



# 4° CORSO RESIDENZIALE EEG e POTENZIALI EVOCATI

22 – 27 NOVEMBRE 2021

Con il Patrocinio di



**Neuromonitoraggio in area critica**

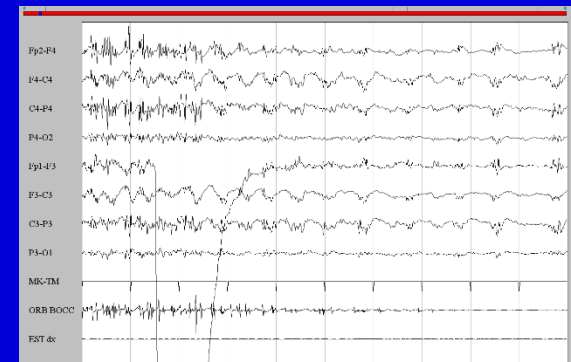
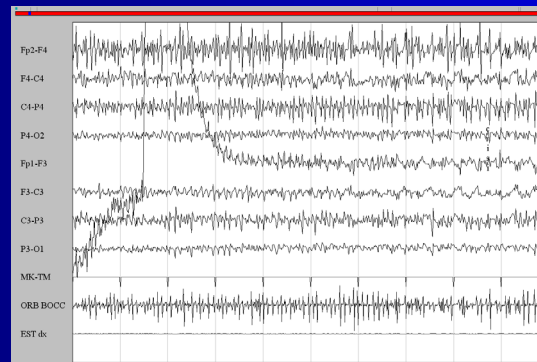
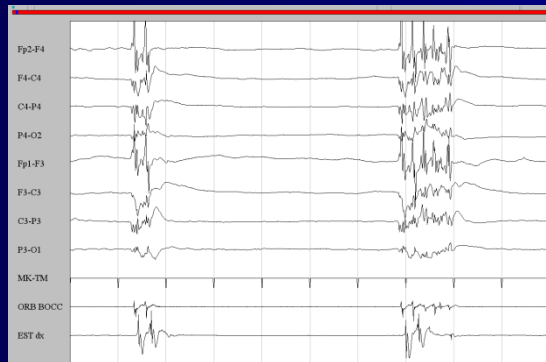
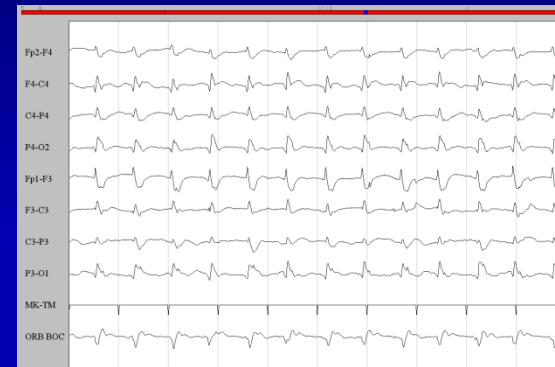
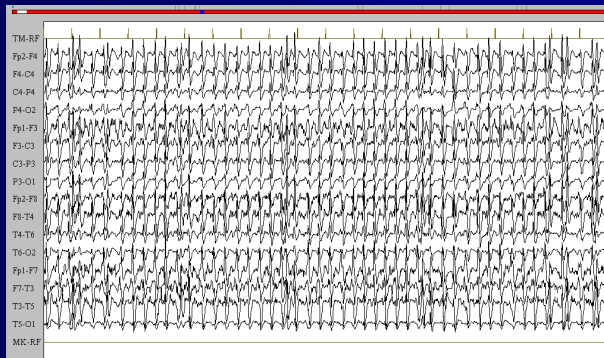
**A.Amantini - A.Grippo  
(Firenze)**



**CASI CLINICI**

# STATO EPILETTICO POST-ANOSSICO

( trattamento delle crisi convulsive, mioclono e SE non convulsivo)



# Trattamento crisi, stato epilettico e mioclono

- LPDs e GPDs da sole non ritenute stato epilettico
- Rimane aperto se definire SENC e trattare GPDs con frequenza maggiore di 2-2.5 c/s....
- Le crisi, lo stato epilettico convulsivo e non convulsivo (se presenti i criteri EEG di pattern critico)
- Il mioclono solo se associato a GPEDs



## SCHEDA VALUTAZIONE COMA POST-ANOSSICO

Sig... **BP** .....Data nascita...

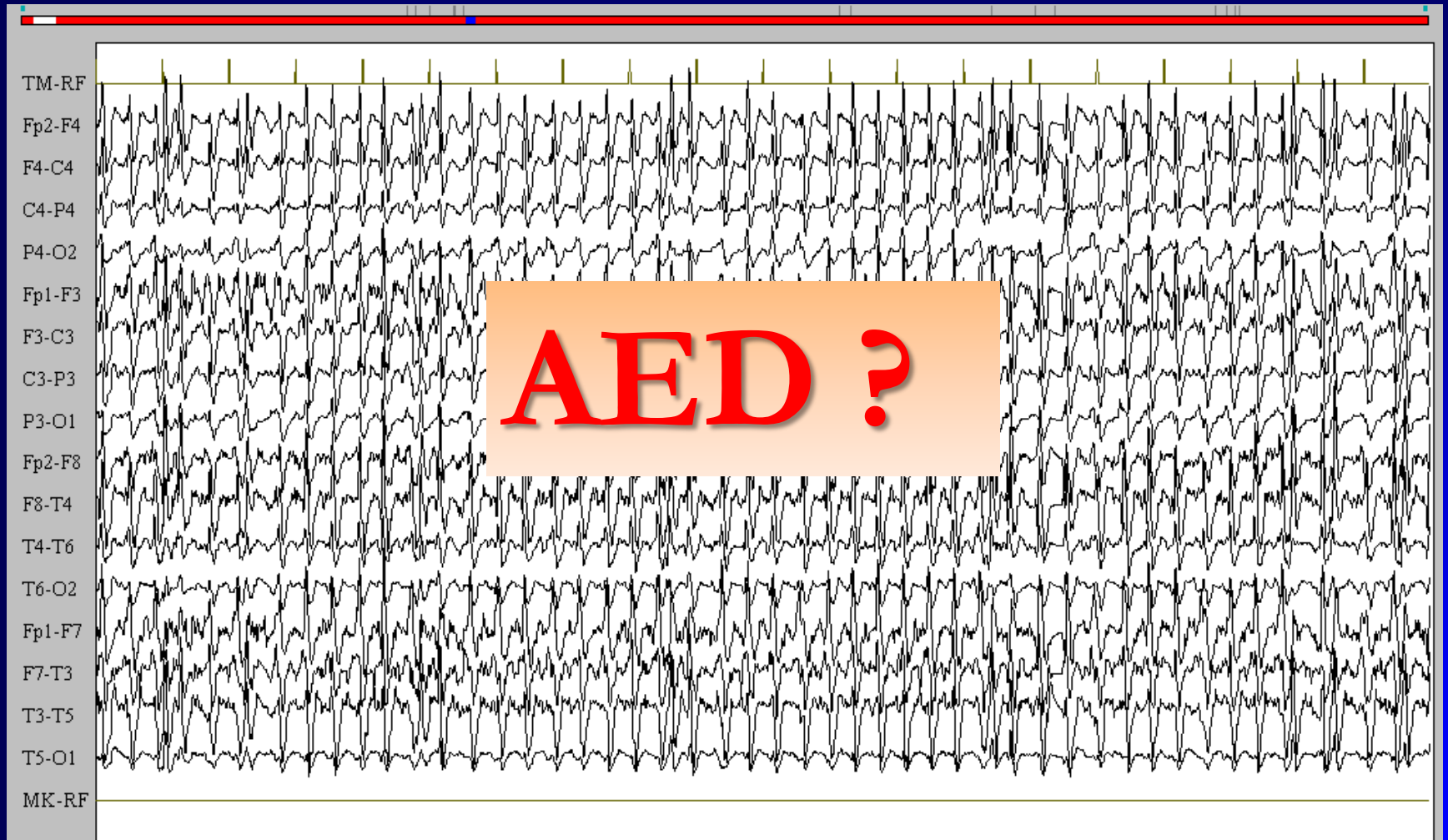
...Età... **80** ..Sesso M....

Reparto: **UTIC**

TIPO	DATI			
DATA ACR	15-03-2013			
Ora ACR	9.00			
LUOGO ACR				<b>EXTRAOSPEDALIERO</b>
CAUSA ACR	TRAUMA	ACIDOSI	<b>ALTRO</b>	
			<b>X</b>	
RITMO ESORDIO	FV/TV	ASISTOL	PEA(Pulseless Electrical Activity)	
	<b>X</b>			
DURATA ARRESTO	45'			
TEMPO STIMATO ROS	45'			
GCS INGRESSO 118/PS	E1	V1	M2	Tot: 4
GCS INGRESSO ICU	E1	V1	M2	Tot: 4
Pupille		Diametro Reattività	Miotiche	media midr midriatiche <b>SI</b> <b>NO</b>
Episodi ipossia peri - arresto	SI	<b>NO</b>		
Episodi ipotensione Protratta (>30')	SI	<b>NO</b>		
INIZIO IPOT DA ACR (ore)	3 h			
DURATA IPOTERMIA (ore)	24 h			

# STATO EPILETTICO POST-IPOSSI/ISCHEMICO

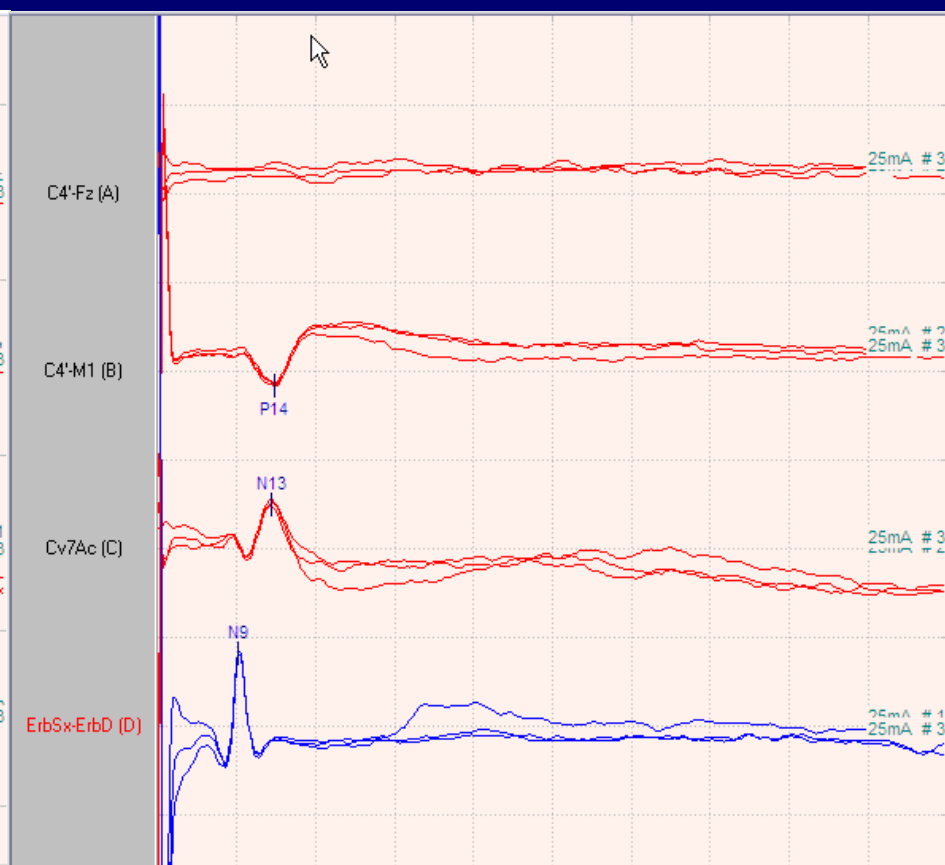
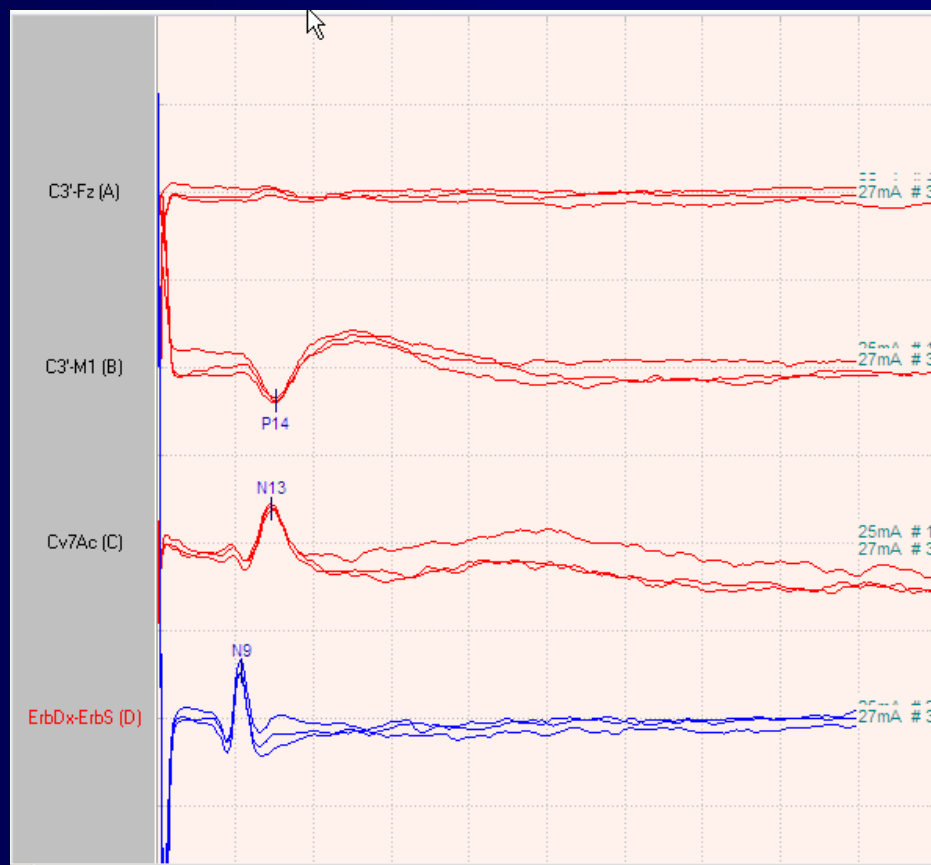
## 24 h



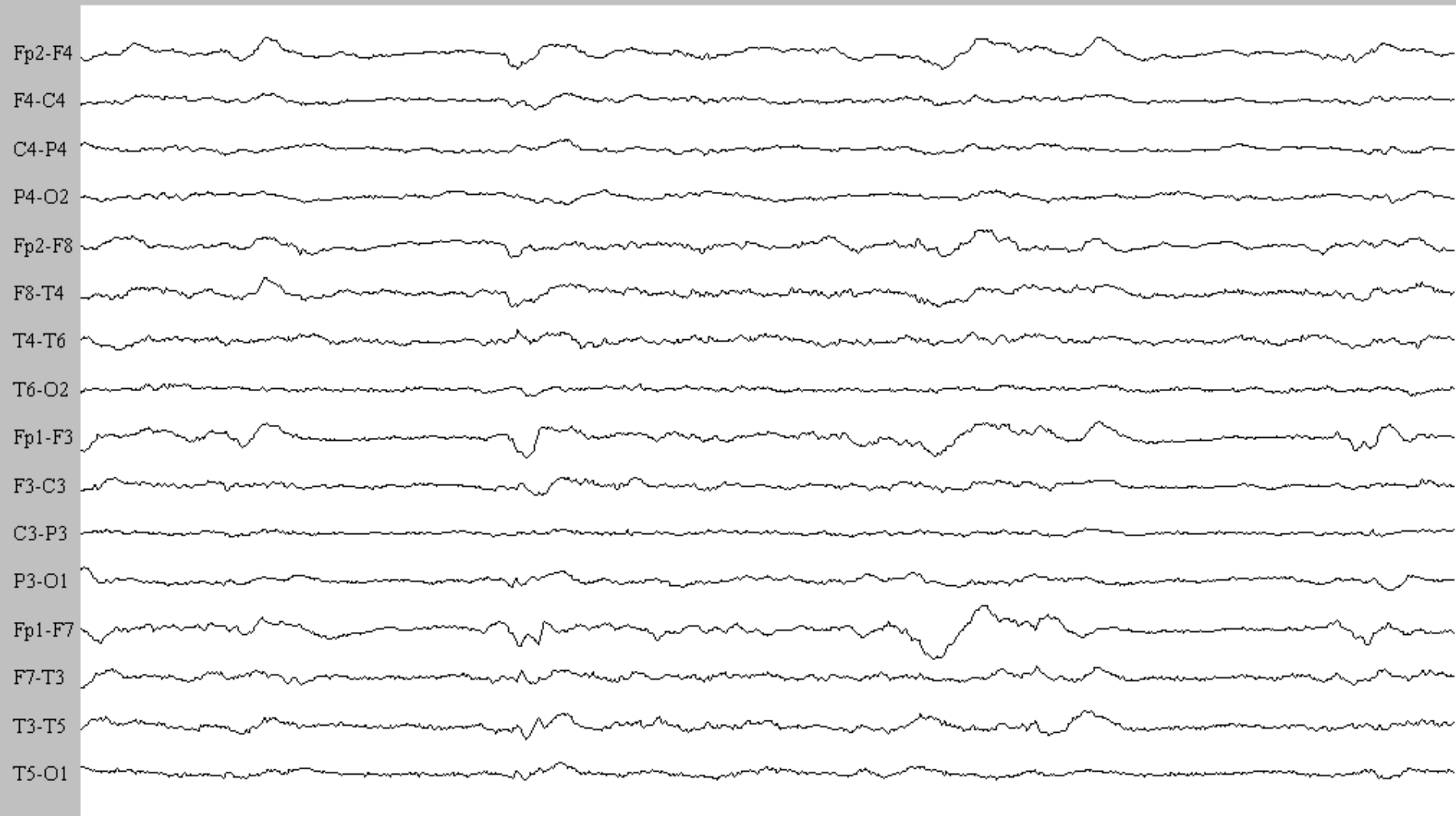
24 h

PES n. mediano Dx

PES n. mediano Sn



# evoluzione (48 h)

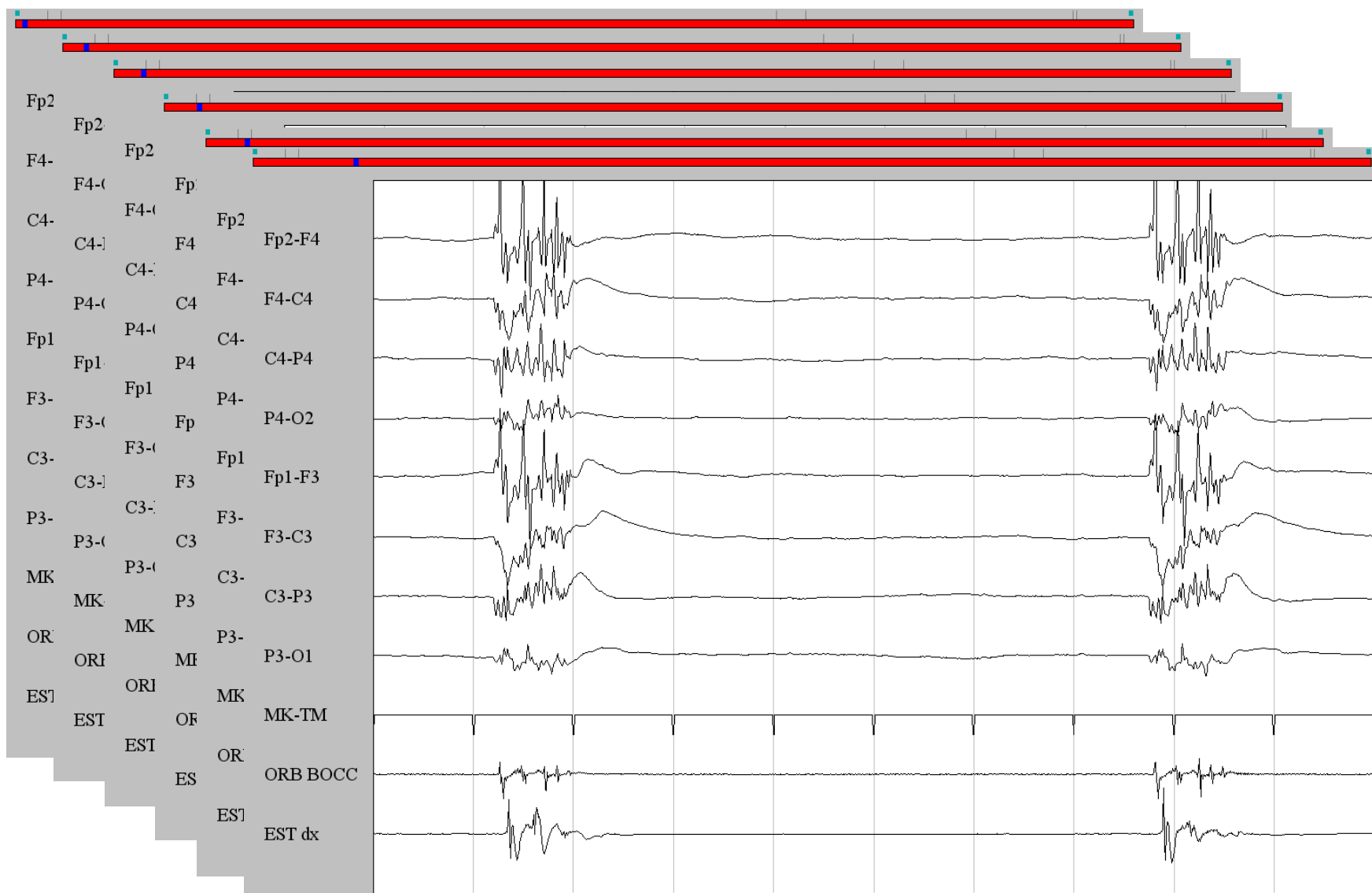




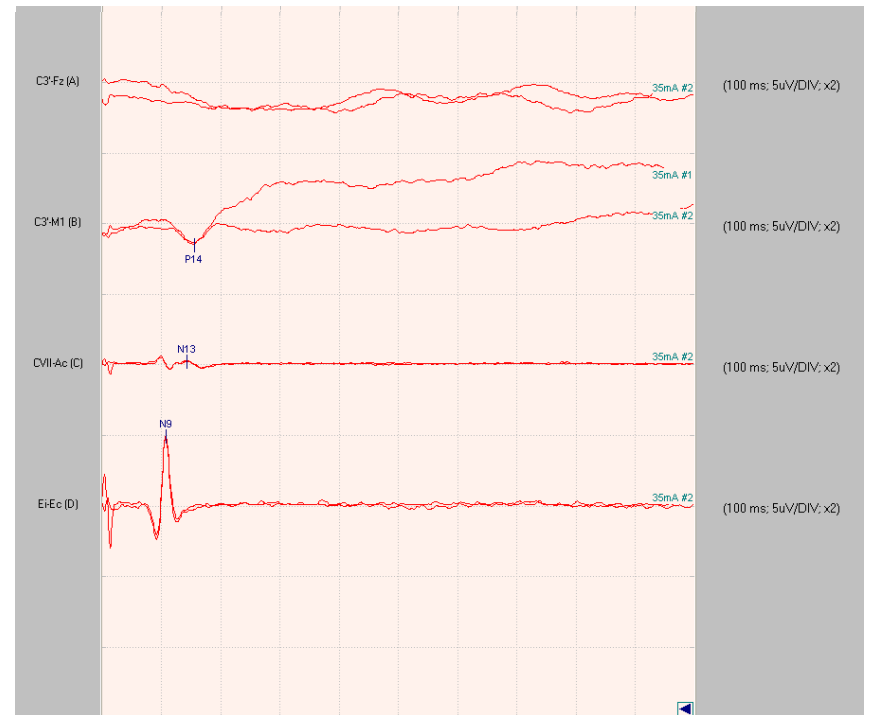
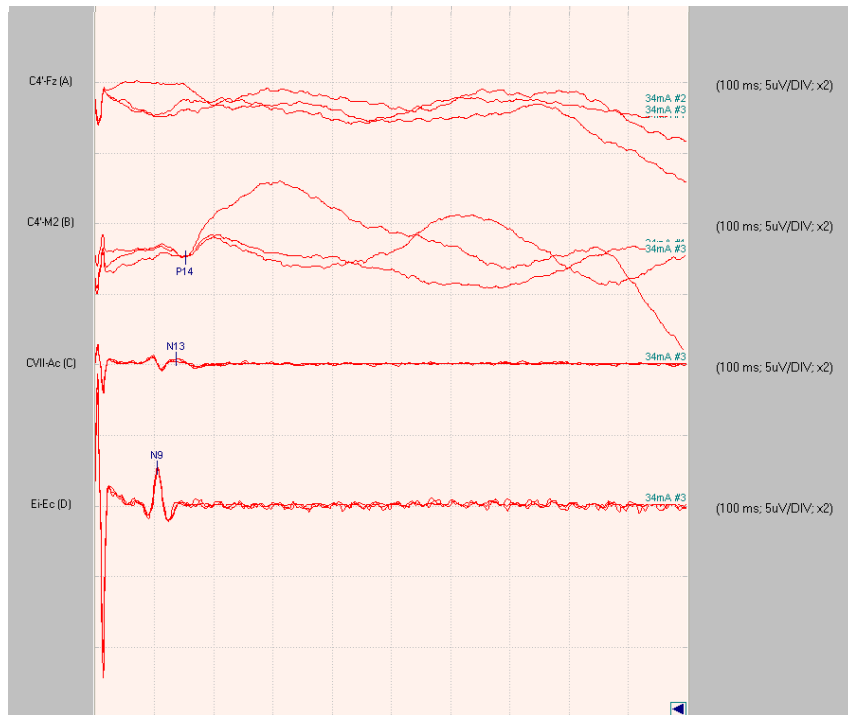
# evoluzione (4°g.)

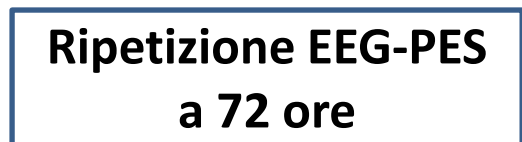
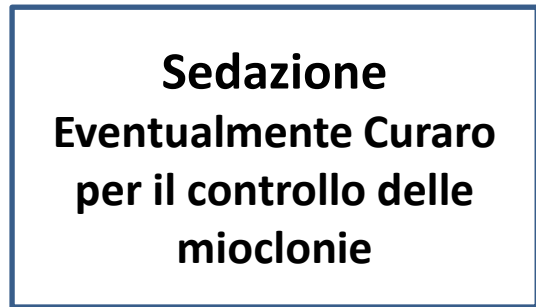
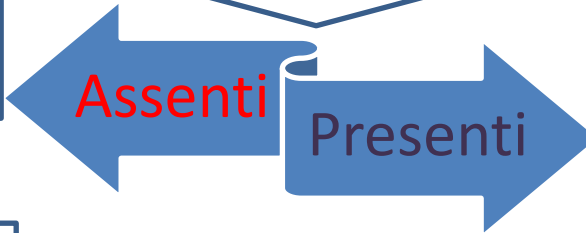


# 62 aa., GCS=4, miosi - 25/11 14h



# PES 25/11/13





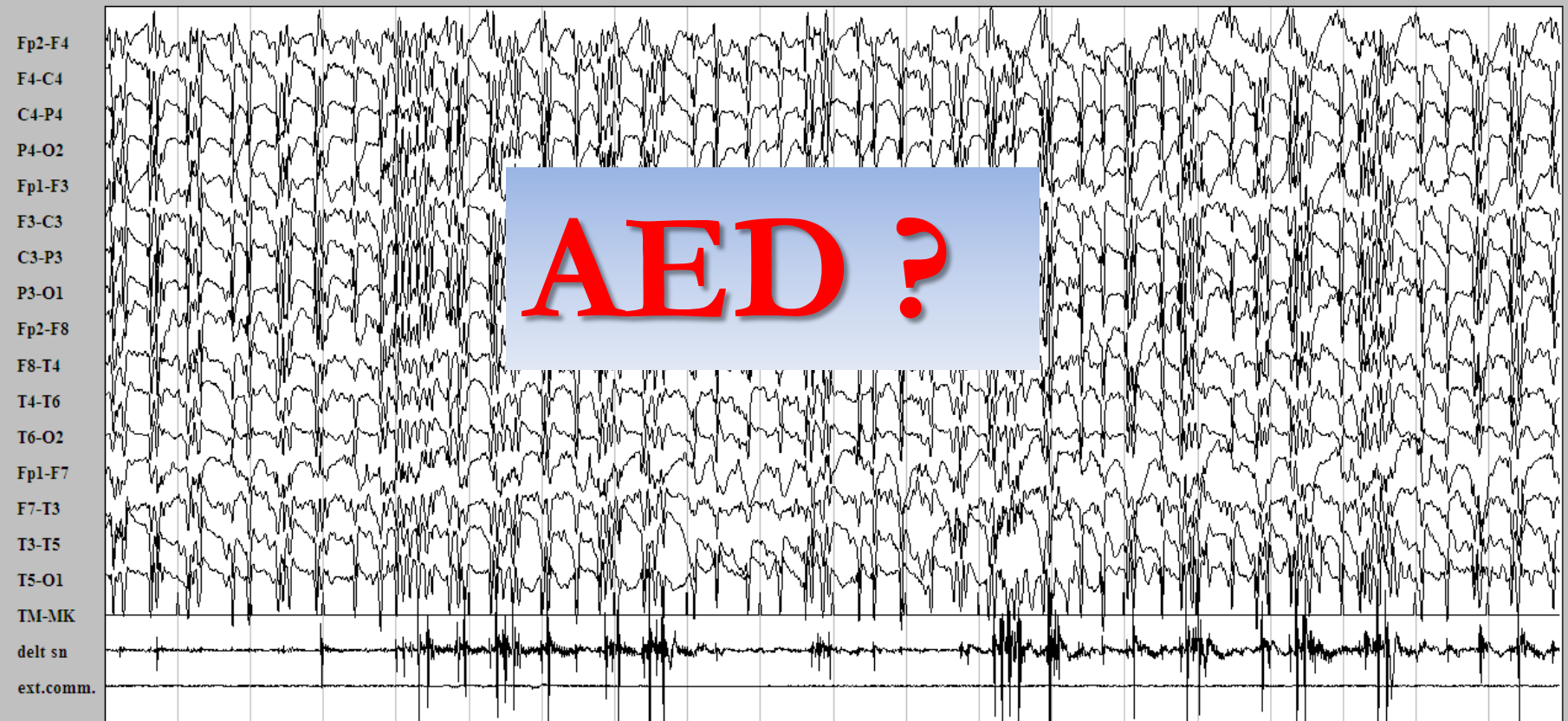
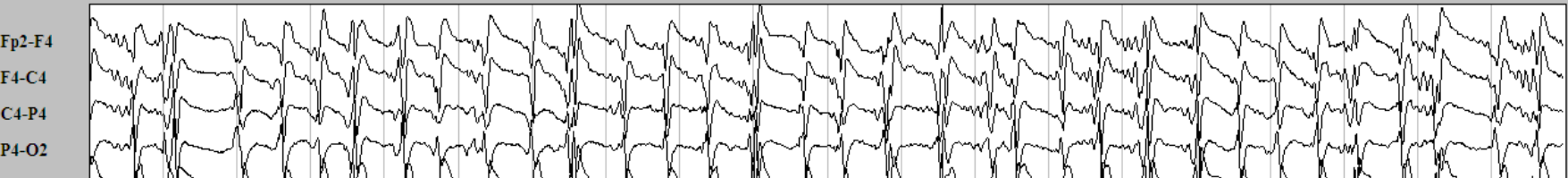
Sig...LP.....Data nascita 07-06-1962.....Età...48.....Sesso: M



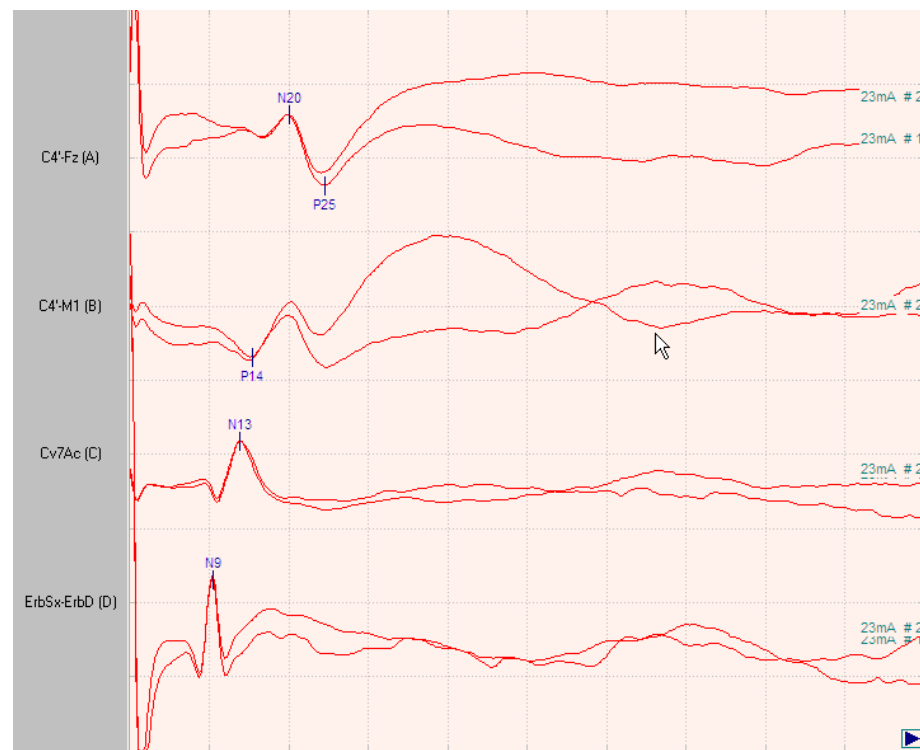
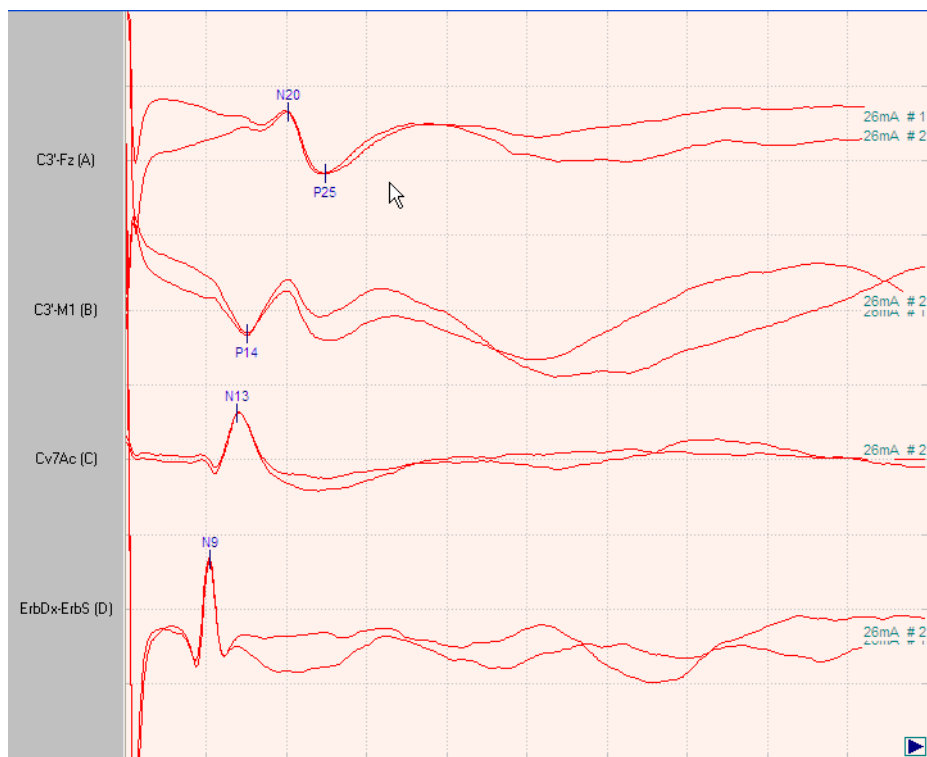
## Scheda Valutazione Coma Post-Anossico

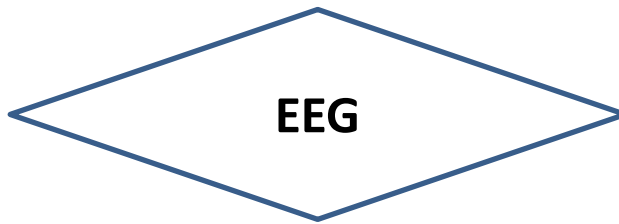
TIPO	DATI				
DATA ACR	14-11-2010	Ora ACR	17.00		
LUOGO ACR			EXTRAOSP		
CAUSA ACR	TRAUMA	ACIDOSI	ALTRO		
			X		
RITMO ESORDIO	FV/TV	ASISTOLIA	Pulseless Electrical Activity		
	X				
DURATA ARRESTO	10	TEMPO STIMATO ROS	25'		
GCS INGRESSO 118/PS	E=1	V=1	M=1	Tot:3	
GCS INGRESSO ICU	E=1	V=1	M=2	Tot:3	
Pupille		Diametro	X Miotiche	media midr	midriatiche
		Reattività	SI	NO	
INIZIO IPOT DA ACR (ore)					
DURATA IPOTERMIA (ore)	24				

# LR - 16.11.2010 (2°gg)

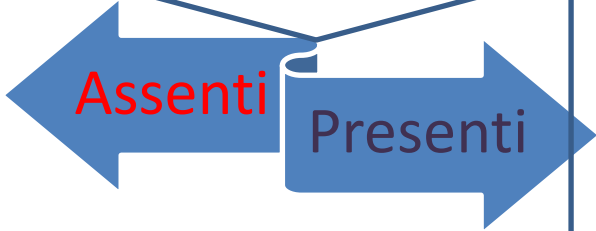


# LR - 16.11.2010 (2°gg)





Attività epilettiforme continua associata o meno a stato mioclonico



Sedazione + AED  
Levetiracetam  
2000mg/ Bolo  
2-3000mg/24h I.V.

Sedazione per 24 h

No Remissione  
Mioclonie

Remissione  
Mioclonie

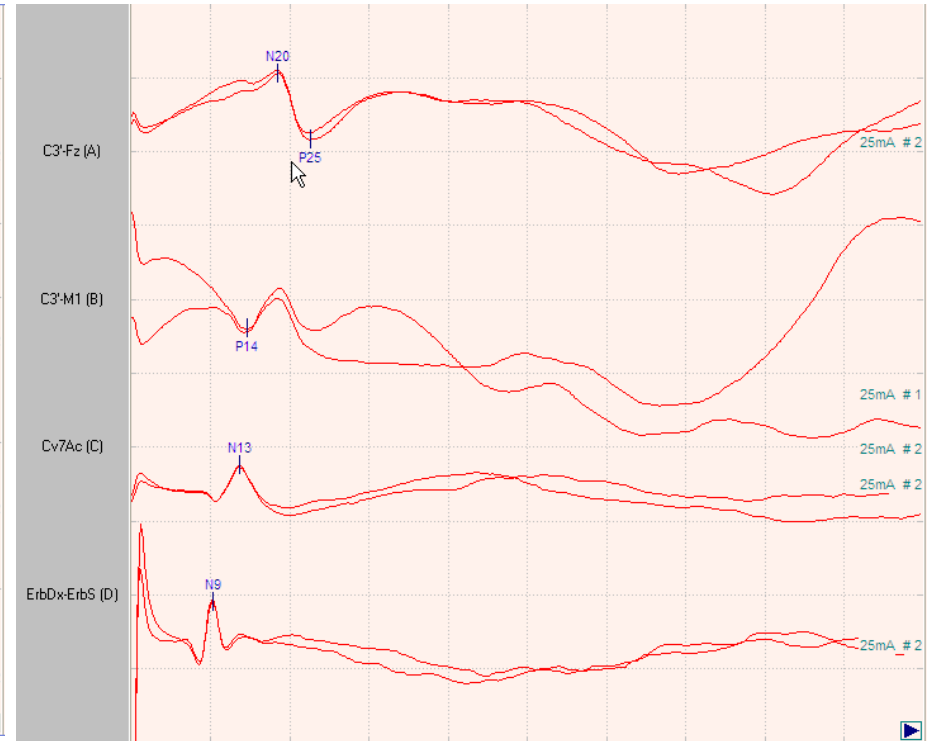
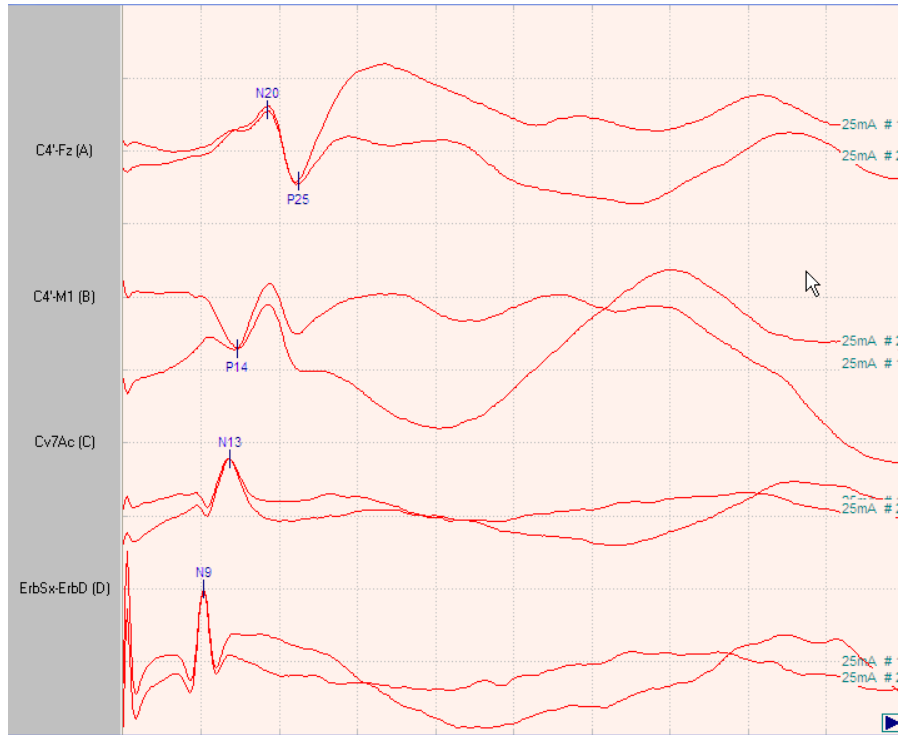
Depakin  
30mg/Kg Bolo  
1-2mg/Kg/h I.V



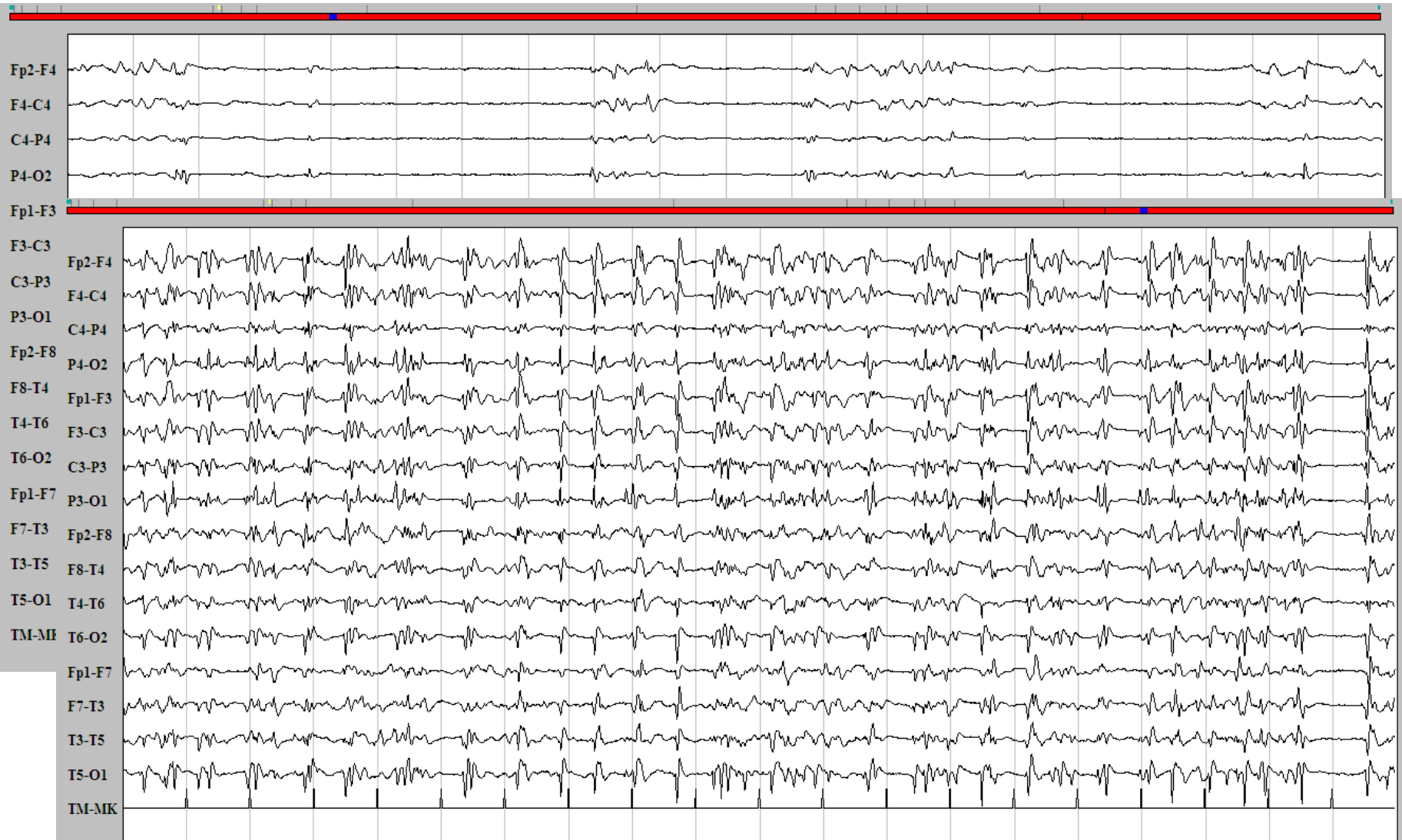
# LR - 19.11.2010 (5° gg)



# LR - 19.11.2010 (5° gg)



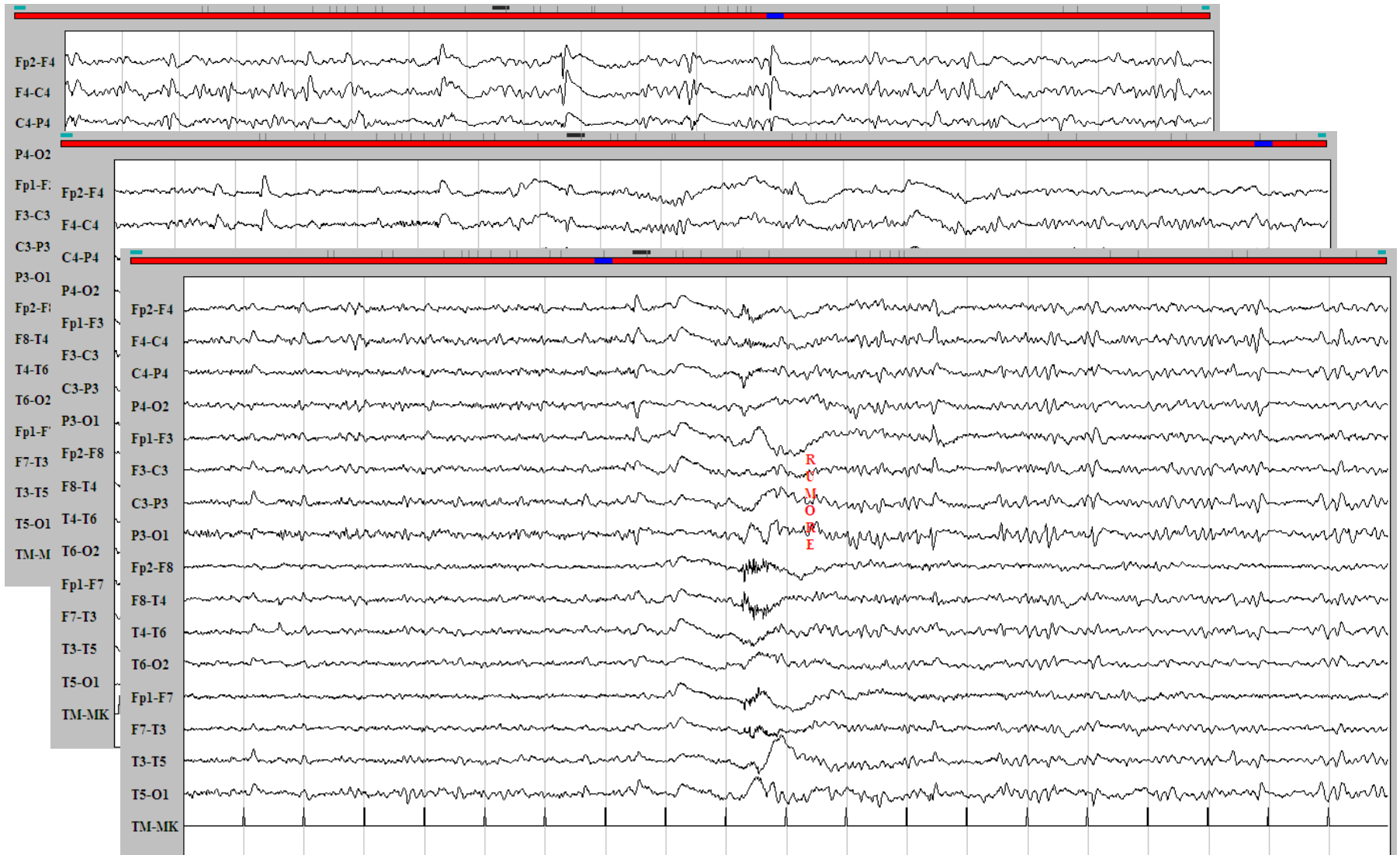
# LR - 23.11.2010 (9°gg)



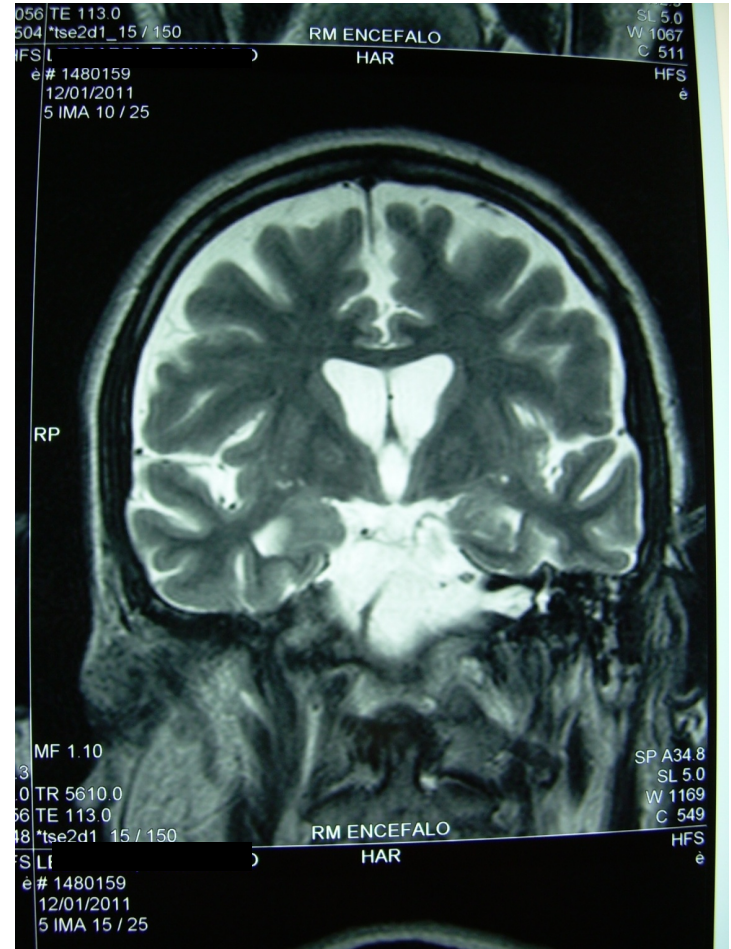
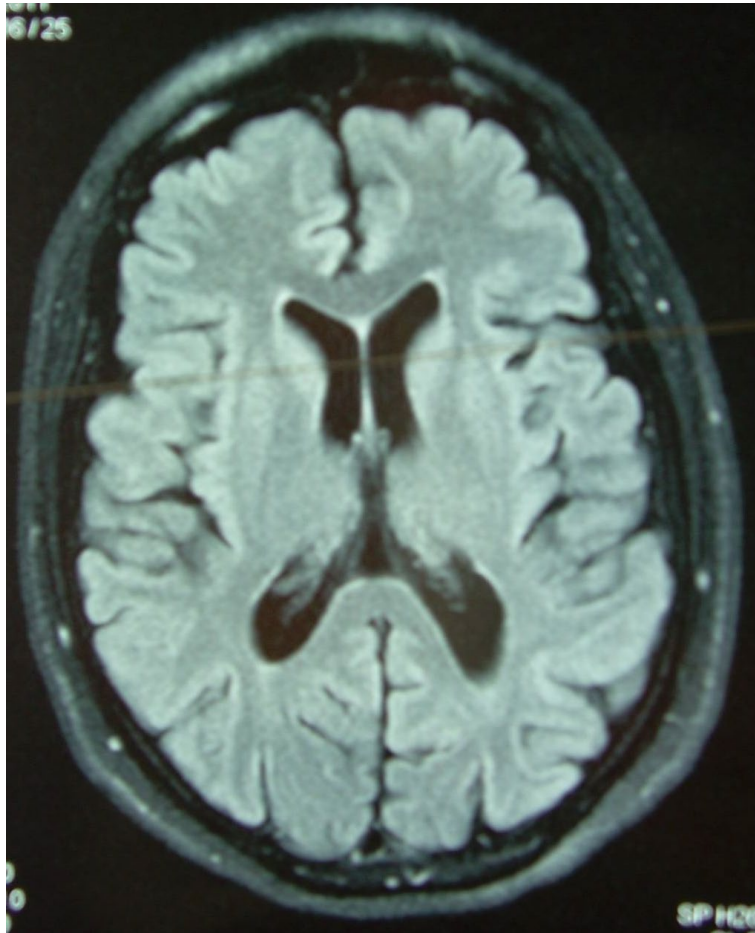
# LR - 30.11.2010 (16° gg)



# LR - 06.12.2010 (23°gg)

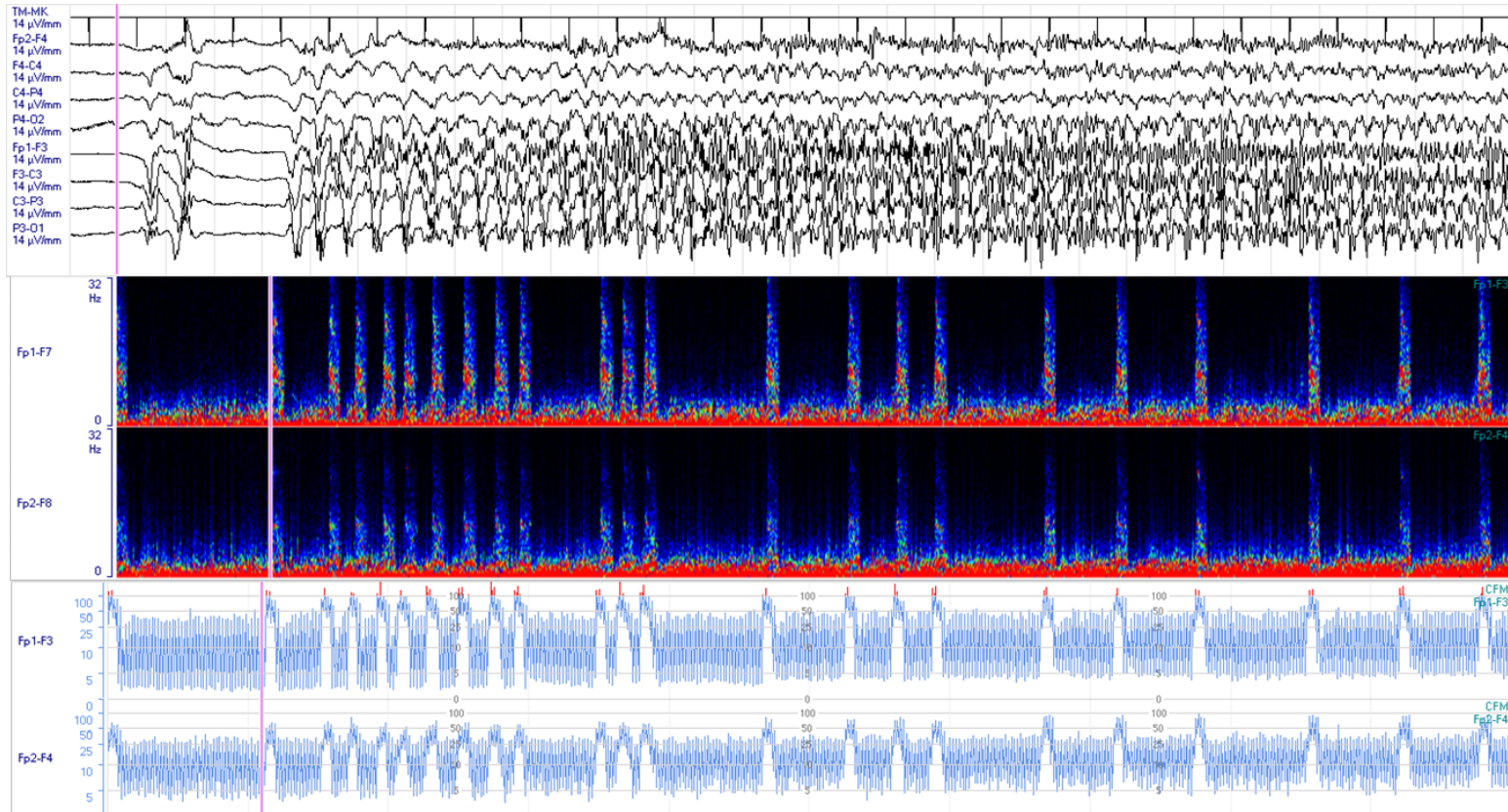


# LR 12-01-2011





# SENC





# t1 and t2 less defined for focal motor SE and NCSE (forms other than GCSE) in ICU



Treatment options different from GCSE and from NCSE after GCSE

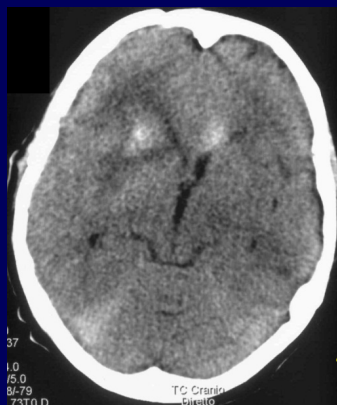
Different clinical settings: pt already intubated and sedated for ABI

NCS/NCSE are they contributing to the patient's clinical status?

cEEG is needed for diagnosis and treatment of NCSE and what the EEG end point during IV anesthetics?

Different times of evaluation of AED efficacy (first and subsequent AED) under EEG guide interpreting the dynamic changes of ictal and periodic discharges

Multimodal approach to managing critical ill patients with periodic or rhythmic patterns in the ictal-interictal continuum



D.A,w, 61 yrs

SHA rupture a.c.a.aneurism and left fronto-mesial hematoma

16.11

AdMISSION IN ICU: GCS=6

intubated and sedated (Propofol 2mg/kg/h)

2 focal to tonic-clonic seizures in 10 minutes ( right facial clonic, right gaze version)

Start treatment with BDZ followed by PHT (Phenytoin) I.V. (bolus + maint.)

17.11

Doubtful facial myoclonic movements, GCS=5 (E1,V1,M3)

starts Video-EEG

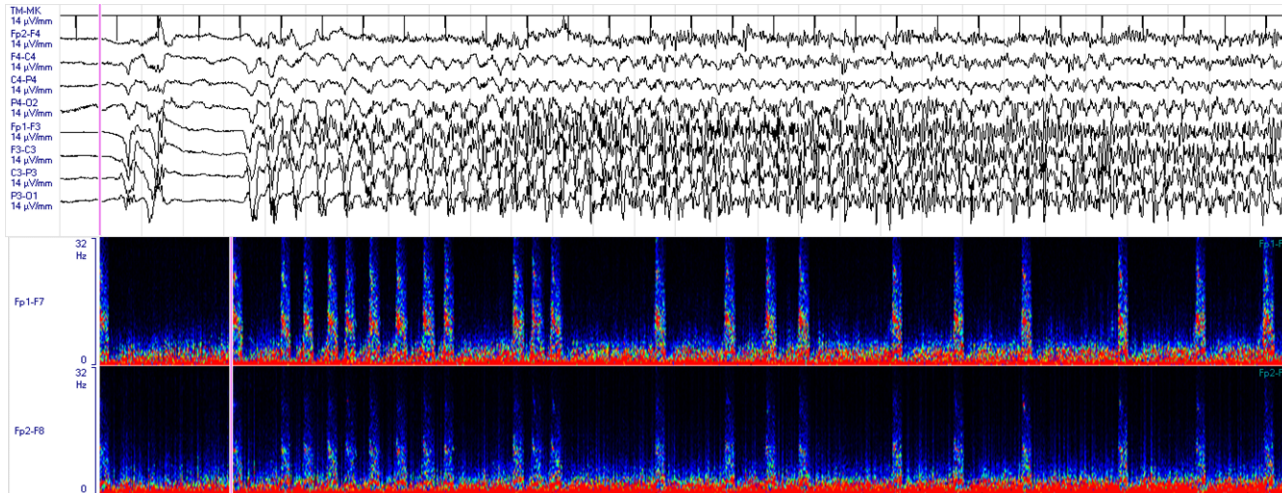








D.A., 61 a



## «subtle» focal NCSE with low propofol and one AED

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- DPH within therapeutic range
  - The pt is already intubated

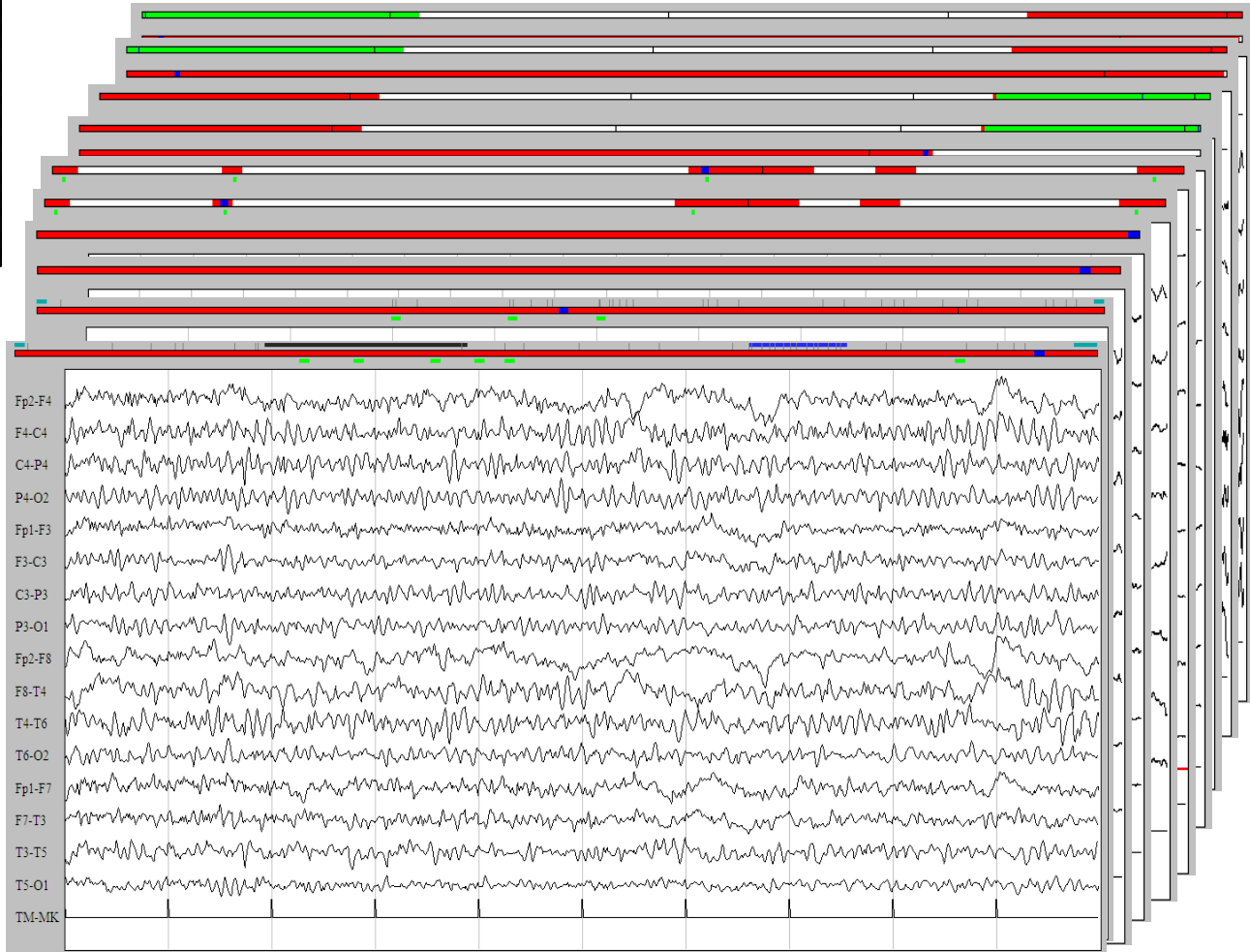
?



BDZ  
2° AED



Increase propofol  
infusion under EEG  
guide





We agree that NCSE need to be treated less aggressive than refractory GCSE, but the ICU setting may provide different options:



*if the patient is already intubated and sedated for ABI and not for NCSE, we think in this condition if we diagnose a NCSE the therapeutic choice is not univocal and is tailored on individual patient and individual type of SE*



# What EEG end-point?

*Suppression of seizures*

*Suppression of seizures and interictal epileptiform activity*

*Suppression of seizures + burst suppression*

*Burst suppression > or < 10 sec*

*EEG suppression of 100%*

“The length of interburst intervals and burst suppression did not predict successful termination of RSE...”  
(Johnson et al., 2016)

***Seizure suppression is the primary end point with eventual brief EEG suppression intervals if possible***

*Maintain seizure suppression for 12/24 hs, then reduce progressively sedation*

*Increase again the sedation if seizures relapse (super-refractory SE)*

## P.M., F – 75 yrs

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▶ 07/08/13:

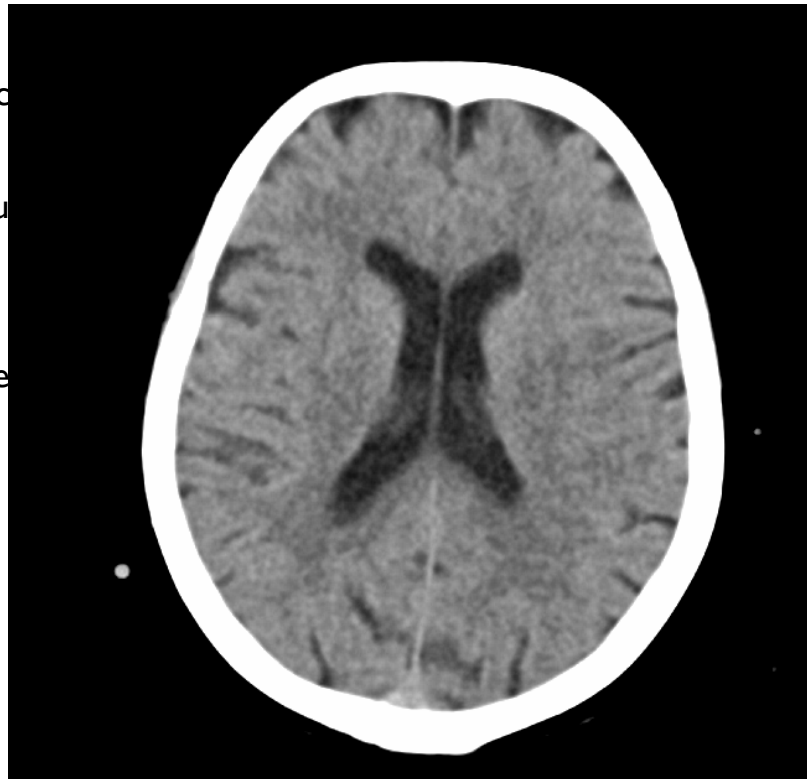
Admission in ICU after right lobectomy

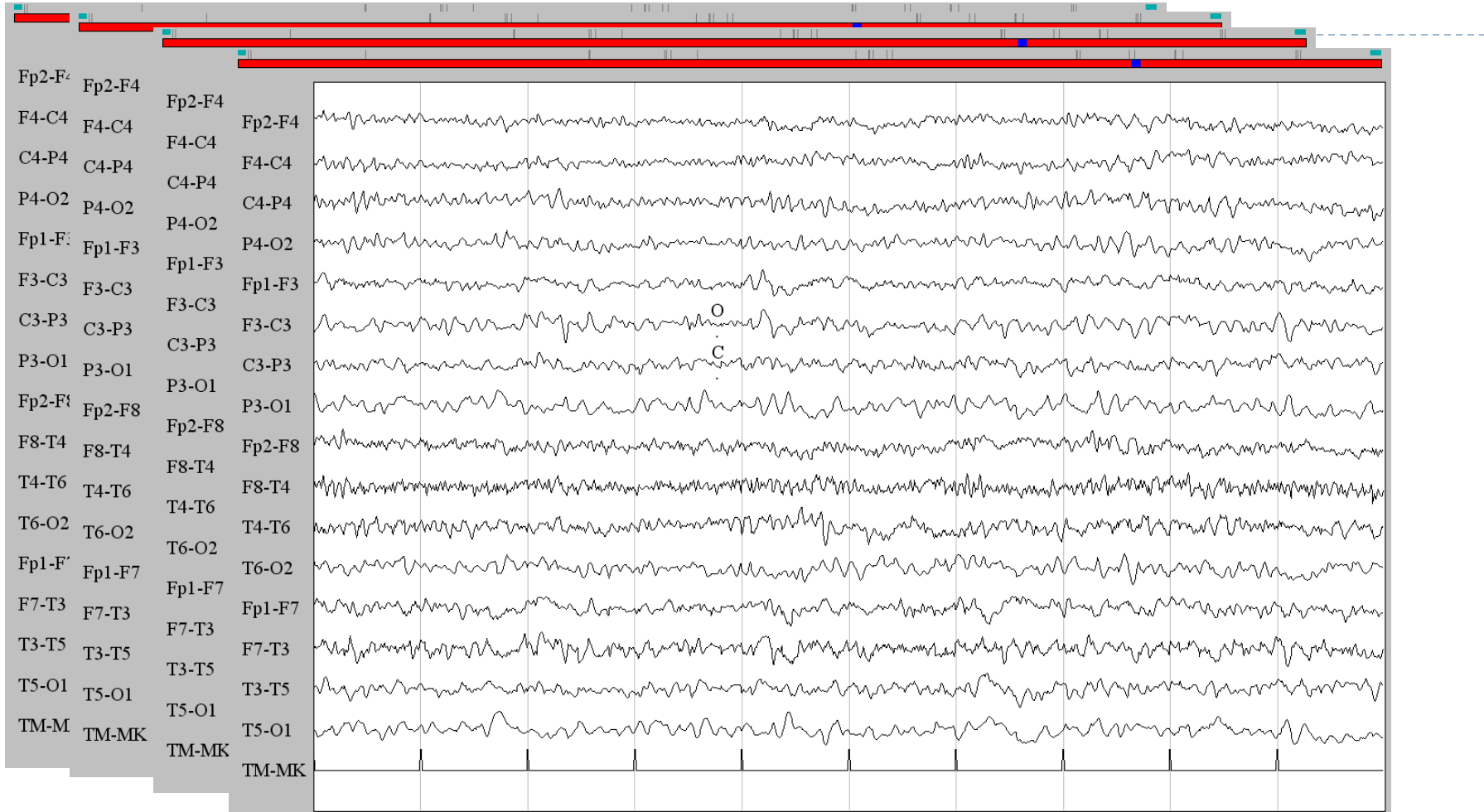
▶ 08/08/13:

a focal motor to tonic-clonic seizure

Brain TC and angio-TC negative.

During EEG recording : drowsy, he





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▶ 9/8/13:

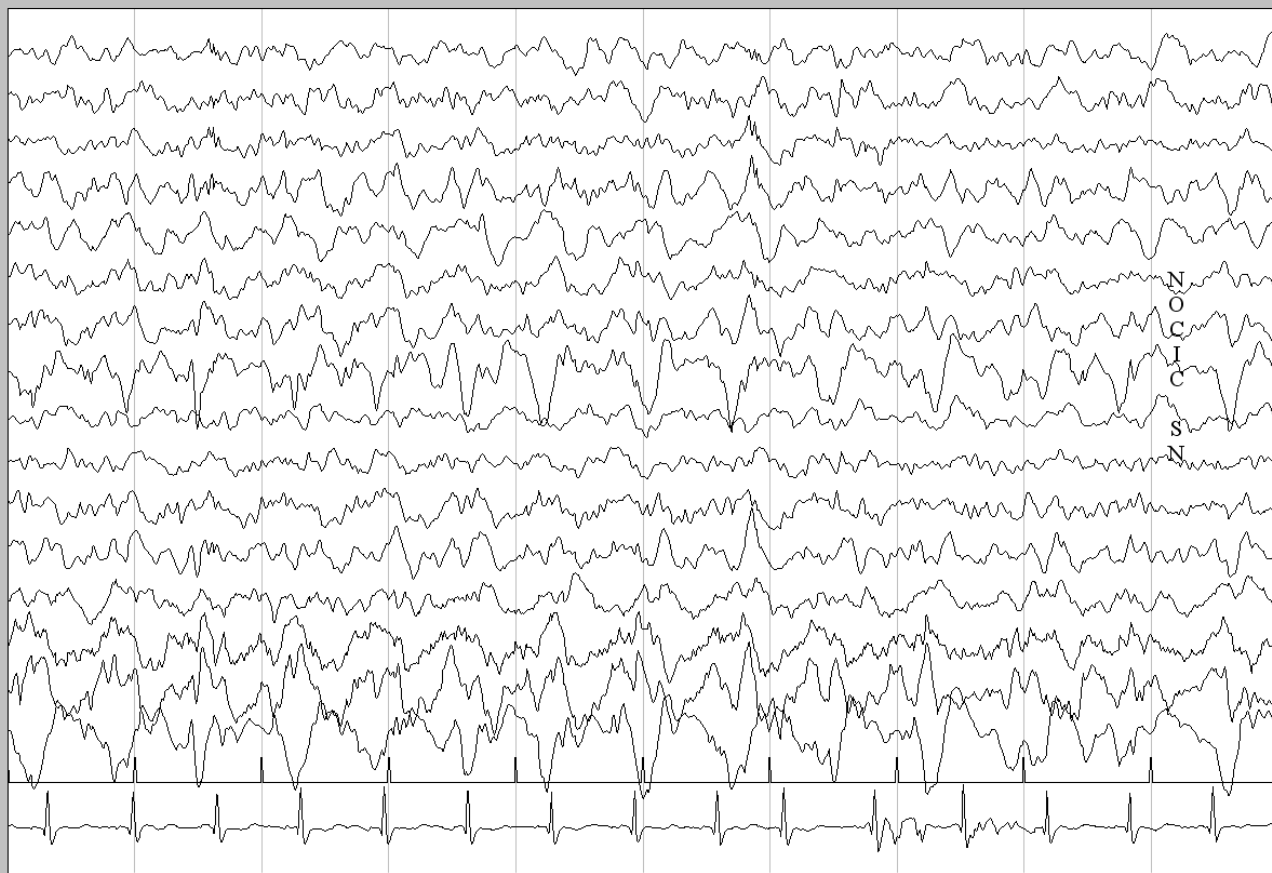
2 focal motor to tonic-clonic seizures in one hour:  
diazepam IV (10mg + 5mg).

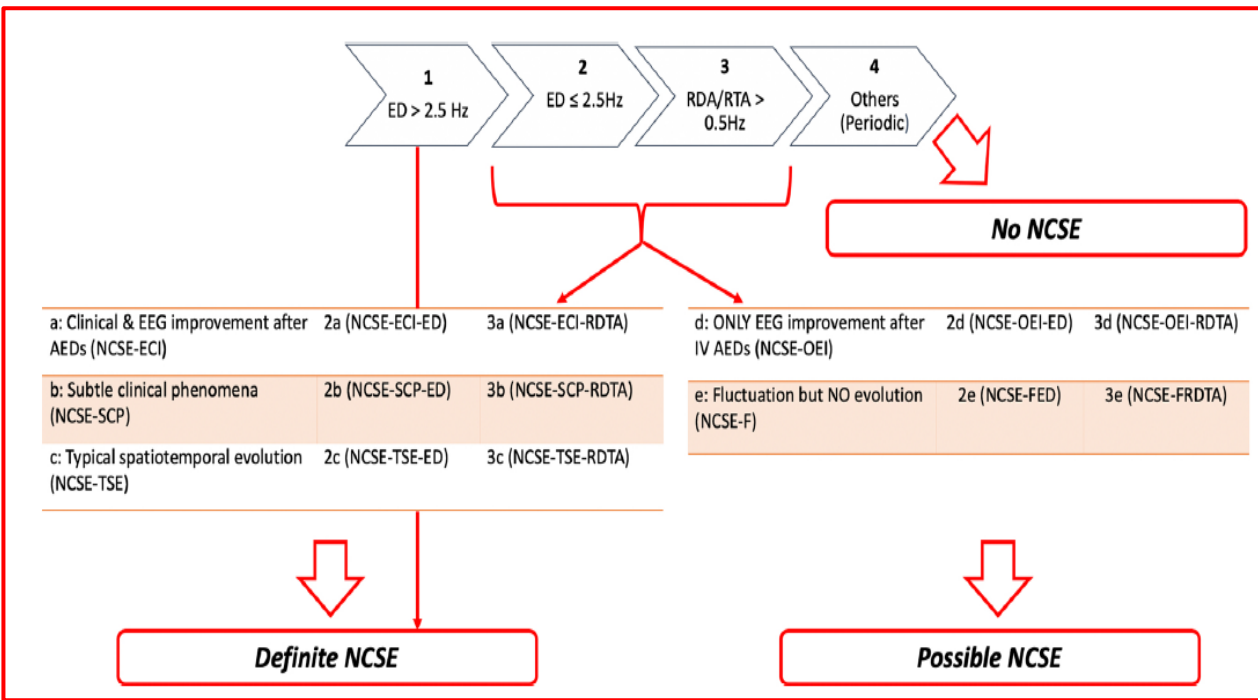
Pt unresponsive, persist right hemiparesis



# EEG 09/08/2013

Fp2-	Fp2-F	Fp2-F4	Fp2-F4	Fp2-F4	Fp2-F4	Fp2-F4
F4-C	F4-C4	F4-C4	F4-C4	F4-C4	F4-C4	F4-C4
C4-F	C4-P4	C4-P4	C4-P4	C4-P4	C4-P4	C4-P4
P4-C	P4-O2	P4-O2	P4-O2	P4-O2	P4-O2	P4-O2
Fp1-	Fp1-F	Fp1-F3	Fp1-F3	Fp1-F3	Fp1-F3	Fp1-F3
F3-C	F3-C3	F3-C3	F3-C3	F3-C3	F3-C3	F3-C3
C3-F	C3-P3	C3-P3	C3-P3	C3-P3	C3-P3	C3-P3
P3-C	P3-O1	P3-O1	P3-O1	P3-O1	P3-O1	P3-O1
Fp2-	Fp2-F	Fp2-F8	Fp2-F8	Fp2-F8	Fp2-F8	Fp2-F8
F8-T	F8-T4	F8-T4	F8-T4	F8-T4	F8-T4	F8-T4
T4-T	T4-T6	T4-T6	T4-T6	T4-T6	T4-T6	T4-T6
T6-C	T6-O2	T6-O2	T6-O2	T6-O2	T6-O2	T6-O2
Fp1-	Fp1-F	Fp1-F7	Fp1-F7	Fp1-F7	Fp1-F7	Fp1-F7
F7-T	F7-T3	F7-T3	F7-T3	F7-T3	F7-T3	F7-T3
T3-T	T3-T5	T3-T5	T3-T5	T3-T5	T3-T5	T3-T5
T5-C	T5-O1	T5-O1	T5-O1	T5-O1	T5-O1	T5-O1
TM-I	TM-M	TM-MK	TM-MK	TM-M	TM-MK	TM-MK
ECG	ECG	ECG	ECG	ECG	ECG	ECG



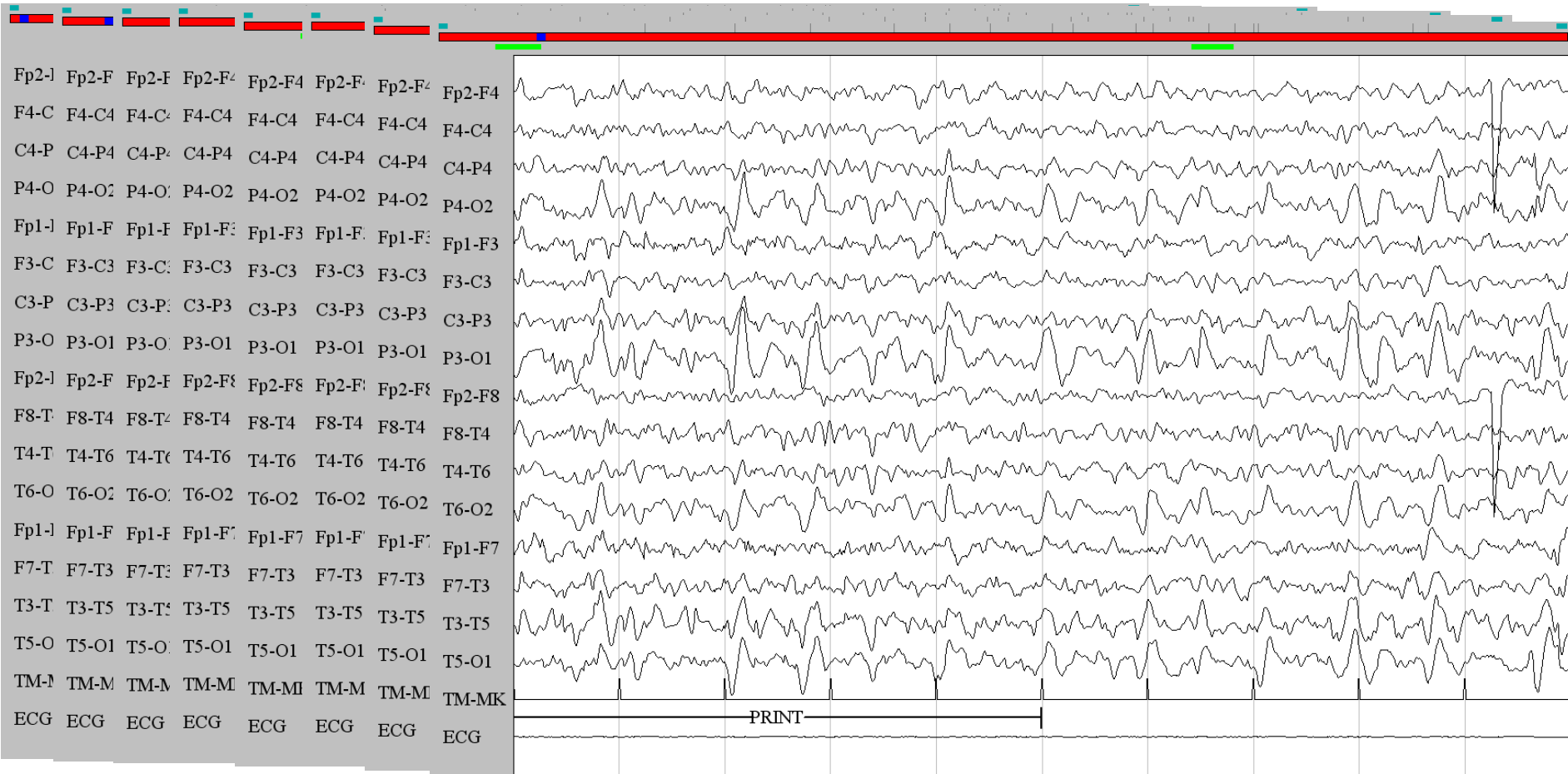


(Othman et al., 2020)

Start AED with VPA (bolus 30mg/kg + infusion 2 mg/kg/h)



10/08/13



- 
- ▶ The EEG shows focal NCSE refractory to BDZ and first AED, but the focal continuous epileptiform activity is fragmented into PDs (LPDs) at low frequency and discrete focal NCS with recruiting rhythm

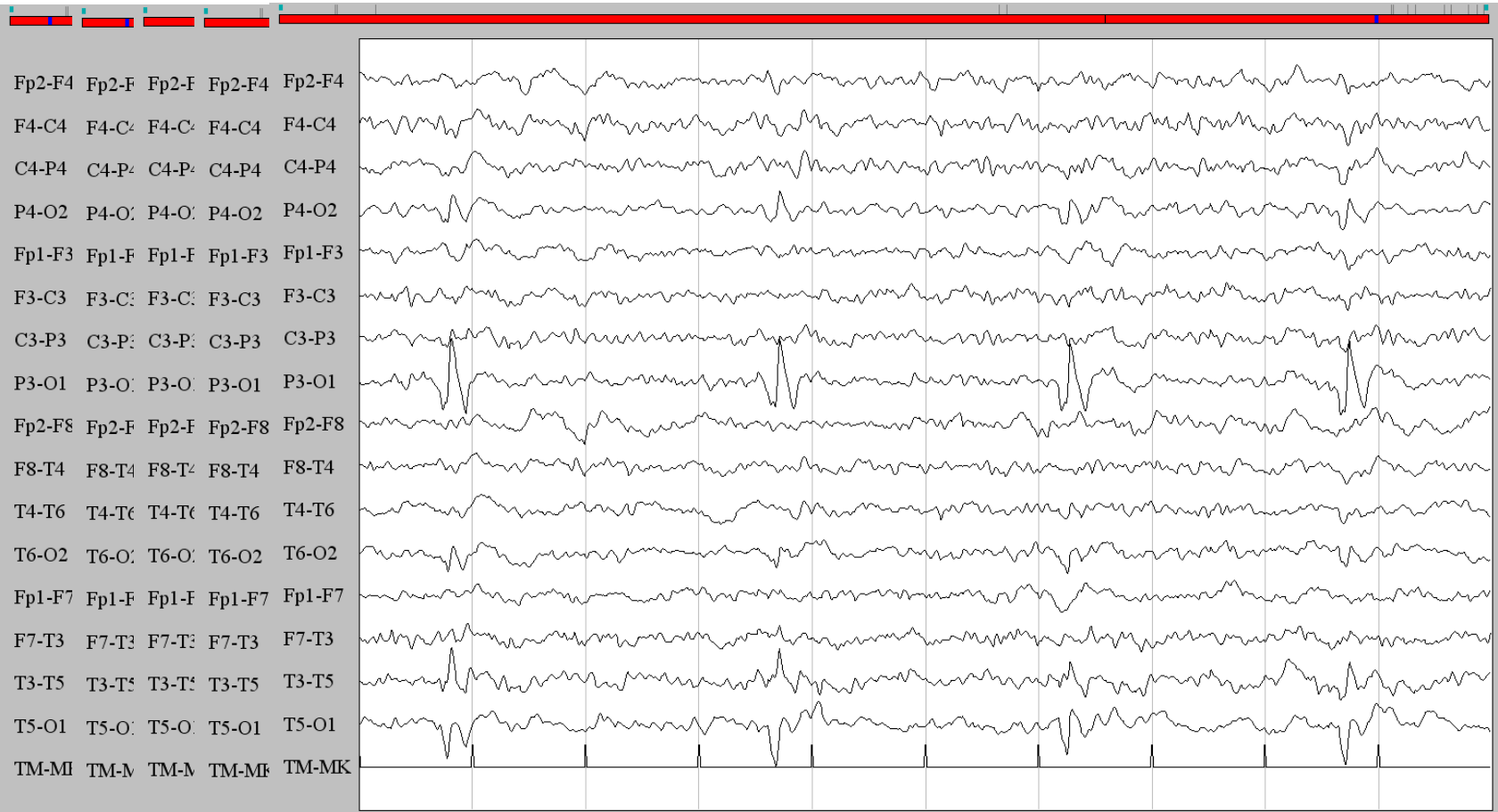
### **What further treatment?**

- ▶ Second AED?
- ▶ Sedation?
- ▶ Otherwise...?
  
- ▶ **Therapy unchanged**

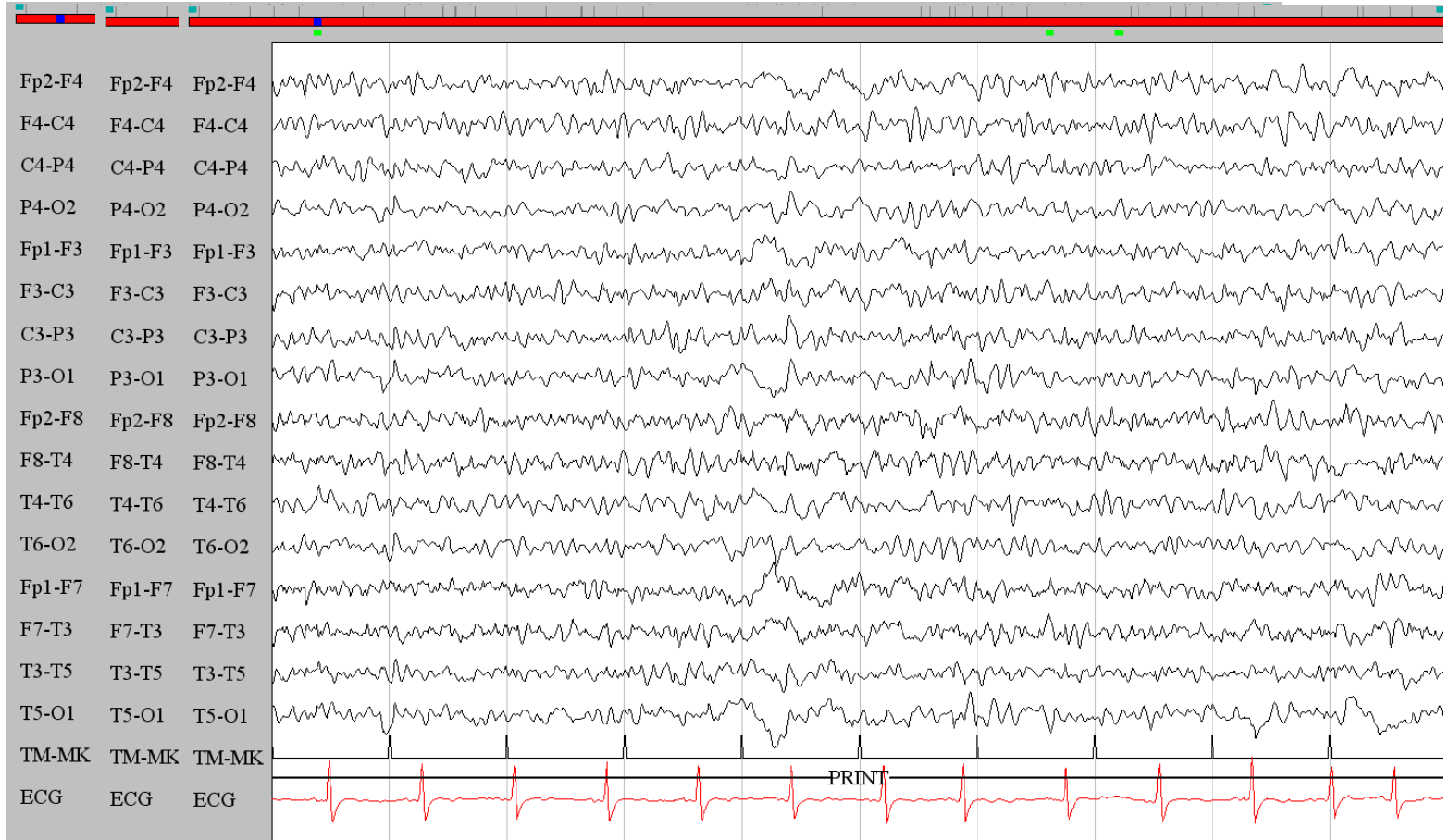




11/08/13



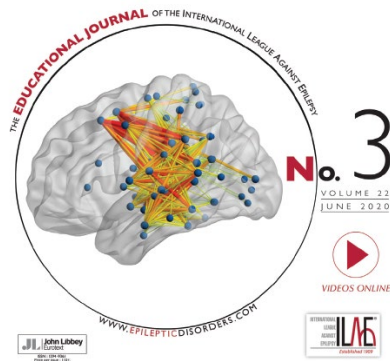
14/08/13



- 
- ▶ In a focal refractory NCSE how long to wait to evaluate the efficacy of first AED before adding a second AED?
  - ▶ We think that in this context is not so urgent the decision and that EEG has an important role, in expert hands, to understand the dynamic changes and evolution of epileptic activity in the absence of clinical manifestations



# Epileptic Disorders



## Clinical commentary

*Epileptic Disord* 2020; 22 (3): 337-41

# A multimodal diagnostic approach for lateralised rhythmic delta activity in the ictal-interictal continuum\*

Maddalena Spalletti<sup>1</sup>, Francesca Pescini<sup>2</sup>, Davide Gadda<sup>3</sup>,  
Benedetta Piccardi<sup>2</sup>, Maenia Scarpino<sup>1,4</sup>, Riccardo Carrai<sup>1,4</sup>,  
Cristina Boccardi<sup>1</sup>, Antonello Grippo<sup>1,4</sup>, Aldo Amantini<sup>1,4</sup>



M.M., 82 yrs

**14.03.18**

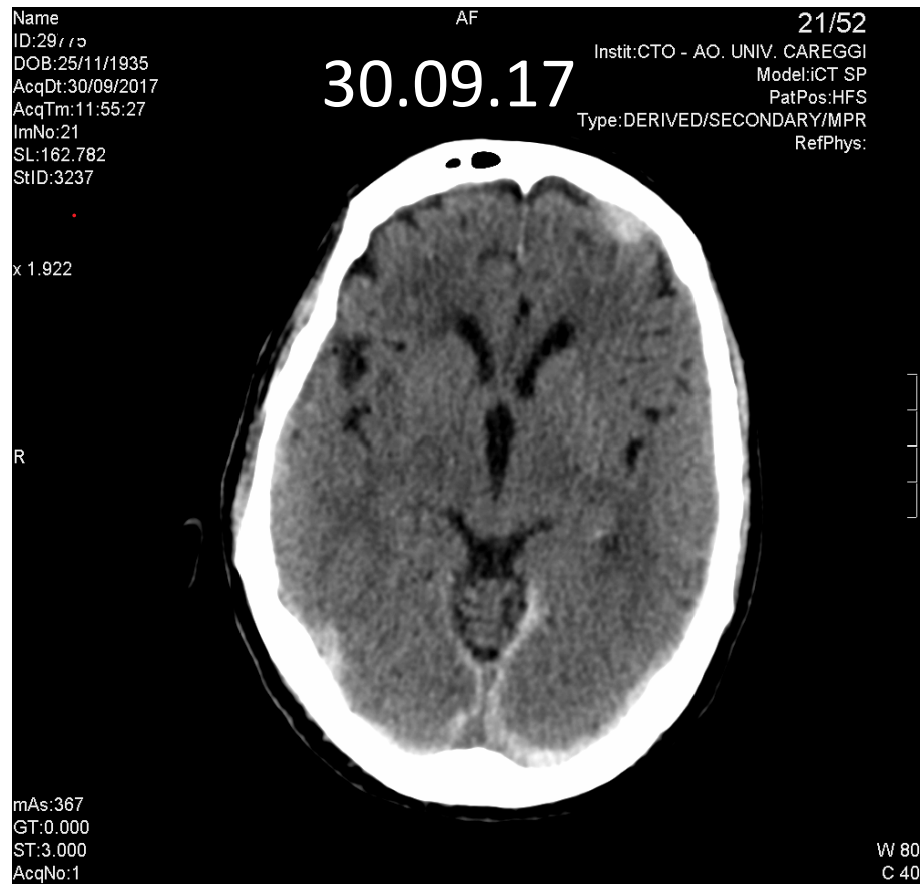
ER admission for acute onset aphasia and confusional state.

Normal metabolic indices

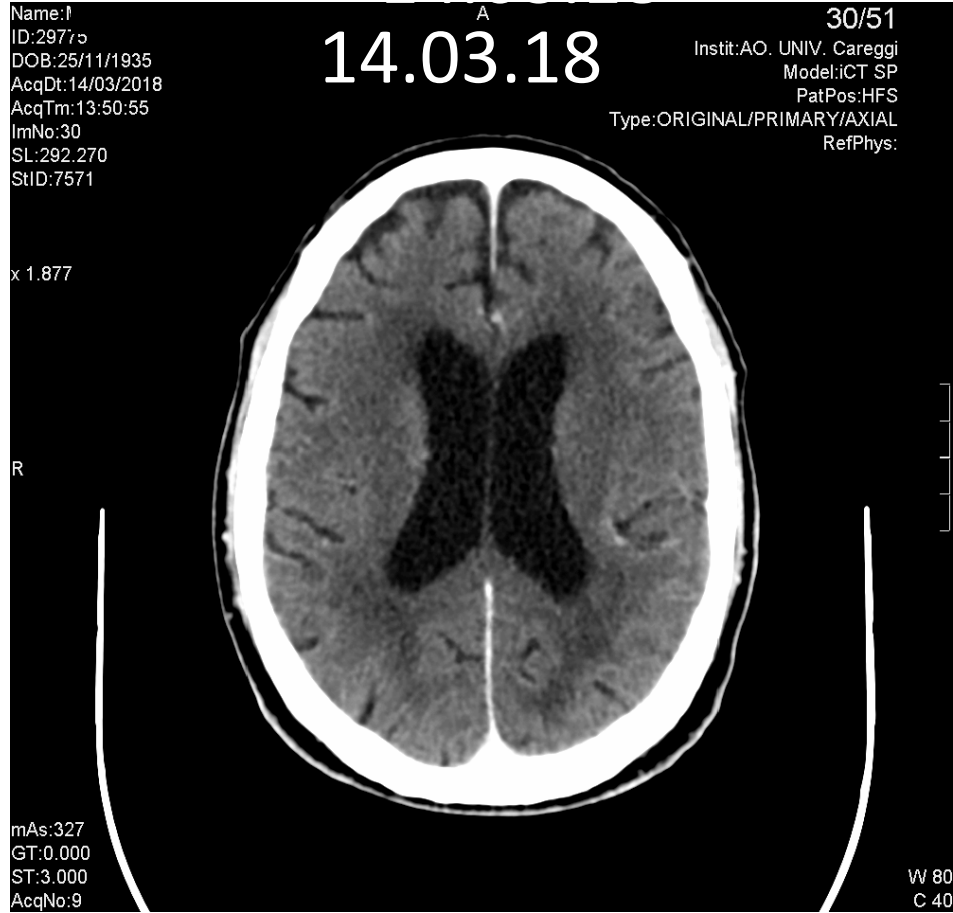
No fever



M.M., 82  
yrs



M.M., 82 yrs



Name: I  
ID: 29775  
DOB: 25/11/1935  
AcqDt: 14/03/2018  
AcqTm: 13:45:06  
ImNo: 4  
SL: 0.000  
StID: 7569

14.03.18

4/5  
Instit: AO. UNIV. Careggi  
Model: iCT SP  
PatPos: HFS  
Type: DERIVED/SECONDARY/3D/MIP  
RefPhys:

x 0.602

R



10 cm

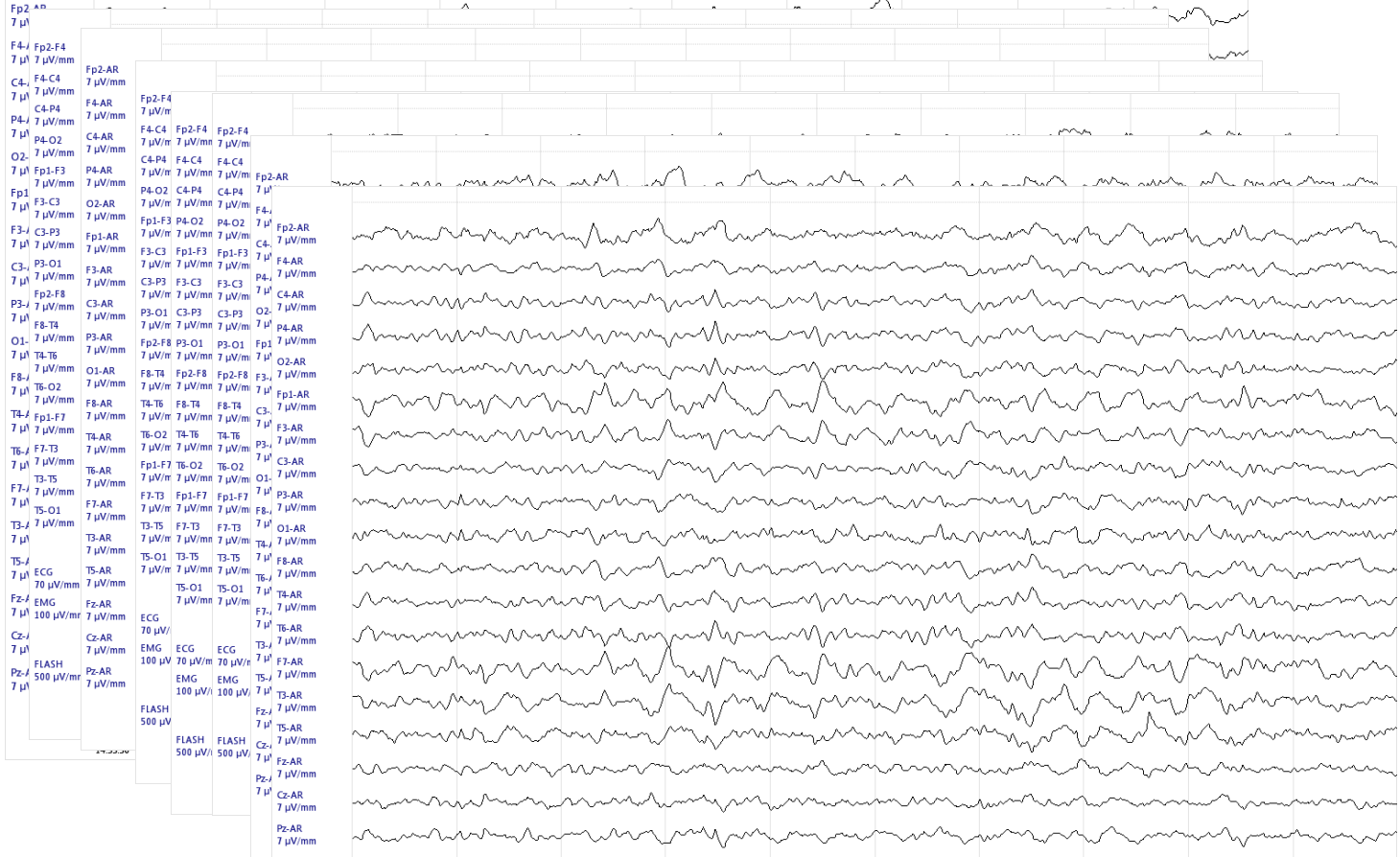


mAs:  
GT: 0.000  
ST: 0.000  
AcqNo: 5

W 741  
C 247



14.03.18 – h.14.48



# RHYTHMIC DELTA ACTIVITY (RDA) (No+/+F/+S/+FS):

Lateralized (Lateralized Rhythmic Delta Activity, LRDA),

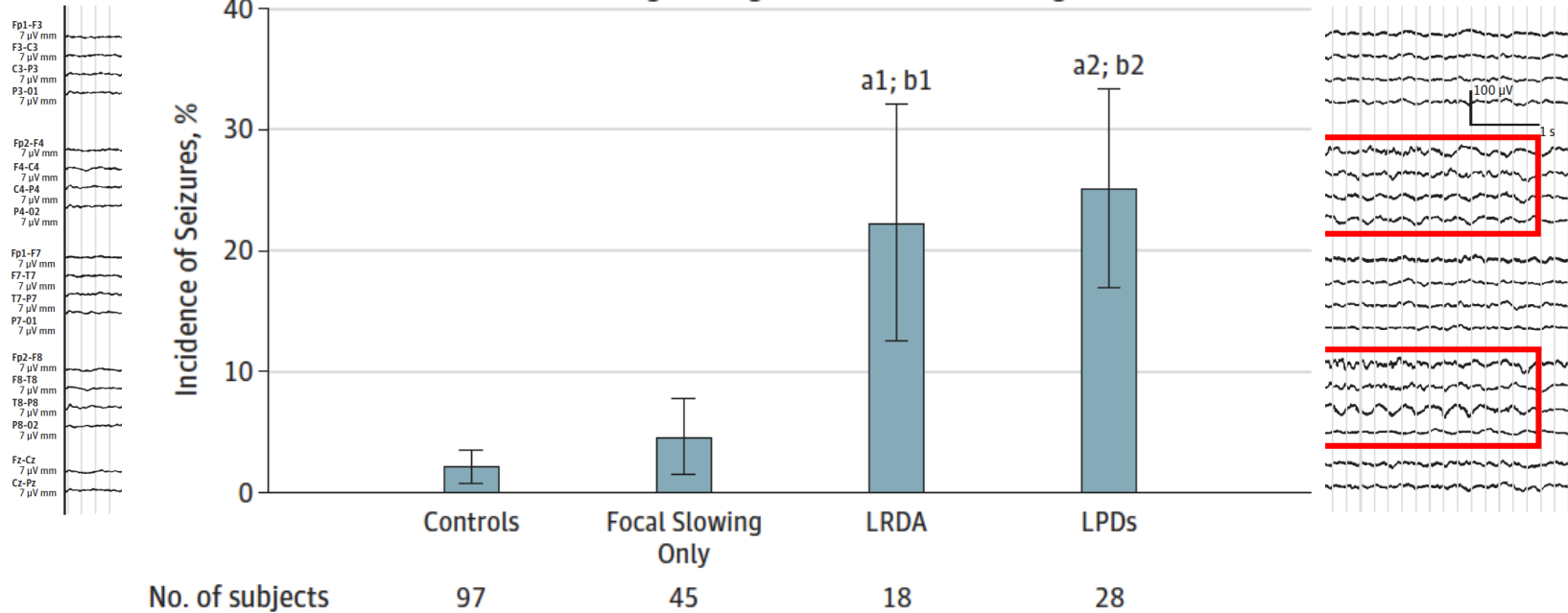
Original Investigation

## Similarity of Lateralized Rhythmic Delta Activity to Periodic Lateralized Epileptiform Discharges in Critically Ill Patients

Caenard et al • JAMA Neurol. 2013;70(10):1288-1295.

B

EEG Findings During First Hour of Monitoring



**Multimodal Approach to Decision to Treat Critically Ill Patients With Periodic or Rhythmic Patterns Using an Ictal–Interictal Continuum Spectral Severity Score**

Gregory Kapinos,\* Eugen Trinka,†‡ and Peter W. Kaplan§

The degree to which the types of PRPs are:

**epileptiform**

**ictal** (representing a seizure)

**ictogenic** (i.e., related to risk of a subsequent acute seizure)

**INVITED REVIEW**

(J Clin Neurophysiol 2018;35: 309–313)

## **Approach to Managing Periodic Discharges**

Andrew Bauerschmidt, Clio Rubinos, and Jan Claassen

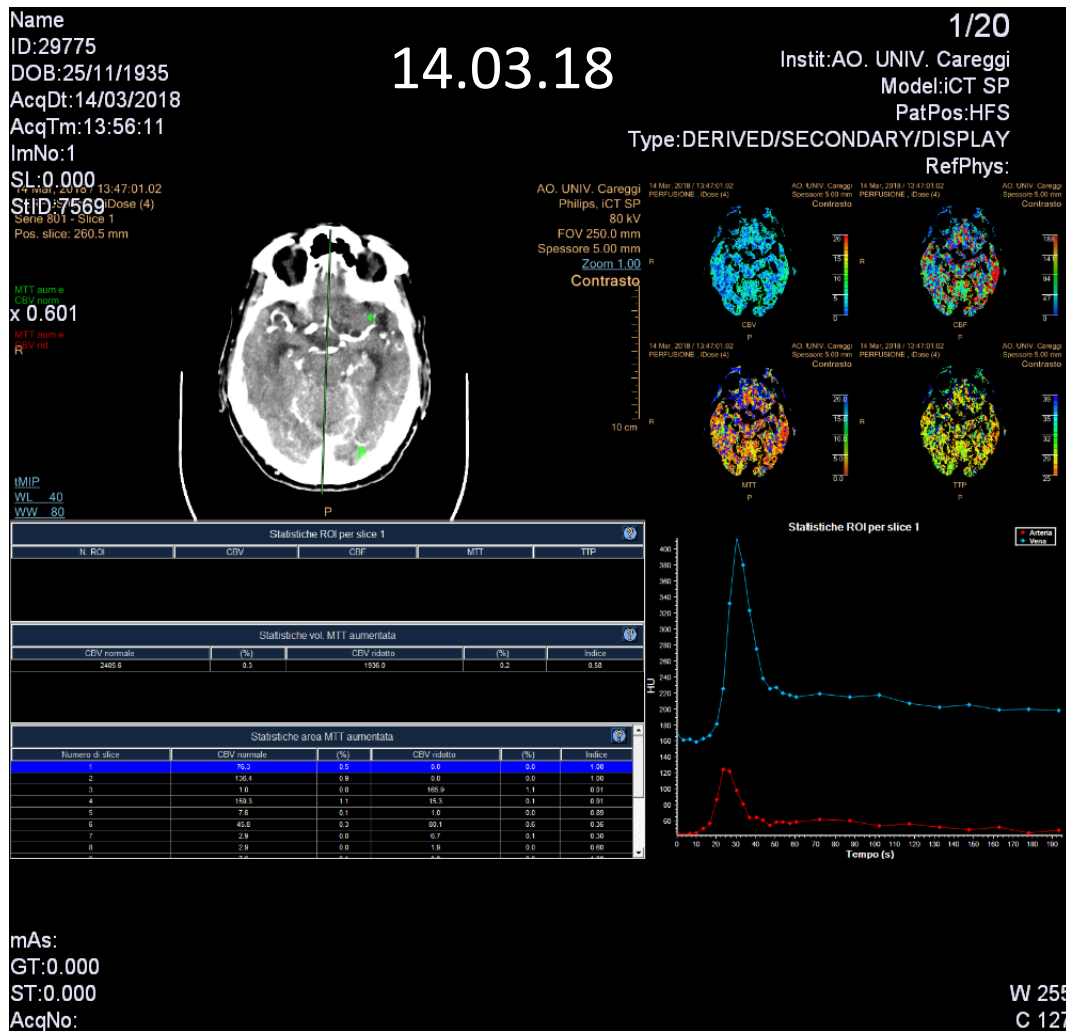
stepwise approach to assessment and management of  
Periodic Discharges:

**(1) clinical assessment including benzodiazepine trial**

**(2) EEG assessment, with a focus on discharge  
frequency**

**(3) integration of adjunctive data such as neuroimaging  
and metabolic data when available.**

# PERFUSION CT: relative increase of blood volumes and blood flow at left cortical temporo-parietal (post-ictal hyperflow?)



# Ictal-interictal continuum

Considering EEG pattern

Considering clinical picture not justified by structural lesions on neuroimaging

Considering left parietal hyperflow on CT perfusion

**LRDA = ictal pattern** likely configuring a focal NCSE

Start therapy with lacosamide 200mg bolus + 100mgx2 at h. 22.30

15.03.18 h. 8.00

Fp2-AR 7 µV/mm  
F4-AR 7 µV/mm  
C4-AR 7 µV/mm  
P4-AR 7 µV/mm  
O2-AR 7 µV/mm  
Fp1-AR 7 µV/mm  
F3-AR 7 µV/mm  
C3-AR 7 µV/mm  
P3-AR 7 µV/mm  
O1-AR 7 µV/mm  
F8-AR 7 µV/mm  
T4-AR 7 µV/mm  
T6-AR 7 µV/mm  
T8-AR 7 µV/mm  
F7-AR 7 µV/mm  
T3-AR 7 µV/mm  
T5-AR 7 µV/mm  
Fz-AR 7 µV/mm  
Cz-AR 7 µV/mm  
Pz-AR 7 µV/mm  
FLASH 500 µV/mm  
Fp2-F4 7 µV/mm  
F4-C4 7 µV/mm  
C4-P4 7 µV/mm  
F4-AR 7 µV/mm  
P4-O2 7 µV/mm  
C4-AR 7 µV/mm  
Fp1-F3 7 µV/mm  
P4-AR 7 µV/mm  
F3-C3 7 µV/mm  
C3-P3 7 µV/mm  
Fp1-AR 7 µV/mm  
F3-AR 7 µV/mm  
F3-C3 7 µV/mm  
C3-P3 7 µV/mm  
P3-O1 7 µV/mm  
Fp2-F8 7 µV/mm  
F8-T4 7 µV/mm  
T4-T6 7 µV/mm  
T6-O2 7 µV/mm  
Fp1-F7 7 µV/mm  
F8-AR 7 µV/mm  
F7-T3 7 µV/mm  
T4-AR 7 µV/mm  
T3-T5 7 µV/mm  
T6-AR 7 µV/mm  
T5-O1 7 µV/mm  
F7-AR 7 µV/mm  
T3-AR 7 µV/mm  
T3-T5 7 µV/mm  
T5-AR 7 µV/mm  
Fz-AR 7 µV/mm  
Cz-AR 7 µV/mm  
Pz-AR 7 µV/mm  
FLASH 500 µV/mm  
Cz-AR 7 µV/mm  
Pz-AR 7 µV/mm  
FLASH 500 µV/mm  
Fp2-F4 7 µV/mm  
C4-AR 7 µV/mm  
F4-C4 7 µV/mm  
P4-AR 7 µV/mm  
O2-AR 7 µV/mm  
Fp1-F3 7 µV/mm  
P4-AR 7 µV/mm  
F3-C3 7 µV/mm  
C3-P3 7 µV/mm  
Fp1-AR 7 µV/mm  
F3-AR 7 µV/mm  
F3-C3 7 µV/mm  
C3-P3 7 µV/mm  
P3-O1 7 µV/mm  
Fp2-F8 7 µV/mm  
F8-T4 7 µV/mm  
T4-T6 7 µV/mm  
T6-O2 7 µV/mm  
Fp1-F7 7 µV/mm  
F8-AR 7 µV/mm  
F7-T3 7 µV/mm  
T4-AR 7 µV/mm  
T3-T5 7 µV/mm  
T6-AR 7 µV/mm  
T5-O1 7 µV/mm  
F7-AR 7 µV/mm  
T3-AR 7 µV/mm  
T3-T5 7 µV/mm  
T5-AR 7 µV/mm  
Fz-AR 7 µV/mm  
Cz-AR 7 µV/mm  
Pz-AR 7 µV/mm  
FLASH 500 µV/mm  
Cz-AR 7 µV/mm  
Pz-AR 7 µV/mm  
FLASH 500 µV/mm

ARTICLE CLASS OF EVIDENCE

# Can perfusion CT unmask postictal stroke mimics?

A case-control study of 133 patients

Margot G.A. Van Cauwenberge, MD, Sven Dekeyzer, MD, Omid Nikoubashman, MD, Manuel Dafotakis, MD, and Martin Wiesmann, MD

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**Correspondence**  
Dr. Van Cauwenberge  
margot.vancauwenberge@gmail.com

## Conclusion

VPCT can differentiate ictal stroke mimics with hyperperfusion from acute ischemic stroke, but not postictal patients who display perfusion patterns overlapping with ischemic stroke.

