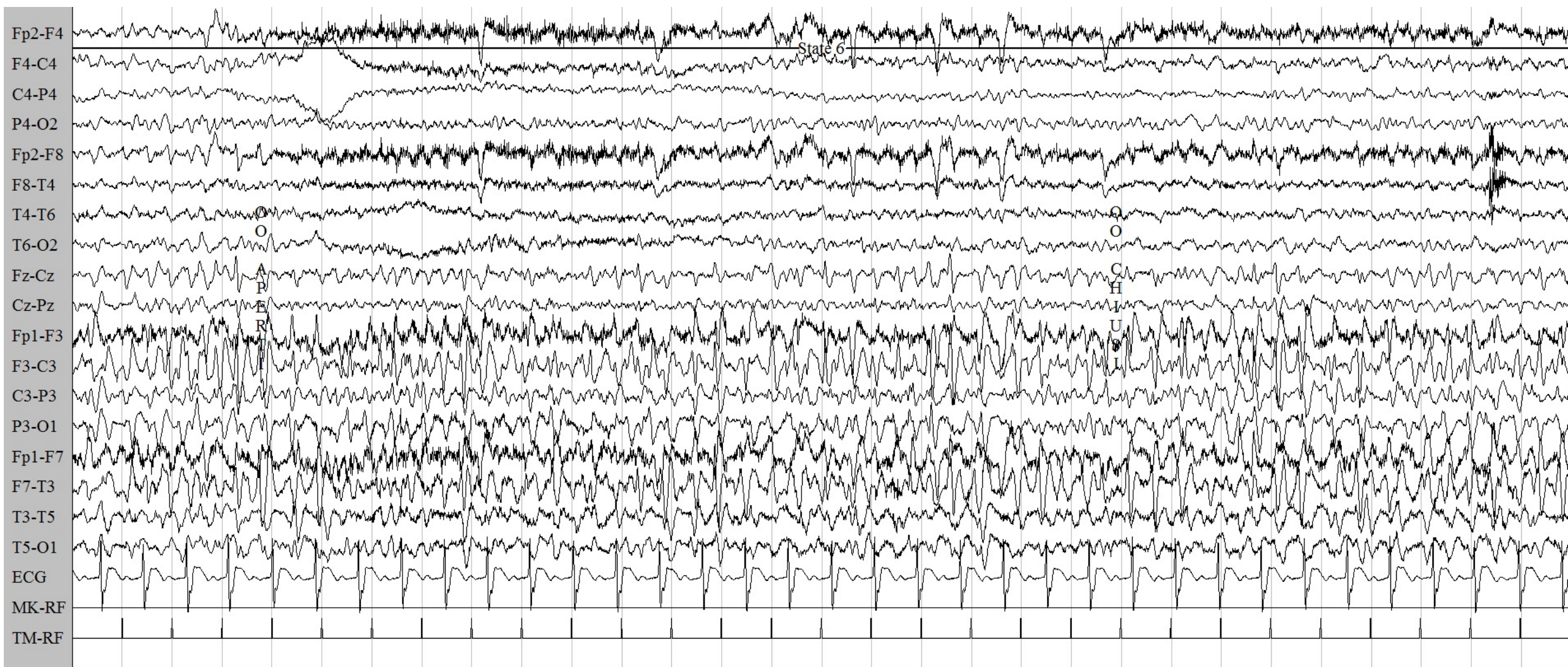
Diagnosi: Salzburg EEG criteria



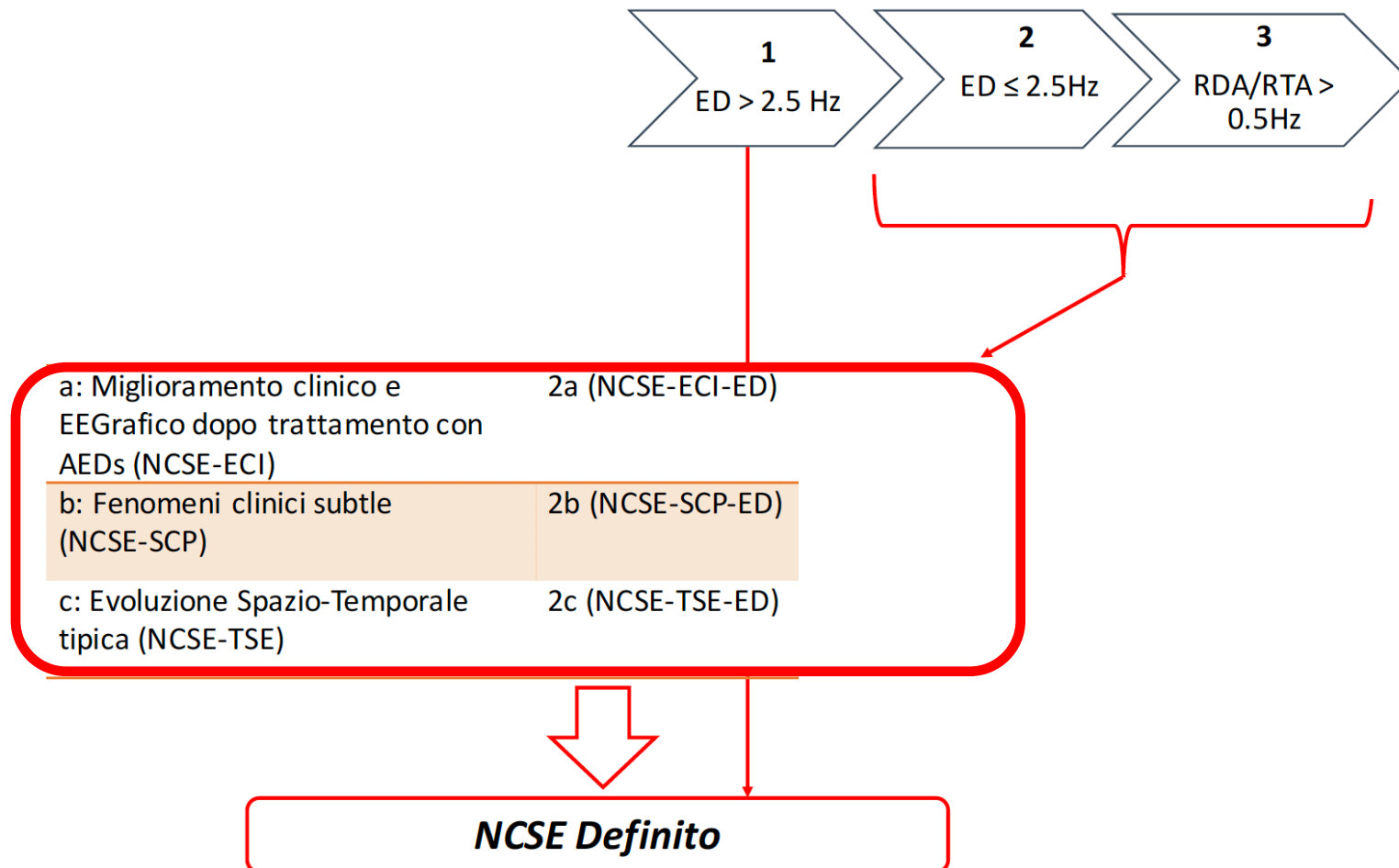
Diagnosi: Salzburg EEG criteria



ED > 2.5 Hz NCSE definito



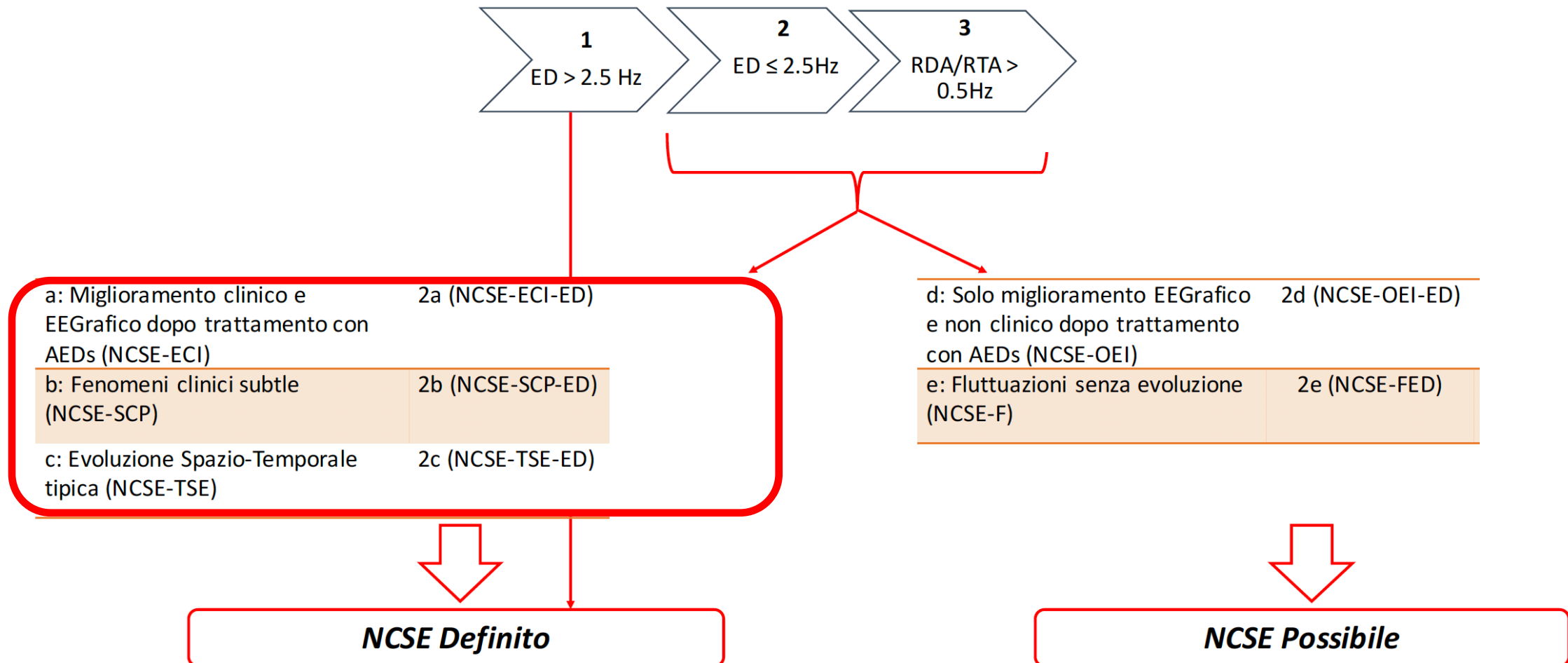
Diagnosi: Salzburg EEG criteria



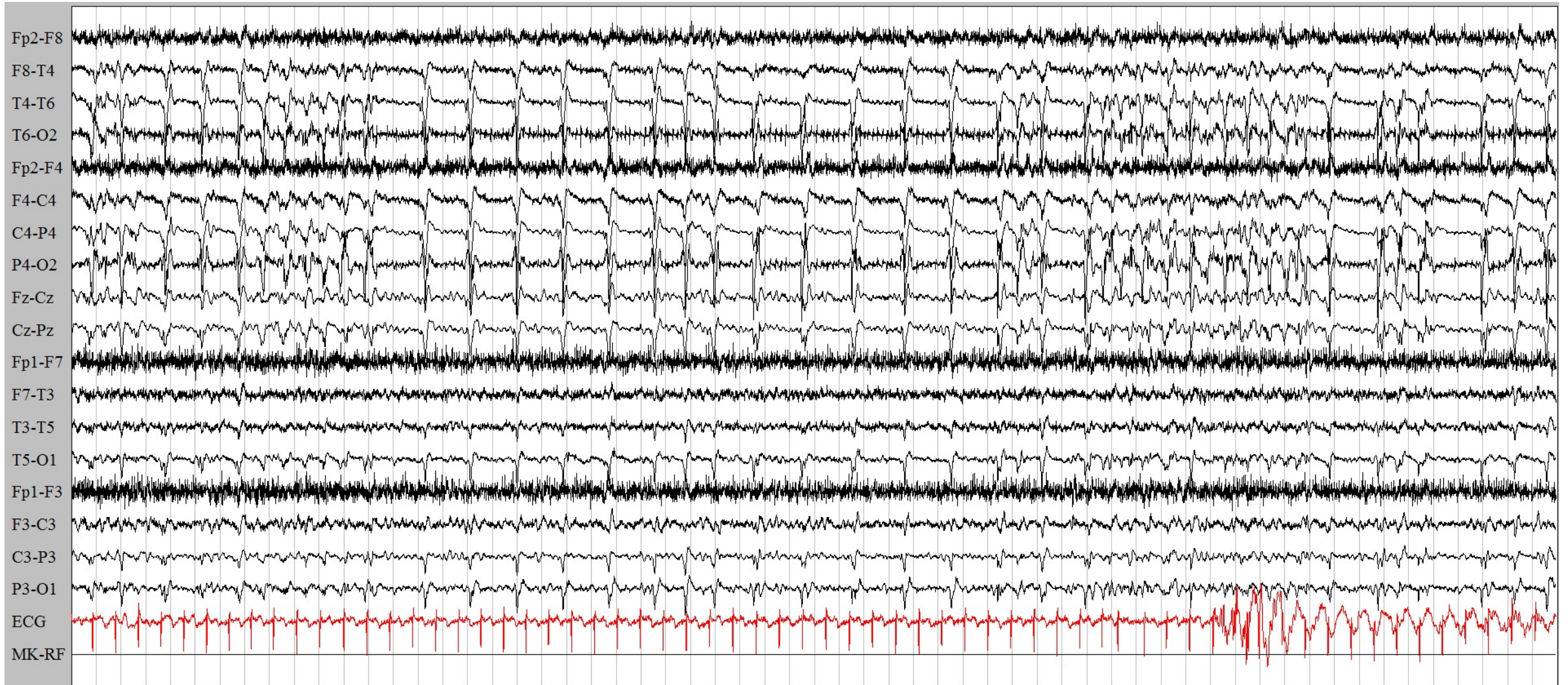
Crisi discrete – evoluzione ST: NCSE definito



Diagnosi: Salzburg EEG criteria

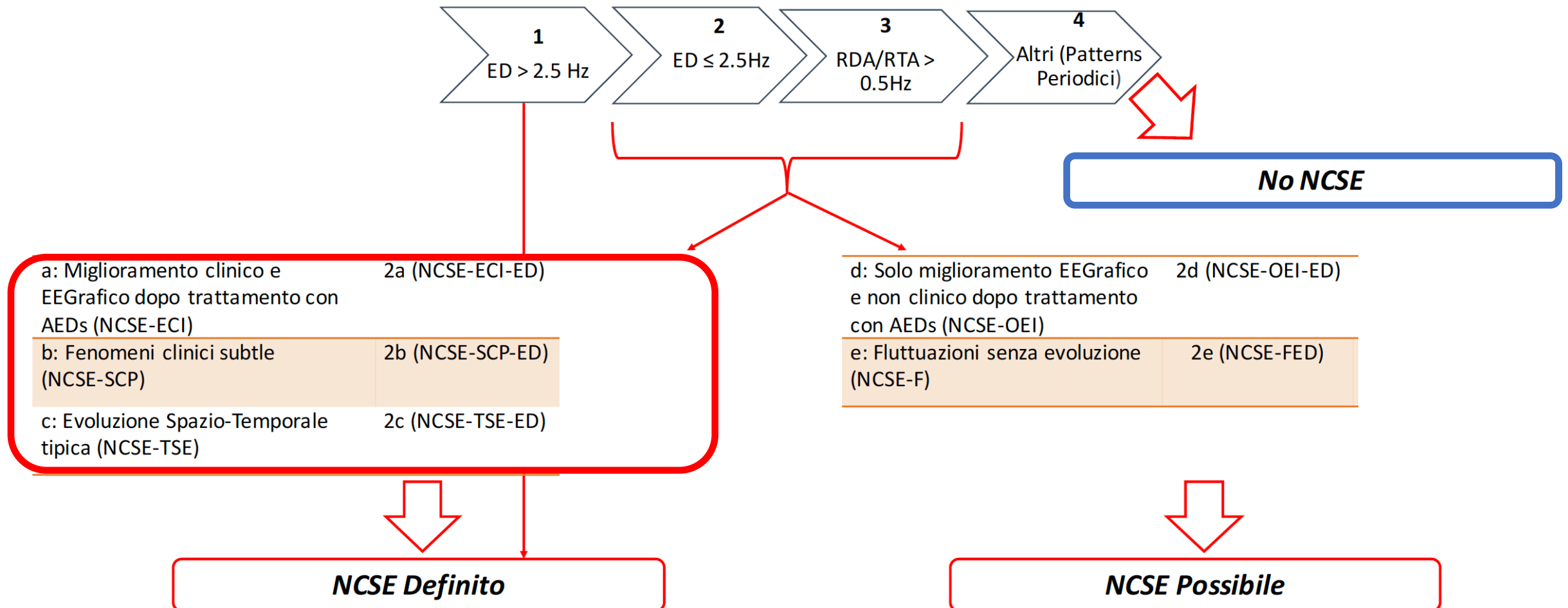


ED < 2.5 Hz – fluttuazione ma non evoluzione ST

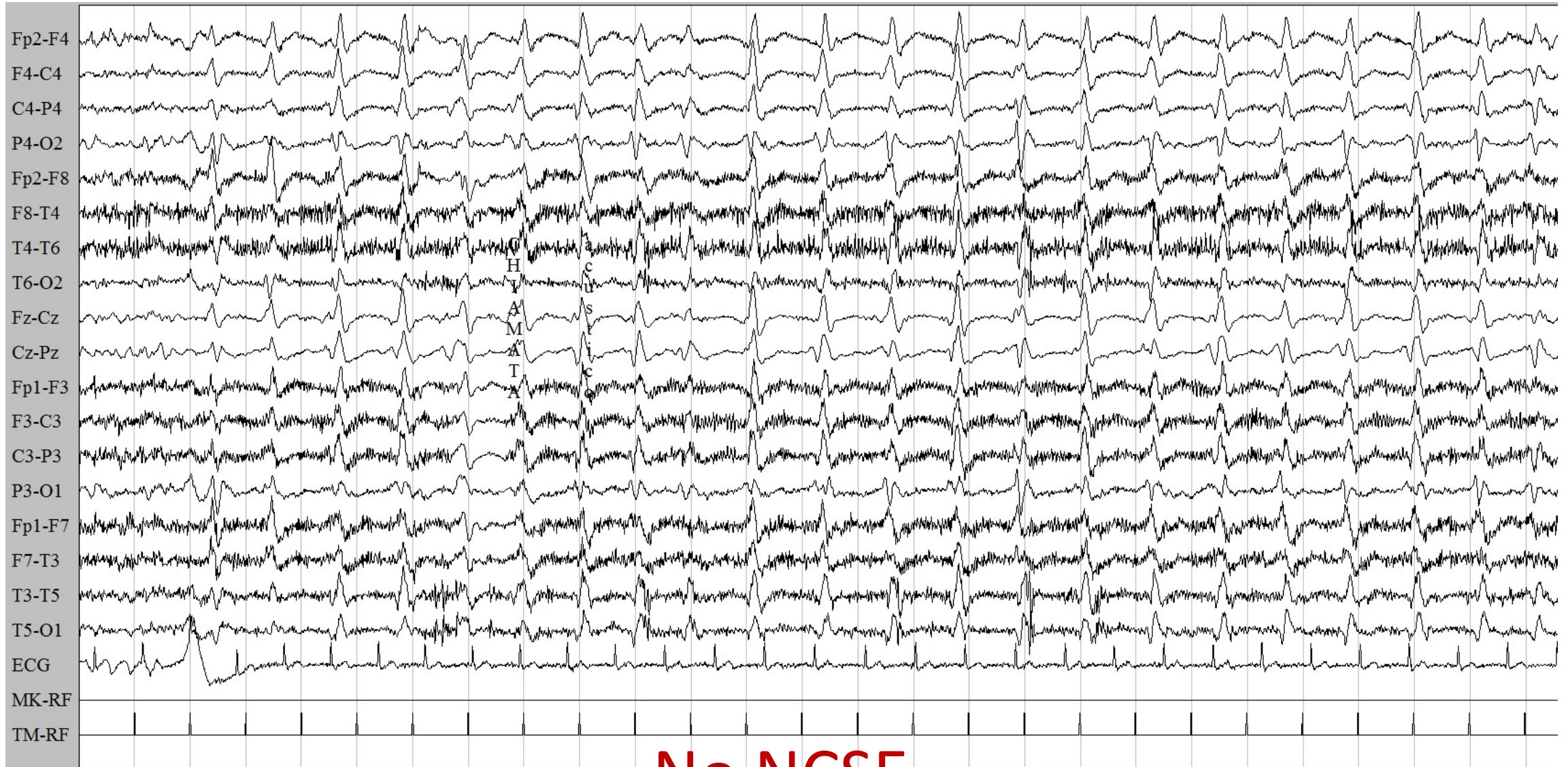


Possible NCSE

Diagnosi: Salzburg EEG criteria





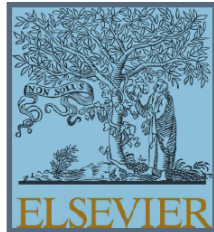
ED 1Hz - no fluttuazioni



No NCSE

BRIEF COMMUNICATION**The difficulty of diagnosing NCSE in clinical practice; external validation of the Salzburg criteria**

Rianne J. M. Goselink^{1,2}  | Jeroen J. van Dillen¹ | Marjolein Aerts¹ | Johan Arends² |
Charlotte van Asch² | Inge van der Linden² | Jaco Pasman¹ | Christiaan G. J. Saris¹ |
Machiel Zwarts²  | Nens van Alfen¹

Contents lists available at [ScienceDirect](#)**Seizure: European Journal of Epilepsy**journal homepage: www.elsevier.com/locate/seizure

The EEG diagnosis of NCSE: Concordance between clinical practice and Salzburg Criteria for NCSE

Alshimaa S. Othman^{a,d}, Stefano Meletti^{a,b,*}, Giada Giovannini^{b,c}



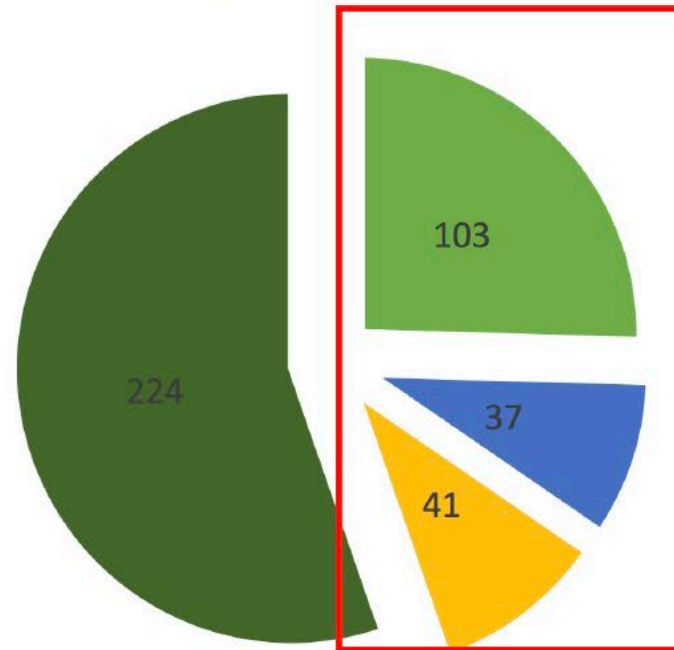
2020

Modena 01 gen – 31 mar 2018

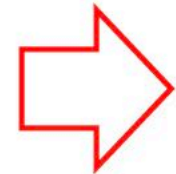
N = 405

- Alterazioni qualitative della coscienza ad esordio acuto
- Stati confusionali acuti
- Alterazioni quantitative della coscienza ad esordio acuto
- Pazienti senza alterazioni di coscienza dopo un sospetto episodio comiziale

Categorie di EEG urgente

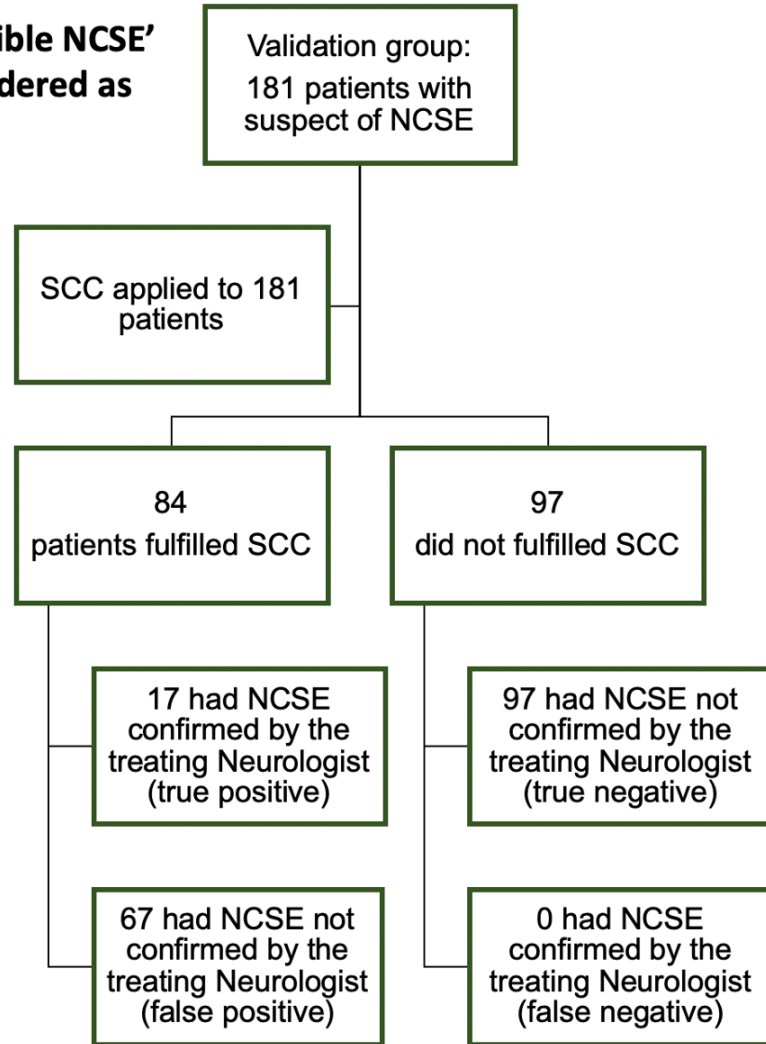


N=181
Sospetti SE presi in considerazione nelle analisi successive



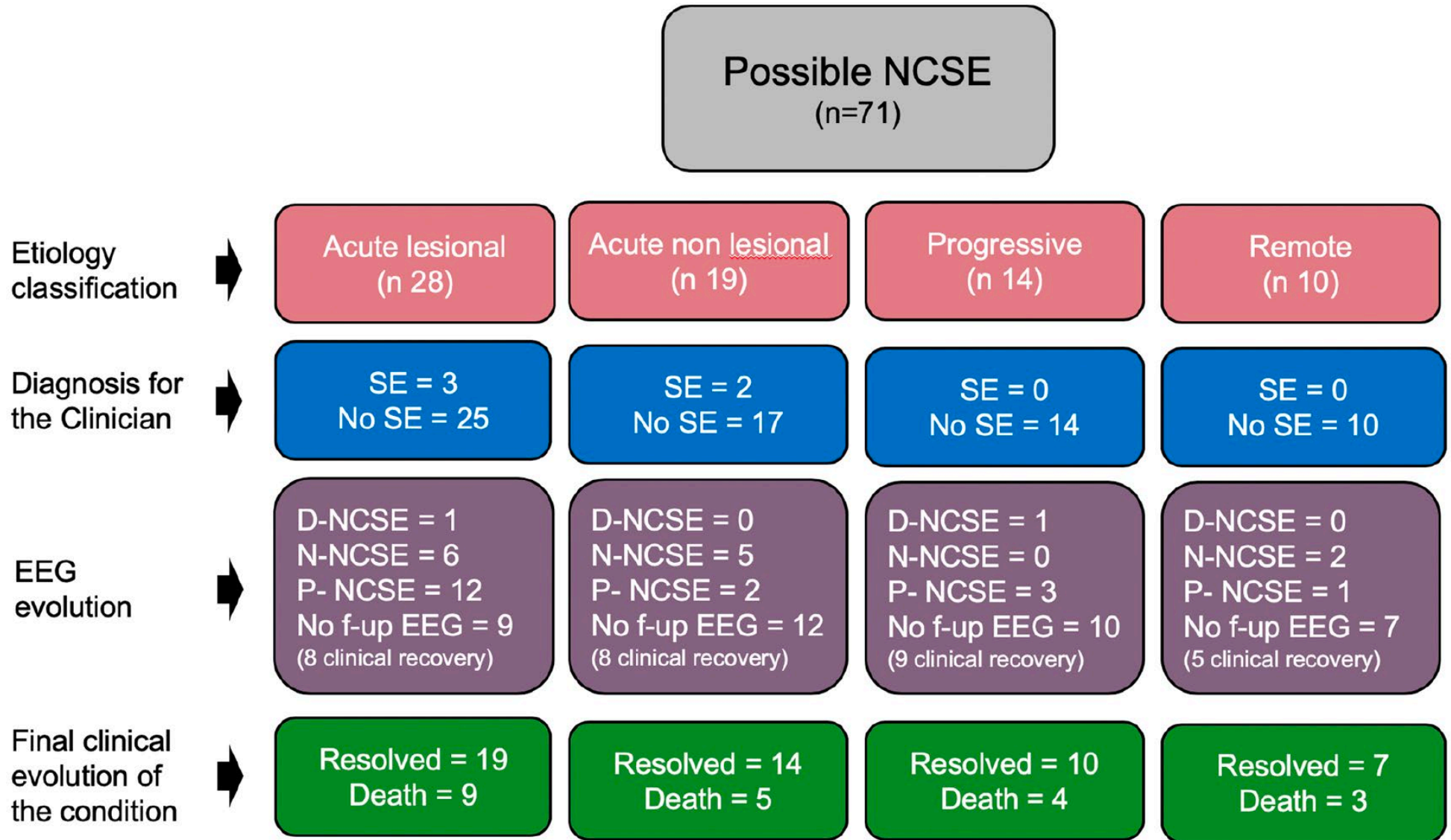
La diagnosi EEG concordanza SC

'Possible NCSE' considered as NCSE



Concordance between the reference standard (treating physician) and SCC depending on the consideration of possible NCSE as “SE” or “Not SE”.

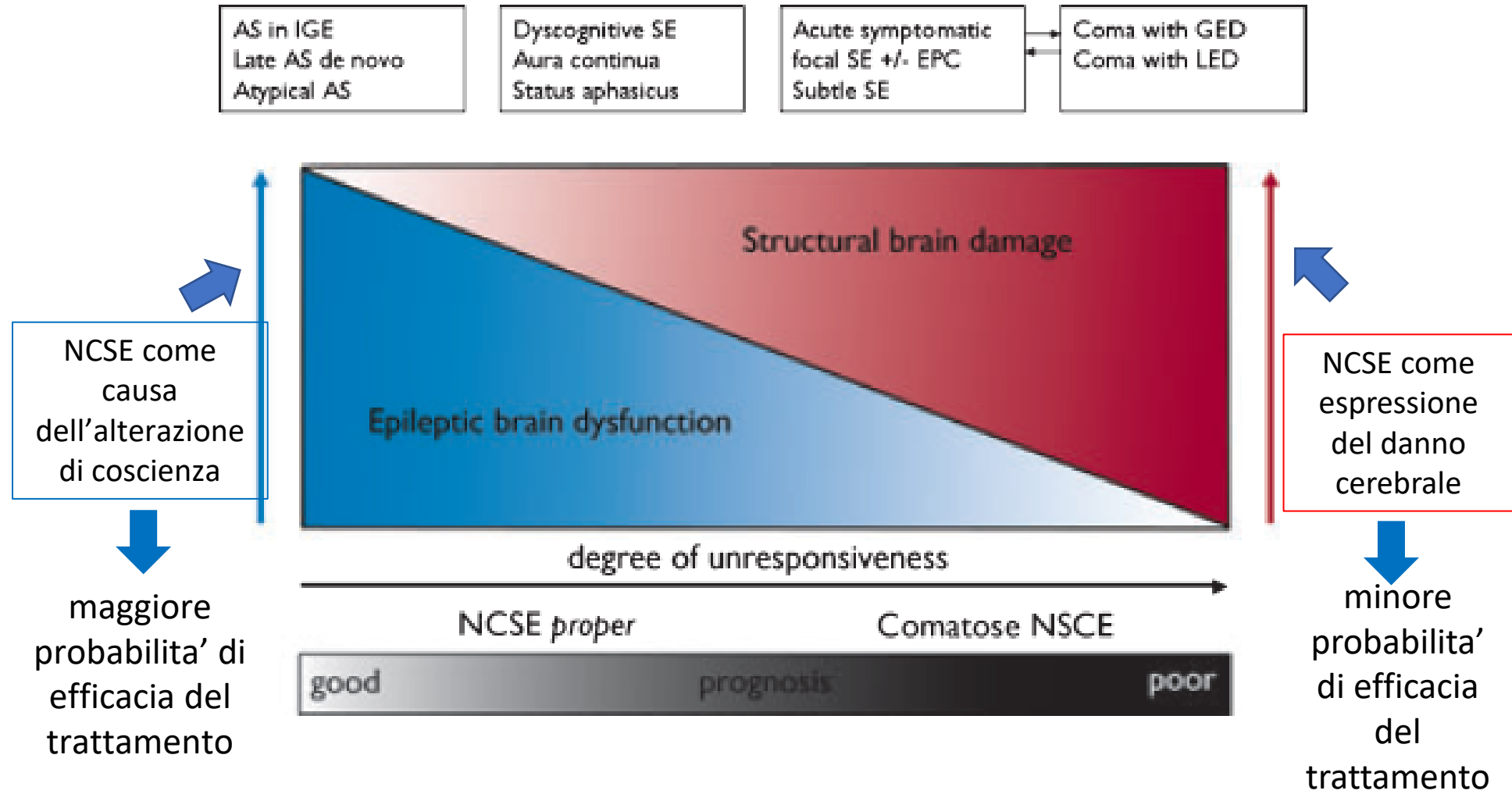
	NCSE for the treating Neurologist	No NCSE for the treating Neurologist	Total	PPV	NPV
Possible NCSE considered as definite SE					
NCSE for SCC	17	66	83	20.5 %	
No NCSE for SCC	0	98	98		100 %
Total	17	164	181		
Sensitivity	100 %				
Specificity		59.8 %			
Possible NCSE considered as no SE					
NCSE for SCC	12	0	12	100 %	
No NCSE for SCC	5	164	169		97 %
Total	17	164	181		
Sensitivity	70.6 %				
Specificity		100 %			



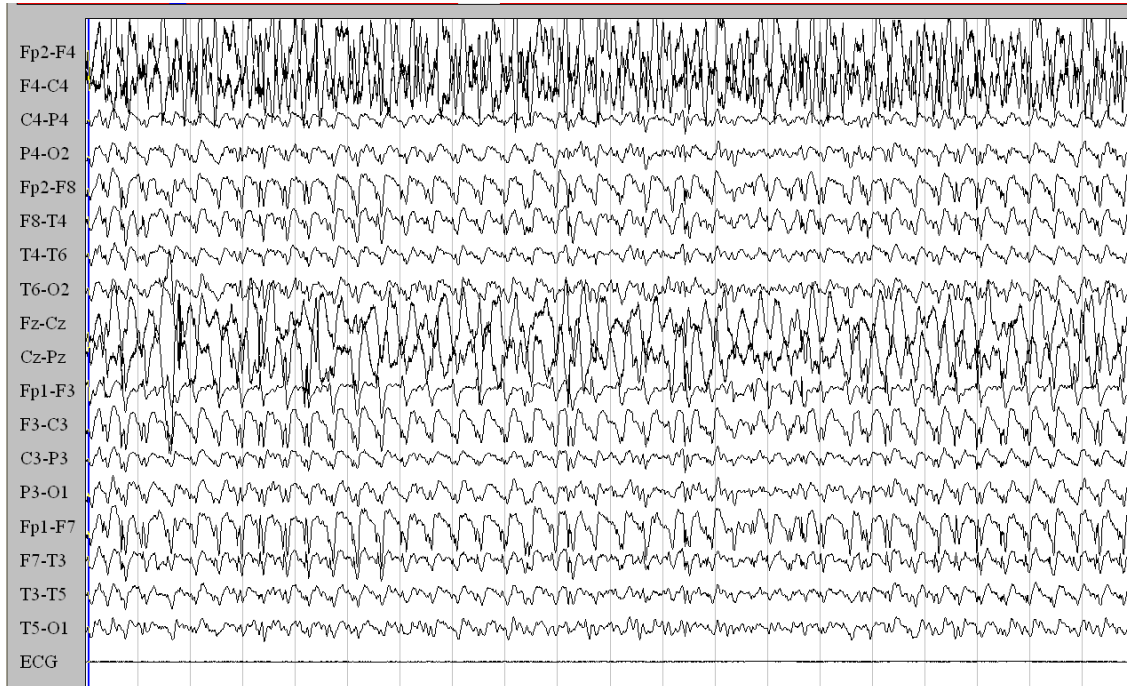
UNIMORE NCSE IN COMA: QUANDO e quanto TRATTARE?

- Nella valutazione del **trattamento** di un sospetto NCSE in coma le **domande fondamentali** da porsi sono:
 1. Il **coma e' causato dalle anomalie epilettiformi osservate** o queste sono un epifenomeno dell'eziologia sottostante?
 2. **Quanto contribuisce** l'attivita' epilettiforme osservata **al grado di coma?**
 3. La continua attivita' epilettiforme **peggiora la prognosi?**
 4. Deve essere trattata qualsiasi attivita' epilettiforme riscontrata e quando instaurato quanto deve essere aggressivo il trattamento?

NCSE IN COMA: QUANDO e quanto TRATTARE?



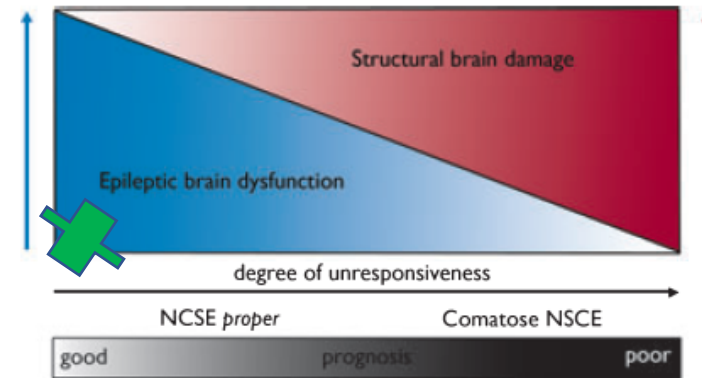
15 aa, esordio acuto di rallentamento ideo-motorio



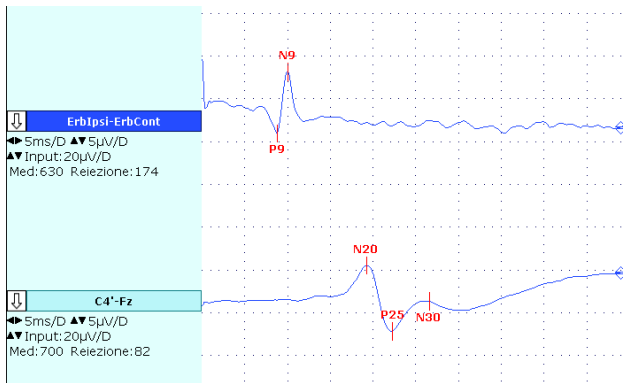
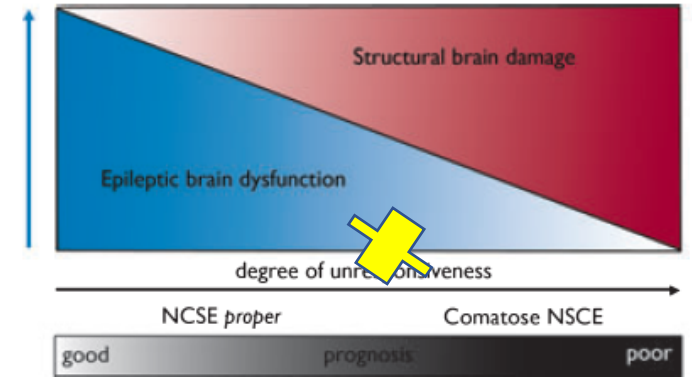
MRI e CT negative



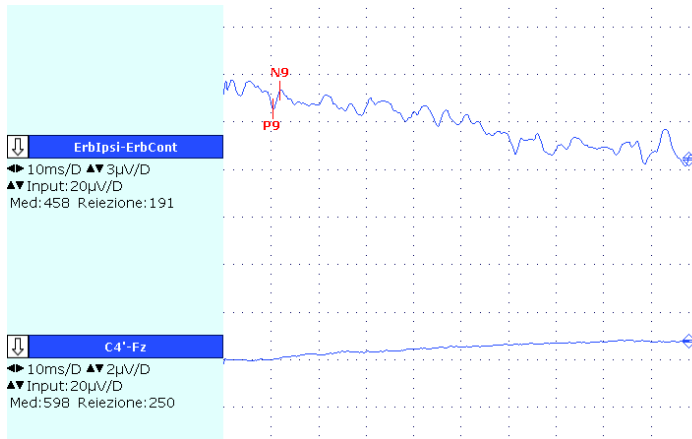
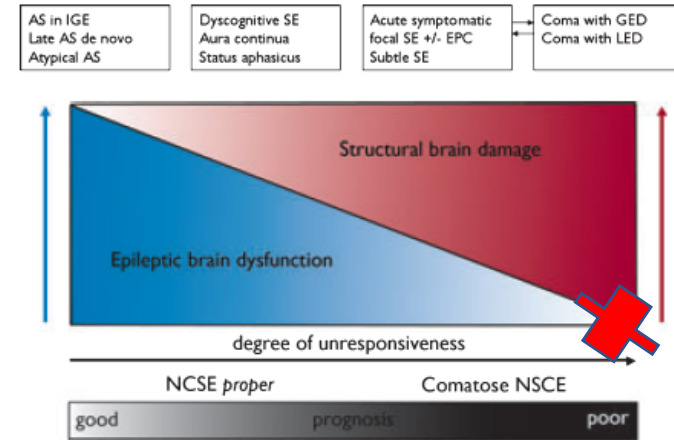
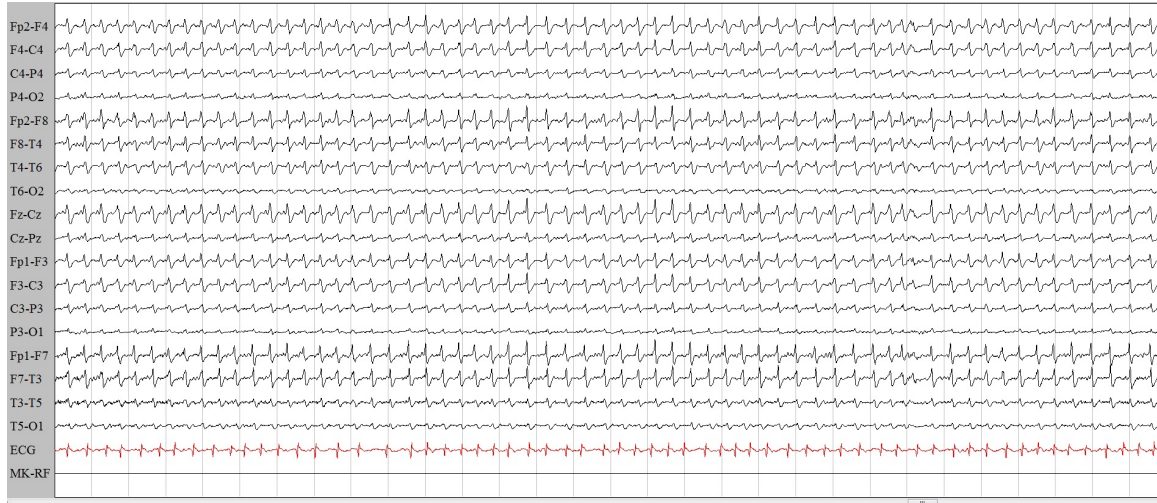
SE DI ASSENZA COME ESORDIO DI IGE



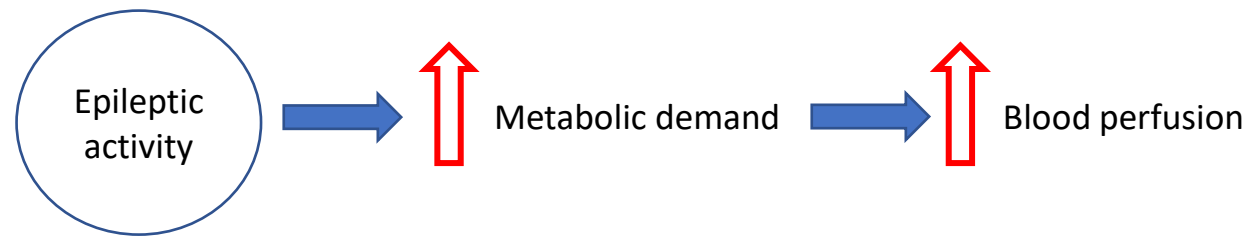
Encefalopatia post-anossica, 3 giornata



Encefalopatia post-anossica, 5 giornata



4 neuroimaging



Neuroimaging di perfusione

Presentazione clinica

Uomo, 65 aa

Esordio acuto di:

- afasia
- Emiparesi destra

Donna, 63 aa

Esordio acuto di:

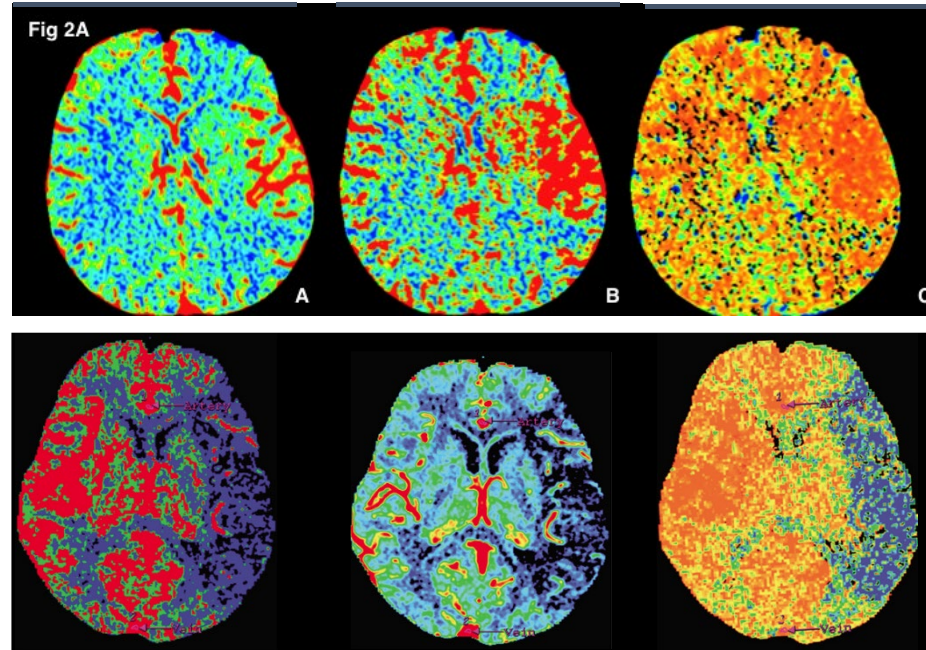
- afasia
- Emiparesi destra

Caratteristiche della CTP

rCBV

rCBF

MTT



Diagnosi

SE
(*perfusione
ictale*)

Stroke

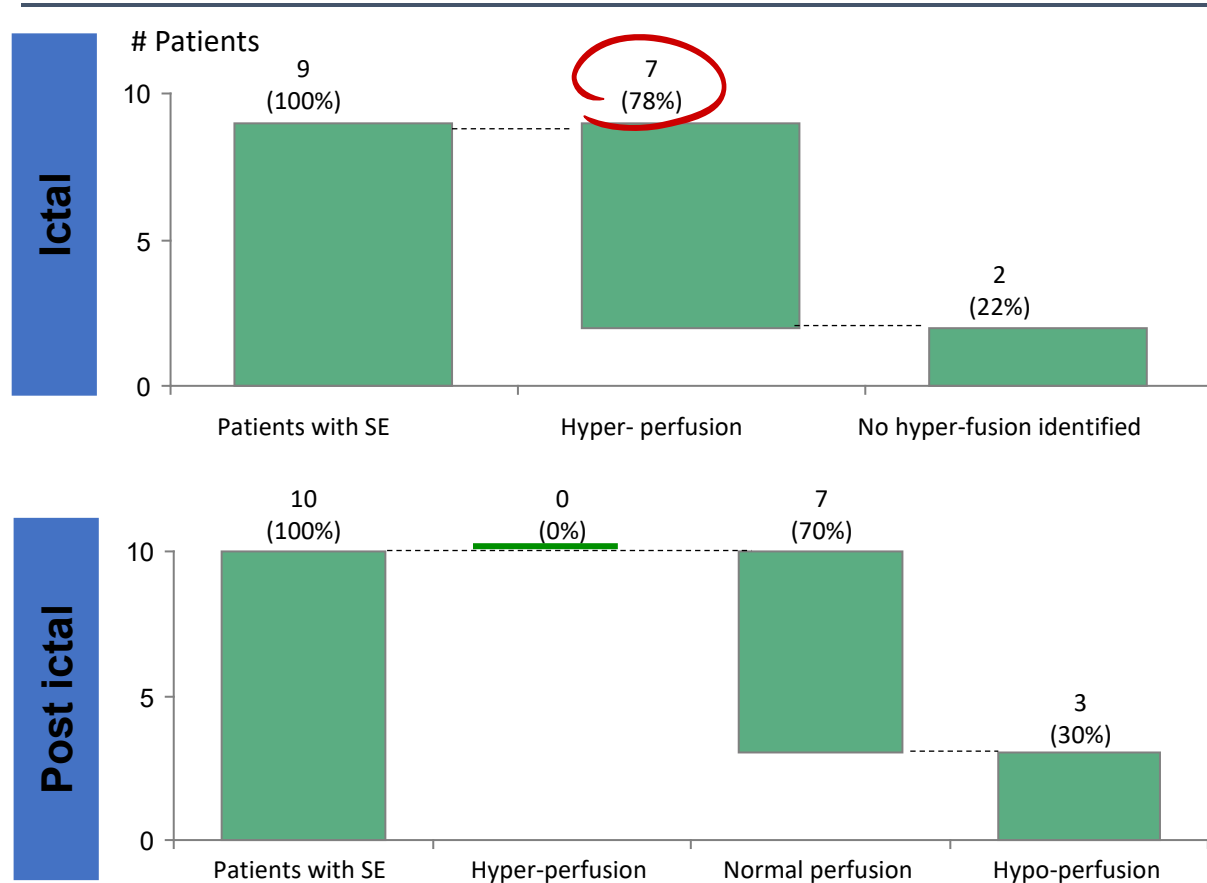
Simile presentazione
clinica

Differenti pattern di CTP

*Diagnosi
differenziale*

Neuroimaging di perfusione

Analisi visiva della CTP identifica il 78% dei pz con NCSE



Considerazioni chiave sulla CTP in NCSE

- + Procedura standard in PS nel sospetto ictus
- + Rapidità di acquisizione
- + Permette una rapida diagnosi e quindi un rapido e corretto trattamento (evitando trattamenti riperfusivi inutili e potenzialmente dannosi)

- Tecnica invasiva (tracciante esogeno)
- Analisi spesso limitata ad un definito volume

CT perfusion & NCSE



ELSEVIER

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Seizure: European Journal of Epilepsy

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



Cortical and thalamic hyper-perfusion in non-convulsive status epilepticus: Relationship between perfusion CT patterns and Salzburg EEG patterns

Giada Giovannini^{a,b,c}, Marcella Malagoli^d, Giulia Turchi^b, Alice Miani^a, Nicolina Anna Elisabetta Vaudano^b, Stefano Meletti^{a,b,*}

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Usefulness of brain perfusion CT in focal-onset status epilepticus

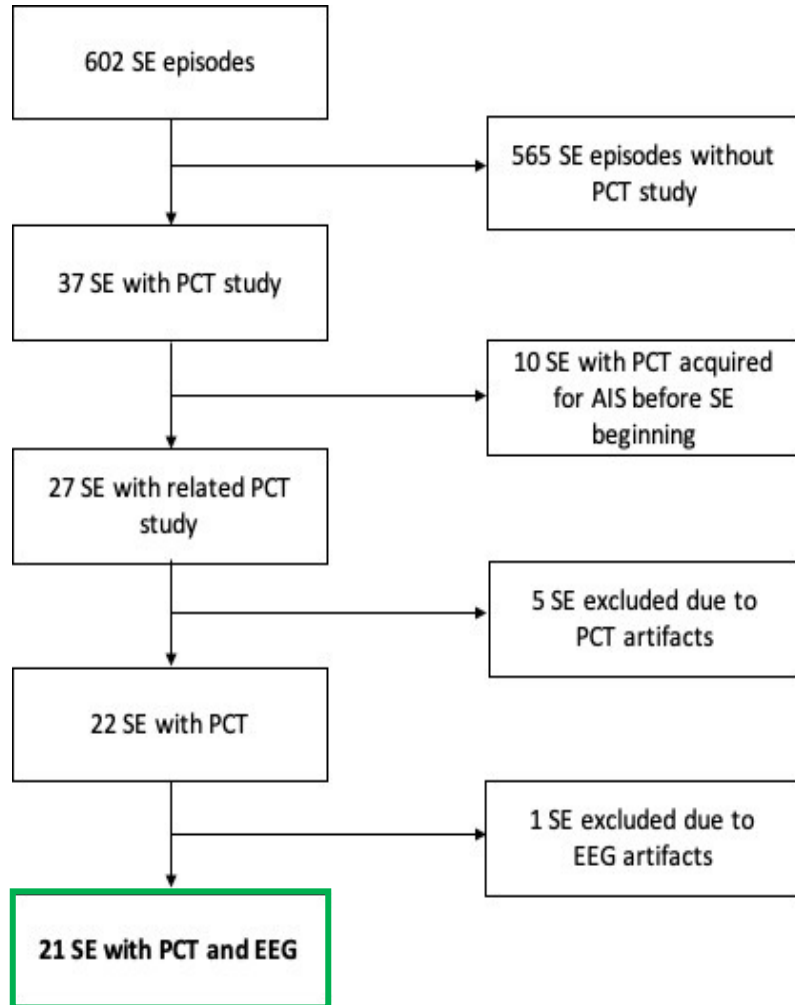
Montserrat González-Cuevas^{1,2}  | Pilar Coscojuela³ | Estevo Santamarina^{1,2}  | Deborah Pareto³ | Manuel Quintana^{1,2} | María Sueiras⁴ | Lorena Guzman⁴ | Silvana Sarria³ | Xavier Salas-Puig^{1,2} | Manuel Toledo^{1,2} | Àlex Rovira³

CT perfusion and EEG patterns in patients with acute isolated aphasia in seizure-related stroke mimics

Paolo Manganotti^{a,*}, Giovanni Furlanis^a, Miloš Ajčević^{a,b}, Paola Polverino^a, Paola Caruso^a, Mariana Ridolfi^a, Roberta Antea Pozzi-Mucelli^c, Maria Assunta Cova^c, Marcello Naccarato^a



CT perfusion & NCSE



1 CTP evaluation

-Hyperperfusion pattern:

↑ rCBF ↑ rCBV ↓ MTT in the affected hemisphere

-Hypoperfusion pattern:

↓ rCBF ↓ rCBV ↑ MTT in the affected hemisphere

-Normoperfusion pattern:

= rCBF, rCBV and MTT in both hemispheres

2 EEG evaluation

SCC

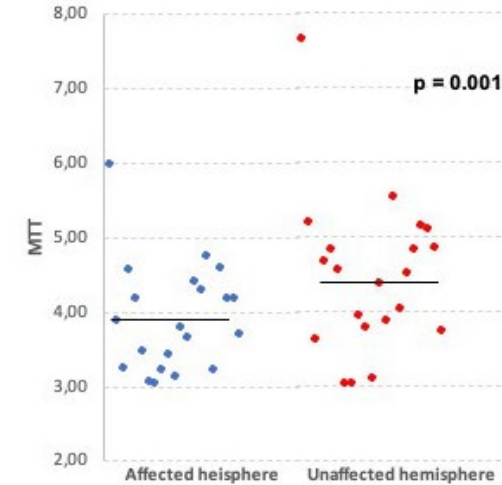
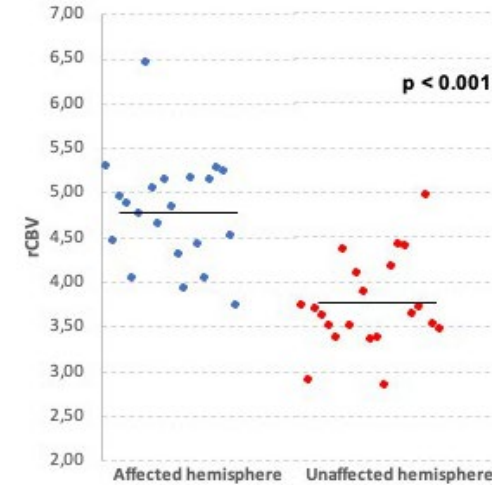
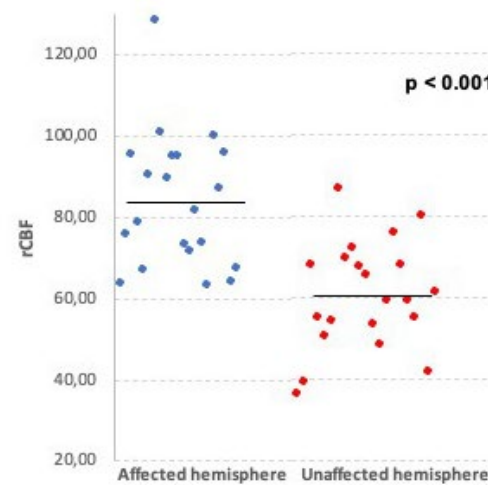
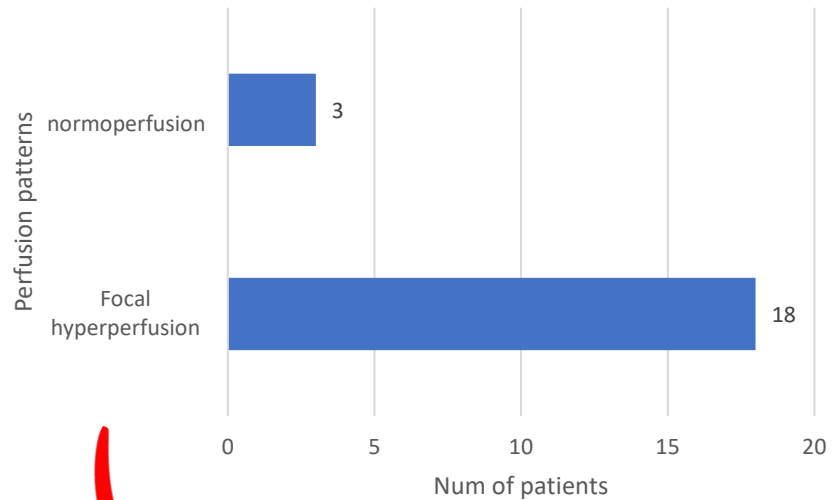
Continuous (CP) or
Discontinuous Patterns (WWP)

Presence or absence of LPD

3 CTP- EEG correlation

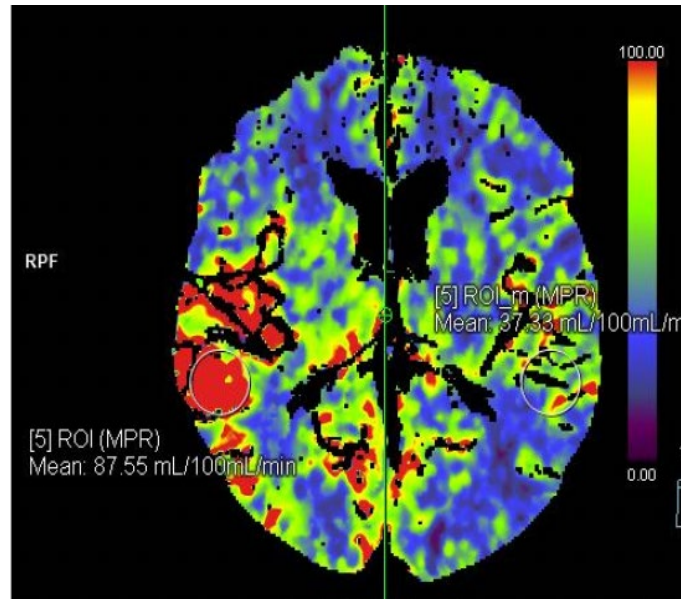
CT perfusion & NCSE

- Average time between symptoms onset and CTP acquisition: 3.30 h (range: 55 min-6.30 h); 5 patients with wake-up symptoms.
- Qualitative – quantitative analysis concordance: 100%

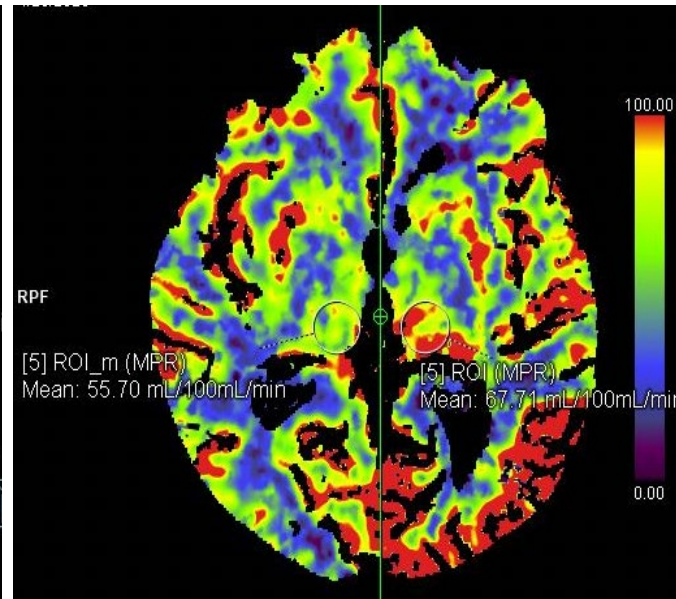


The presence of CTP abnormalities did not correlate with SE duration ($\rho = -0.17$, $p = 0.54$), gender ($\tau = -0.03$, $p = 0.89$) or age at SE presentation ($\rho = -0.34$, $p = 0.13$).

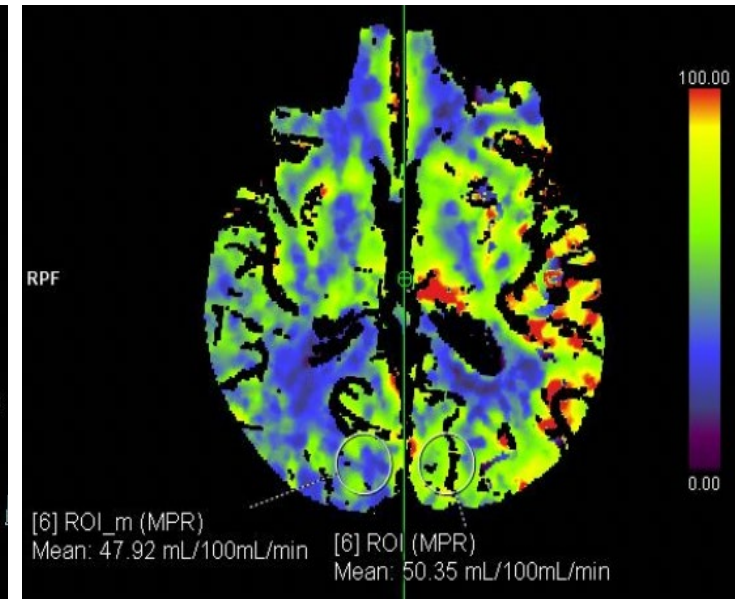
CT perfusion & NCSE



Cortical hyperperfusion only
10 (55%)

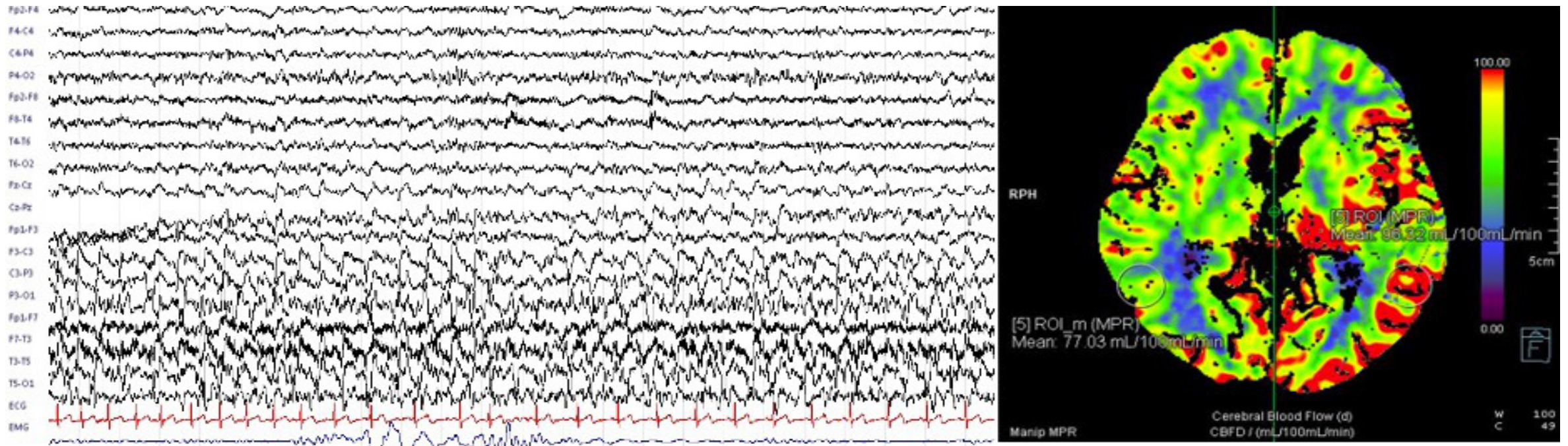


Cortical + Thalamus hyperperfusion
7 (39%)



Thalamus hyperperfusion
only
1 (6%)

CT perfusion & NCSE



CT perfusion & NCSE


- CTP could facilitate the **differential diagnosis** and **speed-up** the **diagnostic process** of NCSE in emergency situation
- NCSE defined by SCC are highly associated with **multi-lobar cortical hyper-perfusion located in the area of maximum EEG activity** and in some cases associated with a hyper-perfusion in the **homolateral thalamus**
- The presence of a CTP pattern with **cortical** acute-onset and enduring alteration of
- Our results suggest that **hyper-perfusion** abnormal **continuous/sustained ictal p**

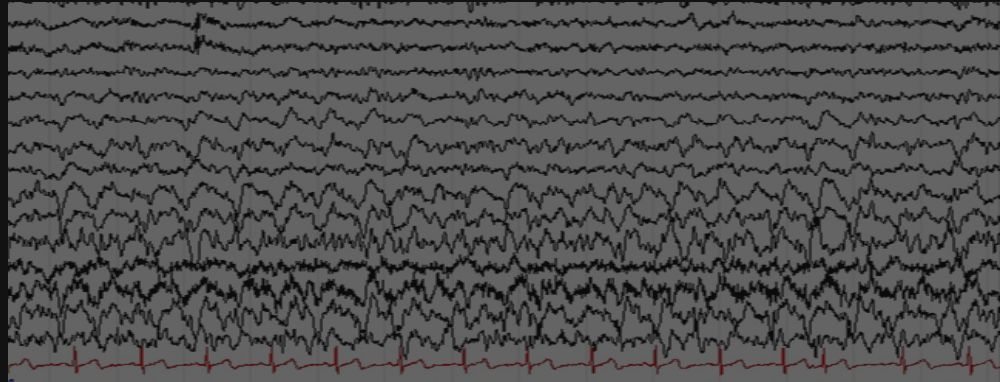
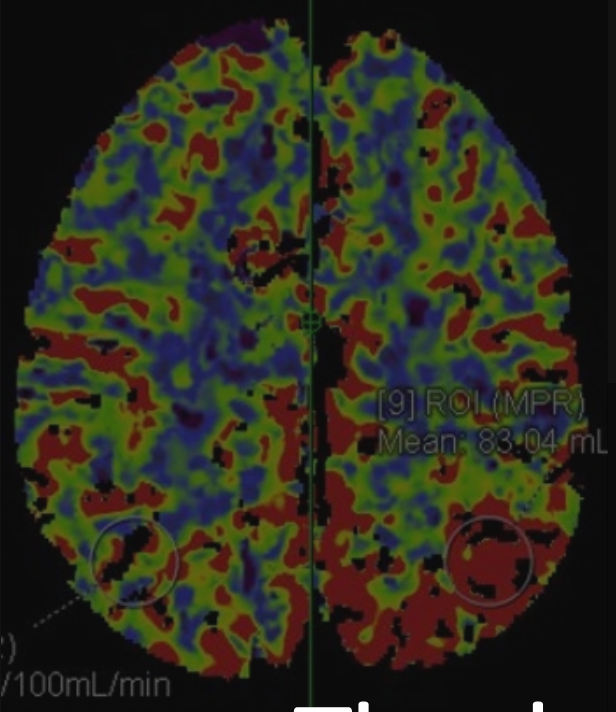
doi:10.1093/braincomms/fcaa223

BRAIN COMMUNICATIONS 2020: Page 1 of 16 | 1

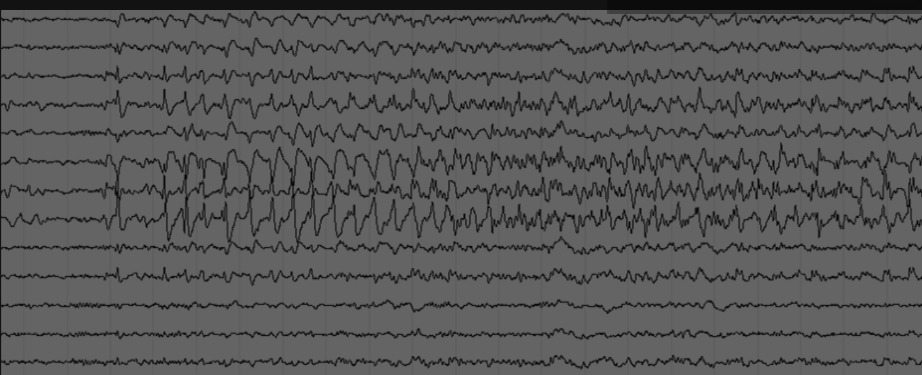
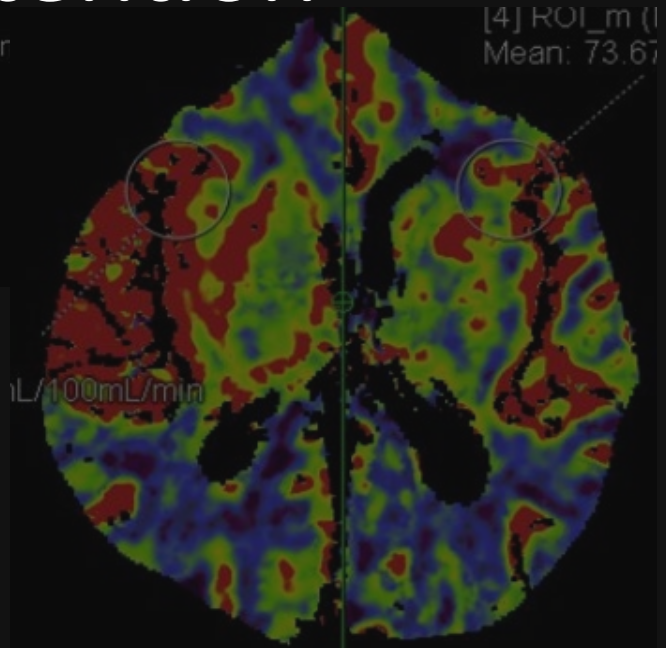
BRAIN COMMUNICATIONS

Hyperperfusion in the thalamus on arterial spin labelling indicates non-convulsive status epilepticus

 Satoru Ohtomo,^{1,2,3} Hiroshi Otsubo,^{4,5} Hiroaki Arai,¹ Yoshiteru Shimoda,^{1,2} Yoichiro Homma⁶ and Teiji Tominaga²



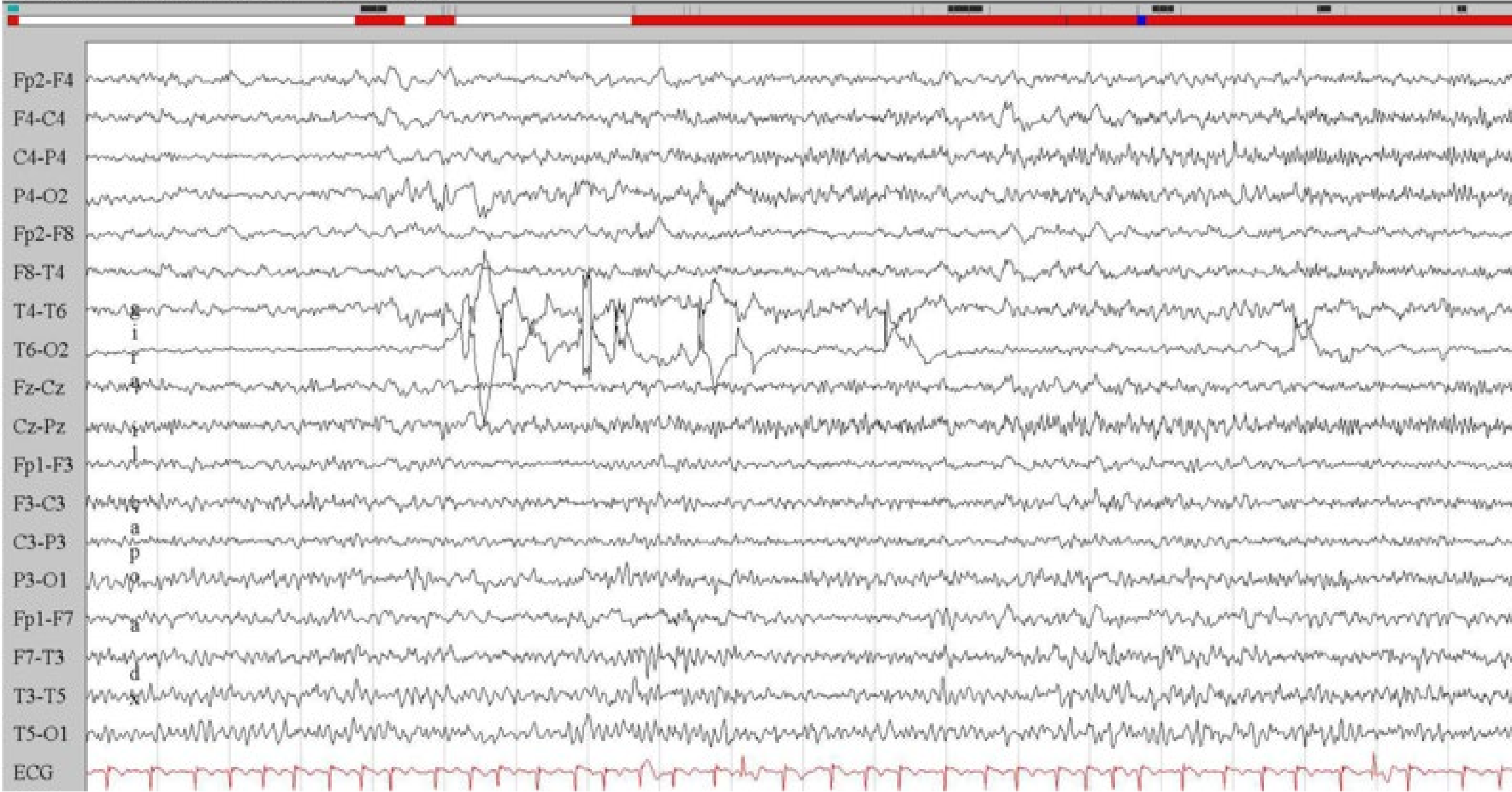
Thank you for you attention

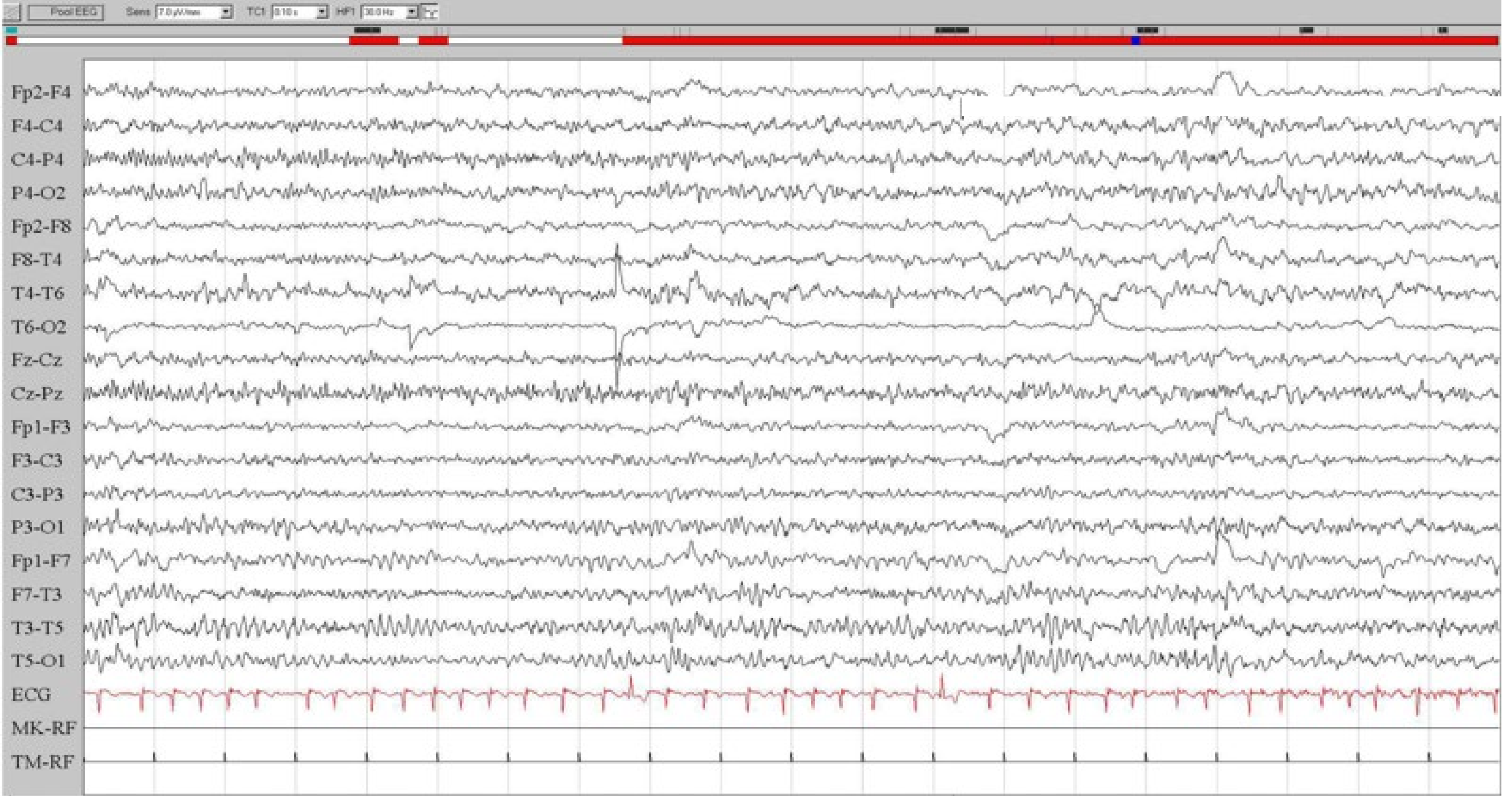


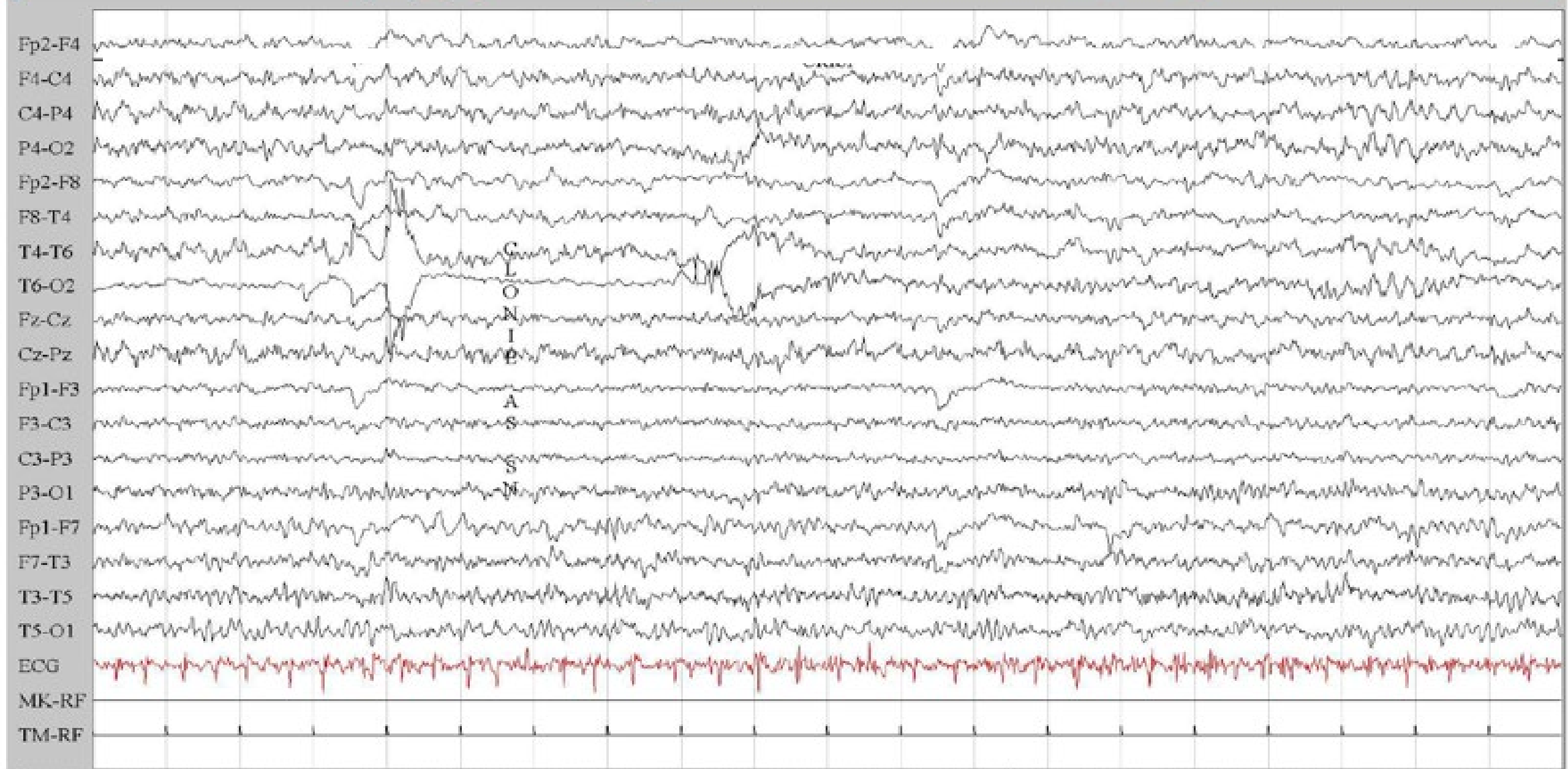
Casi clinici

Caso N. 1

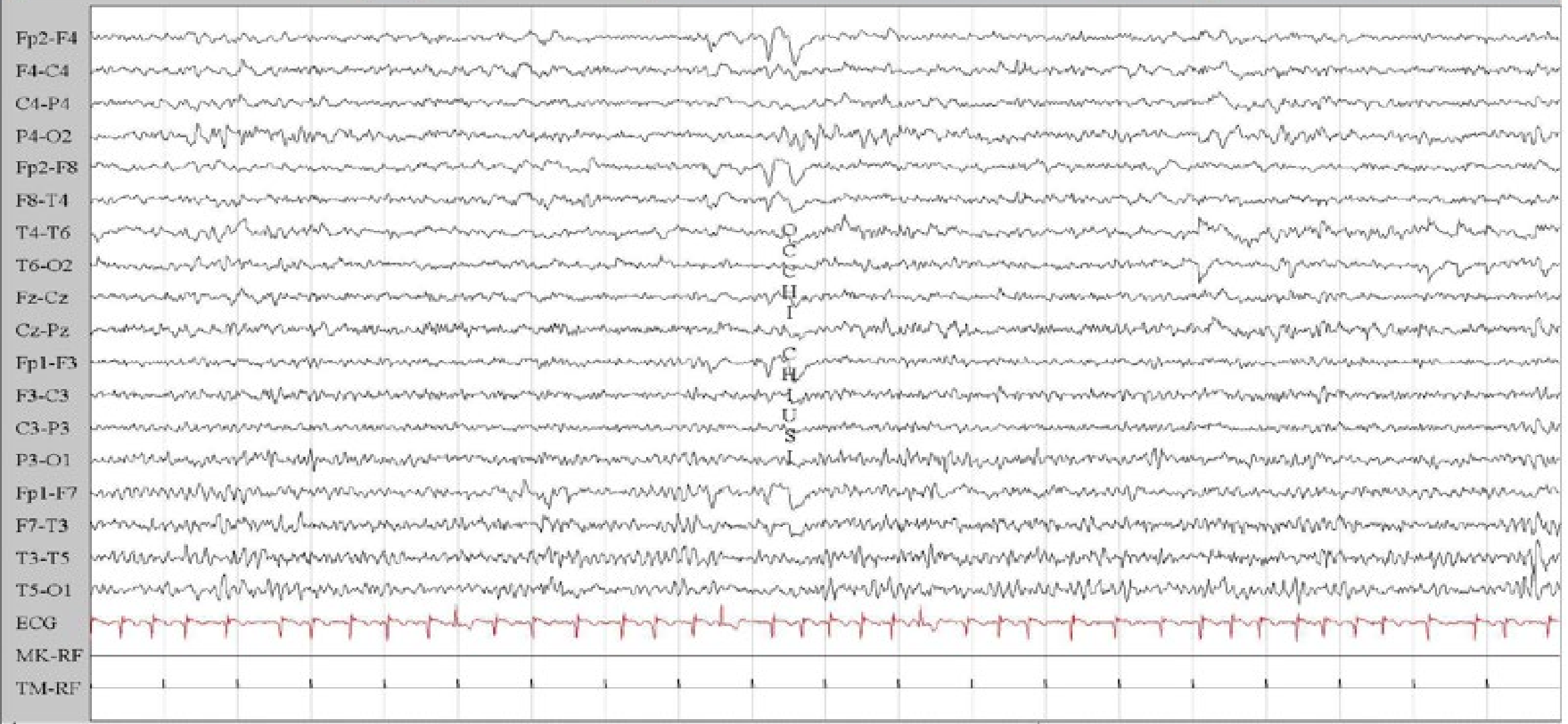
- C.G., ♀, 79 anni.
- **APR**: FA in TAO, ipertensione arteriosa, IRC, pregresso ictus ischemico parietale destro con completa ripresa, pregresso episodio di SE motorio da sospensione di barbiturico. In terapia antiepilettica cronica con VPA.
- **APP**: giunge in PS per caduta a terra per cedimento dell'arto inferiore sinistro
- **EON in PS**: emianopsia laterale sinistra e riscontro di atassia sensitiva







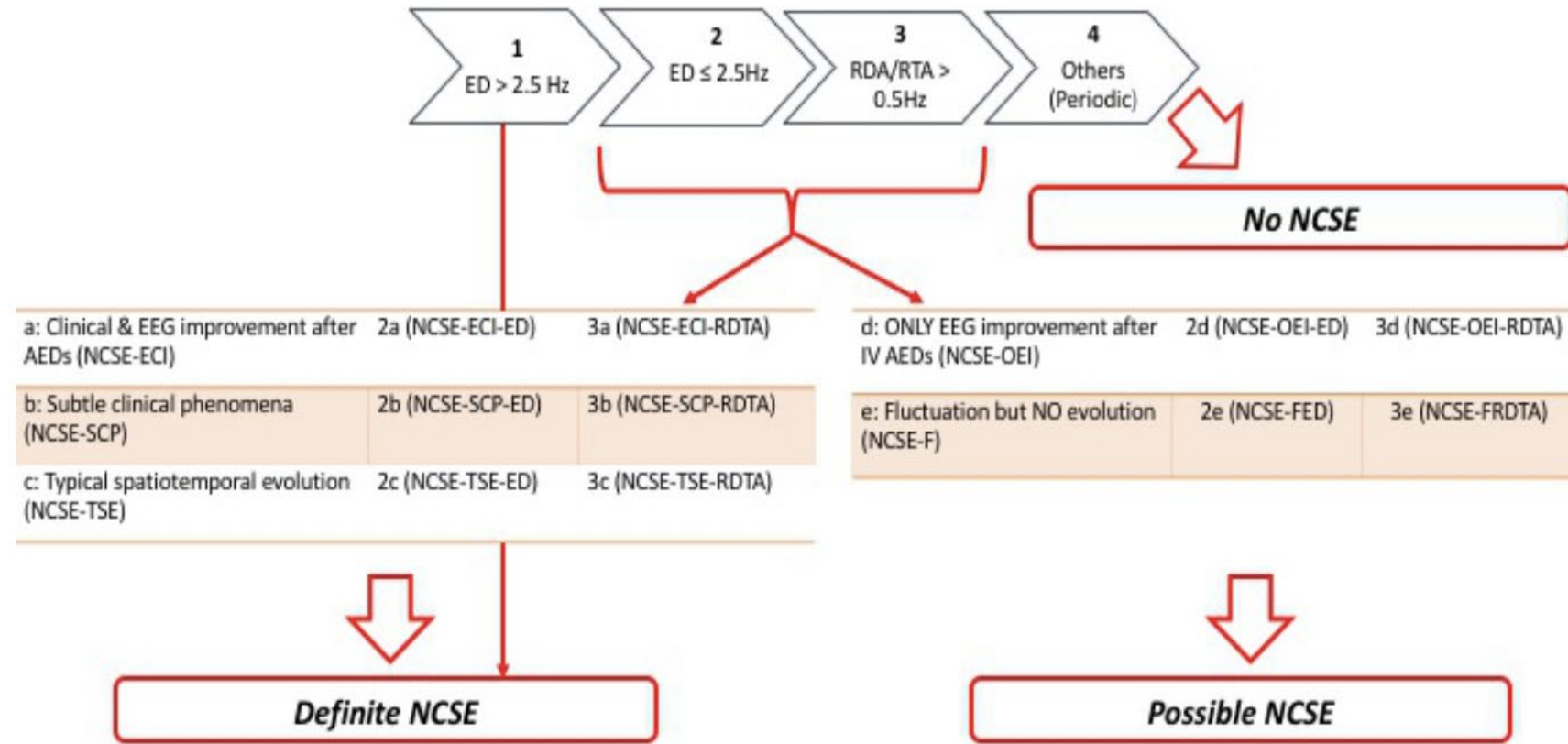




DIAGNOSI

SCC pattern:

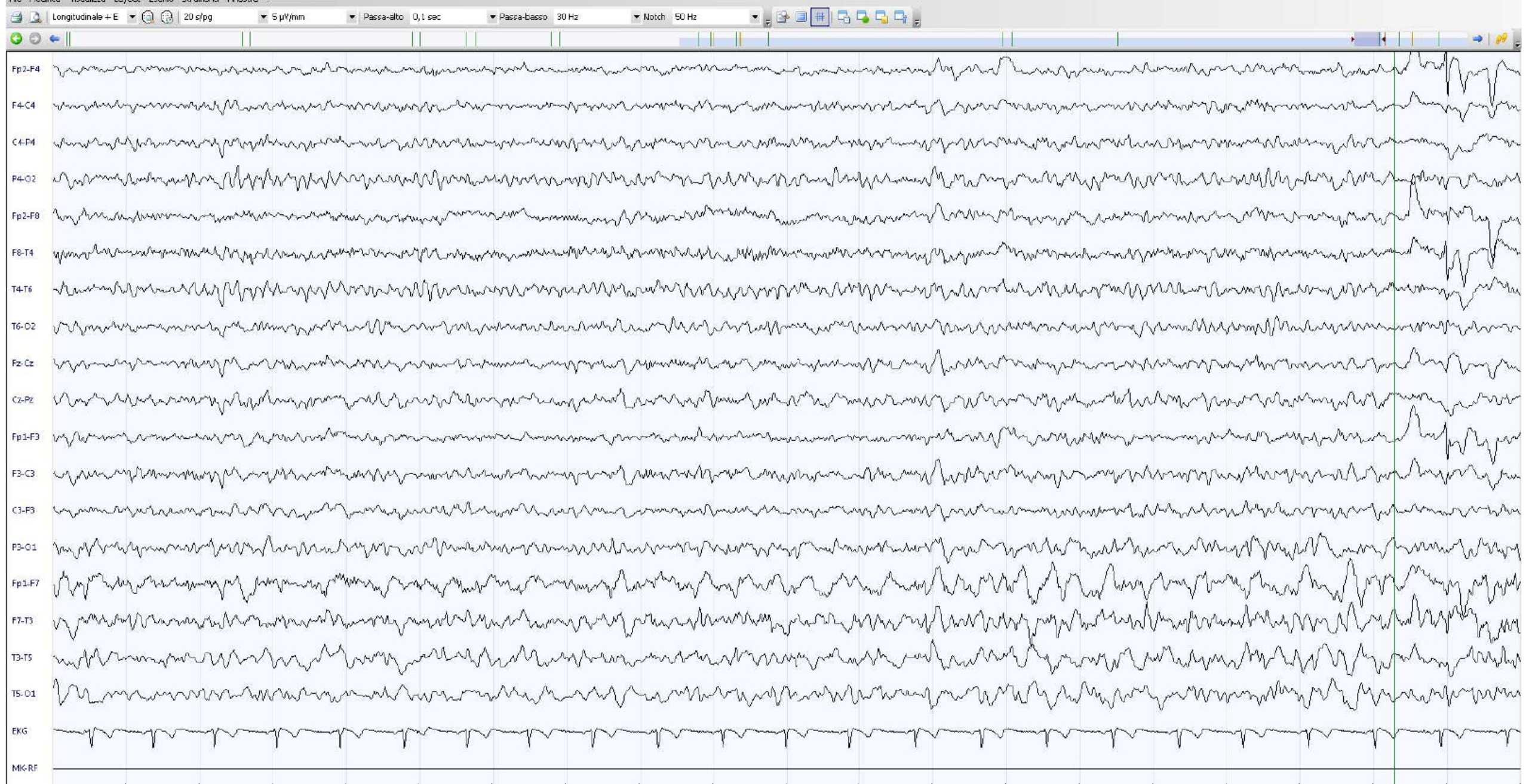
pattern 1 +2C +
2b (clonie
parcellari dell'AS
sx)

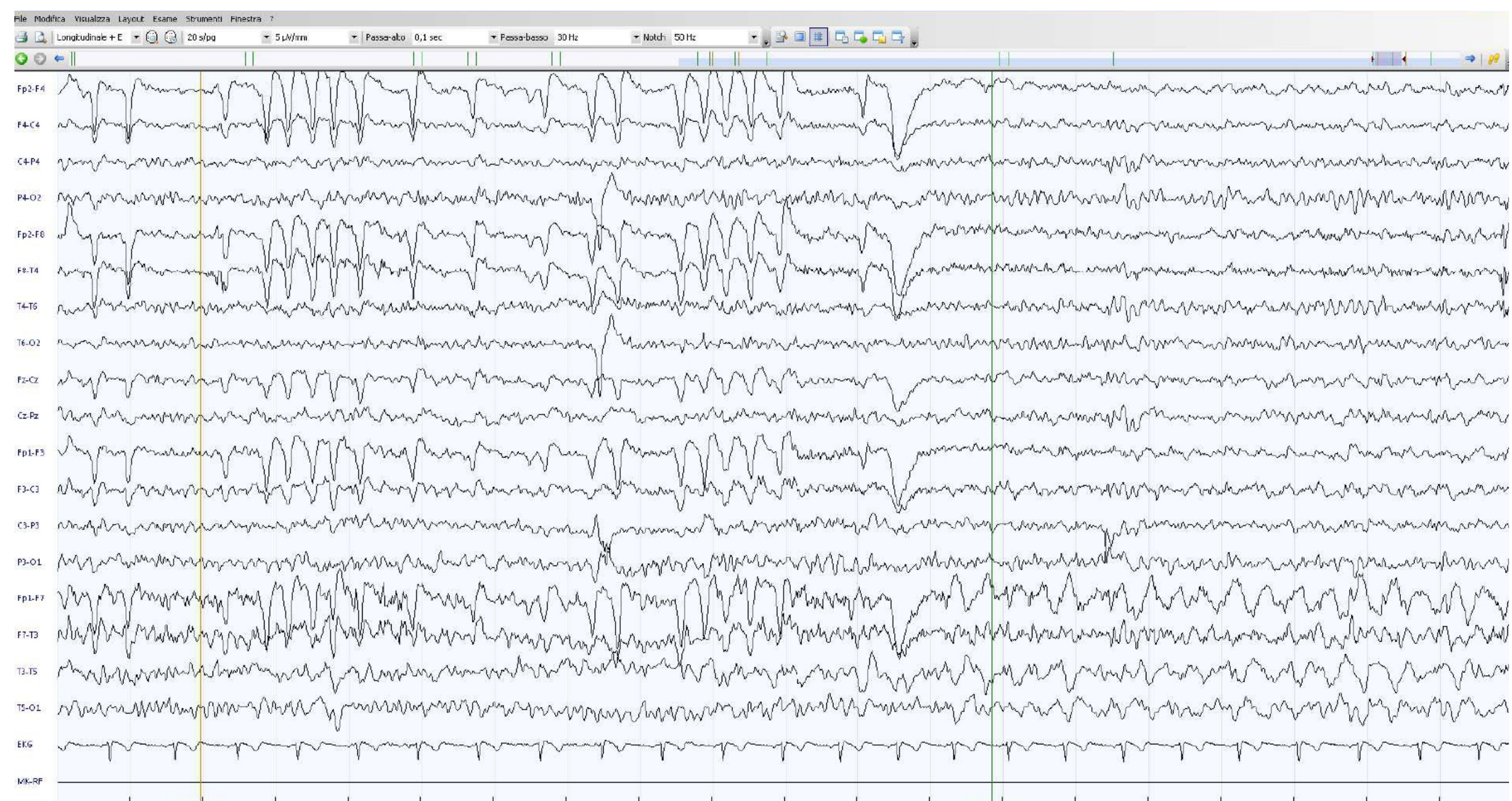


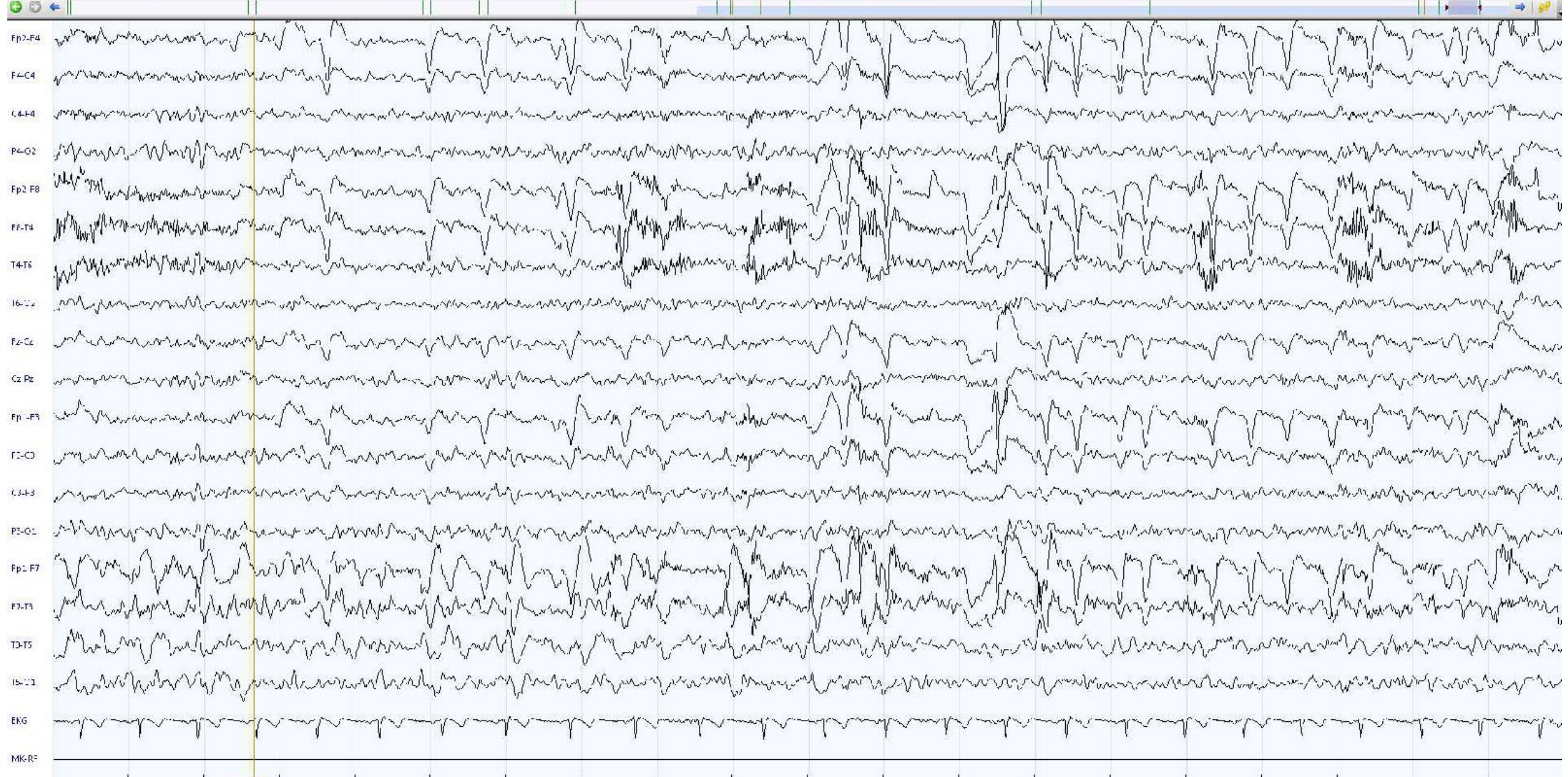
- SENC sintomatico remoto di esito ischemico parietale destro
- Terapia: BRV 100 mg in bolo ev poi 100 mgx2/die con risoluzione.

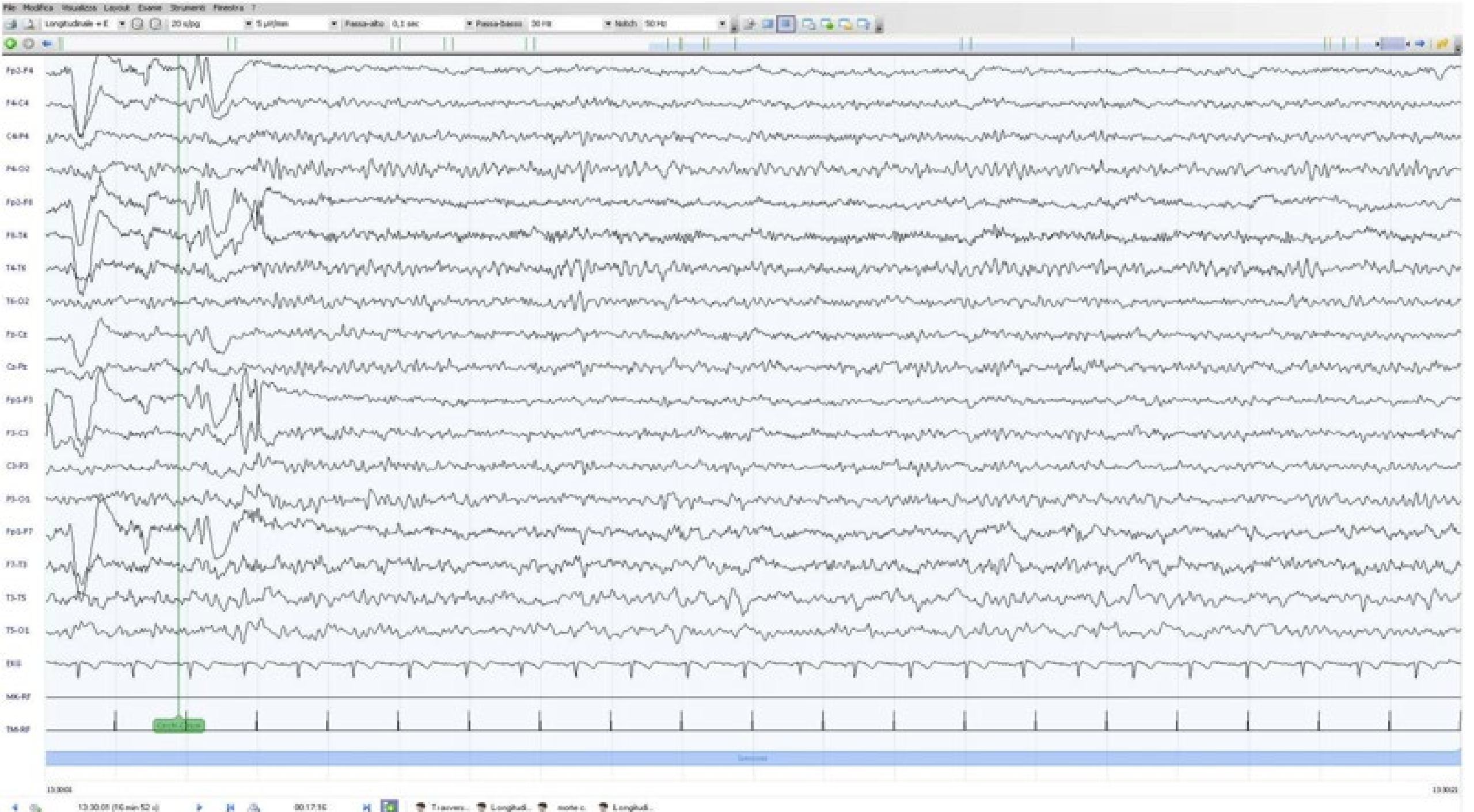
Caso N. 2

- P.G, ♂ , 56 anni.
- **APR**: AOCP, pregressa angina sottoposta a rivascolarizzazione con stenting e PTCA, fumatore, ipertensione arteriosa, dislipidemia, ipotiroidismo.
- **APP**: esordio acuto di episodi subentranti caratterizzati da sensazione di intenso calore a livello del capo con irradiazione verso il basso, fino alle gambe, cui si associava disturbo dell'eloquio (difficoltà nel trovare le parole);
- **EON** in PS: pz afasico, confabulante





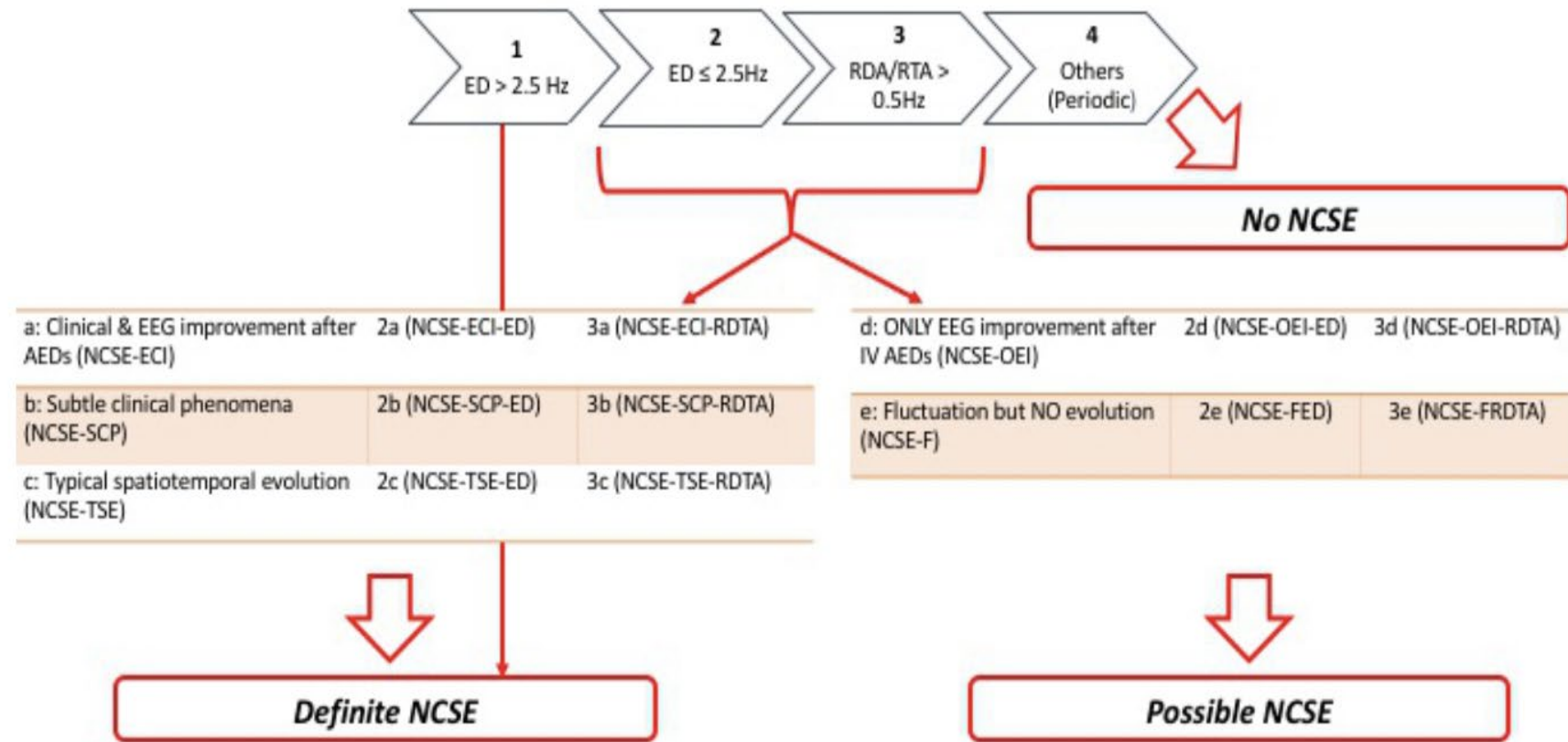




DIAGNOSI

SCC Pattern:

pattern 2c



- Diagnosi: SENC sintomatico progressivo di GBM FT sx.
- Terapia: BDZ bolo, poi LCS 200 mg in bolo ev poi 100 mgx2/die con risoluzione.

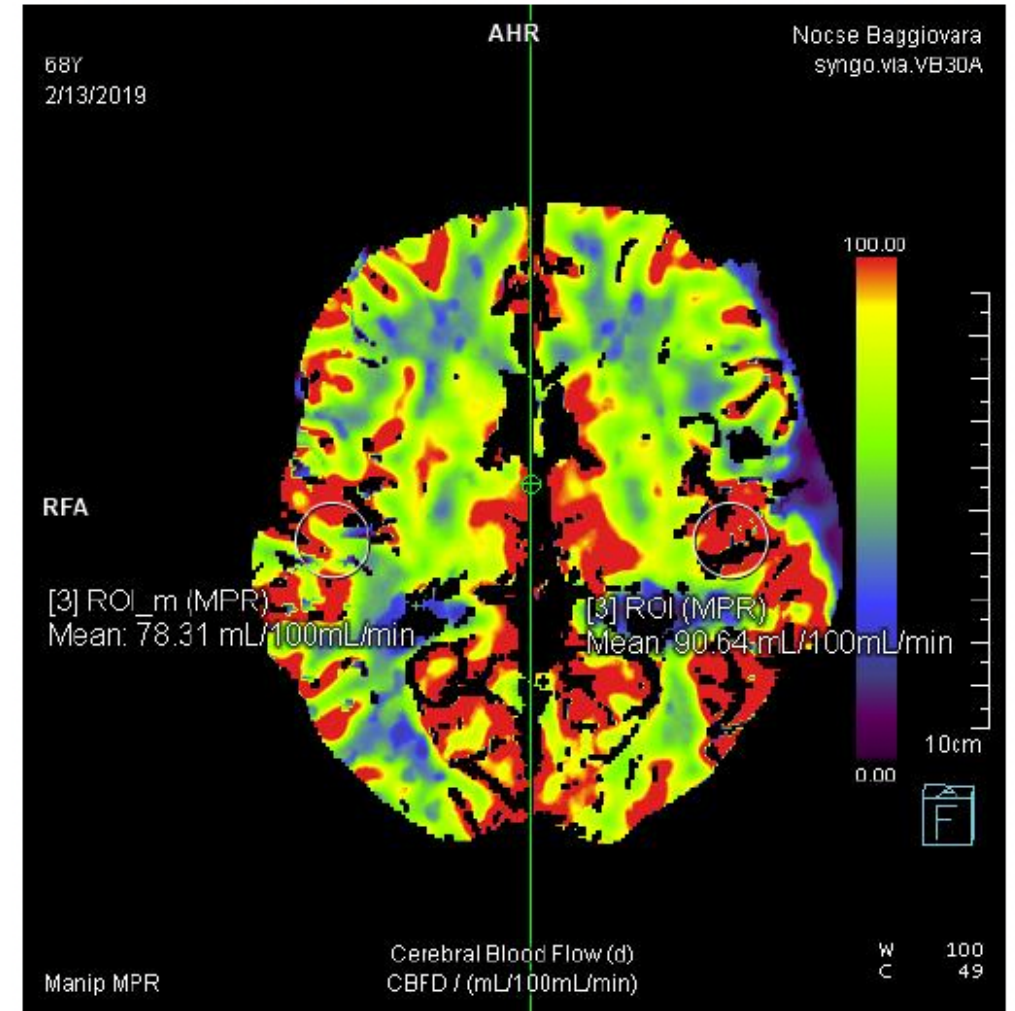
Caso N.3

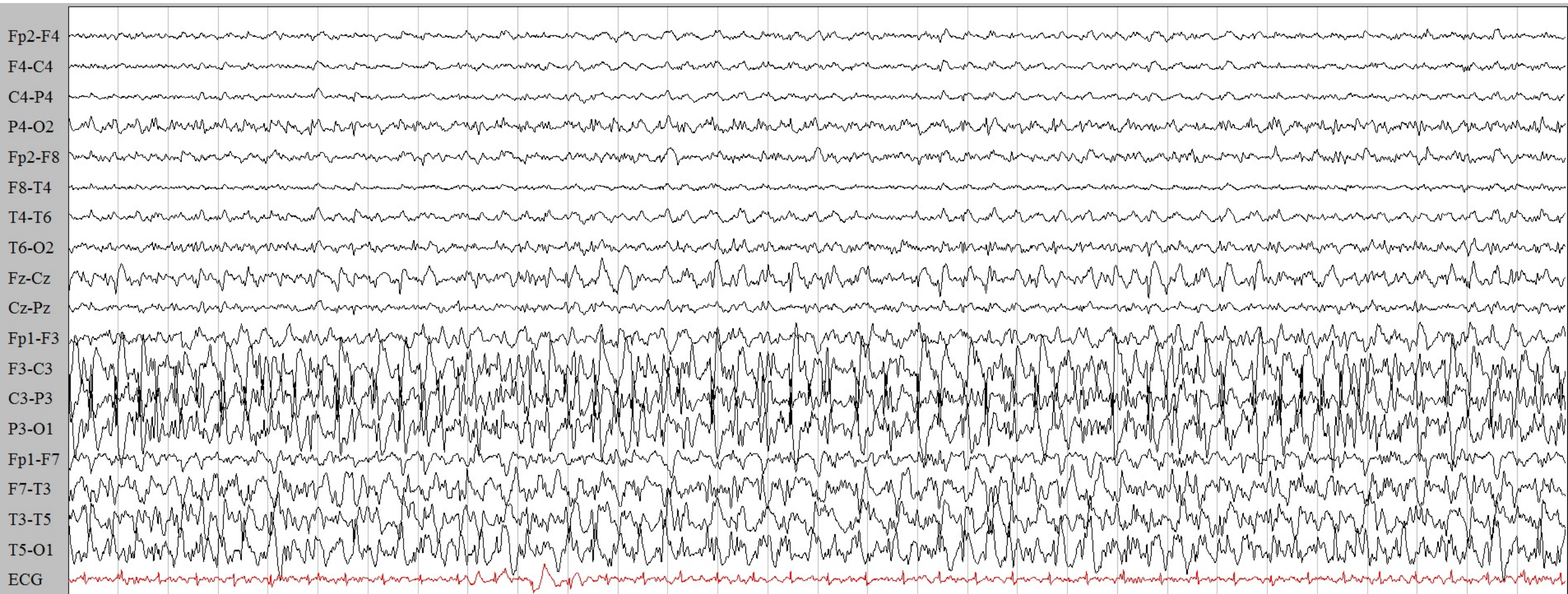
- B.S ♀, 68 anni.
- **APR**: pregresso carcinoma della tiroide, pregressa asportazione di meningioma P sx.
- **APP**: esordio improvviso di sopore, afasia e deficit di forza agli arti di dx.
- **EON in PS**: afasia globale, emiparesi dx, emianopsia dx, emi-ipoestesia dx, GCS 10. Comparsa di crisi convulsiva.

TC in urgenza

Esiti di craniotomia fronto-parietale sn
encefalomalacia fronto-temporo-
parietale;

CTP: iperperfusione PO sx

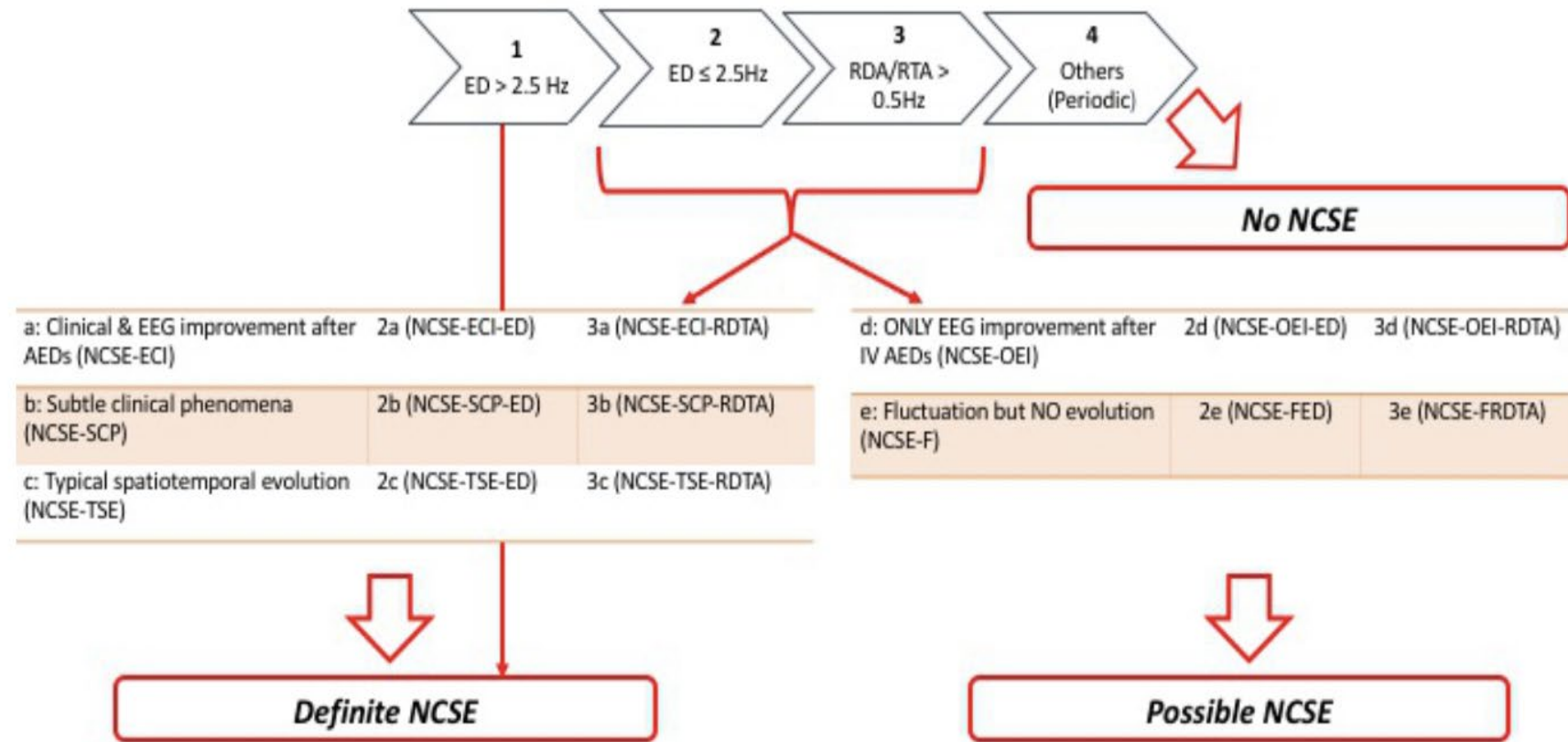




DIAGNOSI

SCC Pattern:

pattern 1



- Diagnosi: SENC focale P sx sintomatico remoto di progressiva asportazione di meningioma
- Trattamento: trattato con DZP 10 mg ev e LEV 3000 mg in bolo ev poi 3000 mg/die con risoluzione

Caso N.4

- C.F, ♂ , 54 anni.
- **APR**: macroadenoma ipofisario trattato chirurgicamente, ipertensione arteriosa, cardiopatia ipertensiva, dislipidemia, iperuricemia, ipovitaminosi. Storia di agitazione e stato ansioso per cui è seguito dal CSM; ad ottobre 2020 riscontro di esiti ischemici lacunari del circolo posteriore.
- **Da Gennaio 2021** ricoverato per polmonite bilaterale da SarsCov2 → ricoverato in TI
- **Marzo 2021**: in seguito a sospensione della sedazione presenta una crisi Tónico-clonica >
LEV 2000

[A] LONGITUDINALE 30 sec/pg

Pool EEG Sens 10.0 μ V/mm TC1 0.10 s HF1 30.0 Hz



[A] LONGITUDINALE 30 sec/pg

Pool EEG Sens 10.0 μ V/mm TC1 0.10 s HF1 30.0 Hz



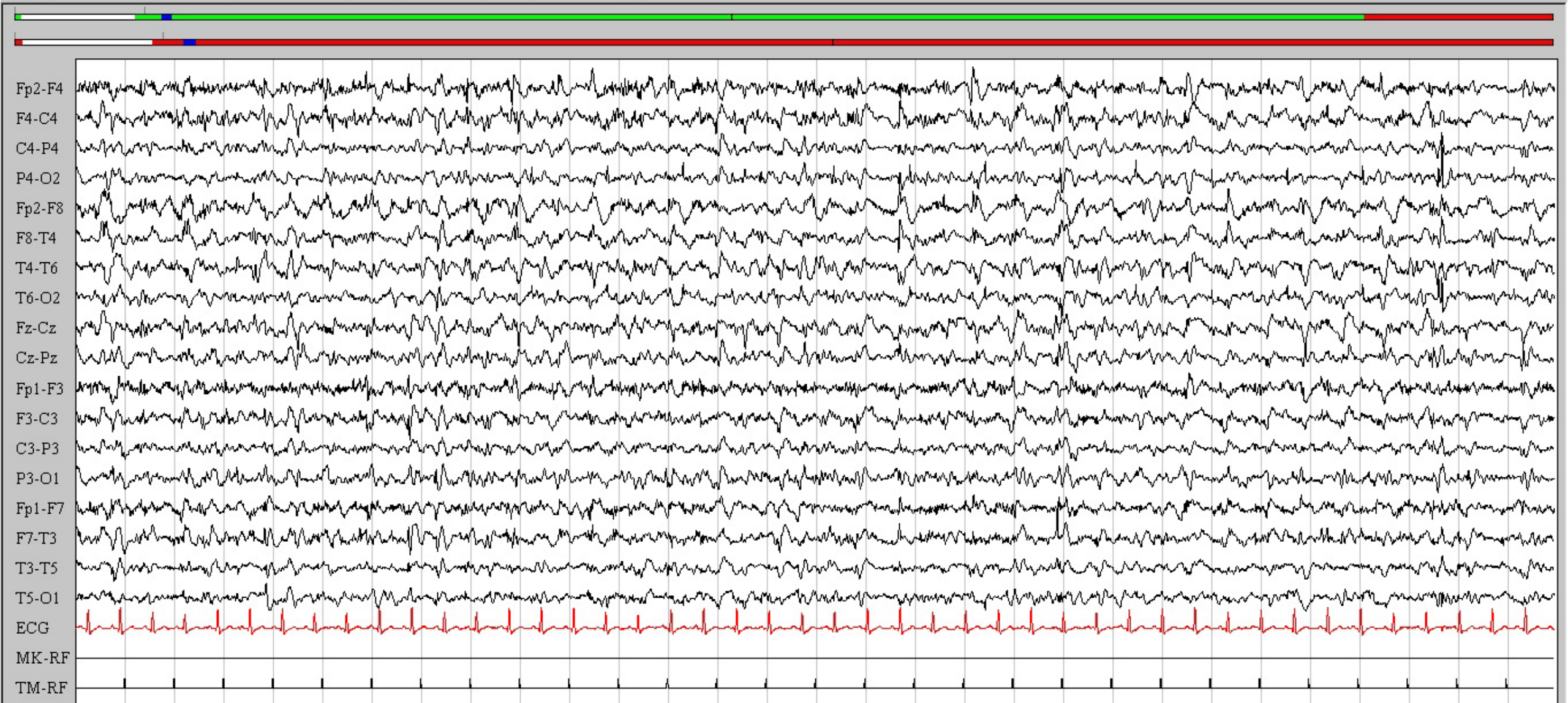
[A] LONGITUDINALE 30 sec/pg

Pool EEG Sens 10.0 μ V/mm TC1 0.10 s HF1 30.0 Hz



[A] LONGITUDINALE 30 sec/pg

Pool EEG Sens 10.0 μ V/mm TC1 0.10 s HF1 30.0 Hz

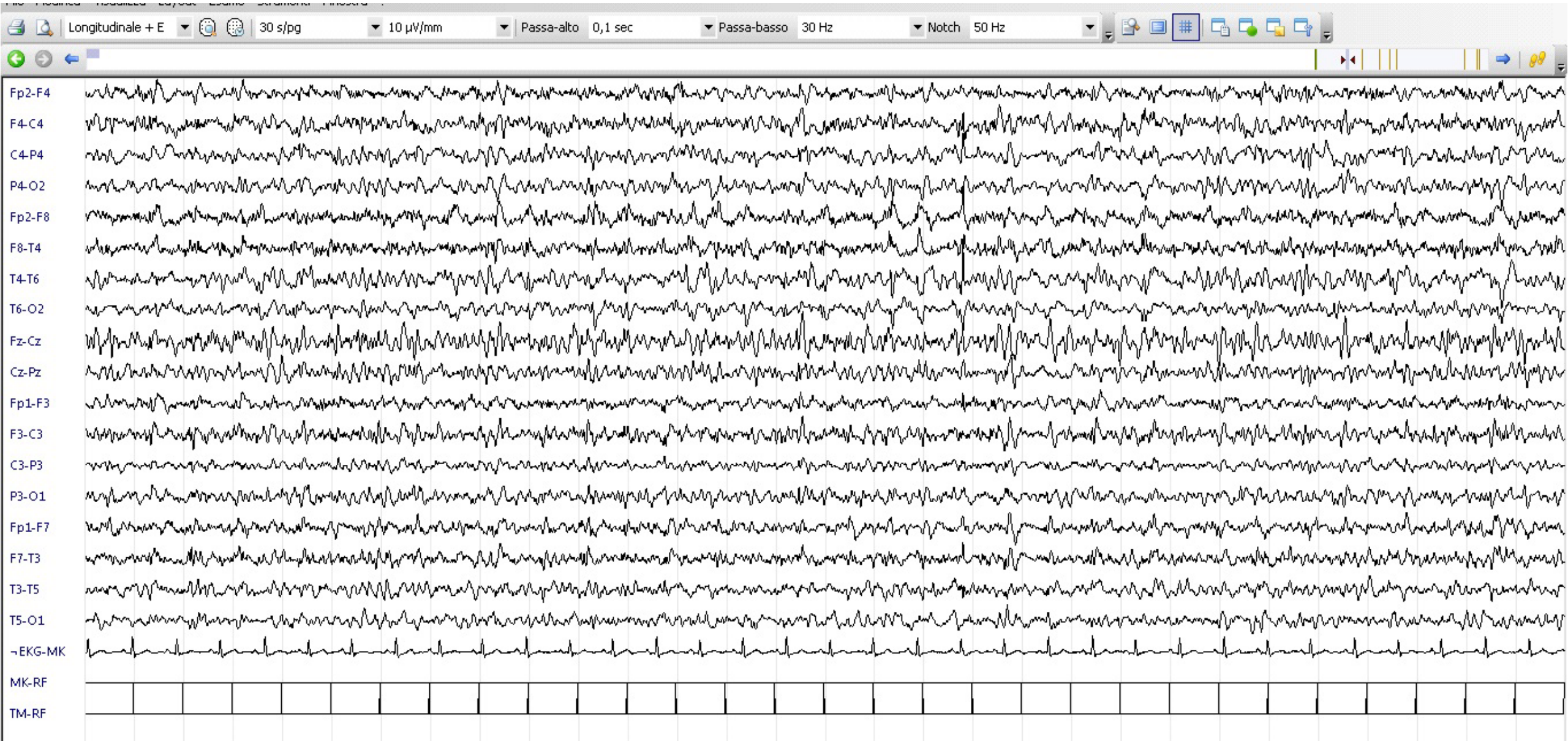


midazolam

[A] LONGITUDINALE 30 sec/pg

Pool EEG Sens 10.0 μ V/mm TC1 0.10 s HF1 30.0 Hz



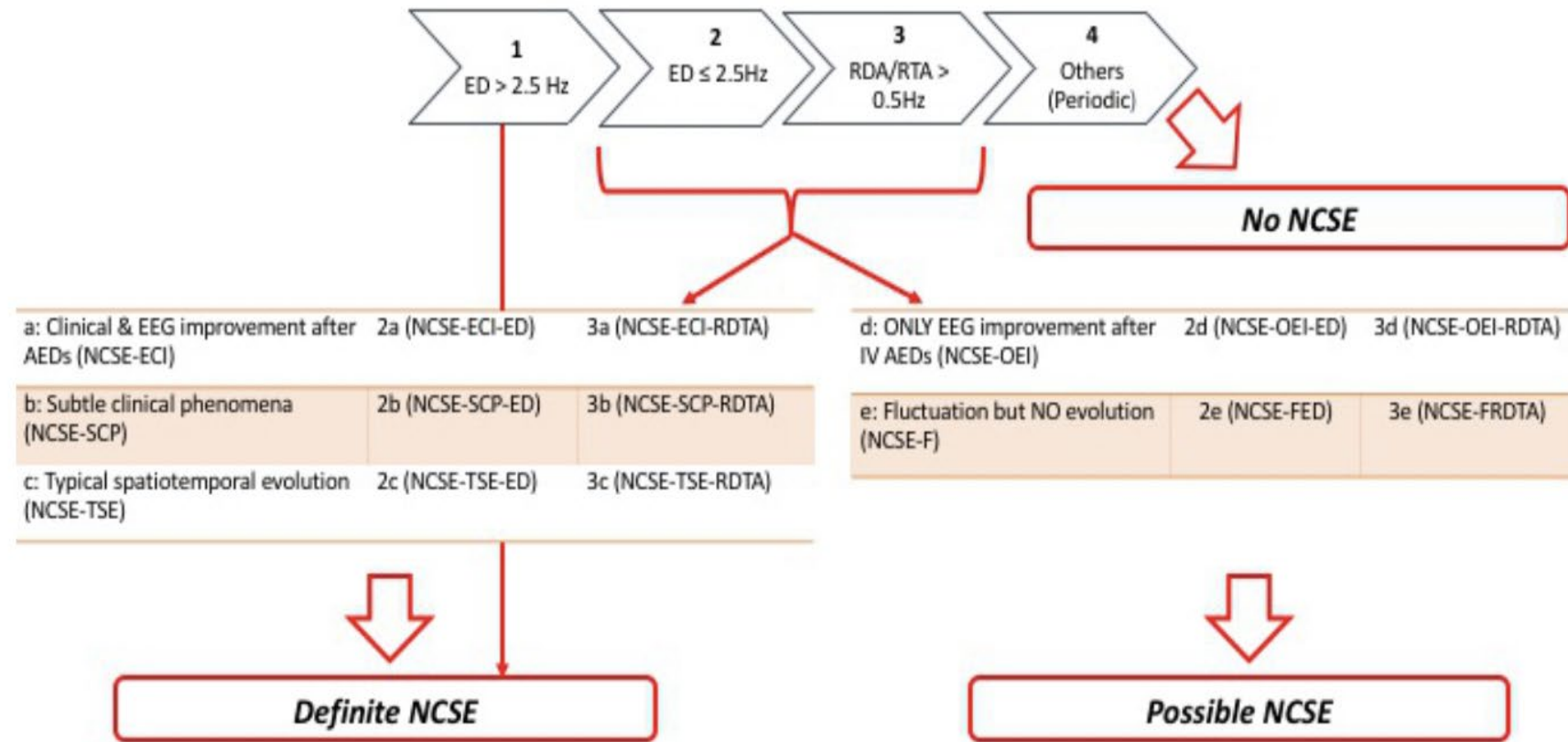


Il giorno dopo

Soluzioni

SCC Pattern:

pattern 1



Diagnosi: SENC in coma

Trattamento: Già in terapia con LEV 3000 mg/die; VPA 2000 mg in infusione continua nelle 24 ore.