Encefalopatie Acute/Subacute

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Definition

The National Institute of Neurological Disorders and Stroke (NINDS) has described encephalopathy as a term for “any diffuse disease of the brain that alters brain function or structure” and says the “hallmark of encephalopathy is an altered mental status.”

NINDS definition complete

The hallmark of encephalopathy is an altered mental state.

Common neurological symptoms are progressive loss of memory and cognitive ability, subtle personality changes, inability to concentrate, lethargy, and progressive loss of consciousness.

Other neurological symptoms/signs may include myoclonus, nystagmus, tremor, muscle atrophy and weakness, dementia, seizures, and loss of ability to swallow or speak

Categories of encephalopathy

There are 2 distinct categories of encephalopathy: acute and chronic

The 2013 Neurocritical Care Society Practice Update states that "acute encephalopathy is synonymous with acute confusional state, acute organic brain syndrome or delirium...[it] describes the clinical presentation of a global cerebral dysfunction induced by systemic factors."

Delirium vs. acute encephalopathy

Delirium and acute encephalopathy are essentially 2 different terms describing the same condition. Delirium represents the mental manifestation while encephalopathy identifies the underlying pathophysiologic process. This is why the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), classifies acute toxic and metabolic encephalopathic states as delirium and does not use encephalopathy in its definitions.

Classification of encephalopathy

Acute encephalopathies are classified using various schemes

- Clinical classification , according to the neurobehavioral/neurocognitive presentation:
  - delirium and coma in the acute setting
  - vegetative state, minimally conscious state, and cognitive impairment in the subacute and chronic setting
- Anatomic classification:
  - primary brain disorders that result from a direct insult to cerebral tissues (e.g., traumatic brain injury, stroke, brain tumors);
  - secondary brain disorders that result from an extracerebral disturbance (e.g., anoxic-ischemic encephalopathy, hepatic encephalopathy, septic encephalopathy)
- Etiologic classification: infectious and postinfectious encephalitis, inflammatory and immune-mediated encephalopathies, anoxic-ischemic encephalopathy, metabolic and toxic encephalopathies, hepatic encephalopathy, uremic encephalopathy, septic encephalopathy

Table 17.1 Etiologic classification of acquired acute encephalopathies

Vascular
- Ischemic stroke
- Intracerebral hemorrhage
- Subarachnoid hemorrhage
- Cerebral venous thrombosis
- Vasculitis
- Posterior reversible encephalopathy syndrome (PRES)

Trauma
- Focal brain lacerations and contusions
- Extra-axial hematomas
- Diffuse axonal injury

Neoplasm
- Primary or secondary brain tumors

Seizures/status epilepticus
- Generalized seizures (convulsive, nonconvulsive)
- Complex partial seizures

Organ failure
- Cardiac arrest (anoxic-ischemic encephalopathy)
- Respiratory (encephalopathies associated with hypoxia, hyperca
- Hepatic encephalopathy
- Uremic encephalopathy

Metabolic
- Severe electrolyte imbalance
- Hypoglycemia; hyperglycemic states
- Cofactor deficiency (Wernicke encephalopathy)

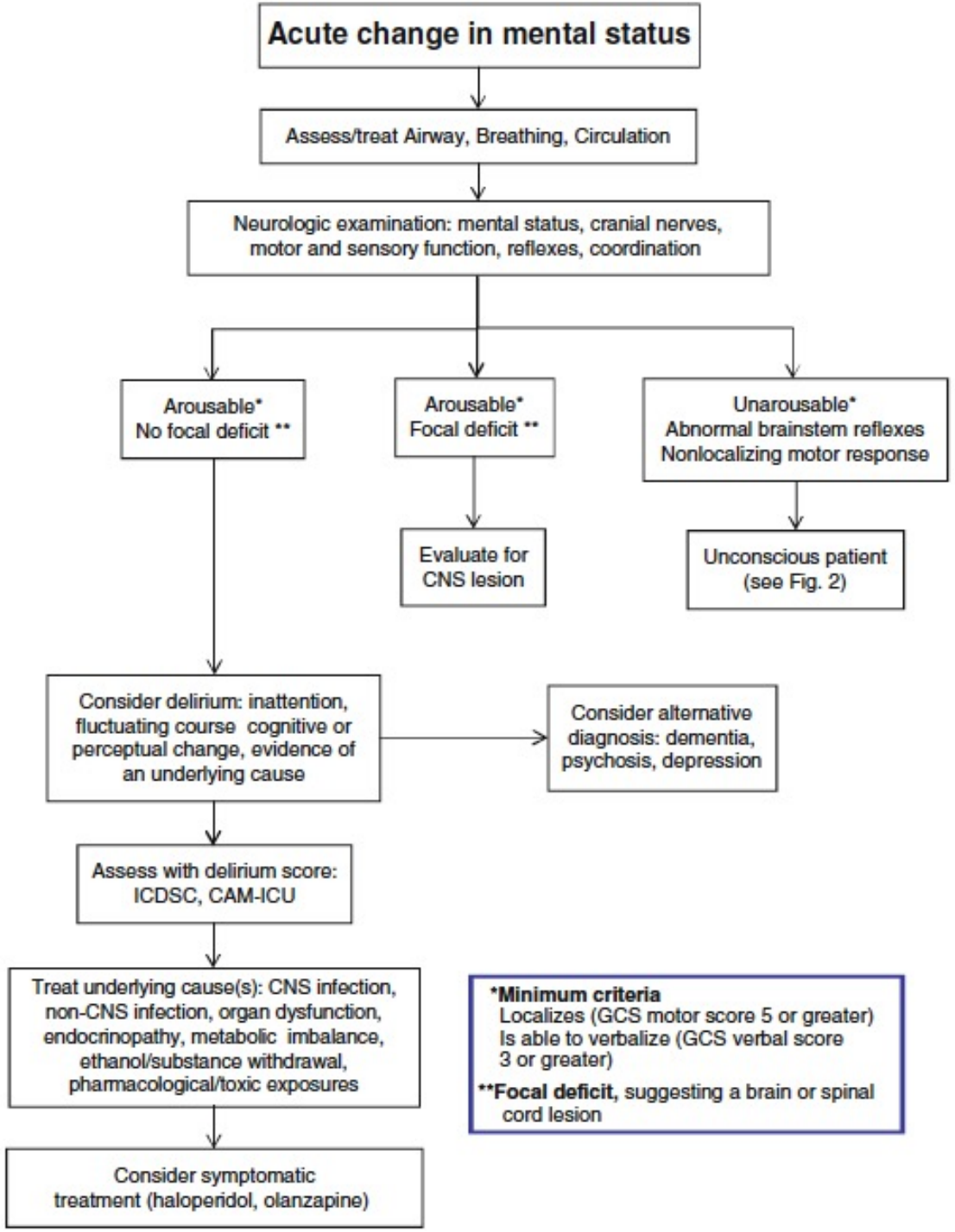
Endocrine
- Hypothalamic and pituitary failure
- Thyroid (myxedema coma, thyrotoxicosis)
- Adrenal (Addison disease)

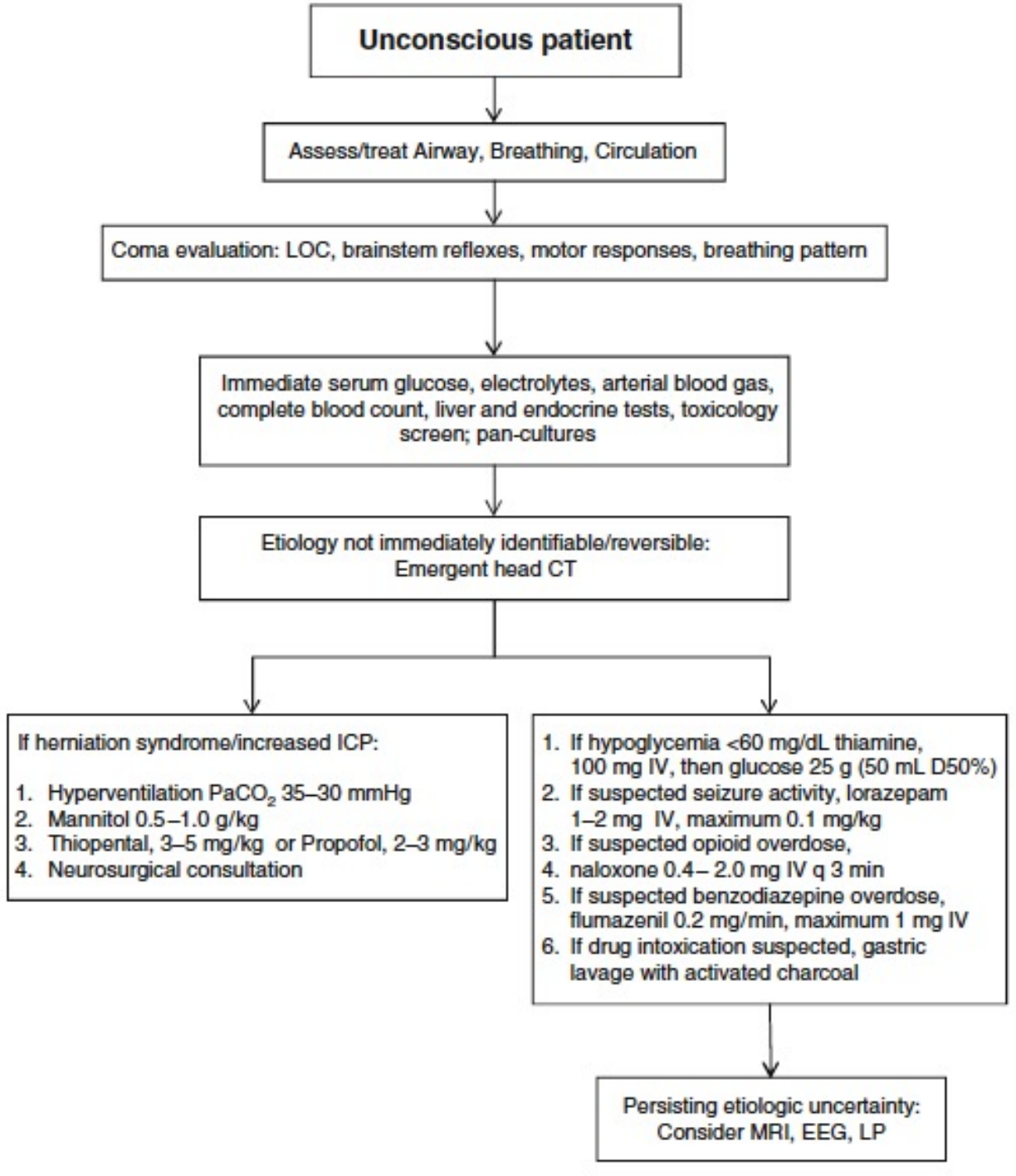
Pharmacologic/toxic
- Prescription medications [opioids, benzodiazepines, barbiturates, tricyclics, neuroleptics, aspirin, SSRIs (selective serotonin reuptake inhibitors), acetaminophen, anticonvulsants]
- Drugs of abuse (opioids, alcohol, methanol, ethylene glycol, amphetamines, cocaine, hallucinogens)
- Environmental exposures (carbon monoxide, heavy metals)

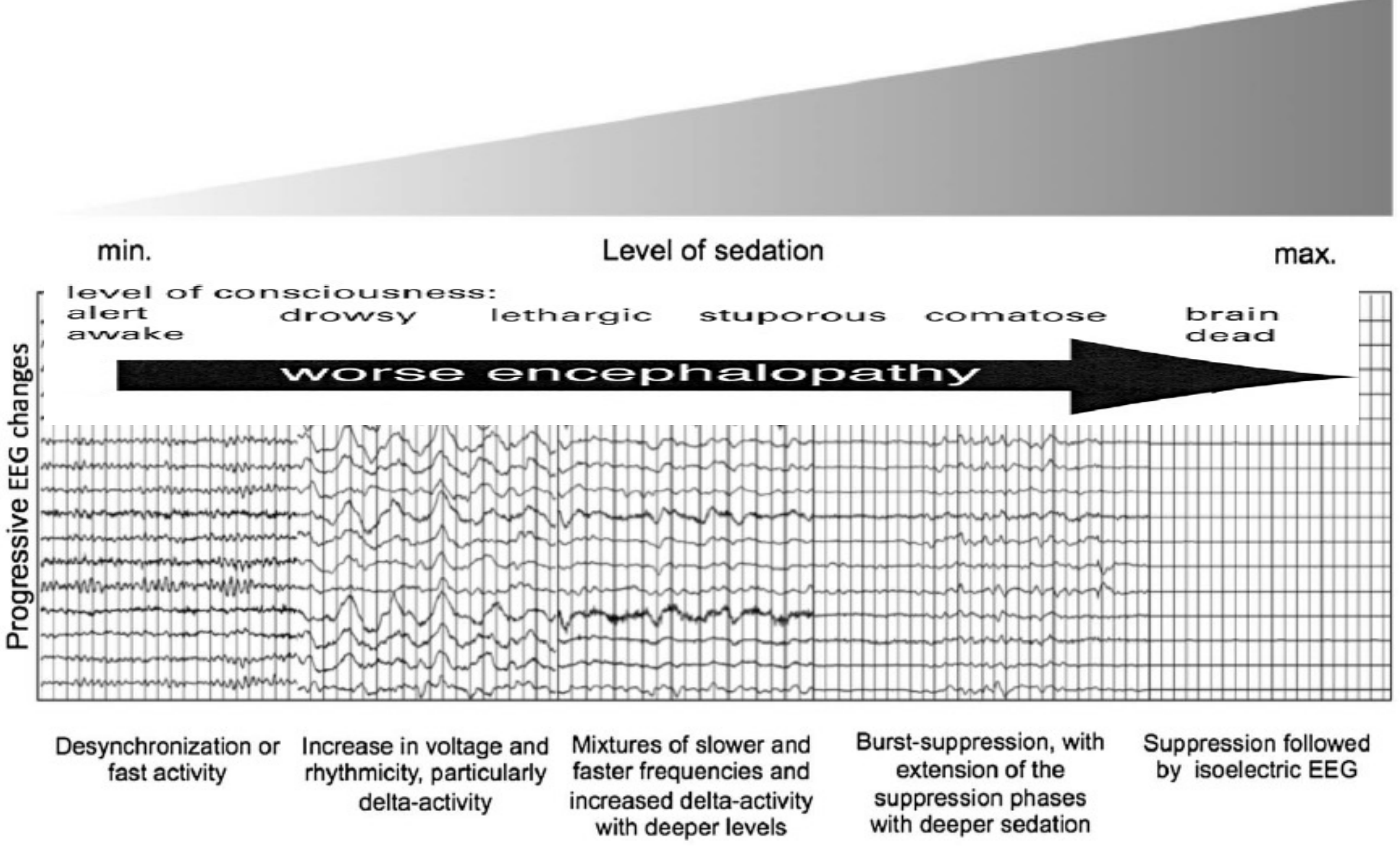
Central nervous system infection
- Meningitis
- Encephalitis

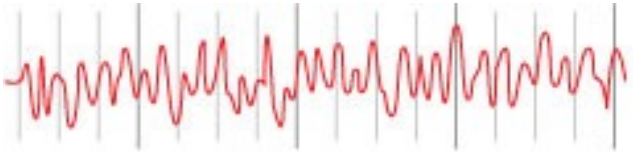
Systemic infection
- Septic encephalopathy

Inflammatory and immune-mediated encephalitis
- Postinfectious encephalitis
- Post-vaccine encephalitis
- Paraneoplastic encephalitis
- Lupus encephalitis
- Neurosarcoidosis
- Acute disseminated encephalomyelitis (ADEM)


Acute change in mental status
Assess/treat Airway, Breathing, Circulation
Neurologic examination: mental status, cranial nerves, motor and sensory function, reflexes, coordination
Arousable*
No focal deficit **
Arousable*
Focal deficit **
Unarousable*
Abnormal brainstem reflexes
Nonlocalizing motor response
Evaluate for CNS lesion
Unconscious patient (see Fig. 2)
Consider delirium: inattention, fluctuating course cognitive or perceptual change, evidence of an underlying cause
Consider alternative diagnosis: dementia, psychosis, depression
Assess with delirium score: ICDSC, CAM-ICU
Treat underlying cause(s): CNS infection, non-CNS infection, organ dysfunction, endocrinopathy, metabolic imbalance, ethanol/substance withdrawal, pharmacological/toxic exposures
Consider symptomatic treatment (haloperidol, olanzapine)
*Minimum criteria
Localizes (GCS motor score 5 or greater)
Is able to verbalize (GCS verbal score 3 or greater)
**Focal deficit, suggesting a brain or spinal cord lesion


Unconscious patient
Assess/treat Airway, Breathing, Circulation
Coma evaluation: LOC, brainstem reflexes, motor responses, breathing pattern
Immediate serum glucose, electrolytes, arterial blood gas, complete blood count, liver and endocrine tests, toxicology screen; pan-cultures
Etiology not immediately identifiable/reversible: Emergent head CT
If herniation syndrome/increased ICP:
1. Hyperventilation $PaCO_2$ 35–30 mmHg
2. Mannitol 0.5–1.0 g/kg
3. Thiopental, 3–5 mg/kg or Propofol, 2–3 mg/kg
4. Neurosurgical consultation
1. If hypoglycemia <60 mg/dL thiamine, 100 mg IV, then glucose 25 g (50 mL D50%)
2. If suspected seizure activity, lorazepam 1–2 mg IV, maximum 0.1 mg/kg
3. If suspected opioid overdose,
4. naloxone 0.4– 2.0 mg IV q 3 min
5. If suspected benzodiazepine overdose, flumazenil 0.2 mg/min, maximum 1 mg IV
6. If drug intoxication suspected, gastric lavage with activated charcoal
Persisting etiologic uncertainty: Consider MRI, EEG, LP

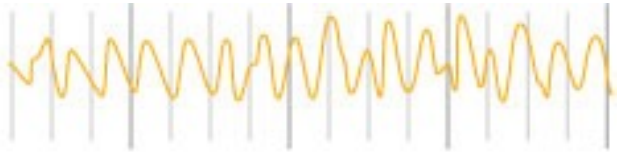

min.
Level of sedation
max.
level of consciousness:
alert
awake
drowsy
lethargic
stuporous
comatose
brain dead
worse encephalopathy
Progressive EEG changes
Desynchronization or fast activity
Increase in voltage and rhythmicity, particularly delta-activity
Mixtures of slower and faster frequencies and increased delta-activity with deeper levels
Burst-suppression, with extension of the suppression phases with deeper sedation
Suppression followed by isoelectric EEG



Beta (14-30 Hz)

Concentration, arousal, alertness, cognition

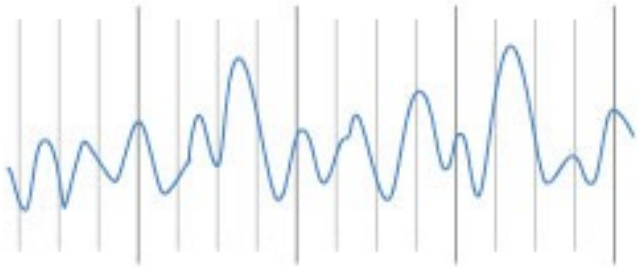
Higher levels associated with Anxiety, disease, feelings of separation, fight or flight



Alpha (8 - 13.9 Hz)

Relaxation, superlearning, relaxed focus, light trance, increased serotonin production

Pre-sleep, pre-waking drowsiness, meditation, beginning of access to unconscious mind

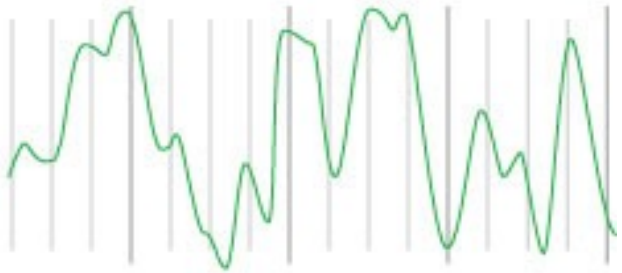


Theta (4-7.9 Hz)

Dreaming sleep (REM sleep)
Increased production of catecholamines (vital for learning and memory), increased creativity

Integrative, emotional experiences, potential change in behavior, increased retention of learned material

Hypnagogic imagery, trance, deep meditation, access to unconscious mind



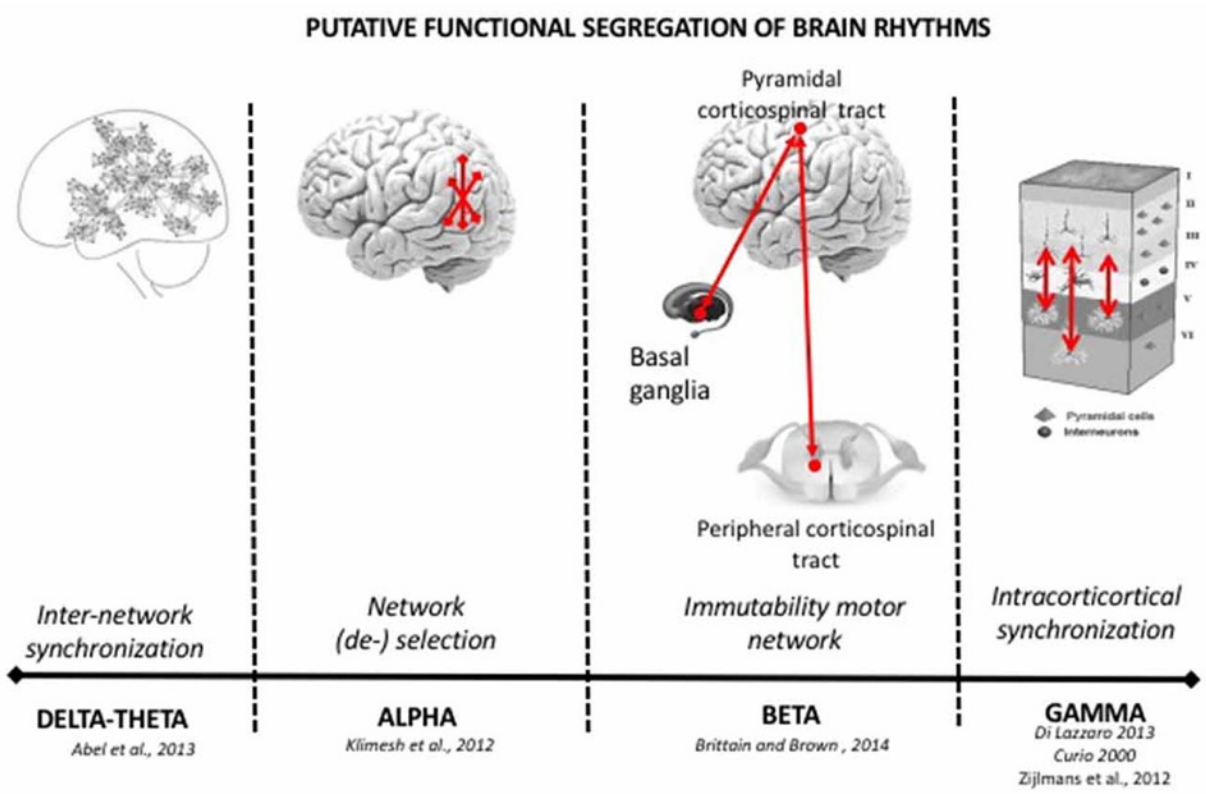
Delta (0.1-3.9 Hz)

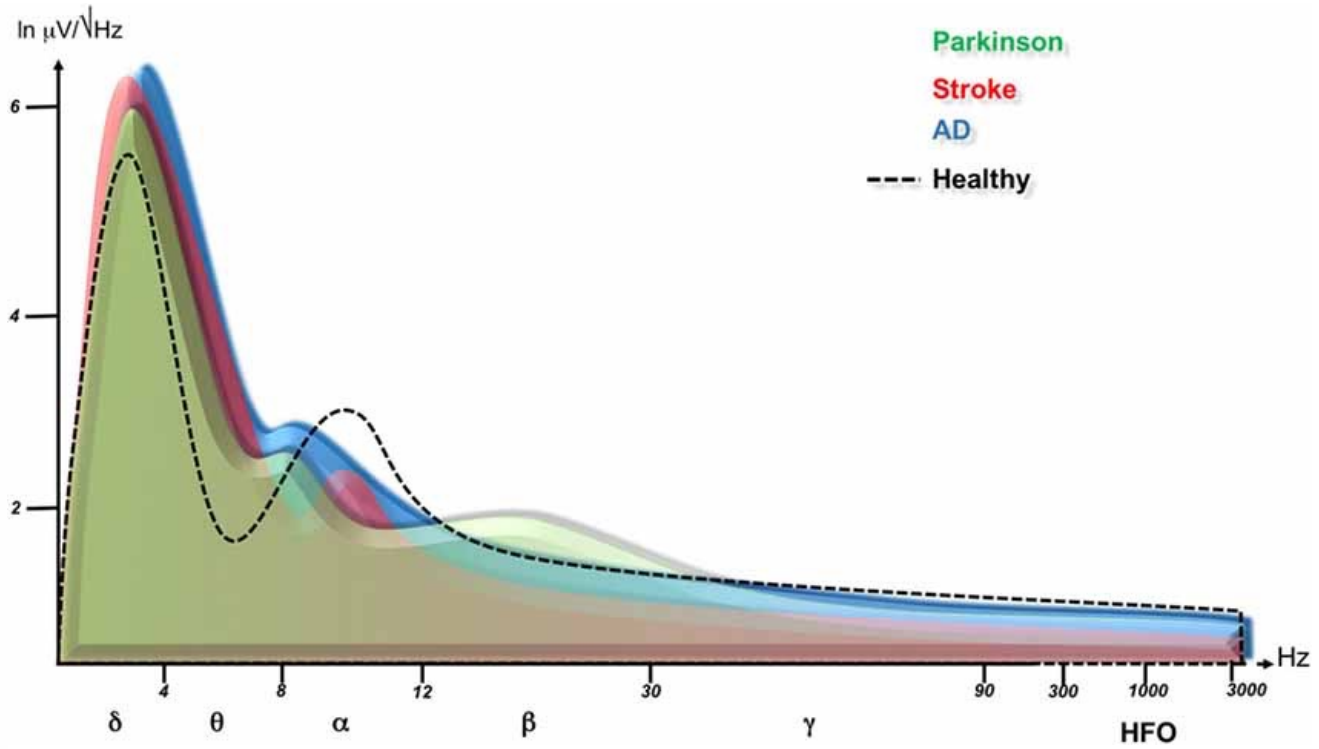
Dreamless sleep
Human growth hormone released

Deep, trance-like, non-physical state, loss of body awareness

Access to unconscious and "collective unconscious" mind,

Assenza et al., 2017





Assenza et al., 2017

TABLE 3. Anatomic Localization and EEG Pattern

Anatomic Localization	EEG Frequency/Pattern
Cortical	Decreased α amplitude Slowing of posterior α background frequency
Subcortical/white matter	Increased polymorphic or arrhythmic δ-activity TWs Frontal intermittent δ-activity
Cortical and subcortical	Slow posterior basic rhythm (background activity) *with* slow-wave intrusion (arrhythmic δ-activity)
Brain stem	Arrhythmic δ-activity, rhythmic δ-activity Impaired arousal patterns Spindle activity

TABLE 5. EEG Frequencies Seen With Different Encephalopathies and Radiologic Features

	α	β	θ	δ
EEG	Mild encephalopathy (e.g., elderly with urinary infection)	Agitation, anxiety	Mild to moderate encephalopathies	Severe encephalopathies
		Benzodiazepines Barbiturates	Dementias	Marked increased intracranial pressure
		Withdrawal states	Systemic infections	Marked white matter disease/ cortical dysfunction (acute)
		Hyperthyroidism		Brain stem dysfunction
MRI/CT	Normal or mild atrophy	Normal	Normal cortical atrophy	Diffuse marked gray/white matter disease

Brain lesions that produce delta waves in the EEG

P. GLOOR, M.D., G. BALL, Ph.D., and N. SCHAUL, M.D.

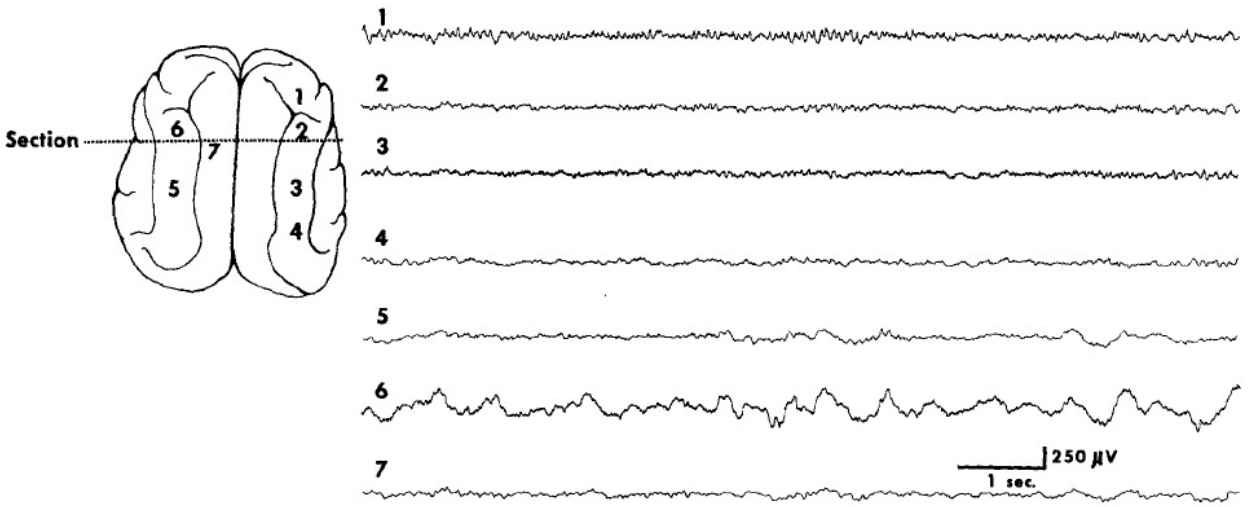
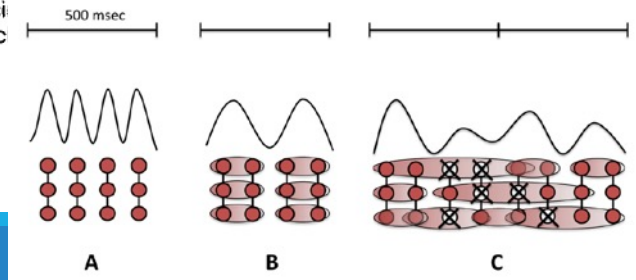
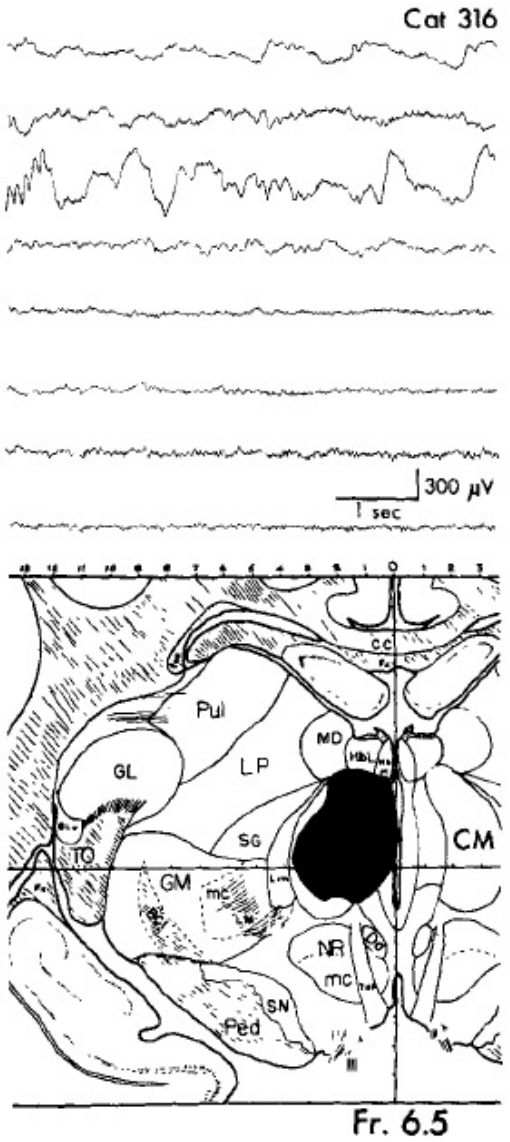


Figure 2. Polymorphic delta activity restricted to an area of cortex overlying a ci
electrode 6). Monopolar EEG. The stippled line indicates the plane along whic
location of the lesion.





Assenza et al., 2015 NRR

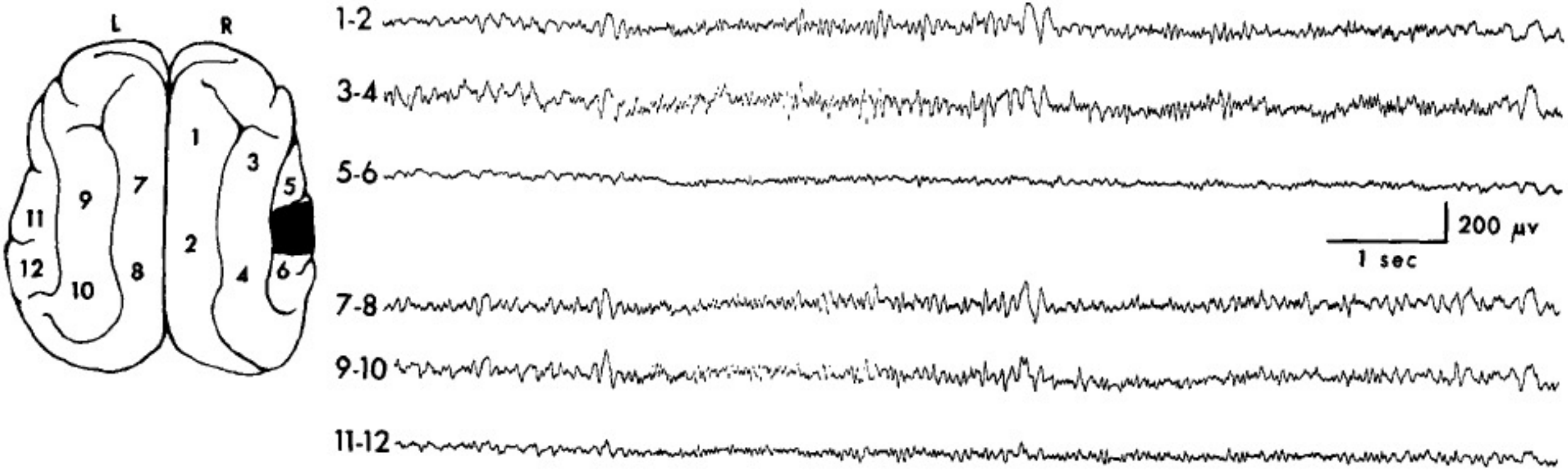
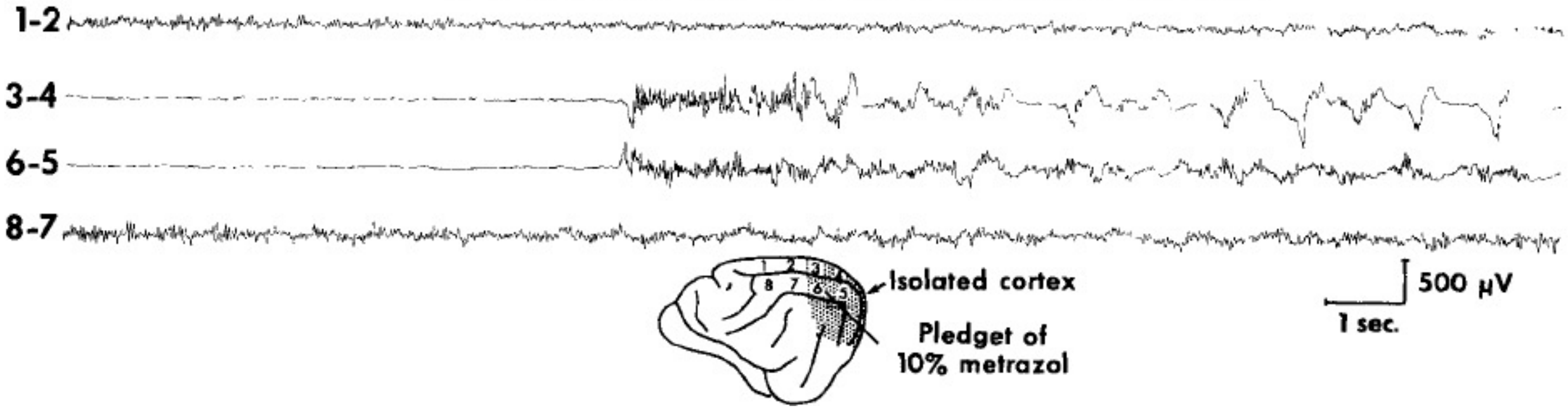


Figure 3. EEG after a purely cortical thermocoagulation lesion of the right middle ectosylvian gyrus. The amplitude of the EEG in the area of the cortical lesion is reduced. Elsewhere the EEG is normal, showing an alternation between an awake and a drowsy pattern.

SLOW WAVES PRODUCED BY ISOLATION OF CORTEX



ACNS Standardized Critical Care EEG Terminology: 2012 version
Reference Chart

Main term 1
G *Generalized* *- Optional : Specify frontally, midline or occipitally predominant*
L *Lateralized* *- Optional: Specify unilateral or bilateral asymmetric* *- Optional: Specify lobe(s) most involved or hemispheric*
BI *Bilateral Independent* *- Optional: Specify symmetric or asymmetric* *- Optional: Specify lobe(s) most involved or hemispheric*
Mf *Multifocal* *- Optional: Specify symmetric or asymmetric* *- Optional: Specify lobe(s) most involved or hemispheric*

Main term 2
PD *Periodic Discharges*
RDA *Rhythmic Delta Activity*
SW *Rhythmic Spike and Wave* *OR* *Rhythmic Sharp and Slow Wave* *OR* *Rhythmic Polyspike and Wave*

Plus (+) Modifier
No +
+F *Superimposed fast activity – applies to PD or RDA only*
+R *Superimposed rhythmic activity – applies to PD only*
+S *Superimposed sharp waves or spikes, or sharply contoured - applies to RDA only*
+FR *If both subtypes apply – applies to PD only*
+FS *If both subtypes apply – applies to RDA only*

REV.

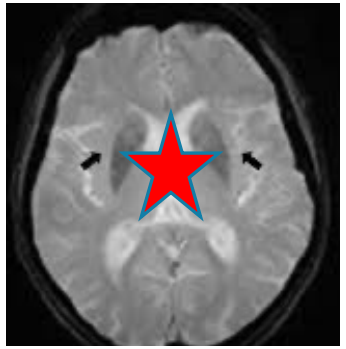
American Clinical Neurophysiology Society's Standardized Critical Care EEG Terminology: 2021 Version

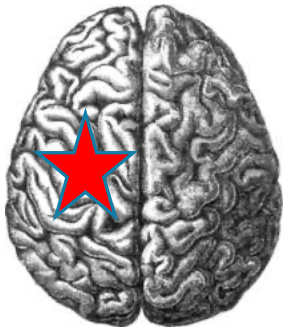
RHYTHMIC AND PERIODIC PATTERNS (RPPs)

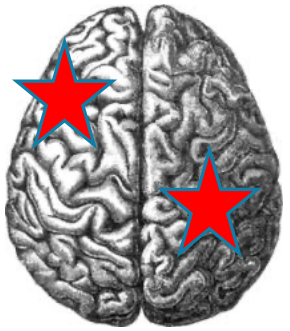
PD= periodic discharges
RDA= rhythmic delta activity

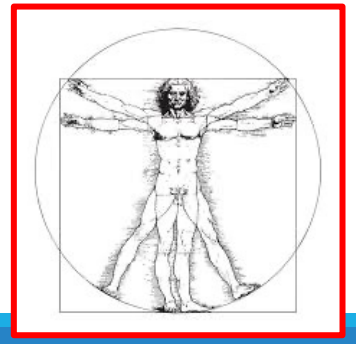
RPPs

- **Generalizzate**
  GRDA /GLPD
- **Lateralizzate**
  LRDA/LPD
- **Bilaterali indipendenti / Multifocali**
  BiRDA /BiPD







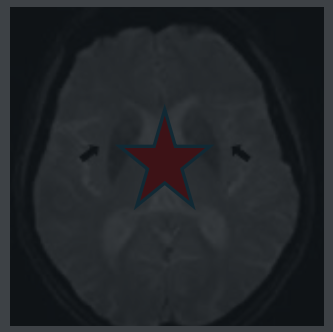


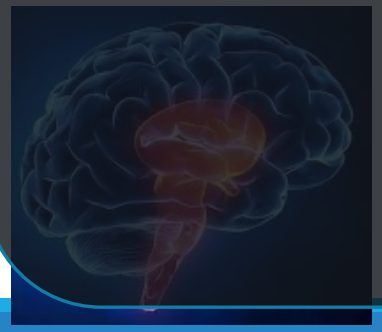
PD= periodic discharges
RDA= rhythmic delta activity

RPPs

Generalizzate

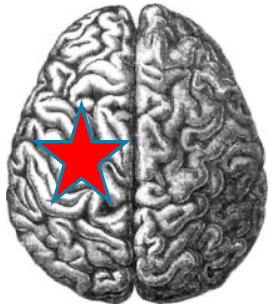
GRDA /GLPD





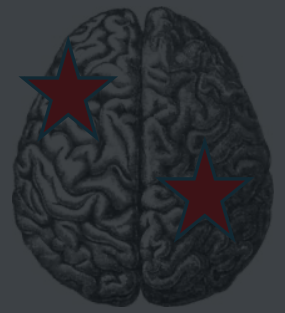
Lateralizzate

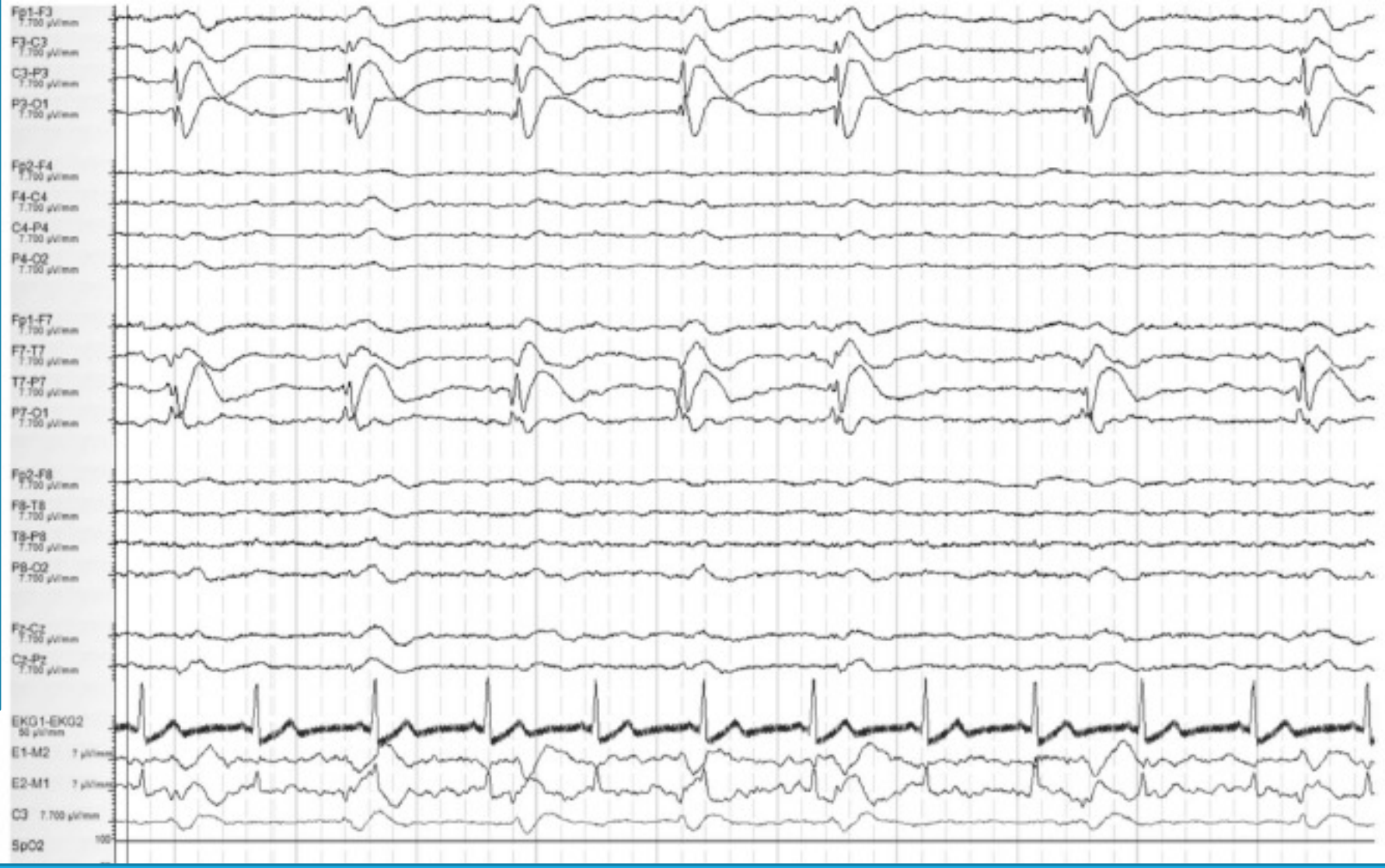
LRDA/LPD



Bilaterali indipendenti / Multifocali

BiRDA /BiLPD




Fp1-F3
F3-C3
C3-P3
P3-O1
Fp2-F4
F4-C4
C4-P4
P4-O2
Fp1-F7
F7-T7
T7-P7
P7-O1
Fp2-F8
F8-T8
T8-P8
P8-O2
Fz-Cz
Cz-Pz
EKG1-EKG2
E1-M2
E2-M1
C3
SpO2

Lateralized periodic discharge (LPD)

- LPD are indicative of an acute unilateral non-specific brain dysfunction

Table 1: Etiology of PLEDs.

Adults	Cases with seizures		Cases without seizures	
CNS infections		11		3
Focal encephalitis[a]	10		2	
Tuberculous meningitis	1		—	
Creutzfeldt–Jakob Disease	—		1	
Cerebrovascular Disease		11		3
Ischemic stroke	8		1	
Intracerebral hemorrhage	—		2	
Cerebral venous occlusion	1		—	
Tuberculous vasculitis	2		—	
Neoplasm		5		—
Undetermined[b]		4		—
Focal cerebral lesion of unknown etiology		2		1
Children		5		—
Progressive neurodegenerative disorders	3		—	
Undetermined[b]	2		—	

N=69

[a] Two with pathologically proven herpes simplex encephalitis; 10 with probable herpes simplex encephalitis (clinically and/or MRI supported); [b] cases with cryptogenic partial epilepsy.

Baykan et al., Seizure 2000

LPD: ictal or interictal?

3 days after GTC status

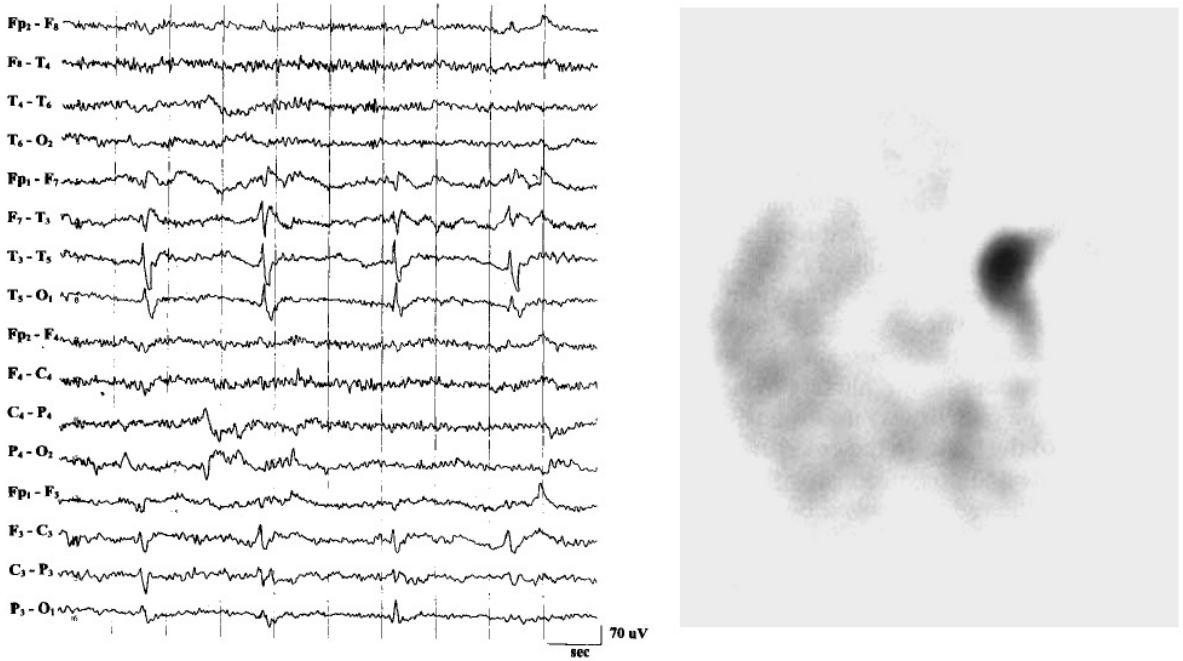


FIG. 2. EEG on day 3, after FDG-positron emission tomography (PET) scan **(right)** showed left-sided temporal-periodic lateralized epileptiform discharges (PLEDs). PLEDs were also evident on days 1 and 2 and on day 3 before the PET scan. The FDG-PET scan image was generated to display the contrast between left temporal lobe and other brain structures. Left mesiotemporal lobe showed markedly increased FDG uptake, compatible with seizure activity. Background cerebral FDG uptake was not altered except in the areas previously damaged by infarct, where uptake was reduced.

after 15 days

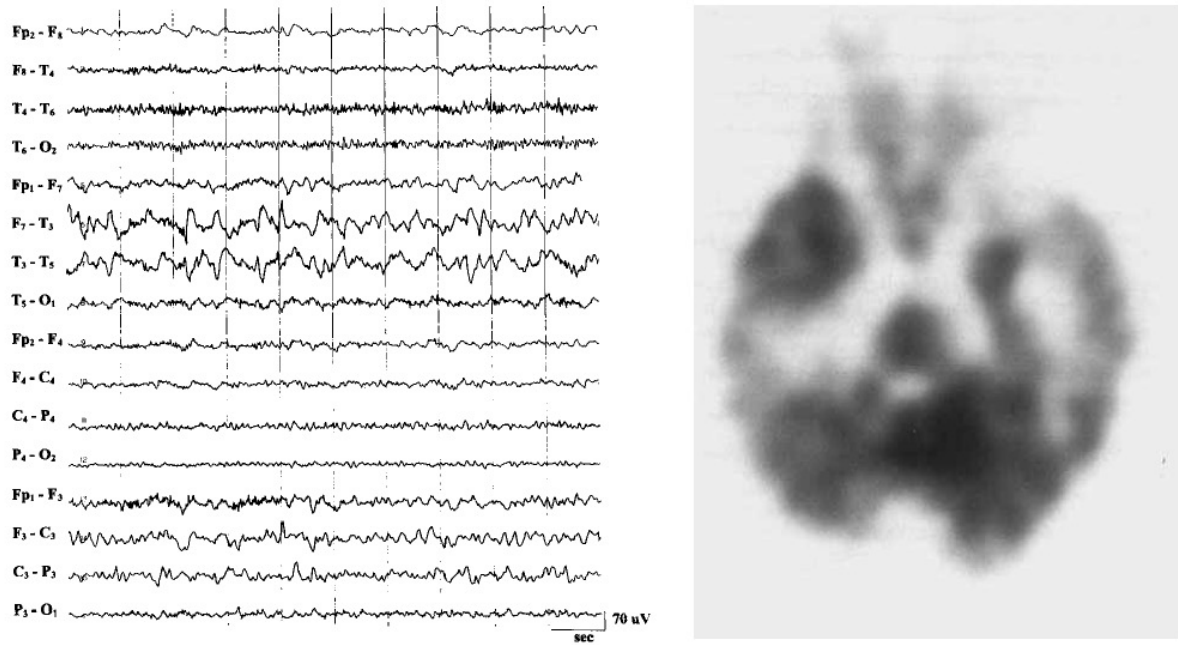


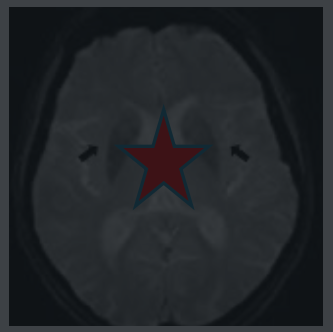
FIG. 3. EEG on day 18 showed considerable irritability, with fluctuating degrees of sharp wave activity, but nonetheless was improved from that of day 3. FDG-positron emission tomography on day 17 demonstrated that left mesiotemporal lobe, although showing glucose metabolism comparable to that of the right side and thus reduced as compared with the previous scan, still displayed more metabolism than the left laterotemporal lobe and therefore remained relatively hypermetabolic.

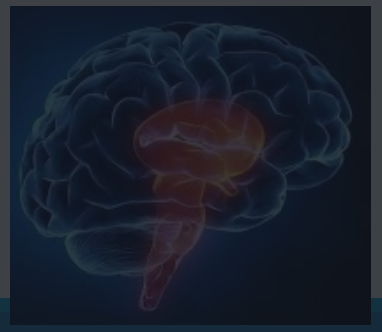
PD= periodic discharges
RDA= rhythmic delta activity

PD/RDA

Generalizzate

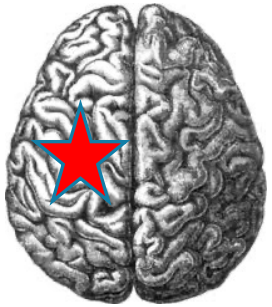
GRDA /GLPD





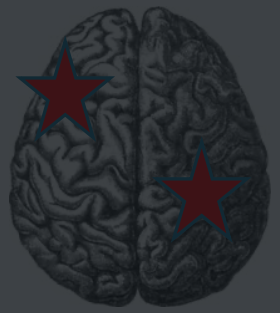
Lateralizzate

LRDA/LPD



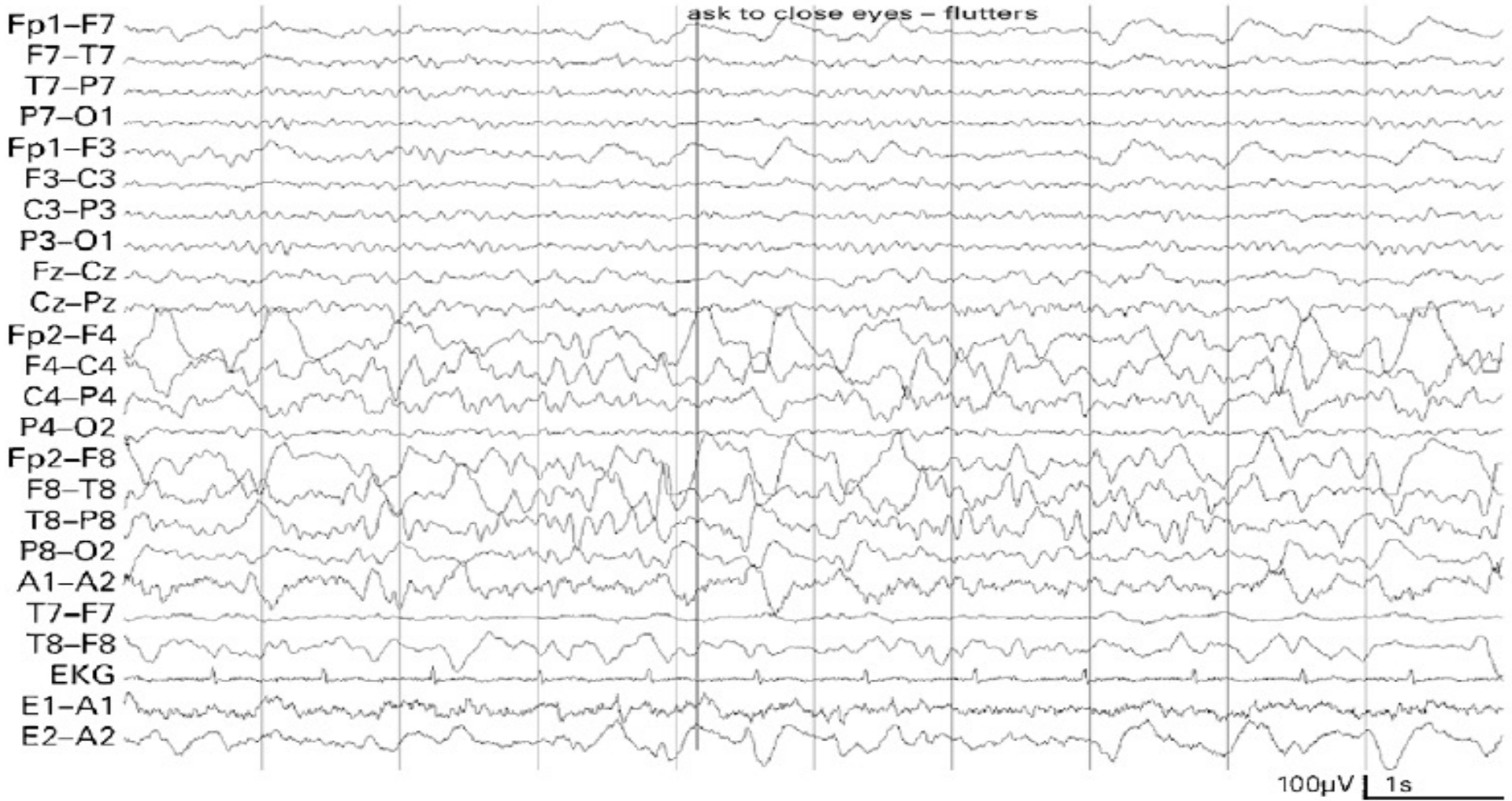
Bilaterali indipendenti / Multifocali

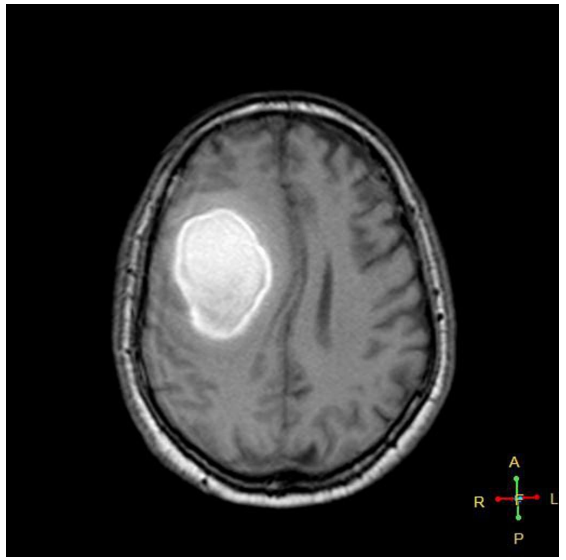
BiRDA /BiLPD



Lateralized rhythmic delta activity (LRDA)

Imaging: nearly all patients with LRDA have a cortical and subcortical focal injury on the side of the rhythmic activity (Gaspard et al., 2013).





RHYTHMIC AND PERIODIC PATTERNS (RPPs)

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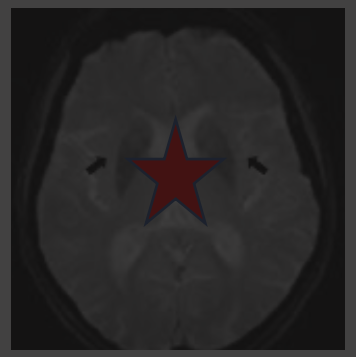
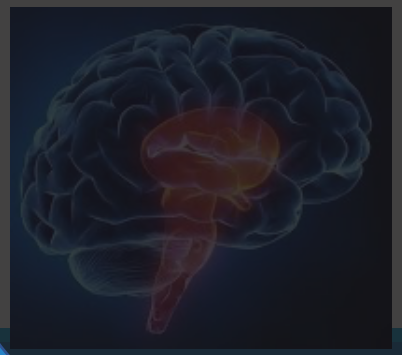
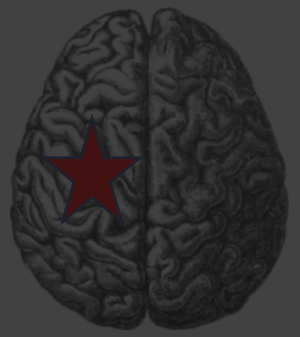
RPPs

Generalizzate
GRDA /GLPD

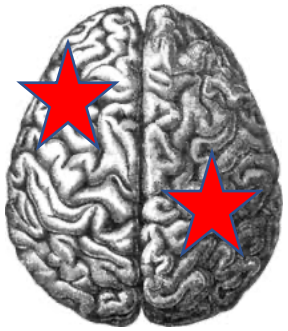




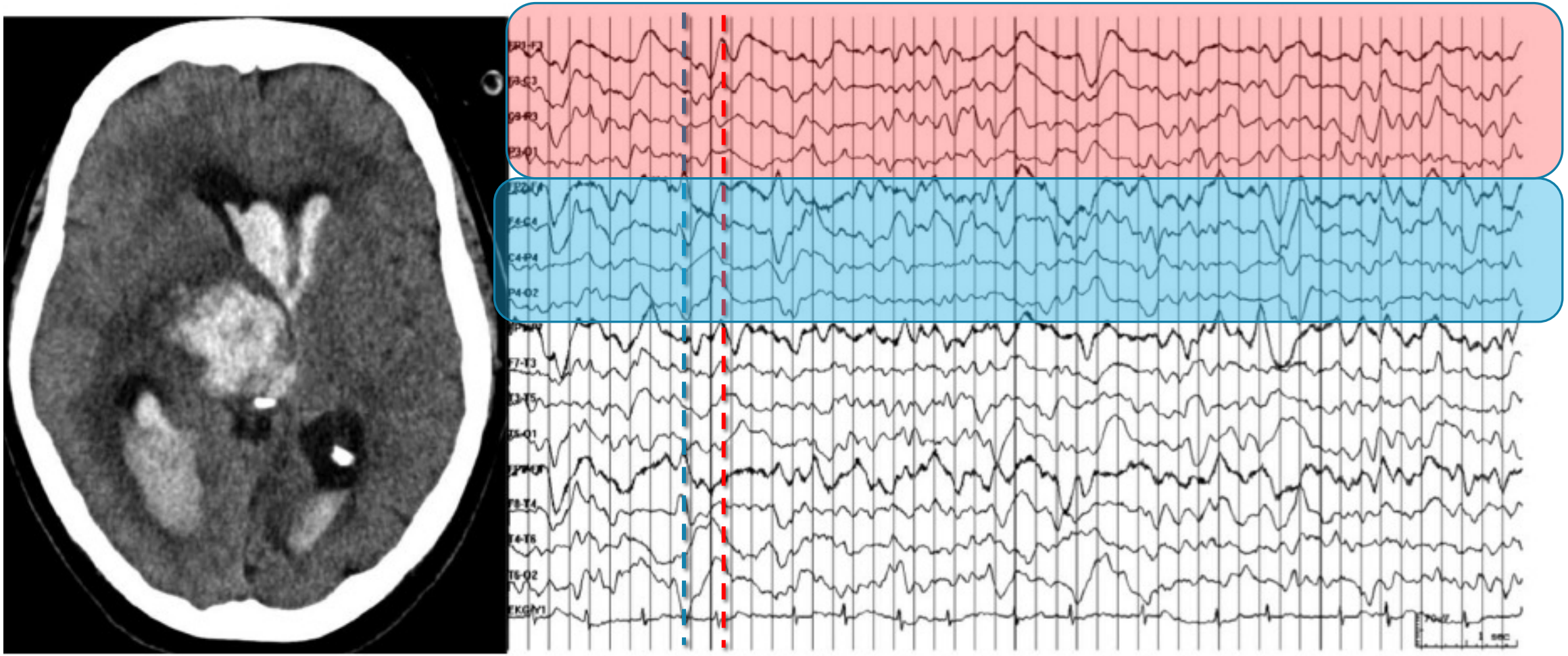
Lateralizzate
LRDA/LPD



Bilaterali indipendenti / Multifocali
BiRDA /BiPD



Bilateral independent periodic discharges (BIPDs)



Bilateral independent periodic discharges (BIPDs)

Imaging

In one study, focal findings on imaging were less common in patients with BIPDs (25%) compared to LPDs (74%) (Pedersen et al., 2013)

Outcome:

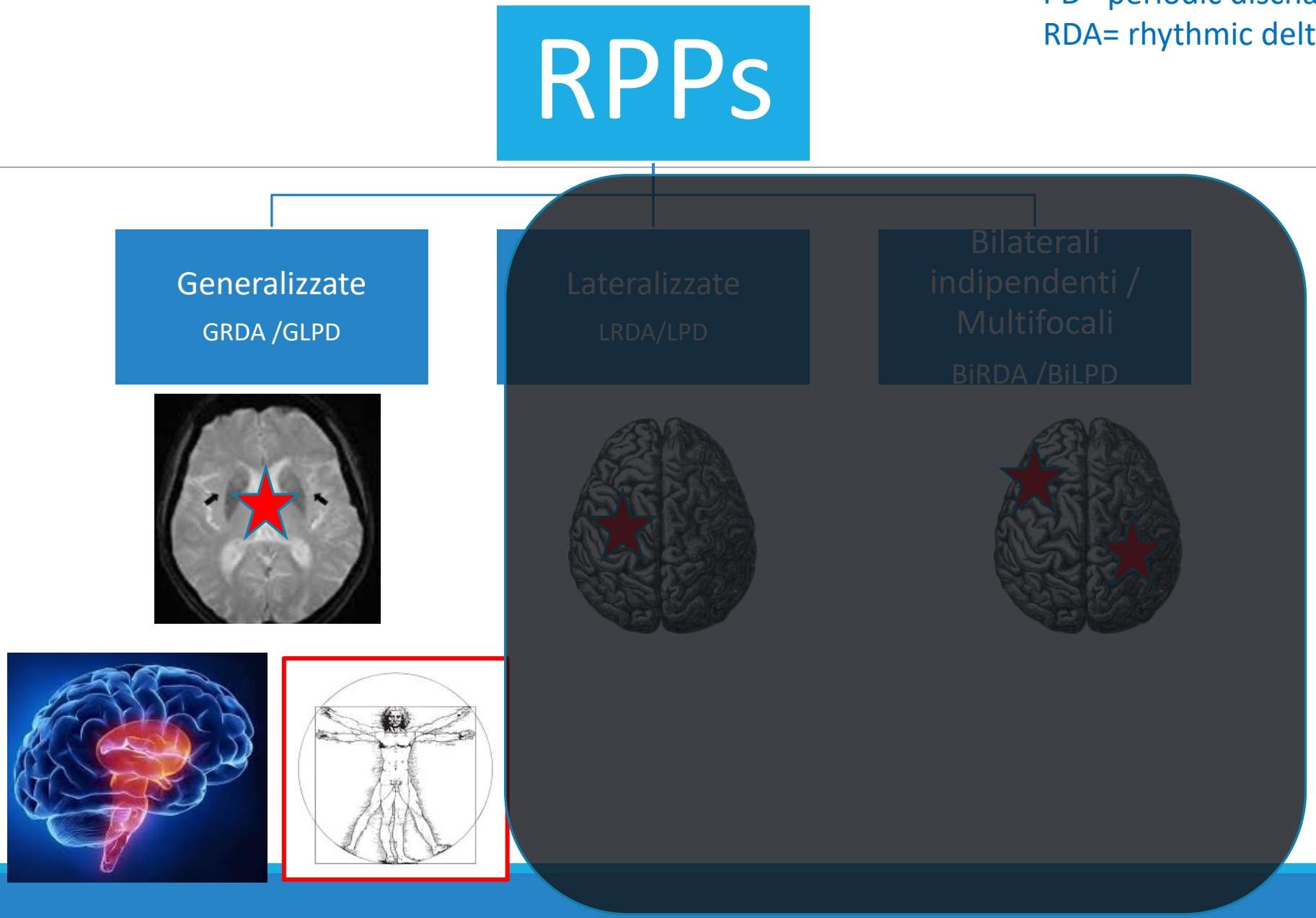
BIPDs have been thought of as a marker of more severe disease and as an indicator of worse prognosis than LPDs.

The reported mortality ranges from 39–100% (Pedersen et al., 2013; San juan Orta et al., 2009).

The largest comparisons of BIPDs to LPDs have shown higher mortality in BIPDs. De la Paz et al. found a 61% mortality in 18 patients with BIPDs, more than twice the 29% found in the 45 patients with LPDs(de la Paz and Brenner, 1981).

RHYTHMIC AND PERIODIC PATTERNS (RPPs)

PD= periodic discharges
RDA= rhythmic delta activity



GPD (Generalized periodic discharges)

Definition

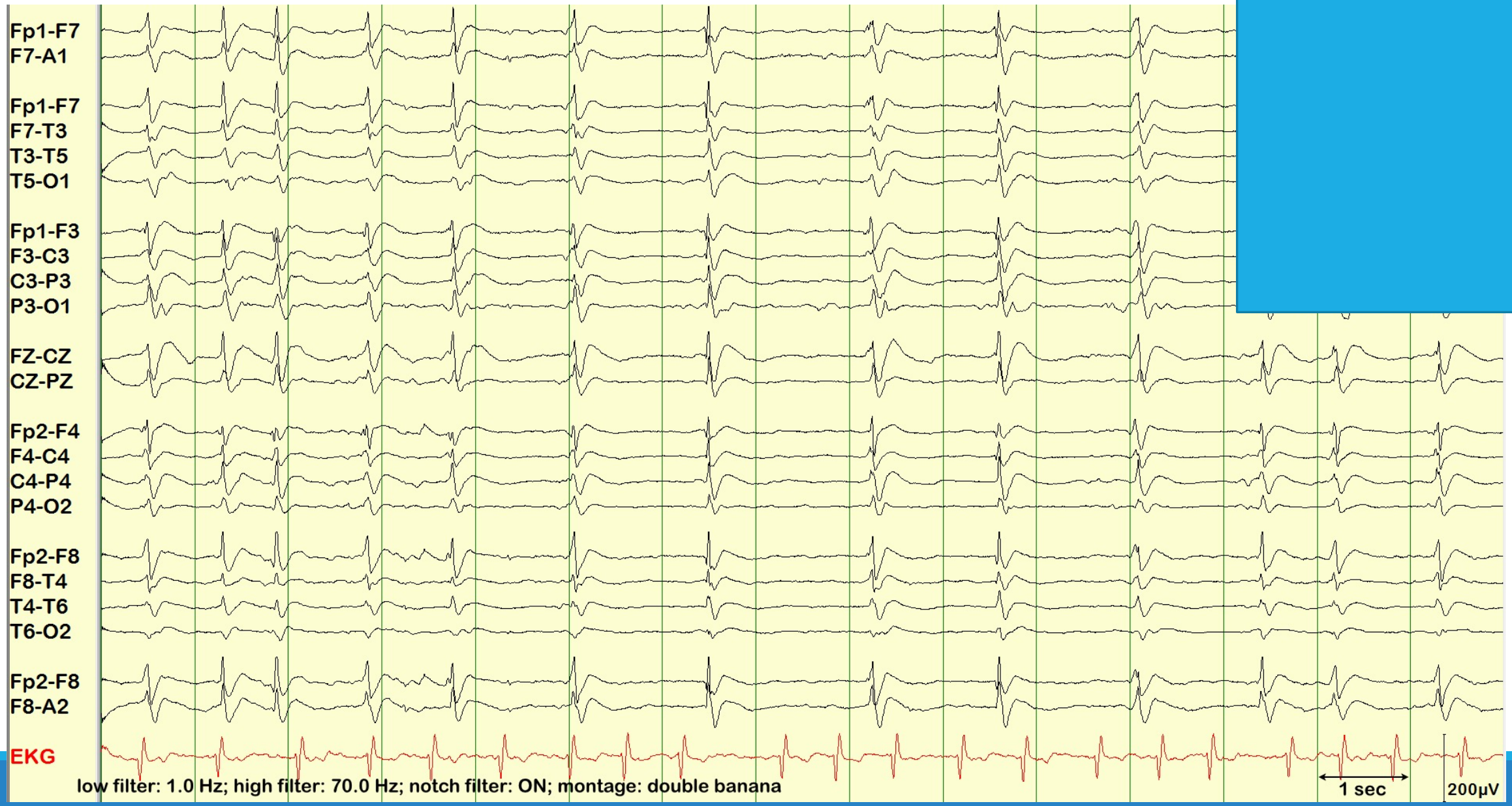
GPDs at first appear closely related to LPDs and BIPDs;

however, metabolic illnesses more commonly give rise to GPDs.

GPDs are bilaterally synchronous, repetitive discharges (often with a sharp or spike morphology), typically with amplitudes >100 uV, repeating at regular intervals at up to 3 per second, with a clear period between adjacent discharges (Hirsch et al., 2013)

Prevalence

A large review of 3064 patients undergoing cEEG found GPDs in 138 (4.5%) (Foreman et al., 2012); other studies have found a much lower prevalence, from 0.8–1.8% (Lee et al., 2016; Swisher et al., 2015). GPDs often coexist with LPDs.


Fp1-F7
F7-A1
Fp1-F7
F7-T3
T3-T5
T5-O1
Fp1-F3
F3-C3
C3-P3
P3-O1
FZ-CZ
CZ-PZ
Fp2-F4
F4-C4
C4-P4
P4-O2
Fp2-F8
F8-T4
T4-T6
T6-O2
Fp2-F8
F8-A2
EKG
low filter: 1.0 Hz; high filter: 70.0 Hz; notch filter: ON; montage: double banana
1 sec
200µV

Triphasic GPD (Generalized Periodic Discharge):

there are three principal phases: the main deflection being downward, representing a surface positive change. This dominant phase is usually preceded by a low-amplitude (often rounded or even absent) negative deflection and followed by a long, slow, broad slow-rising deflection, giving the entire complex a triphasic contour.

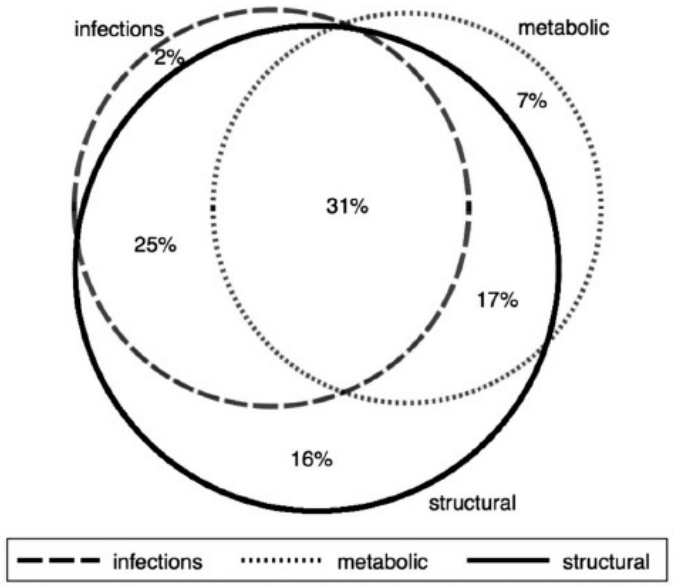


FIG. 3. Proportional distribution of infections, metabolic derangements, and structural brain abnormalities in 105 encephalopathic patients with triphasic waves. Adapted with permission from Sutter et al. (2013*b*).

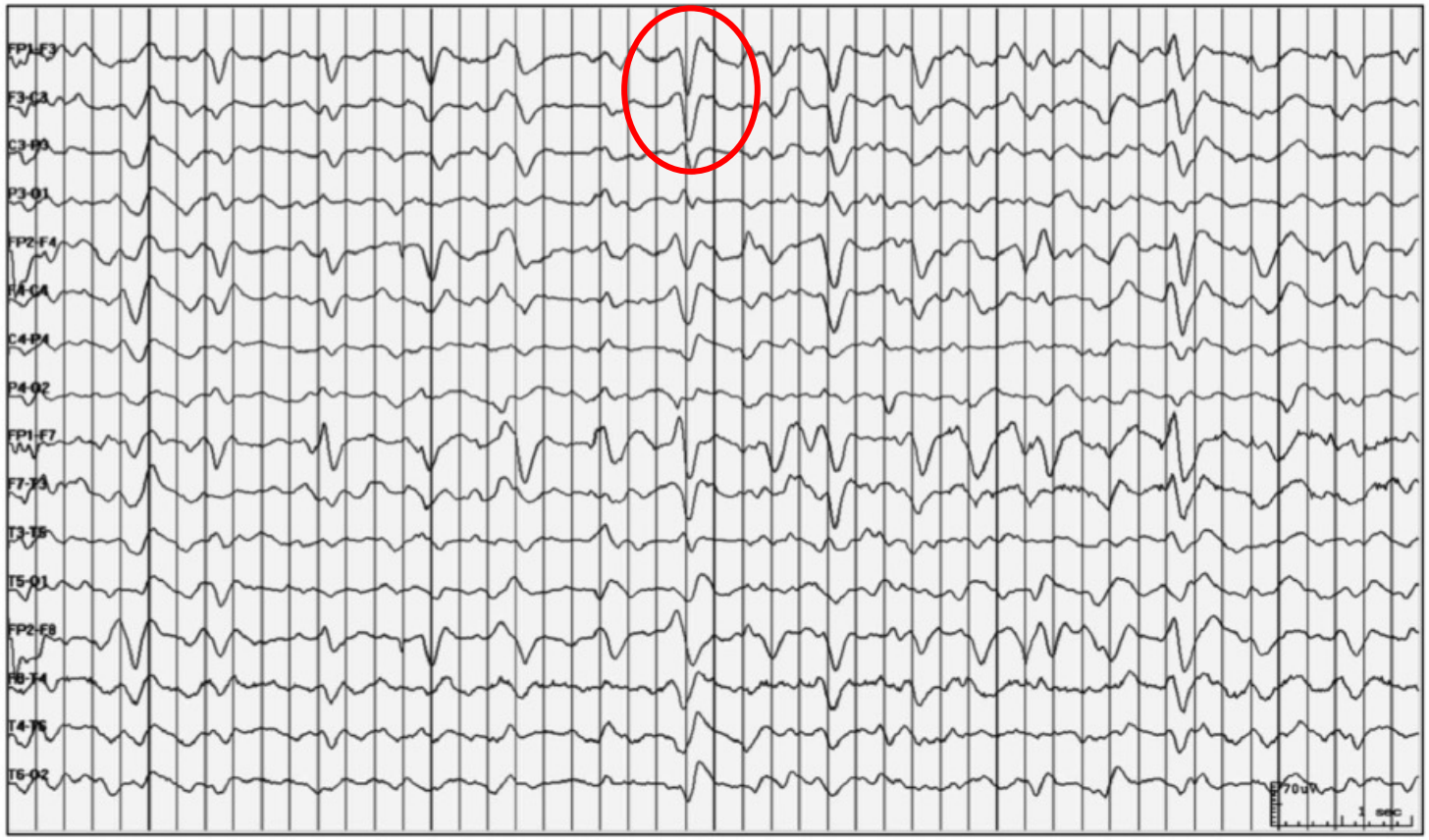
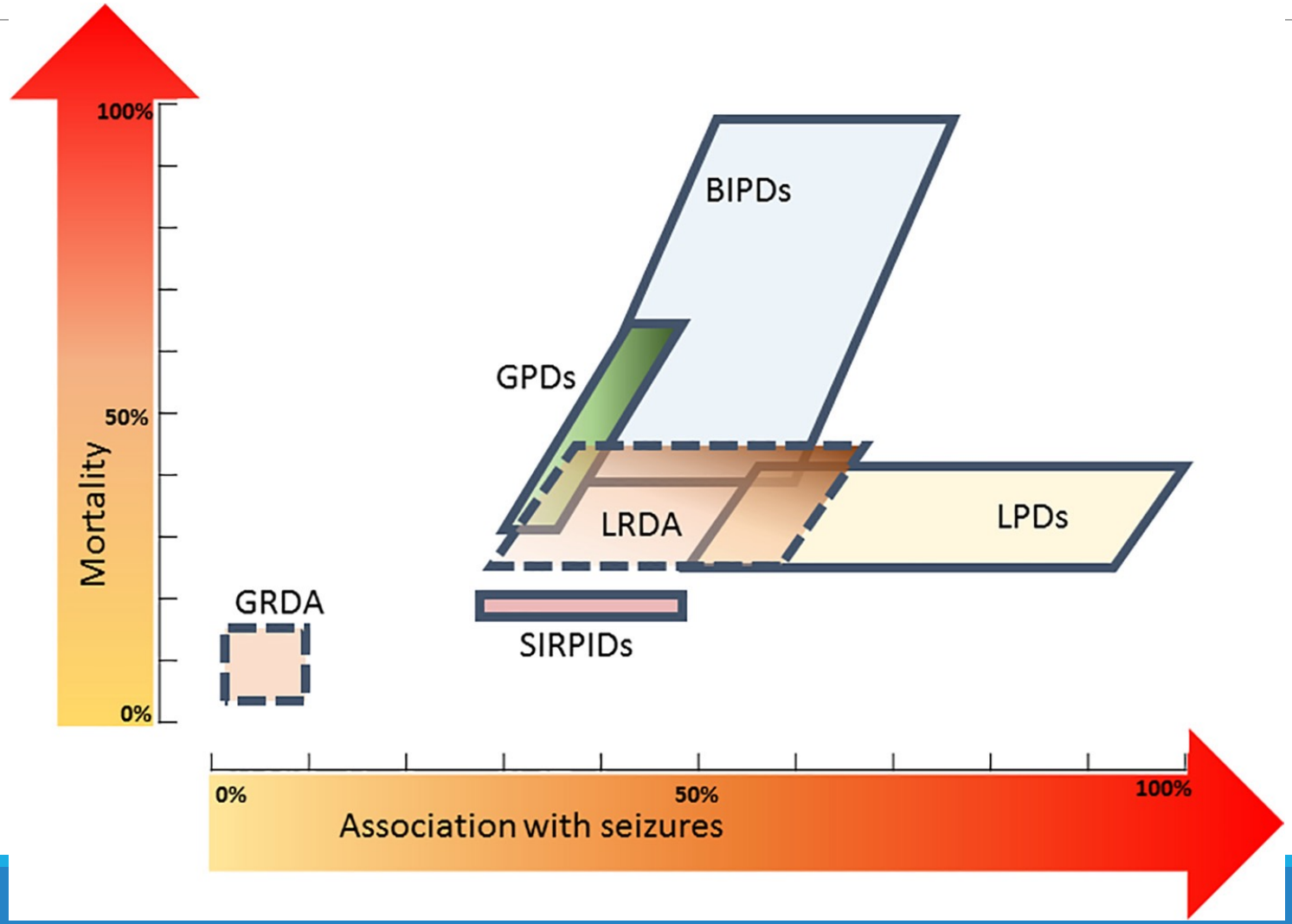


FIG. 5. Triphasic waves in acute encephalopathy. Generalized slowing of background activity with frequencies in the theta (4–
Hz) and delta (<4 Hz) range and bilateral high-voltage (70–100 μV) triphasic waves with a frontocentral maximum and an anterior-posterior or posterior-anterior shift.

Population of the ictal-interictal zone: The significance of periodic and rhythmic activity

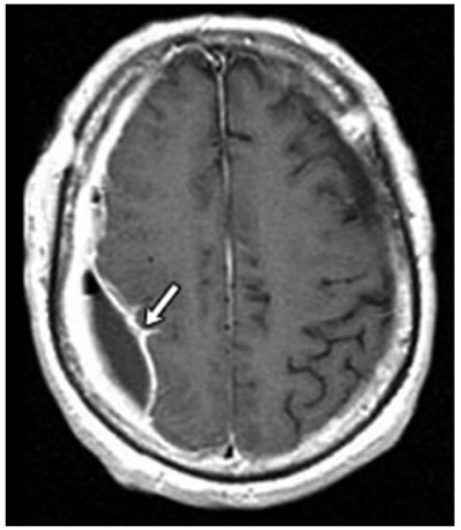
Emily L. Johnson, Peter W. Kaplan, 2017 Clinph practice



Encefalopatie infettive

ENCEFALOPATIE DI ORIGINE INFETTIVA

Empiema Subdurale



PLEDs Evolving into Focal Seizure

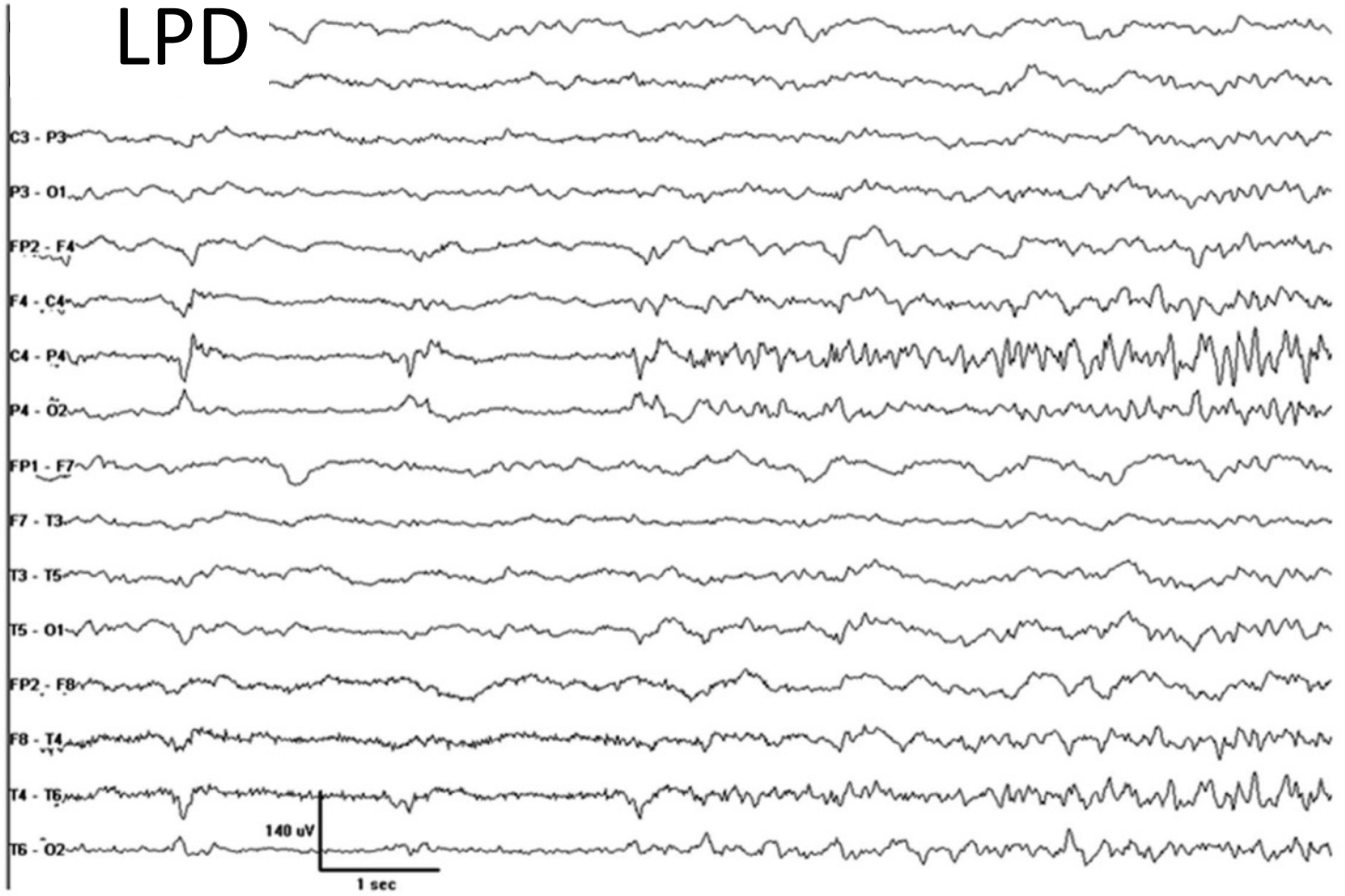


FIG. 2. Seizure evolving beneath the subdural empyema. This is often caused by cortical vein thrombosis.

Encefalite Erpetica

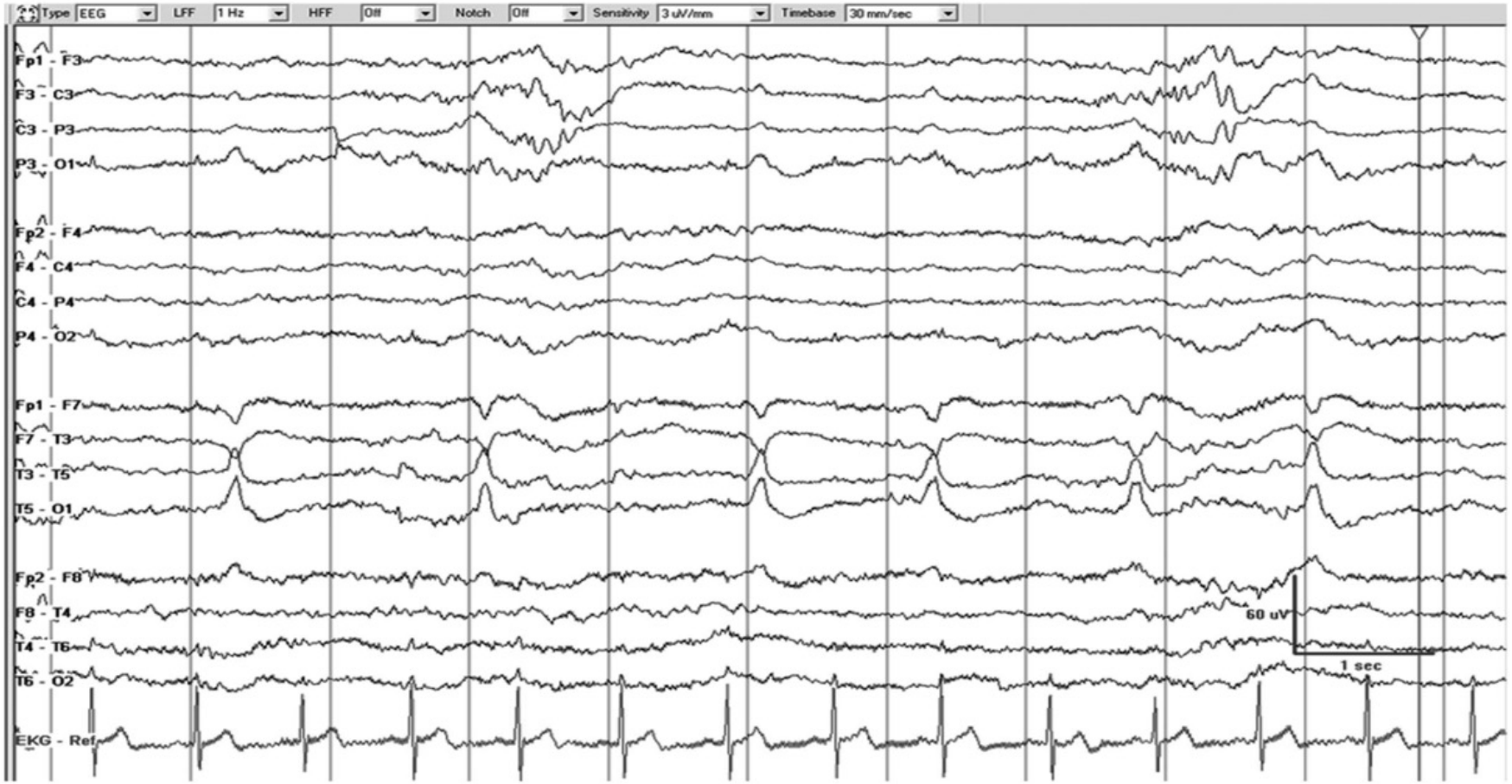
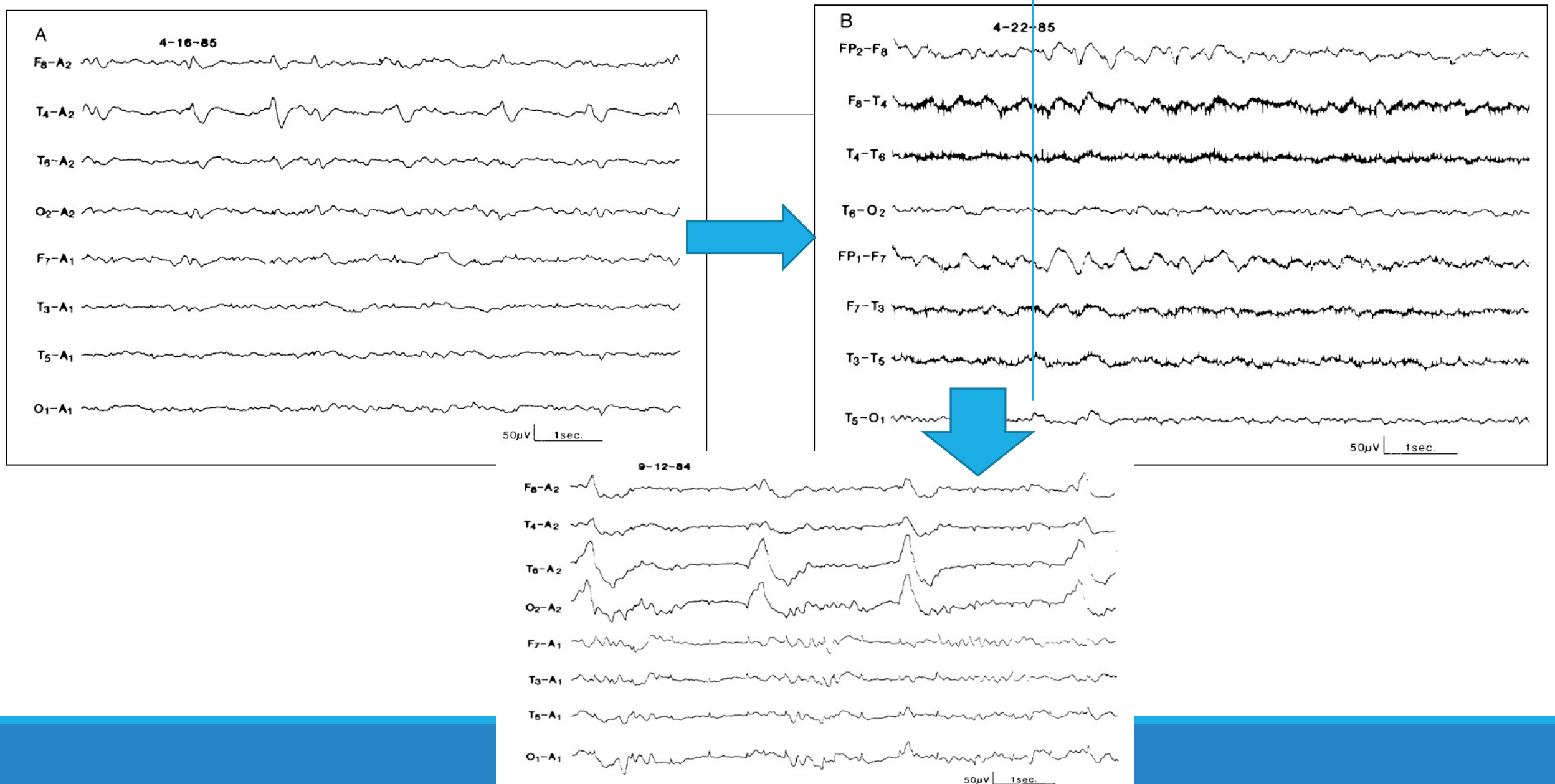


FIG. 3. Herpes simplex encephalitis. Quasiperiodic lateralized periodic discharges maximally expressed in the left midtemporal region occur every one to two seconds.

Encefalite erpetica a evoluzione mortale



Encefalite erpetica con risoluzione

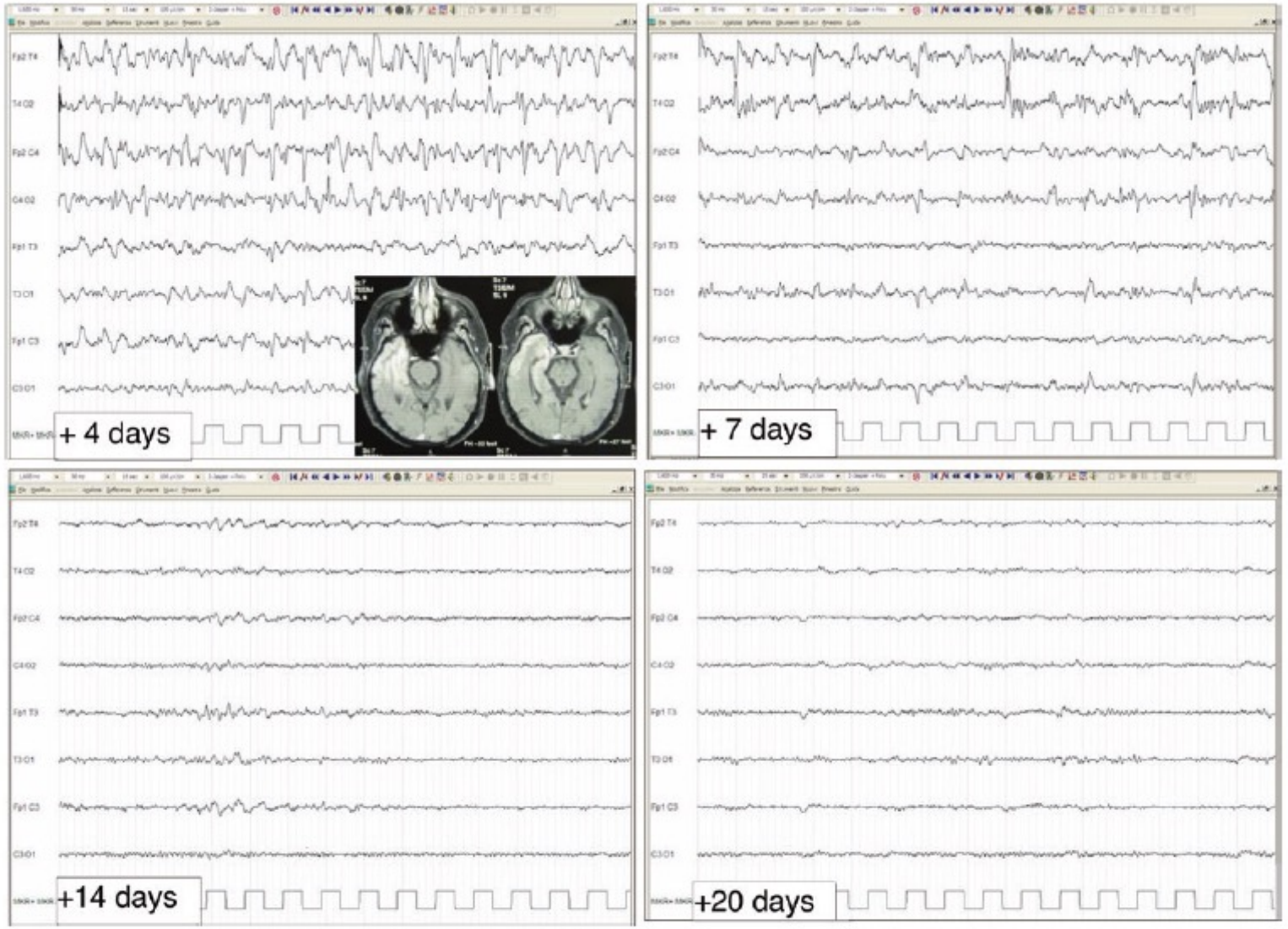


Fig. 39.4 EEG evolution of a 64-year-old male patient with right temporal herpetic encephalitis. Four days after symptoms onset (fever, confusion and focal seizures), EEG showed right quasi-periodic spikes and sharp waves, with a tendency to contralateral transmission. After 7 days, the abnormalities were less represented, maintaining a quasi-periodic recurrence. After 14 days, sporadic slow sequences were only evident, especially in the contralateral hemisphere. After 20 days, EEG normalization was observed. MRI showed in the small boxes.

Mecarelli 2019

Electroencephalography for diagnosis and prognosis of acute encephalitis ☆

Raoul Sutter [a,b,c,d,*], Peter W. Kaplan [b], Mackenzie C. Cervenka [e], Kiran T. Thakur [f], Anthony O. Asemota [f], Arun Venkatesan [f], Romergryko G. Geocadin [a,f]

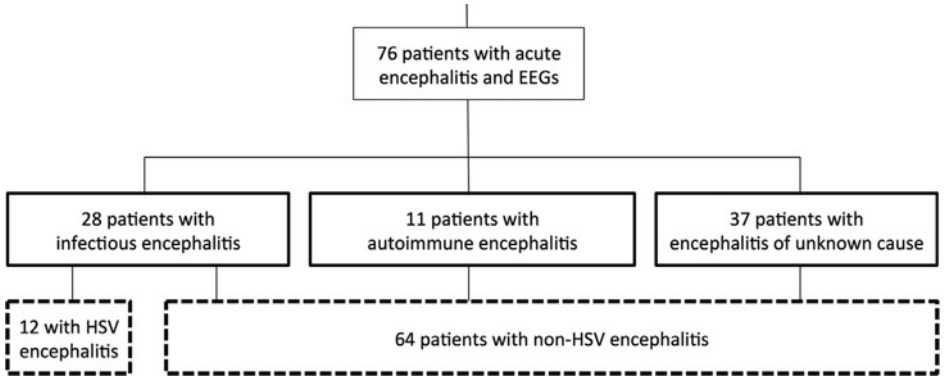


Table 2
Comparisons of clinical and EEG characteristics between HSV and non-HSV encephalitis ($n = 76$).

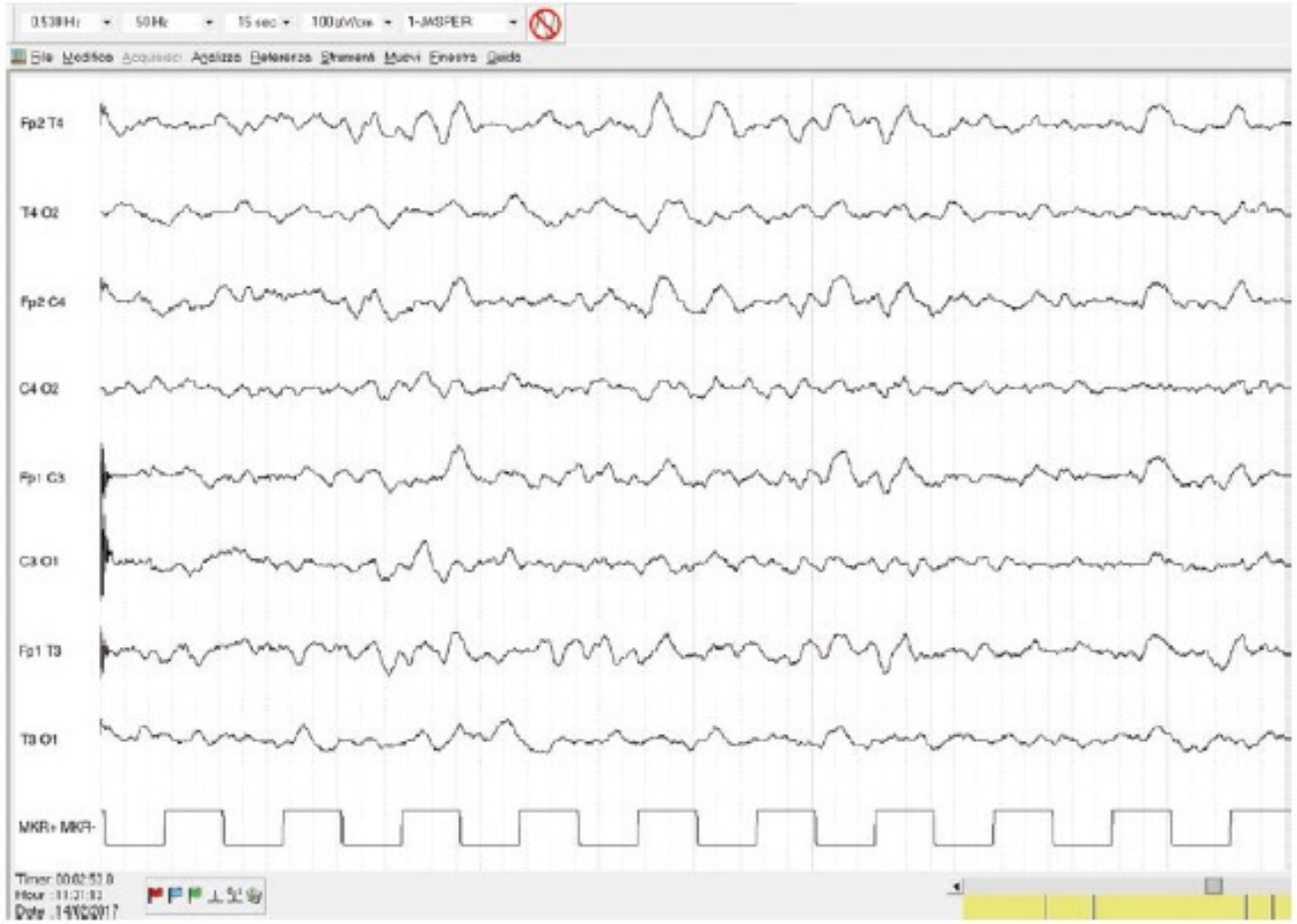
	HSV encephalitis ($n = 12$)		Non-HSV encephalitis ($n = 64$)		p-Value[#]
Demographics					
Gender, male (n, %)	4	33.3	35	54.7	0.217
Age, years (mean, SD)	57.9 ± 12.1		48.8 ± 17		0.079
Clinical features					
GCS on admission (median, IQR)	12	9–14	10	6–13	0.071[§]
Comatose on admission, GCS ⩽8 (n, %)	2	16.7	28	43.8	0.11
Charlson comorbidity index (median, IQR)	3	2–4	1.5	0–5	0.265[§]
Global cerebral edema (n, %)	2	16.7	8	12.7	0.657
Immunosuppression (n, %)	4	33.3	16	25	0.722
Mechanical ventilation (n, %)	5	41.7	42	65.6	0.194
EEG characteristics					
Background frequency ranges (n, %)					0.259
Alpha	8	66.7	24	37.5	
Alpha/theta	1	8.3	5	7.8	
Theta	2	16.7	15	23.4	
Theta/delta	0	0	14	21.9	
Delta	1	8.3	6	9.4	
Focal slowing (n, %)					0.129
Frontal	1	10	3	4.7	
Temporal	2	20	3	4.7	
Central	0	0	3	4.7	
Parietal	0	0	1	1.6	
Occipital	2	20	0	0	**0.017**
Episodic transients (n, %)					
FIRDA	0	0	2	3.1	
TWs	0	0	4	6.3	
PDs	3	30.0	3	4.7	**0.029**
Epileptic activities (n, %)					
Seizures	1	10	3	4.7	
Status epilepticus	1	10	5	7.8	
Nonreactive EEG background activity (n, %)	1	10	14	22.6	0.676

Table 3
Comparisons of early clinical and EEG characteristics between survivors and non-survivors with acute encephalitis ($n = 76$).

	Survivors ($n = 61$)		Non-survivors ($n = 15$)		p-value[a]
Demographics					
Gender, male (n, %)	29	47.5	10	66.7	0.252
Age, years (mean, SD)	49.1 ± 16.4		54.7 ± 17.3		0.248
Clinical features					
GCS on admission (median, IQR)	11	7–14	6	3–11	**0.01**[§]
Comatose on admission, GCS ⩽8 (n, %)	20	32.8	10	66.7	**0.021**
Charlson comorbidity index (median, IQR)	2	0–4	4	0–6	0.103[§]
Global cerebral edema (n, %)	5	8.3	5	33.3	**0.023**
Immunosuppression (n, %)	14	23	6	40	0.201
Mechanical ventilation (n, %)	33	54.1	14	93.3	**0.006**
EEG characteristics					
Normal EEG (n, %)	18	29.5	0	0	**0.01**
Background frequency ranges (n, %)					0.965
Alpha	26	42.6	6	40	
Alpha/theta	5	8.2	1	6.7	
Theta	14	23	3	20	
Theta/delta	11	18	3	20	
Delta	5	8.2	2	13.3	
Focal slowing (n, %)					0.192
Frontal	4	6.7	0	0	
Temporal	5	8.3	0	0	
Central	3	5	0	0	
Parietal	1	1.7	0	0	
Occipital	2	3.3	0	0	
Episodic transients (n, %)					
FIRDA	2	3.3	0	0	
TWs	3	5	1	7.1	
PDs	6	10	0	0	0.587
Epileptic activities (n, %)					
Seizures	3	5	1	7.1	
Status epilepticus	5	8.3	1	7.1	
Nonreactive EEG background activity (n, %)	11	18.3	4	33.3	0.258

GCS = Glasgow Coma Scale; PDs = periodic discharges; EEG = electroencephalography; SD = standard deviation; IQR = inter quartile range. Bold p-values are considered significant.

Pneumococcal encephalitis



22-year-old patient had listeria rhombencephalitis, HIV, and toxoplasmosis.

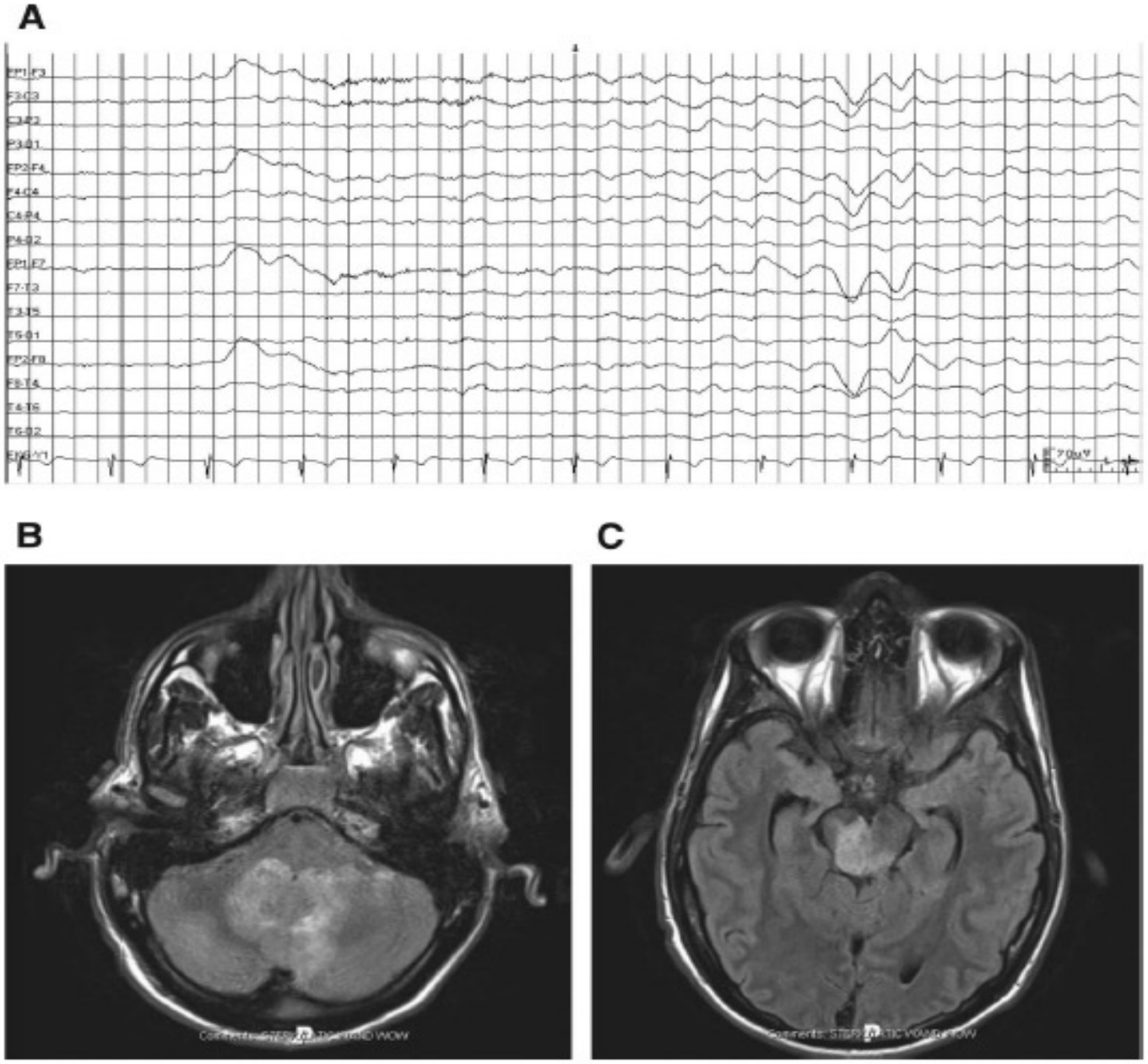


FIG. 10. A, EEG showing slow activity with rhythmic diffuse δ brought out by arousal seen after the eye movement in the third second and frontal muscle artifact in the fourth second. B and C, The MRIs showing the brain stem enhancement of rhombencephalitis.

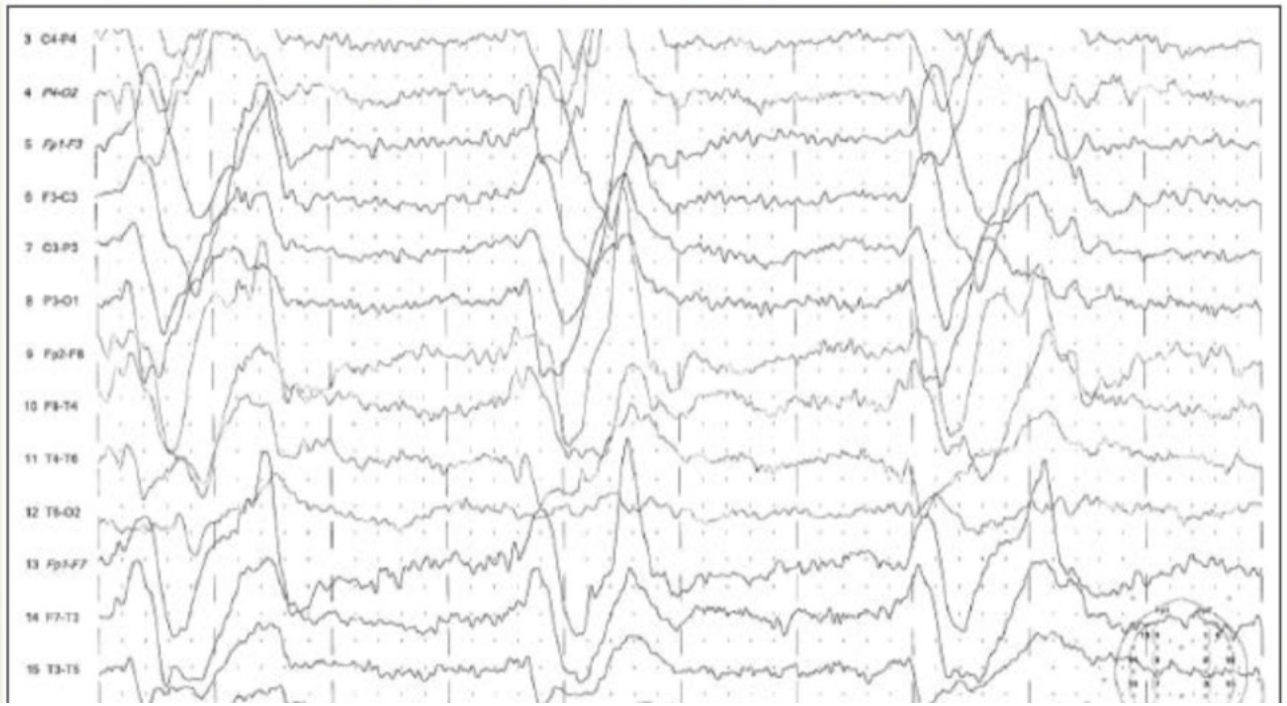
S Afr Med J 2015;105(9):xxxx. DOI:10.7196/SAMJnew.

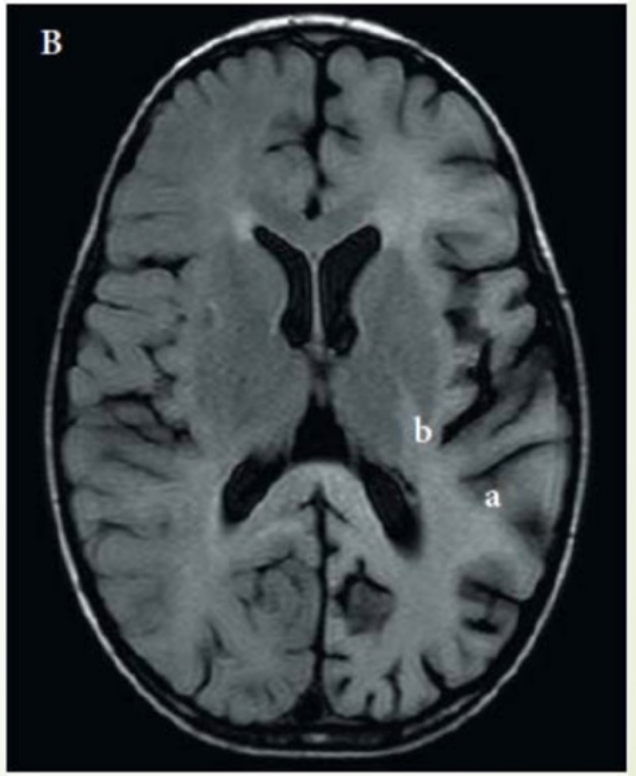
Patognomonico

CLINICAL ALERT

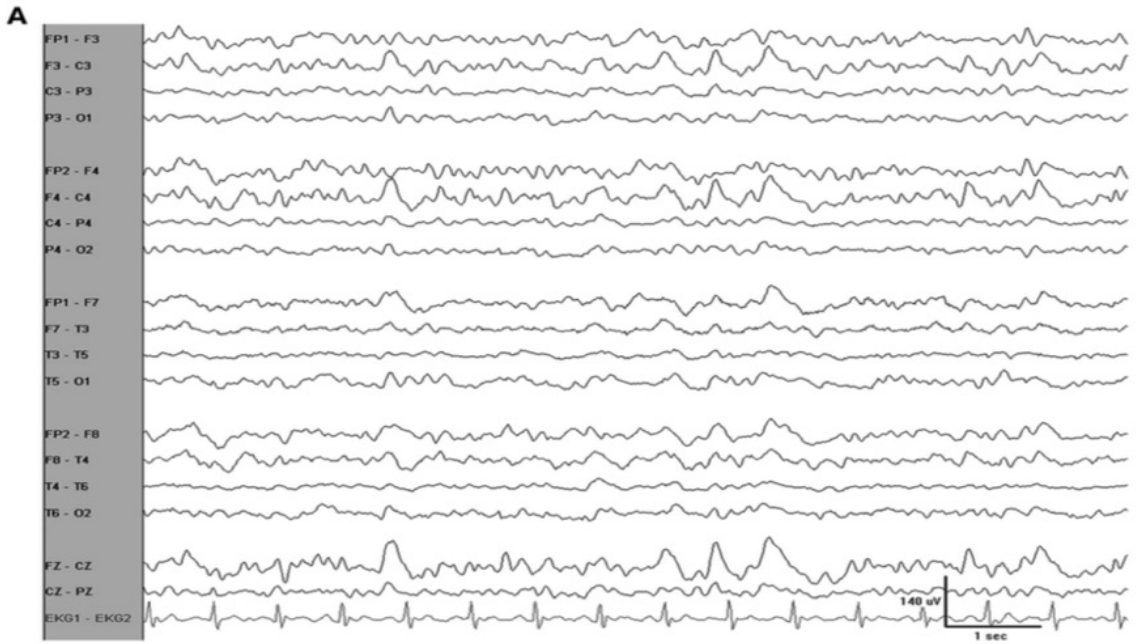
Subacute sclerosing panencephalitis in South African children following the measles outbreak between 2009 and 2011

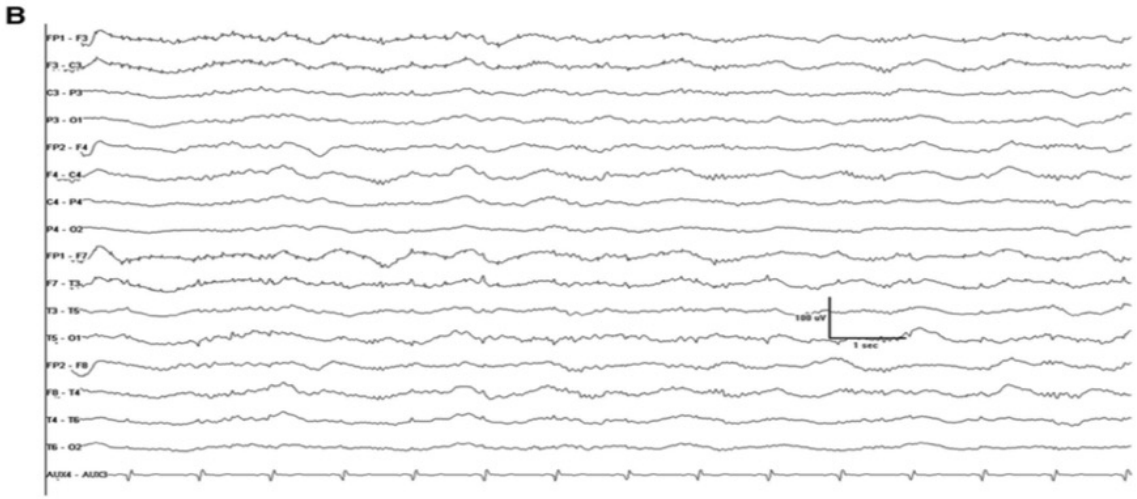
E Kija, A Ndondo, G Spittal, D R Hardie, B Eley, J M Wilmshurst

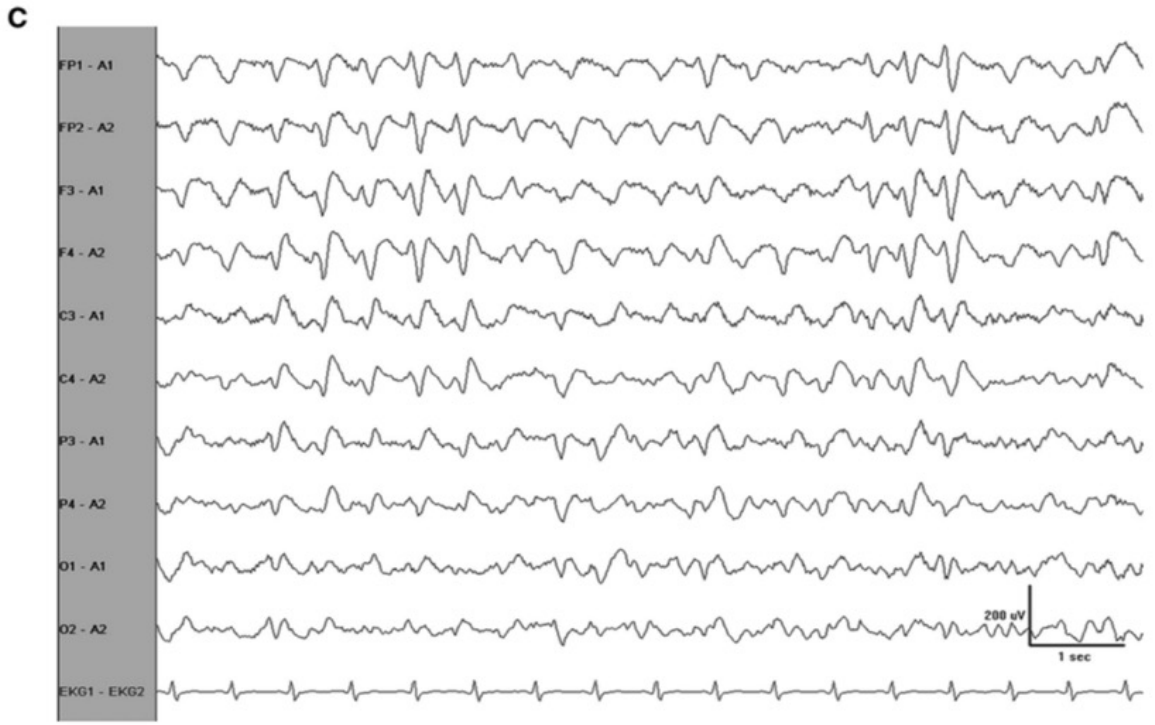




Encefalopatia associata a sepsi ad evoluzione fatale







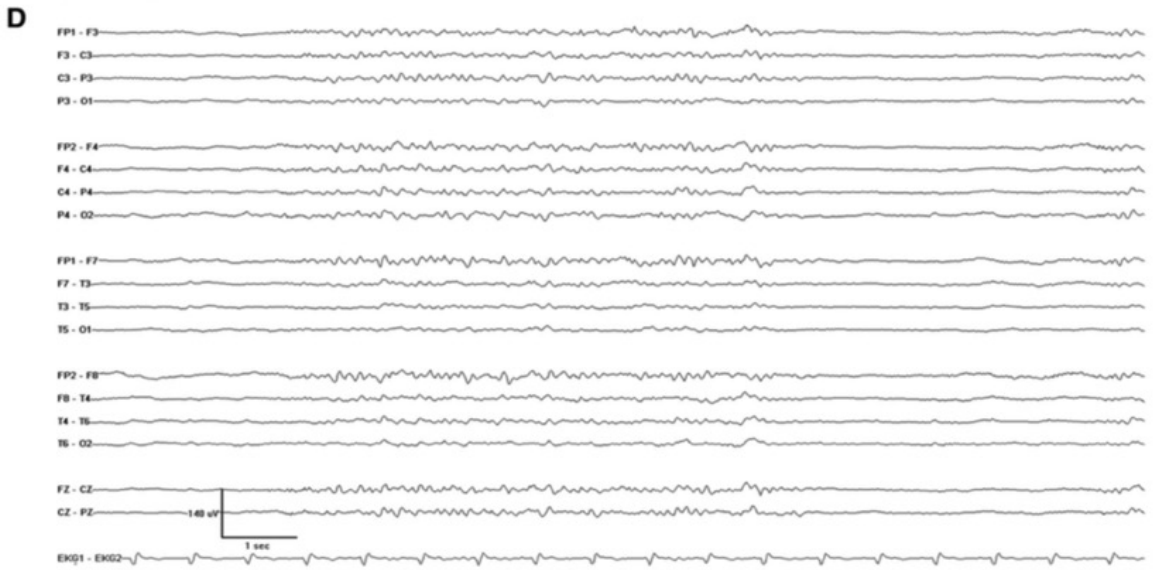


FIG. 6. Progressive changes in patients with sepsis-associated encephalopathy. **A**, Predominant theta frequency with occasional generalized delta bursts. **B**, Continuous rhythmic delta. **C**, Triphasic waves in coma. **D**, A burst suppression in advanced sepsis-associated encephalopathy.

Creutzfeldt-Jacob Disease
Periodic spike and wave complex

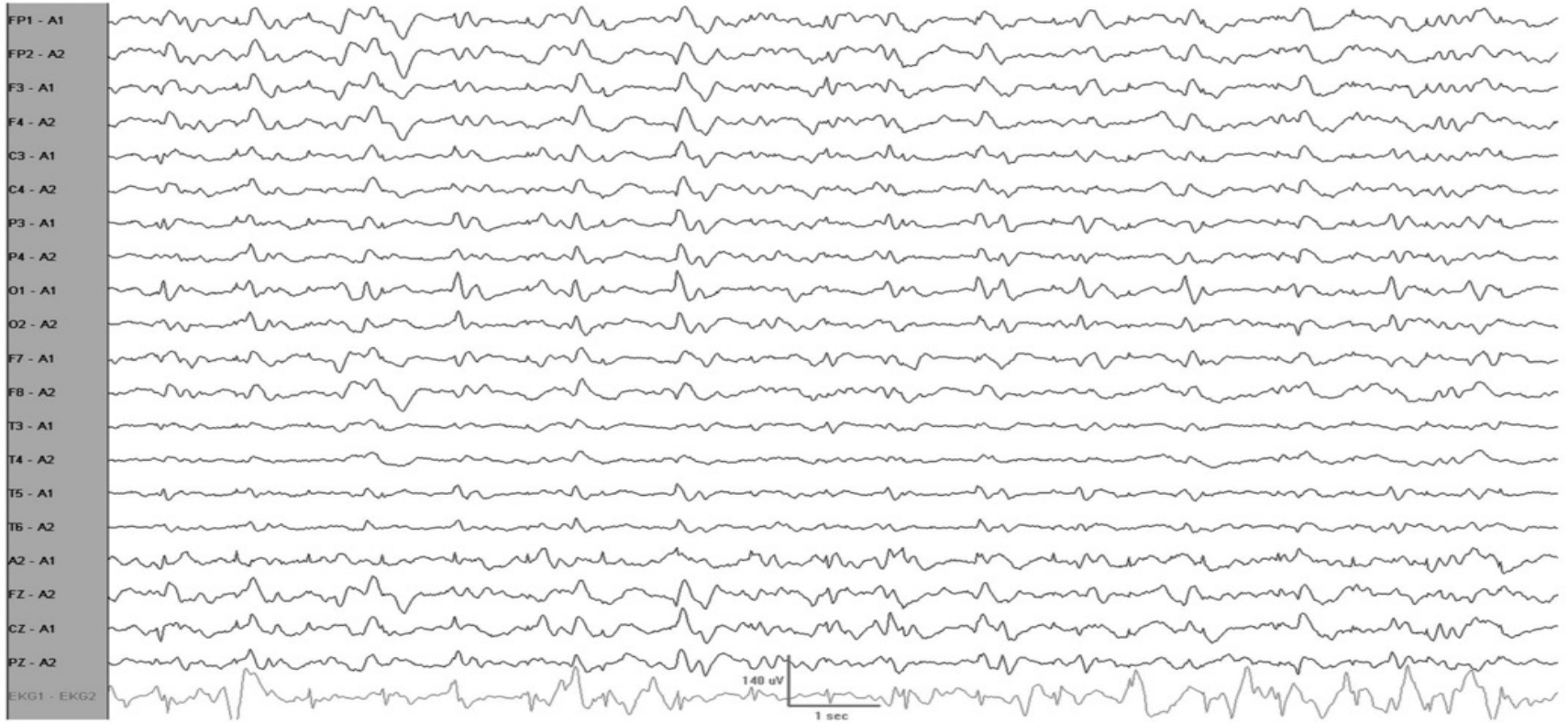
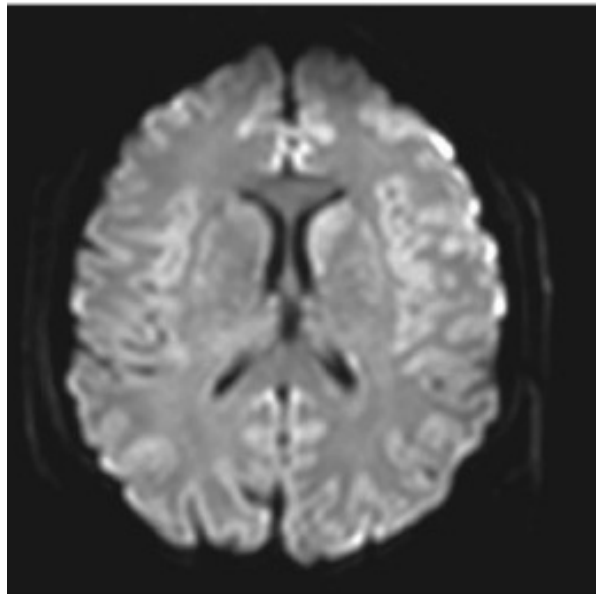
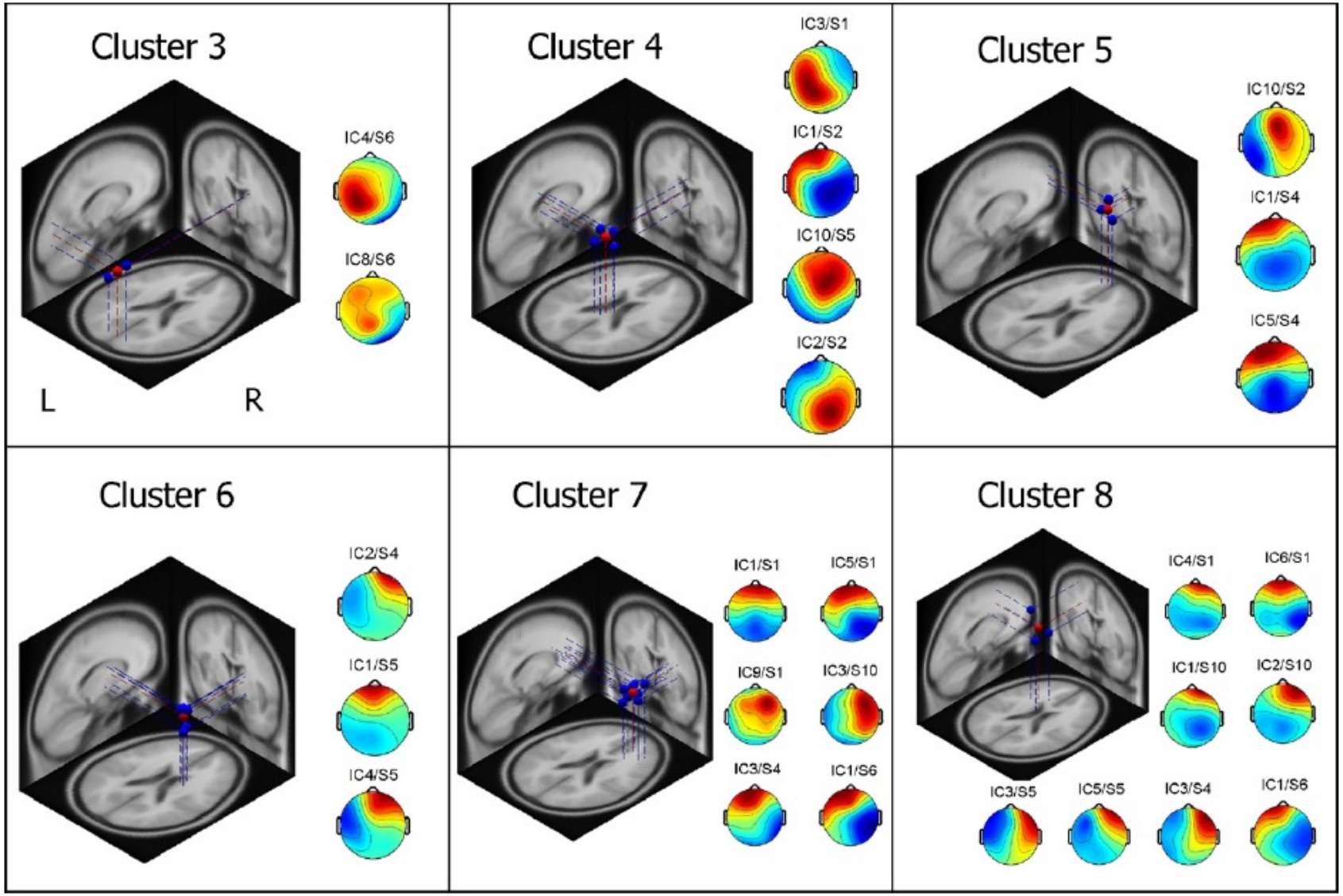


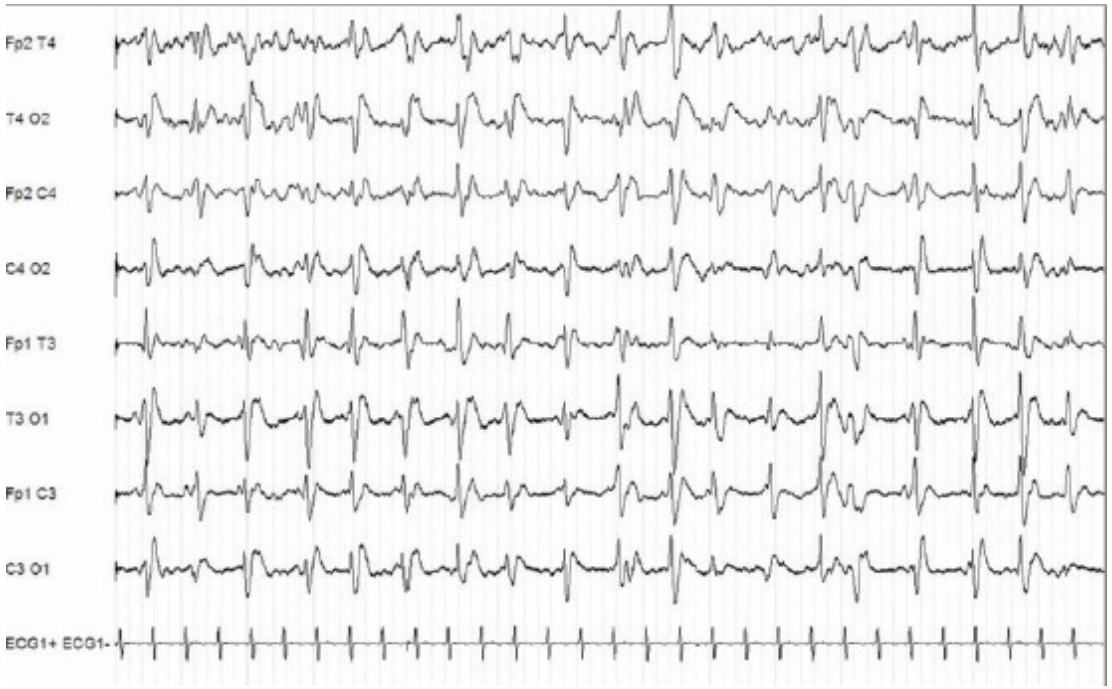
FIG. 5. Creutzfeldt–Jakob disease. The patient was a 65-year-old man with rapidly progressive dementia and myoclonus. MRI scan was diagnostic for sporadic Creutzfeldt–Jakob disease, and the cerebrospinal fluid was positive for 14-3-3 protein.





Clinical neurophysiology
Redefining Periodic Patterns on Electroencephalograms of Patients with Sporadic Creutzfeldt–Jakob Disease
Jung-Won Shin, Byeongsoo Yim, Seung Hun Oh, Nam Keun Kim, Sang kun Lee, Ok-Joon Kim

Creutzfeldt-Jacob Disease



Metabolic encephalopathy

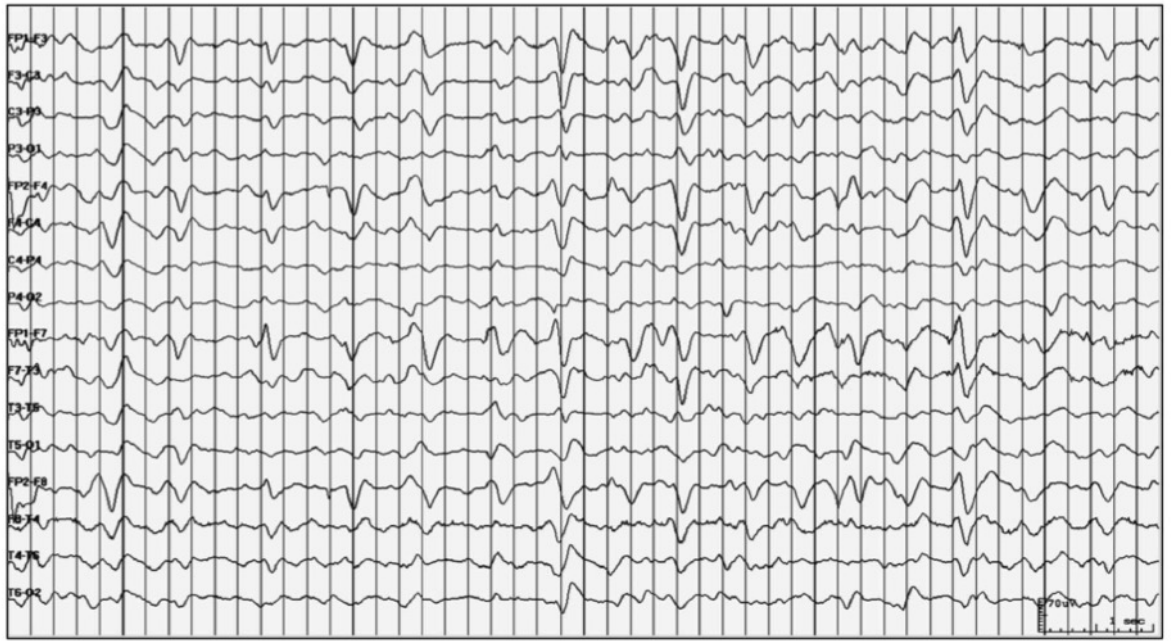
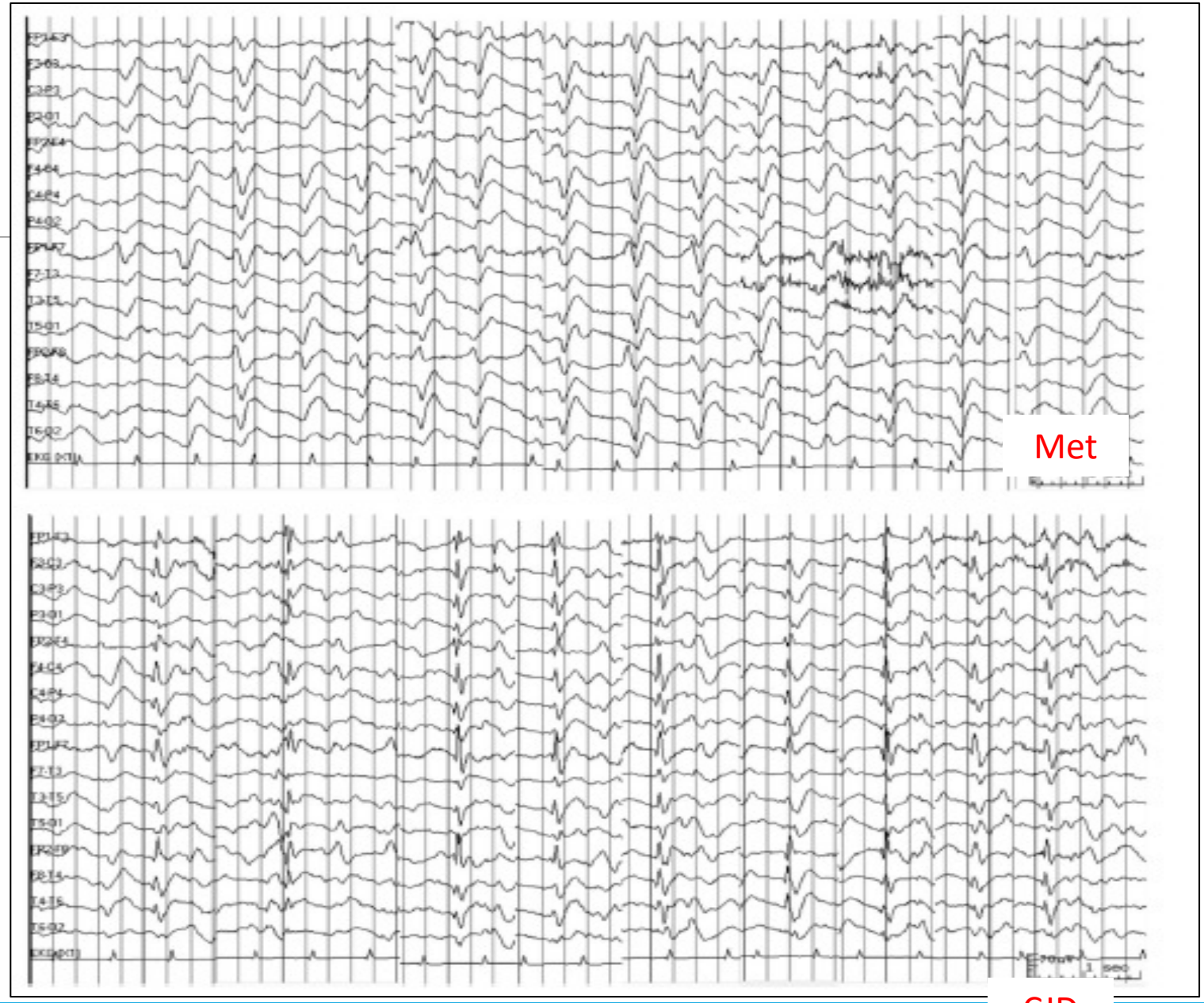


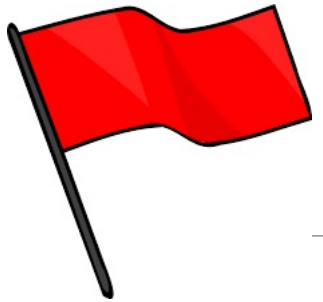
FIG. 5. Triphasic waves in acute encephalopathy. Generalized slowing of background activity with frequencies in the theta (4–7 Hz) and delta (<4 Hz) range and bilateral high-voltage (70–100 μV) triphasic waves with a frontocentral maximum and an anterior-posterior or posterior-anterior shift.

TABLE 1. Triphasic Waves Versus Periodic Epileptiform Discharges

Triphasic Waves	Periodic Epileptiform Discharges
Surface negative, blunted triphasic complexes with (1) low-amplitude, blunted, negative first phase (often wide based); (2) dominant, steep positive second phase; and (3) slow rising third "slow-wave" component. No polyspikes	Surface-negative bi- tri-, or polyphasic discharges with spike, polyspike, sharp wave, or slow-wave complexes or combinations of these
Complex duration: 400–600 milliseconds	Complex duration: 60–600 milliseconds (mean 200 milliseconds)
Amplitude: 100–300 μV on referential montage	Amplitude: 50–300 μV (usually up to 150 μV)
Frequency: 1.0–2.5 Hz (typically 1.8 Hz)	Frequency: 0.2–3 Hz (usually 0.5–2.0 Hz)
Persistence: wax and wane but >10% of a standard 20-minute EEG	Persistence: ≥10 minutes in an EEG recording
Evolution/reactivity: decrease with sleep, drowsiness, or after benzodiazepines; increase and reappear with arousal or stimulation. May exhibit phase-lag, seen best on referential montage	Evolution: static, with only minor variability in waveforms



PSWC vs LPD



Evolution: Unlike PSWC, LPD usually denote a transient EEG phenomenon, which progressively decreases in amplitude and periodicity rate during the disease evolution and often disappear within 2 weeks after the onset of the lesion[19]. Furthermore, LPDs but not PSWC are frequently associated with epileptic seizures[22].

Responsiveness: LPDs are usually not affected by manipulation and sleep, whereas PSWC in CJD are mitigated by external stimulation and disappear during sleep. Actually, PSWC may also be attenuated by sedative medications, in particular with benzodiazepines[23]. Triphasic waves from metabolic[24] and unknown[25] causes and LPD can also be attenuated by benzodiazepines and other non-sedative anti-epileptic drugs. However, in these cases, EEG changes usually do not parallel a consciousness amelioration. In elderly and stuporous patients, drug response is further complicated by the iatrogenic sedation, as in our patients, which obliges to a cautious clinical evaluation.

AAA **Slow periodicity of PSWC** (0.5-2 sec) is another red flag, which should alert the clinician to deepen the clinical examination. Actually, rhythmic PSWC can be easily misinterpreted as epileptiform abnormalities and, according to SC, the periodicity lower then 2.5 Hz obliges the clinician to verify clinical signs and to search for a secondary criterion before formulating NCSE diagnosis.

Panel: Specifications for the Salzburg criteria

Frequency of the epileptiform discharges

Frequency higher than 2·5 cycles per s is considered when more than 25 epileptiform discharges are seen per 10 s epoch.[13]

Continuous (quasi-)rhythmic delta-theta activity

Repetition of waveforms with relatively uniform morphology and duration, and without an interval between consecutive waveforms. The duration of one cycle (ie, the period) of the rhythmic pattern should vary by less than 50% from the duration of the subsequent cycle for most (>50%) cycle pairs to qualify as rhythmic.[9]

Typical spatiotemporal evolution

Sequential change in voltage and frequency, or evolution in frequency and change in location:

- Change in voltage (increase or decrease) with a minimum factor of two of the voltages measured between the first and last graphoelement.
- Change in frequency more than 1 Hz: frequency of the second with highest rate of graphoelements and the second with lowest rate of graphoelements differed by more than 1 Hz.
- Evolution in frequency is defined as at least two consecutive changes in the same direction by at least 0·5 per s.[9]
- Change in location sequential spreading into or out of at least two different standard 10–20 electrode locations.[9]
- To qualify as present, a single frequency or location must persist at least three cycles. The criteria for evolution must be reached without the pattern remaining unchanged in frequency, morphology, or location for 5 min or more.[9]

Fluctuation without definite evolution

Three or more changes, not more than 1 min apart, in frequency (by at least 0·5 per s) or three or more changes in location (by at least one standard interelectrode distance), but not qualifying as evolving.[9]

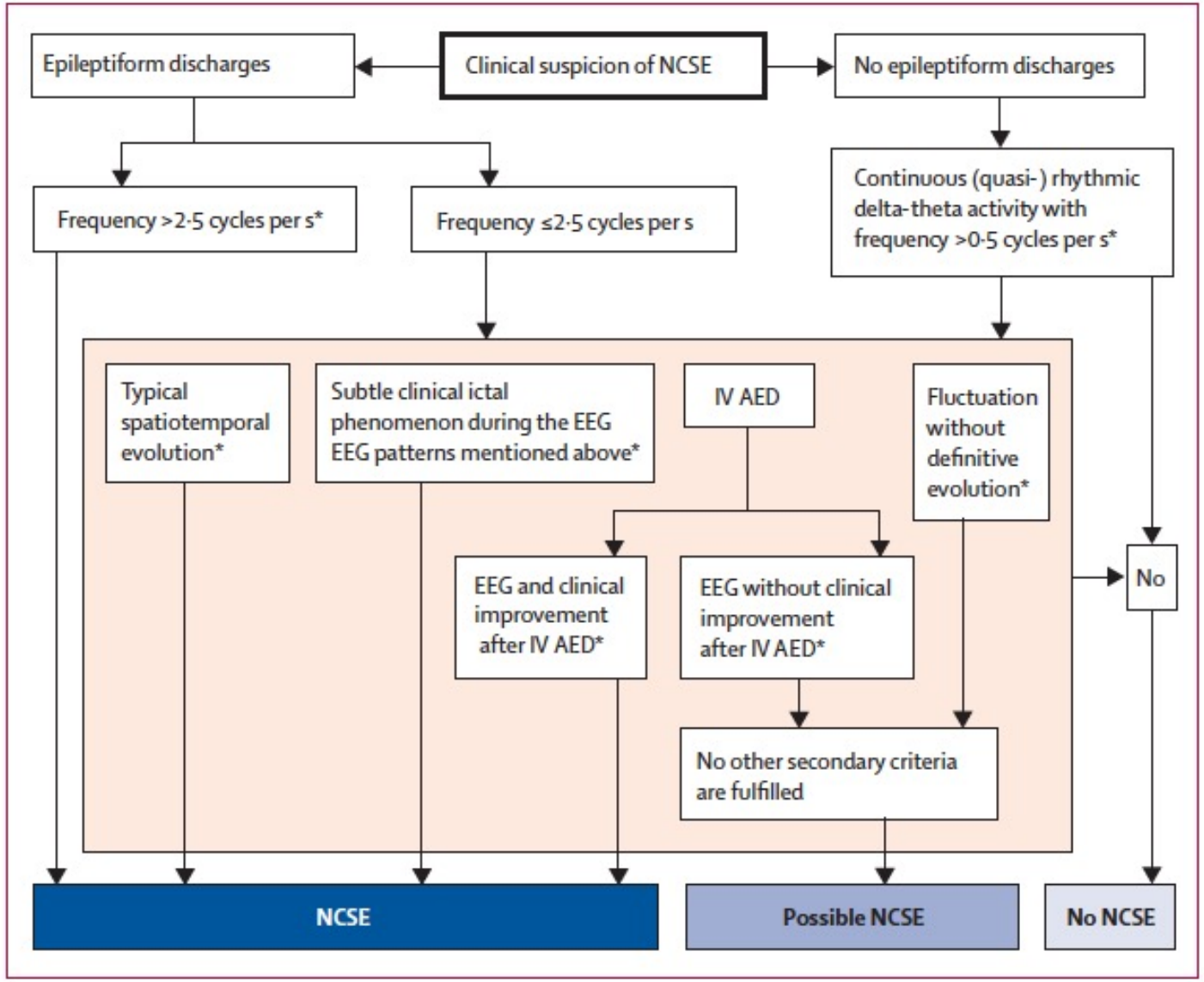
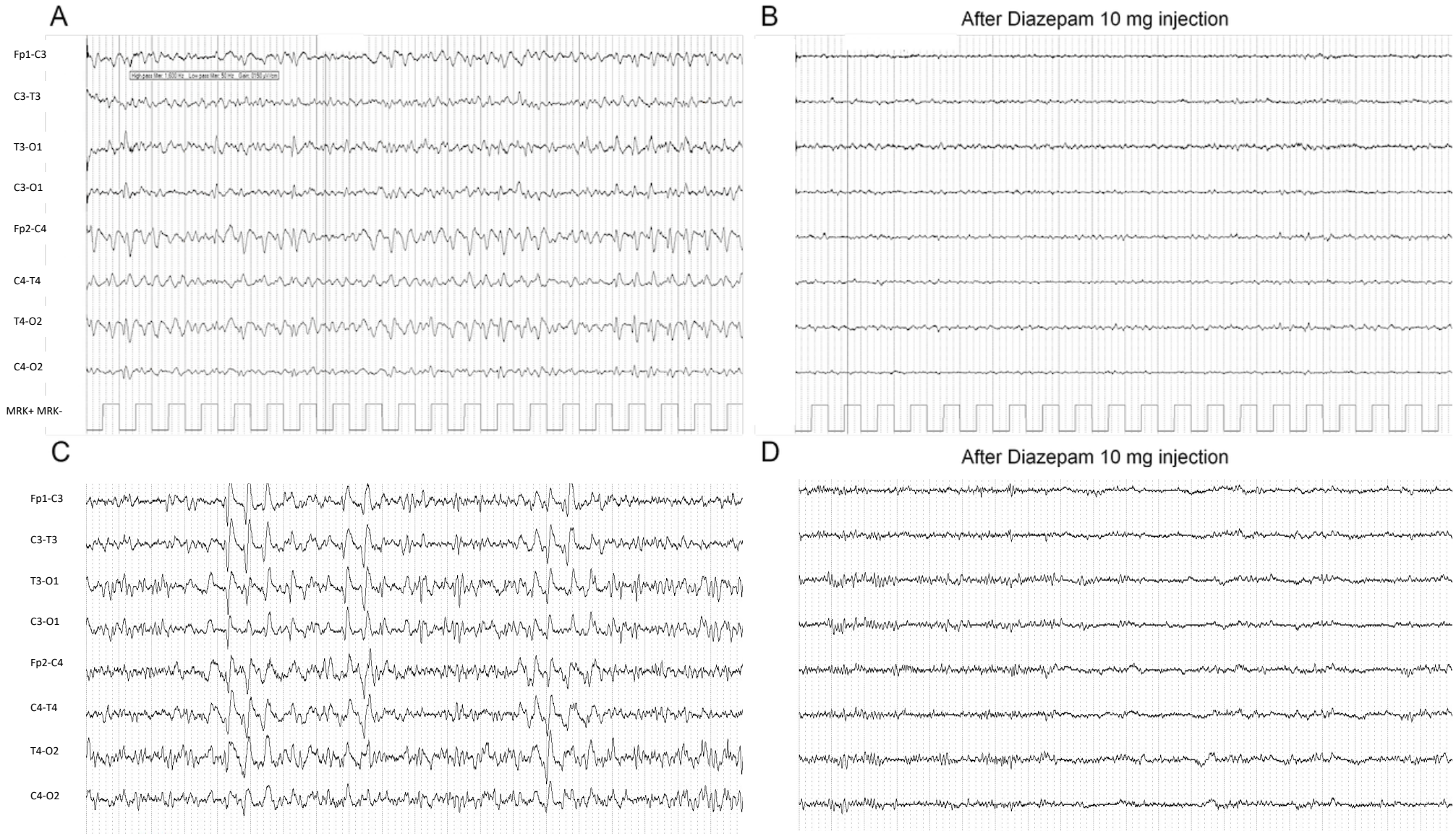


***Figure* 1: Salzburg EEG criteria for the diagnosis of NCSE**

To qualify for a diagnosis of NCSE, the whole EEG recording should be abnormal, and EEG criteria have to be continuously present for at least 10 s. If criteria are not fulfilled at any stage, EEG recording will not qualify for a diagnosis of NCSE or possible NCSE. NCSE=non-convulsive status epilepticus. IV AED=intravenous antiepileptic drug. *Patients with known epileptic encephalopathy should fulfil one of the additional secondary criteria: increase in prominence or frequency of the features above when compared to baseline, and observable change in clinical state; or improvement of clinical and EEG features with IV AEDs (panel).


A
Fp1-C3
C3-T3
T3-O1
C3-O1
Fp2-C4
C4-T4
T4-O2
C4-O2
MRK+ MRK-
B
After Diazepam 10 mg injection
C
Fp1-C3
C3-T3
T3-O1
C3-O1
Fp2-C4
C4-T4
T4-O2
C4-O2
D
After Diazepam 10 mg injection

TW

A

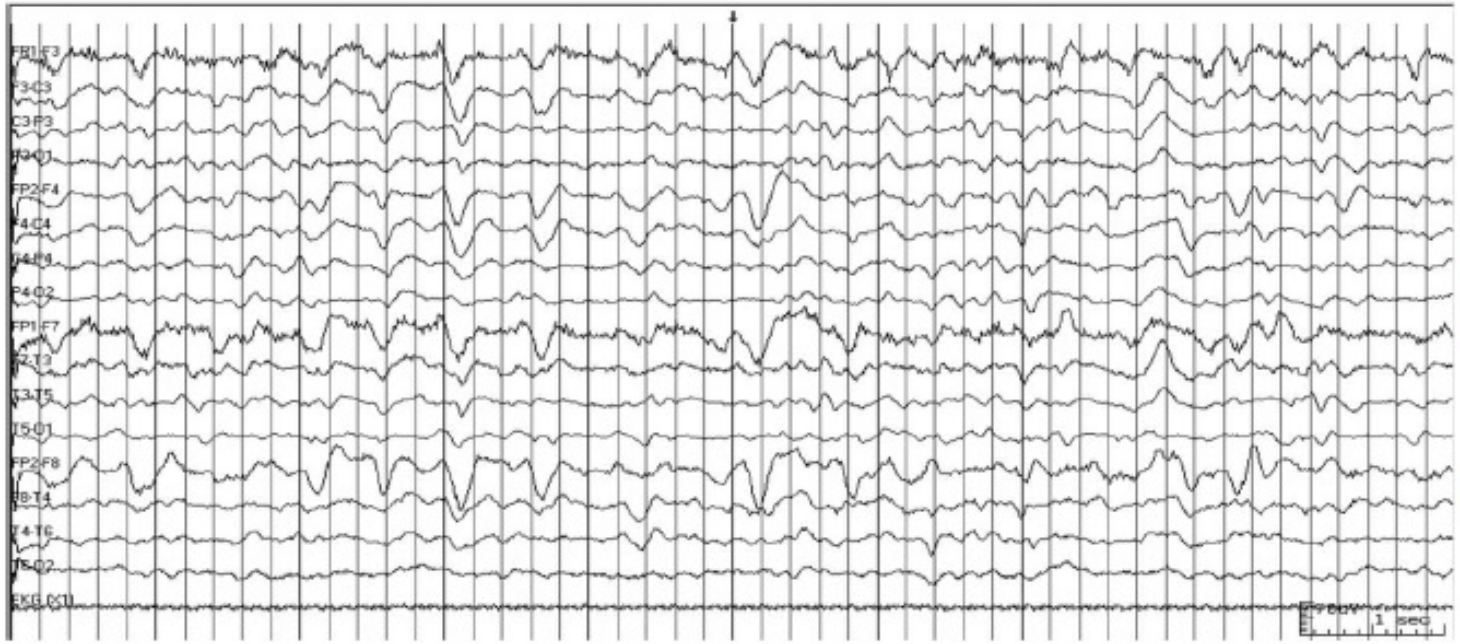
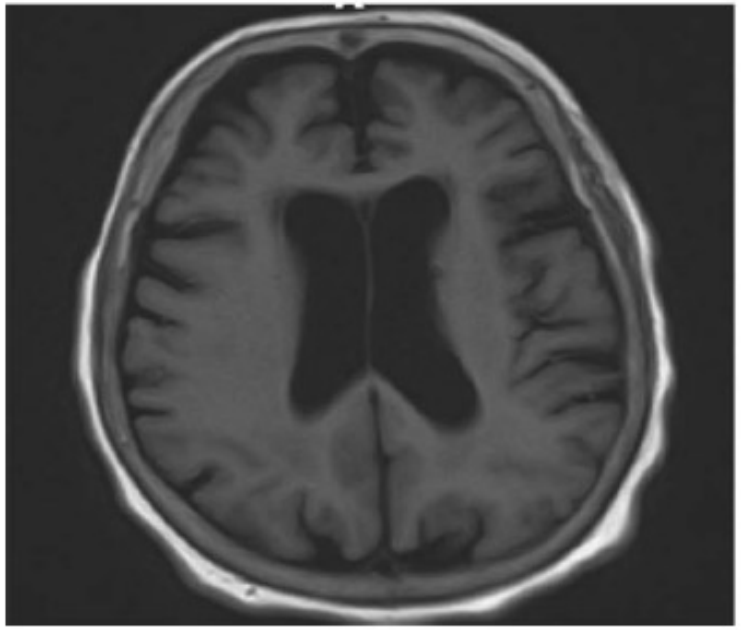


TABLE 4. Clinical Predispositions for TWs	
Without white matter disease/subcortical atrophy	Hepatic encephalopathy, hyperammonemia
	Uremia, other marked electrolyte abnormalities
	Anoxia
	Toxins/medications (e.g., lithium, baclofen)
With white matter disease/subcortical or diffuse atrophy	Mild infections (e.g., urinary tract infection, upper respiratory tract infection)
	Lesser degrees of electrolyte imbalance, toxins

B



C

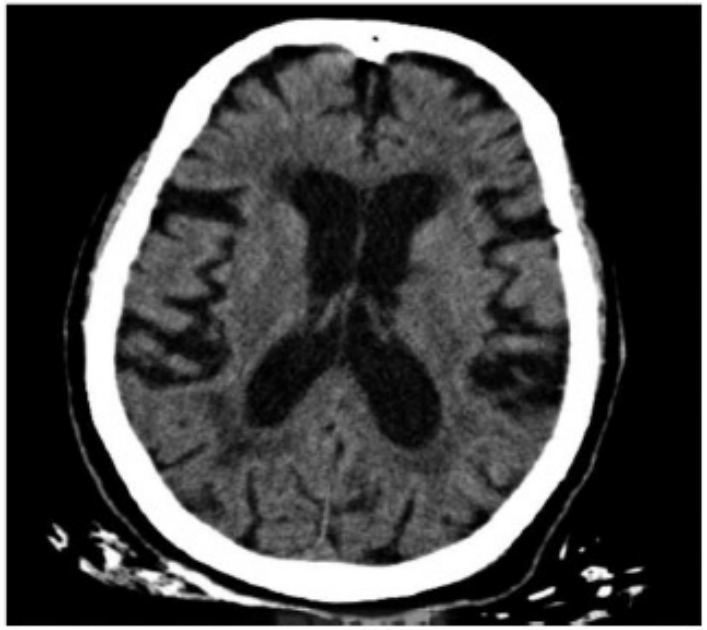


FIG. 12. A, EEG showing intermittent TWs of modest voltage, increased with arousal. B and C, The MRI (B) and head CT (C) revealing white matter disease and ventricular dilation in a patient with *normal* ammonia but with a urinary tract infection. This illustrates that even *without* high ammonia, TWs may occur with white matter disease/diffuse cerebral atrophy along with a relatively minor intercurrent urinary infection (see Table 4).

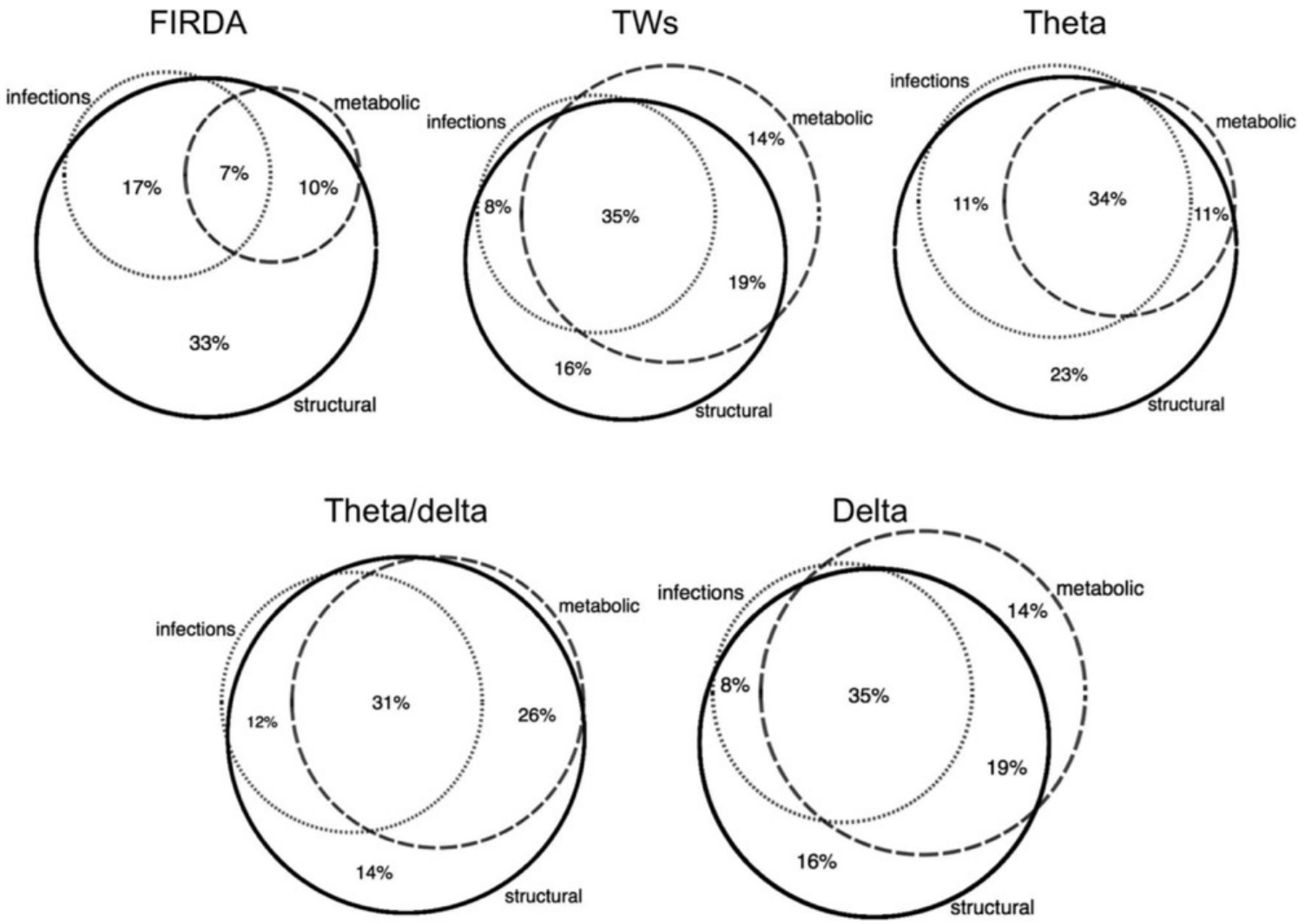
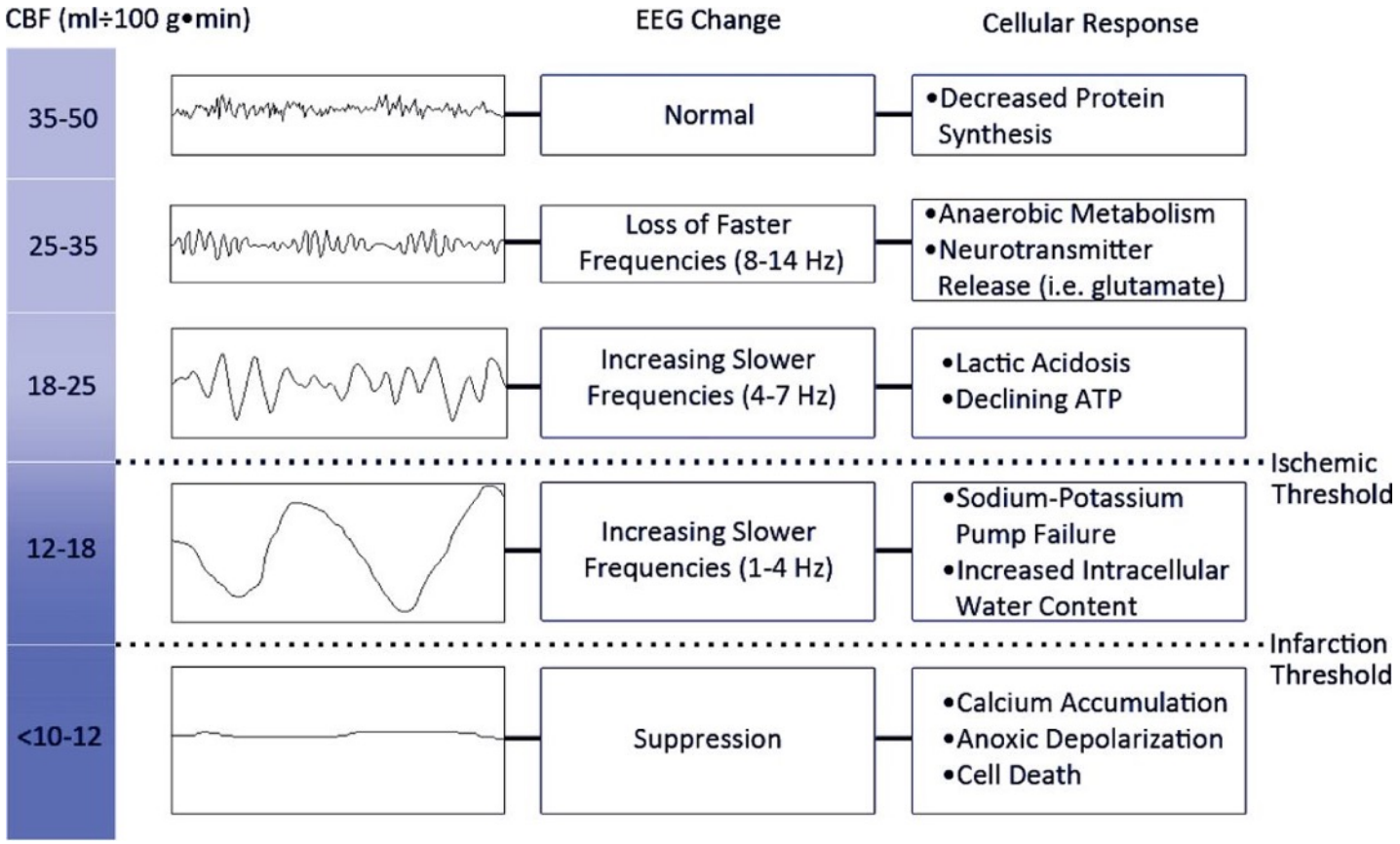
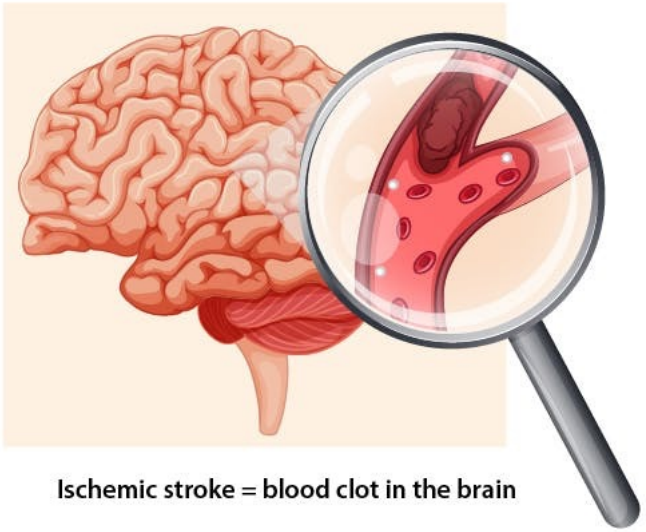


FIG. 2. The presence of clinical, biochemical, and neuroanatomic abnormalities in encephalopathic patients with different EEG patterns. Metabolic problems were renal and/or liver insufficiency. Structural abnormalities included white matter lesions, brain atrophy, cerebral infarcts, intracranial hemorrhage, brain tumors, encephalitis, posterior reversible encephalopathy, and traumatic brain injury. FIRDA, frontal intermittent rhythmic delta activity; TWs, triphasic waves. Adapted with permission from Sutter and Kaplan (2013).

EEG in stroke





Ischemic stroke = blood clot in the brain

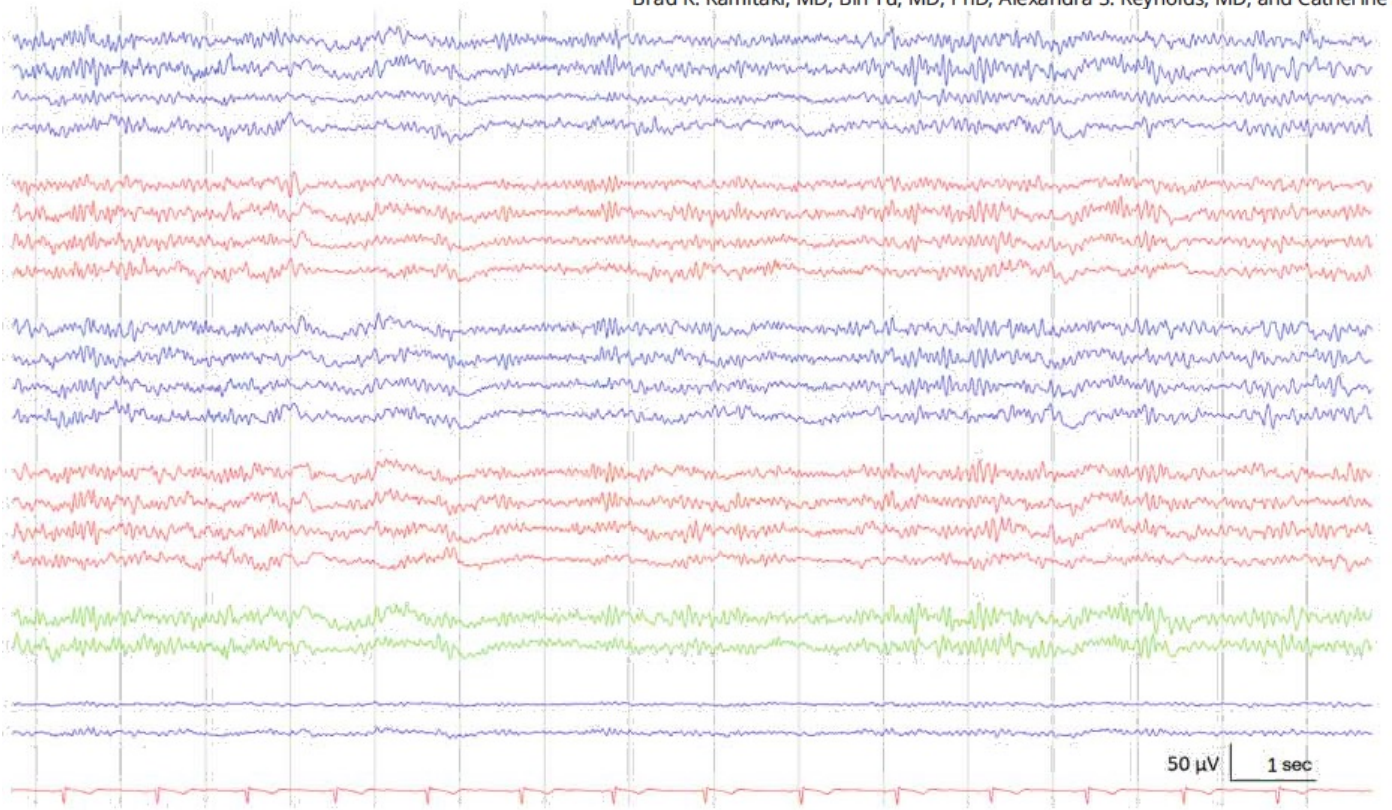
Teaching NeuroImages: Acute stroke captured on EEG in the ICU

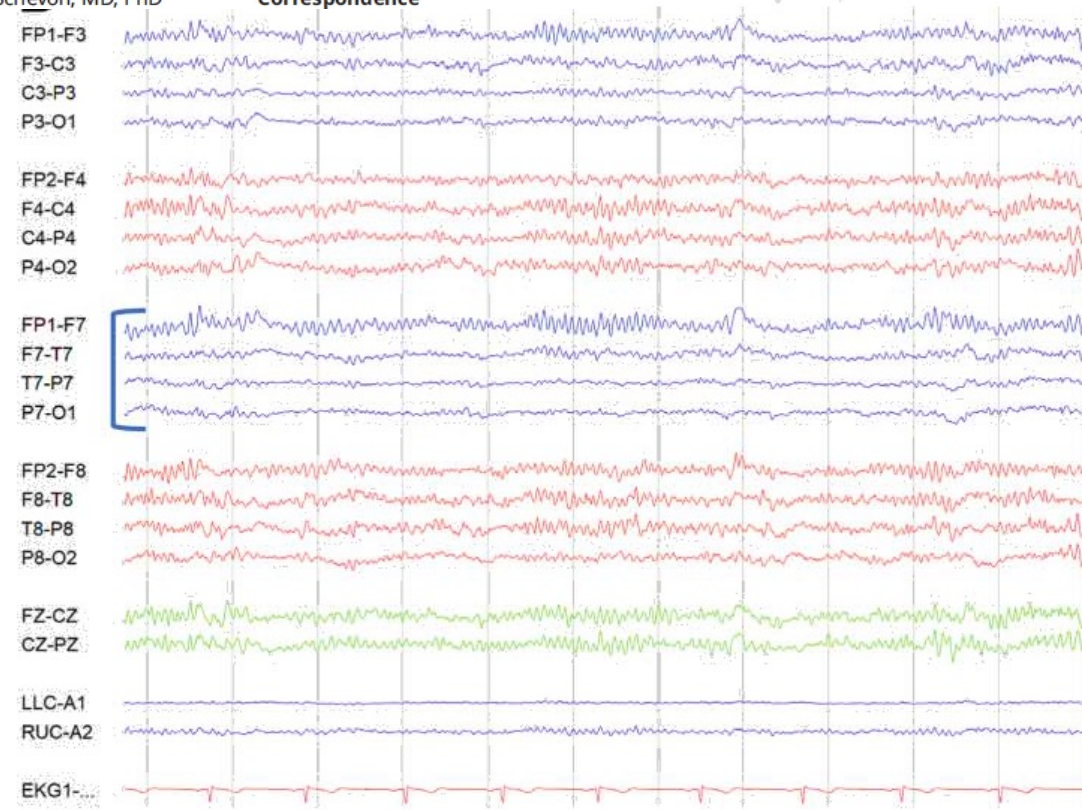
Visual and quantitative analysis

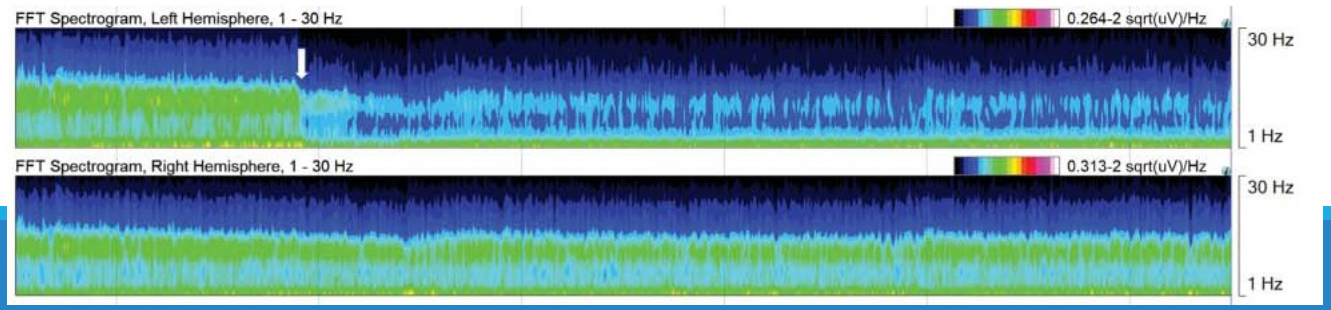
Neurology 2019;92;e626-e627

Brad K. Kamitaki, MD, Bin Tu, MD, PhD, Alexandra S. Reynolds, MD, and Catherine A. Schevon, MD, PhD

Correspondence







EEG in acute stroke
lateralized rhytmic delta activity

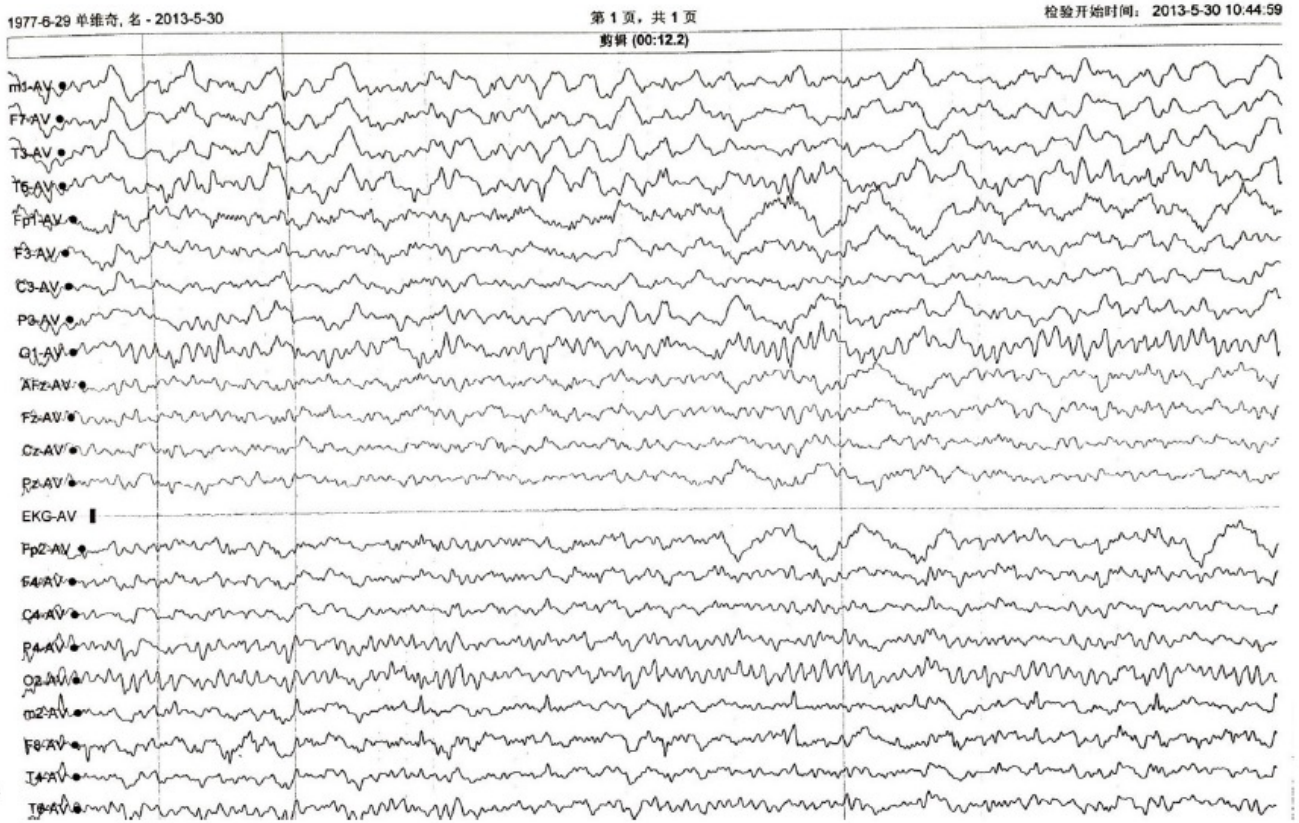


Figure 1: EEG in a 63 years old male patient with acute ischemic stroke shows low to middle amplitude slow waves visualized in the left hemisphere and are more obvious in M1, F7, T3, T5 leads.

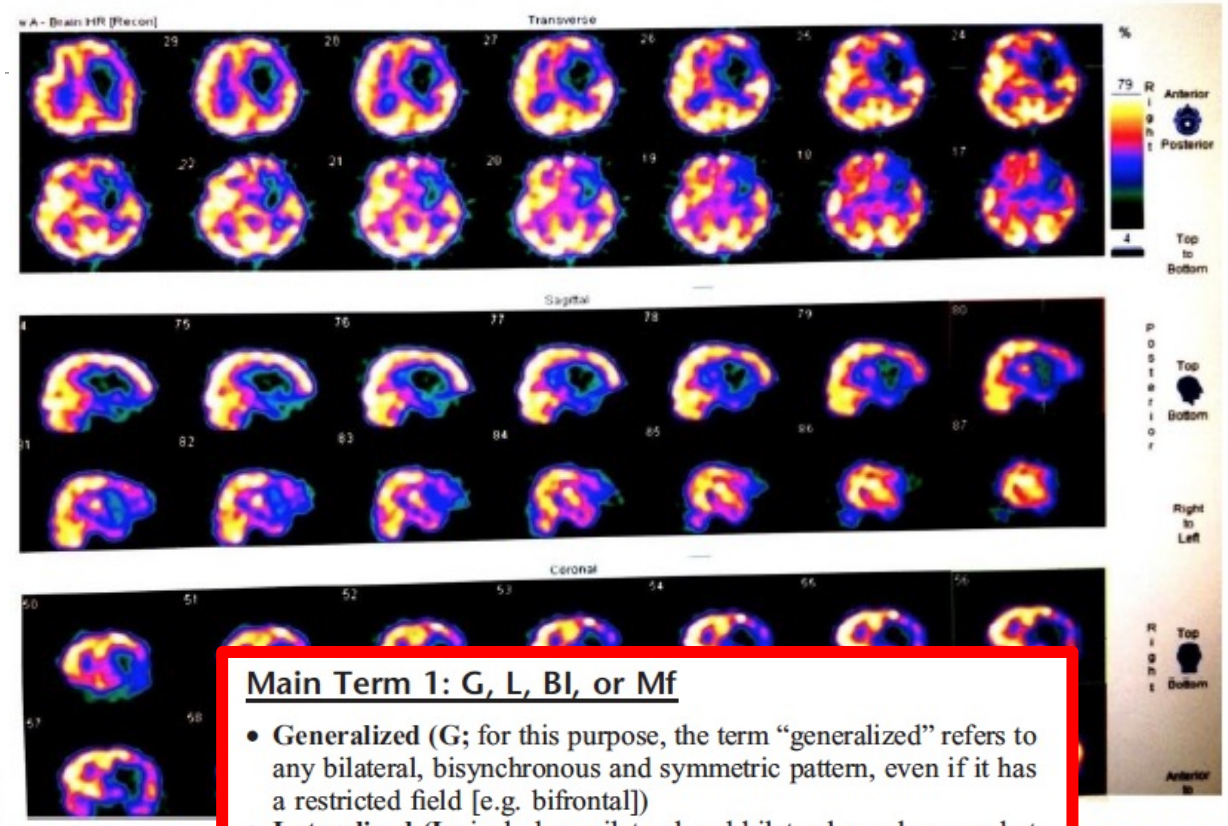
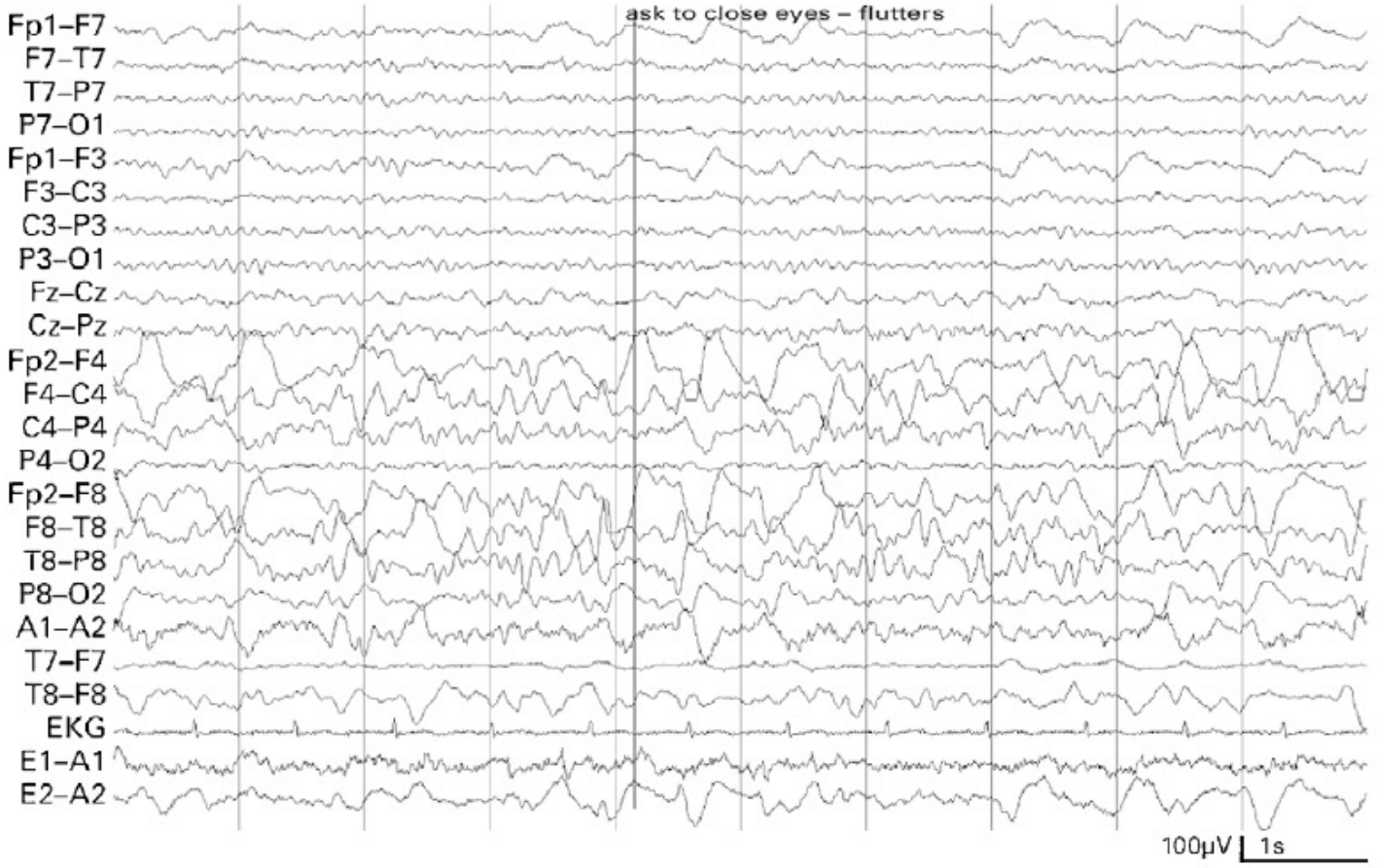


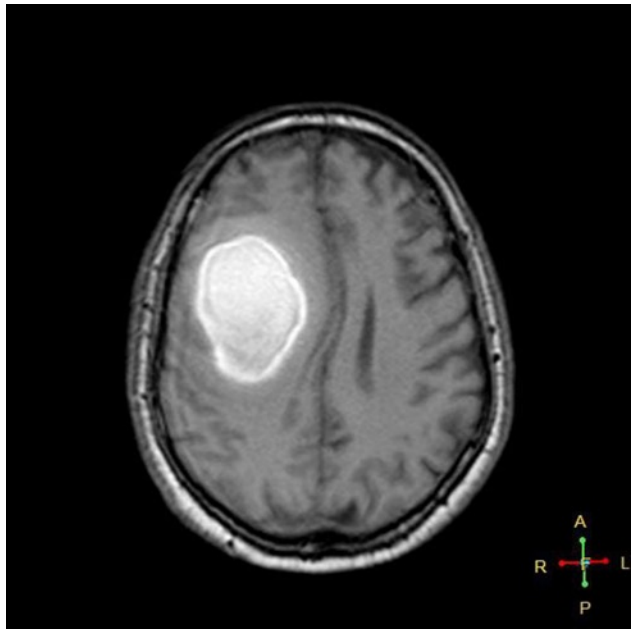
Figure 2: SPECT in a 63 y … left parietal lobe, left insula … e, left frontal lobe,

Main Term 1: G, L, BI, or Mf

- **Generalized (G;** for this purpose, the term "generalized" refers to any bilateral, bisynchronous and symmetric pattern, even if it has a restricted field [e.g. bifrontal])
- **Lateralized (L;** includes unilateral and bilateral synchronous but asymmetric; includes focal, regional and hemispheric patterns)
- **Bilateral Independent (BI;** refers to the presence of 2 independent [asynchronous] lateralized patterns, one in each hemisphere)
- **Multifocal (Mf;** refers to the presence of at least three independent lateralized patterns with at least one in each hemisphere)

Emorragia intraparenchimale





Axial T1

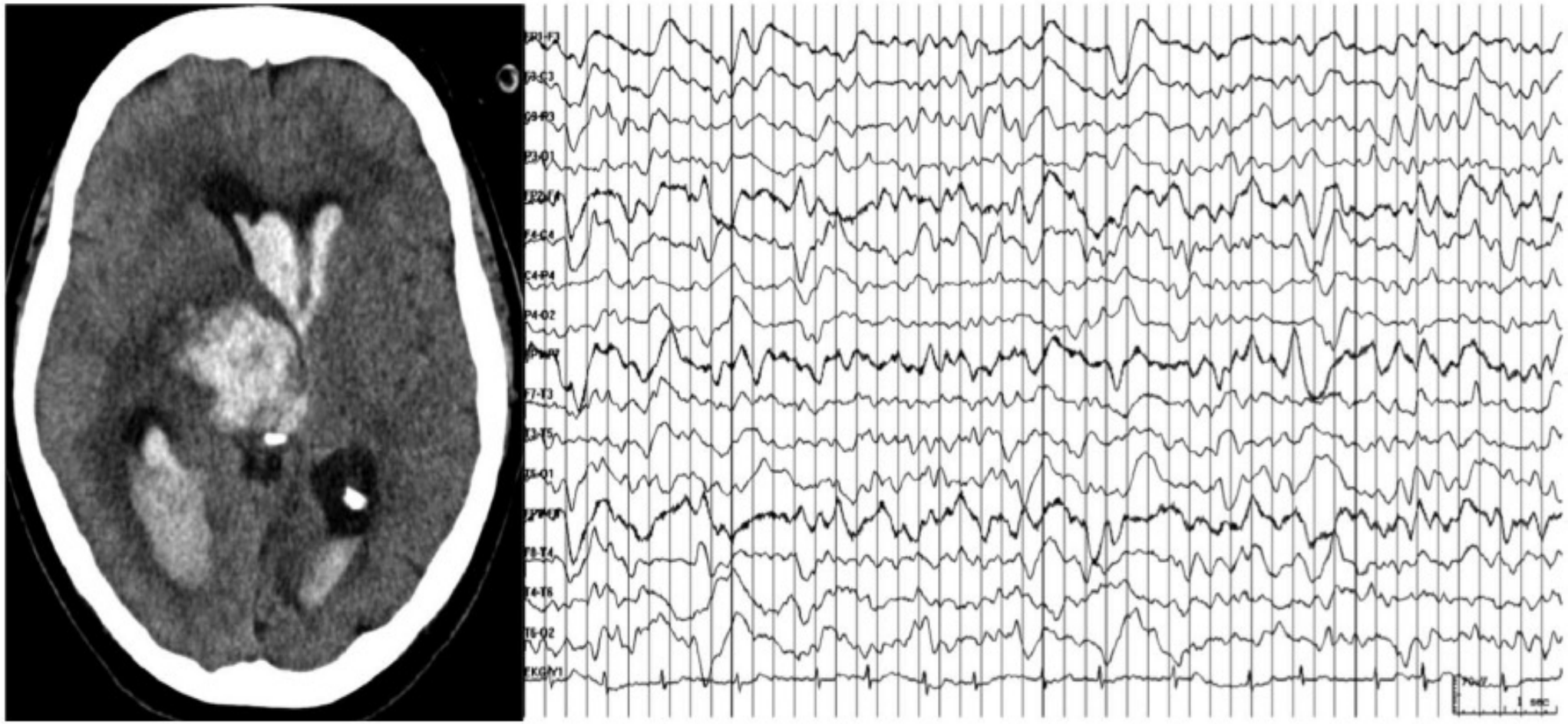
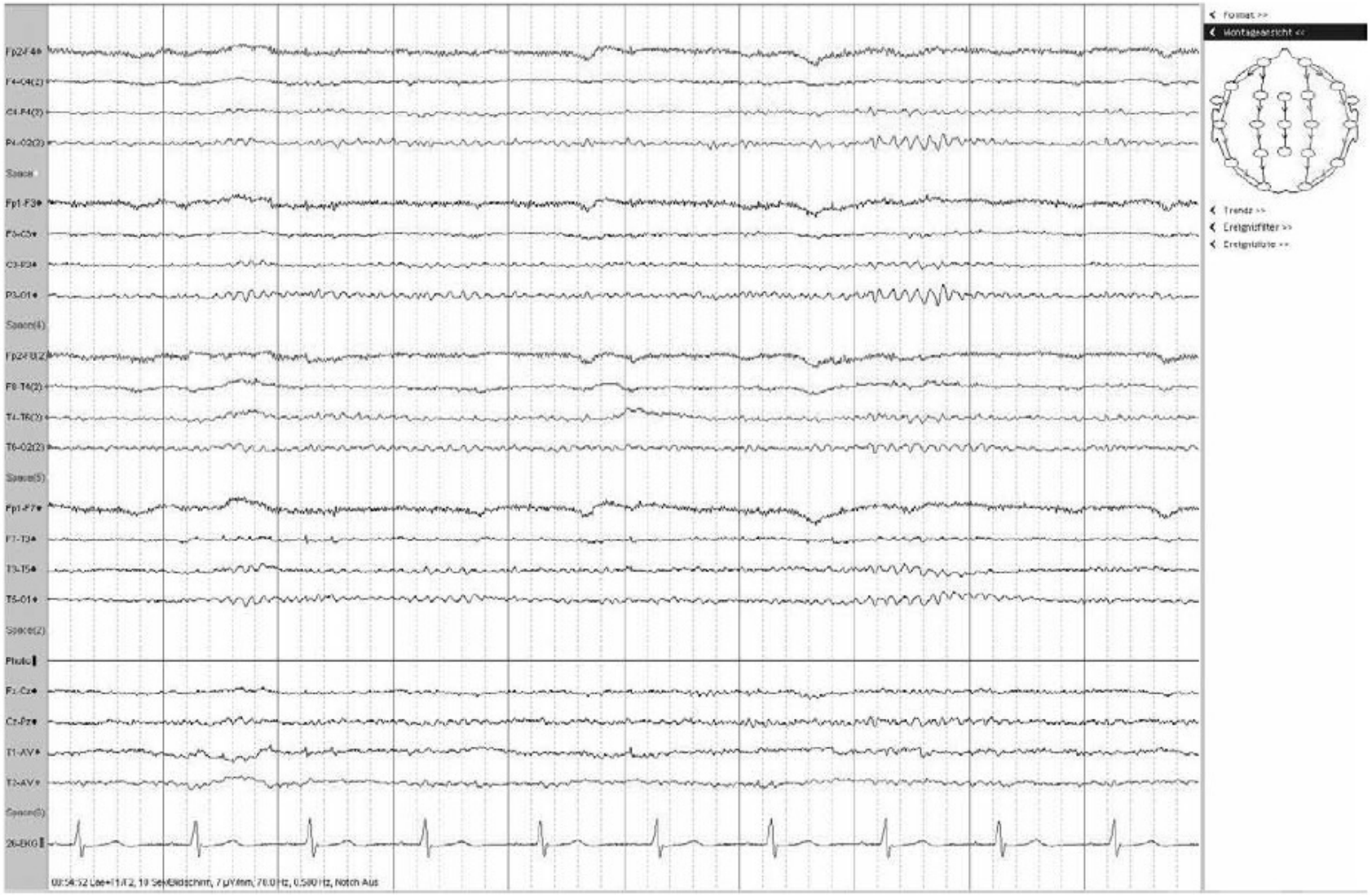
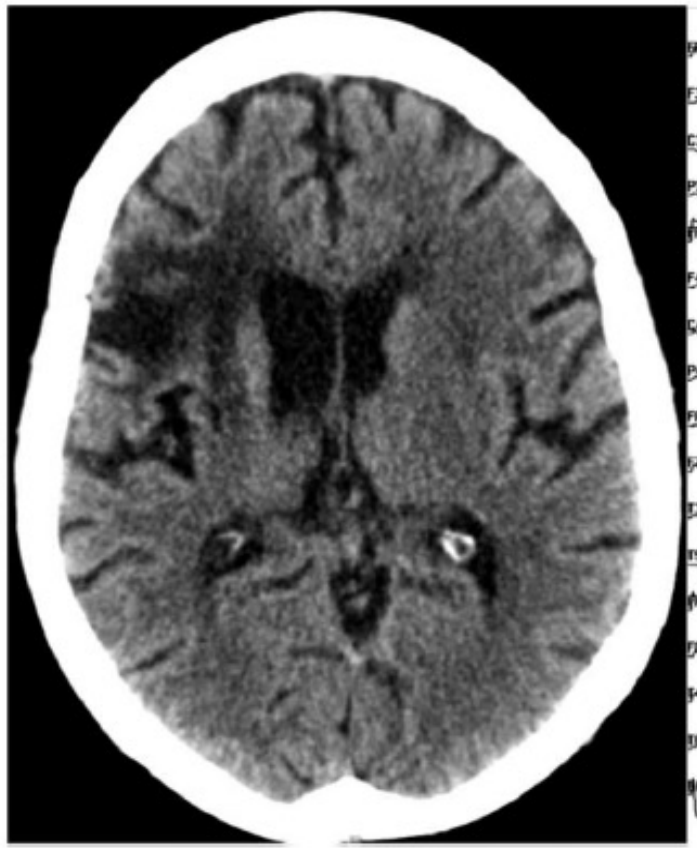


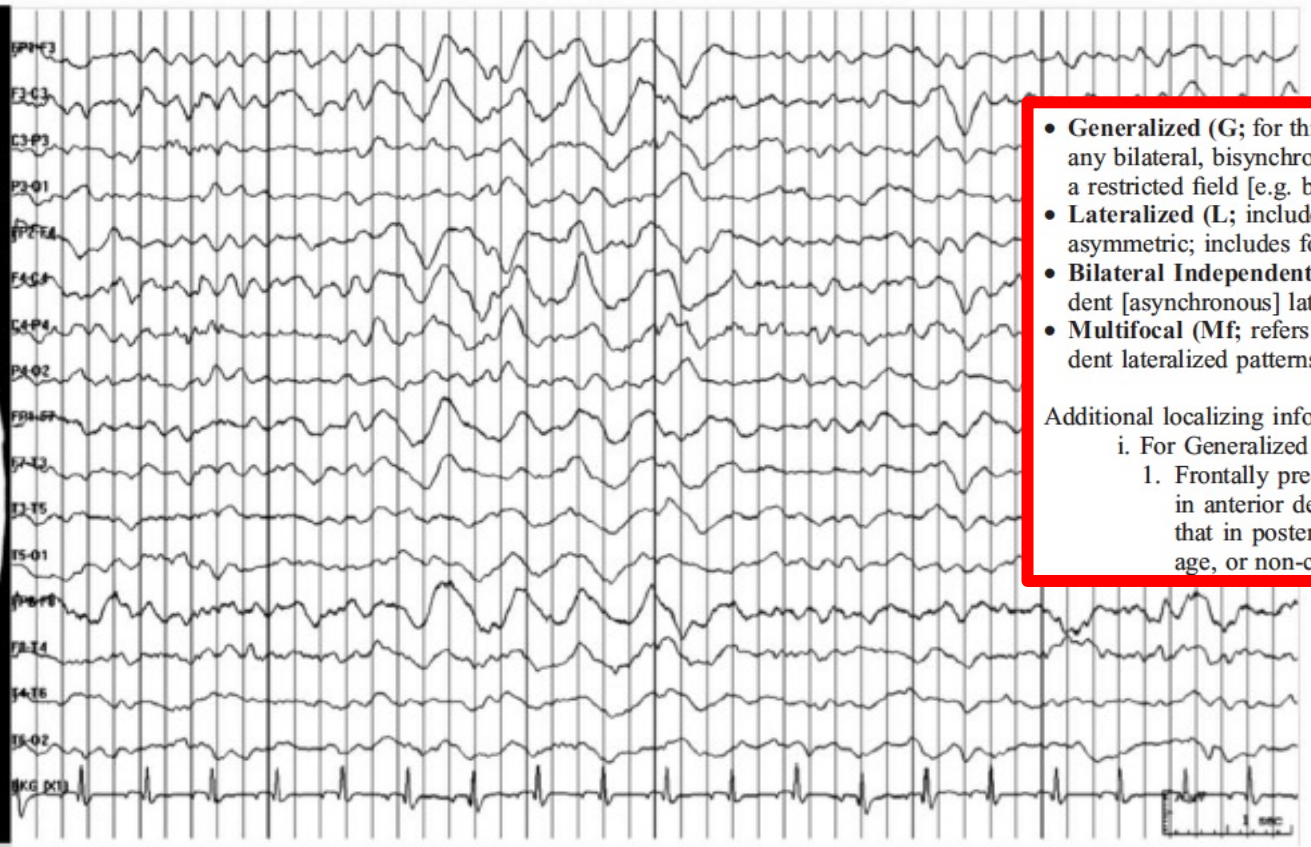
FIG. 3. Theta/delta slowing in a patient with an intracerebral hemorrhage. Axial cerebral CT shows a large hyperdense mass in the right *centrum semiovale* and the right basal ganglia and in both lateral ventricles. There is a marked midline shift to the left with compression of the third ventricle. The EEG reveals a generalized slowing of the background activity with frequencies in the theta (4 to 7 Hz) and delta (≤3 Hz) range. EEG calibration: 1 second per horizontal unit, 70 μV per vertical unit.



Niedermeyer's, Electroencephalography VI ed.2011

Chronic phase stroke: frontally predominant GRDA





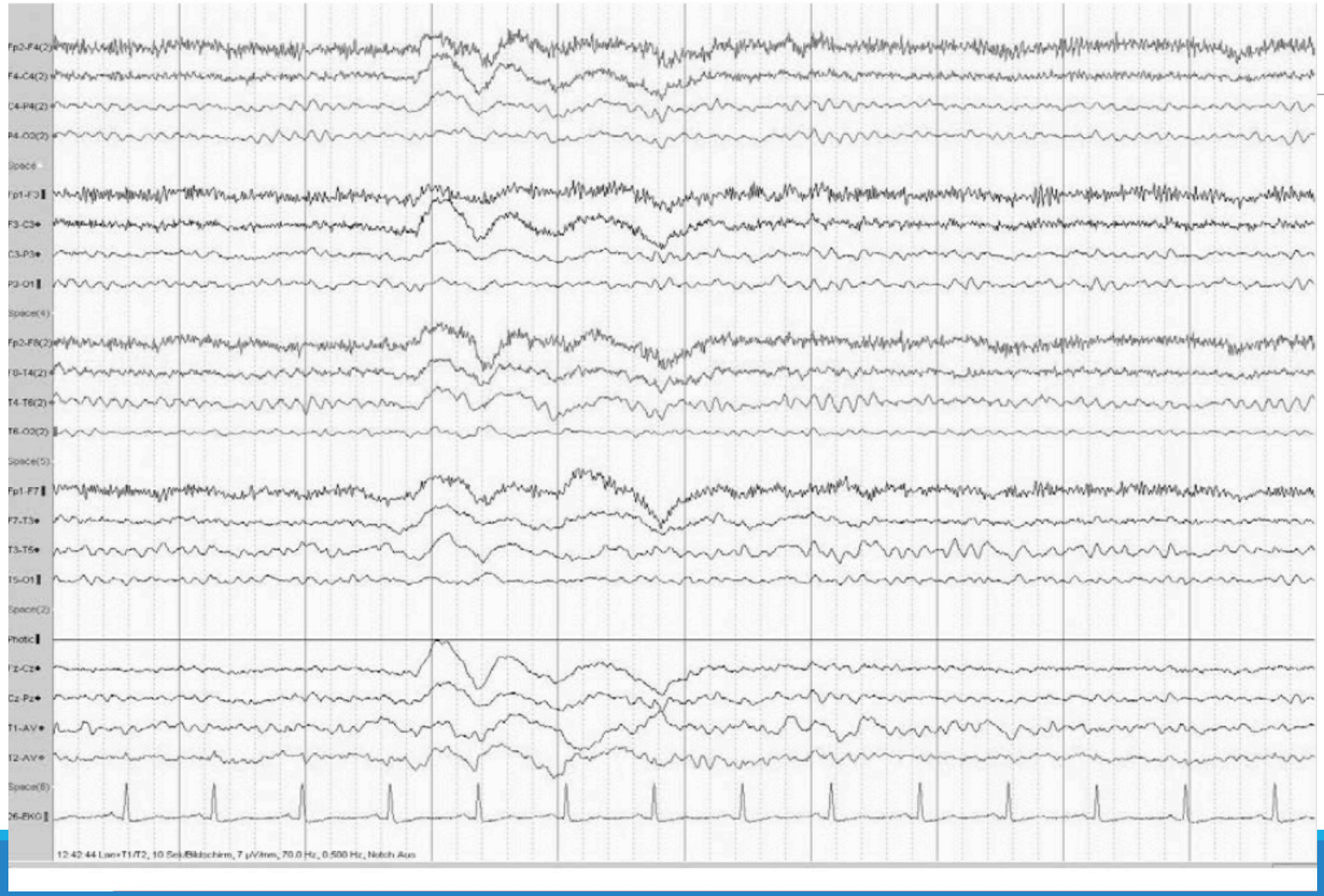
- **Generalized (G;** for this purpose, the term "generalized" refers to any bilateral, bisynchronous and symmetric pattern, even if it has a restricted field [e.g. bifrontal])
- **Lateralized (L;** includes unilateral and bilateral synchronous but asymmetric; includes focal, regional and hemispheric patterns)
- **Bilateral Independent (BI;** refers to the presence of 2 independent [asynchronous] lateralized patterns, one in each hemisphere)
- **Multifocal (Mf;** refers to the presence of at least three independent lateralized patterns with at least one in each hemisphere)

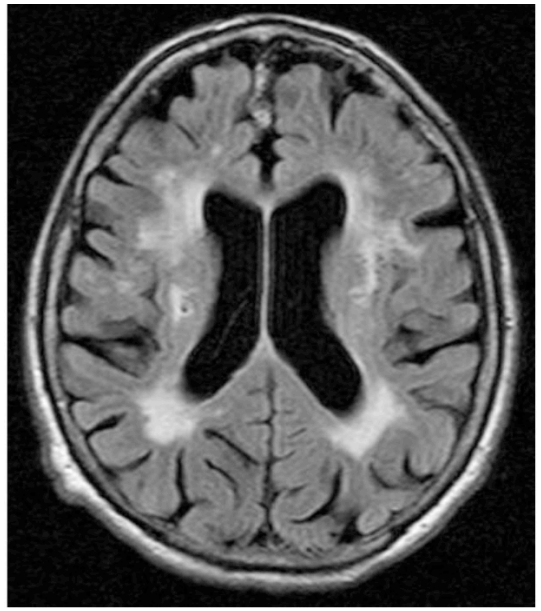
Additional localizing information

i. For Generalized patterns

1. Frontally predominant (defined as having an amplitude in anterior derivations that is at least 50% greater than that in posterior derivations on an ipsilateral ear, average, or non-cephalic referential recording)

TIA e lesioni croniche della bianca





PRES: posterior reversible encephalopathy syndrome

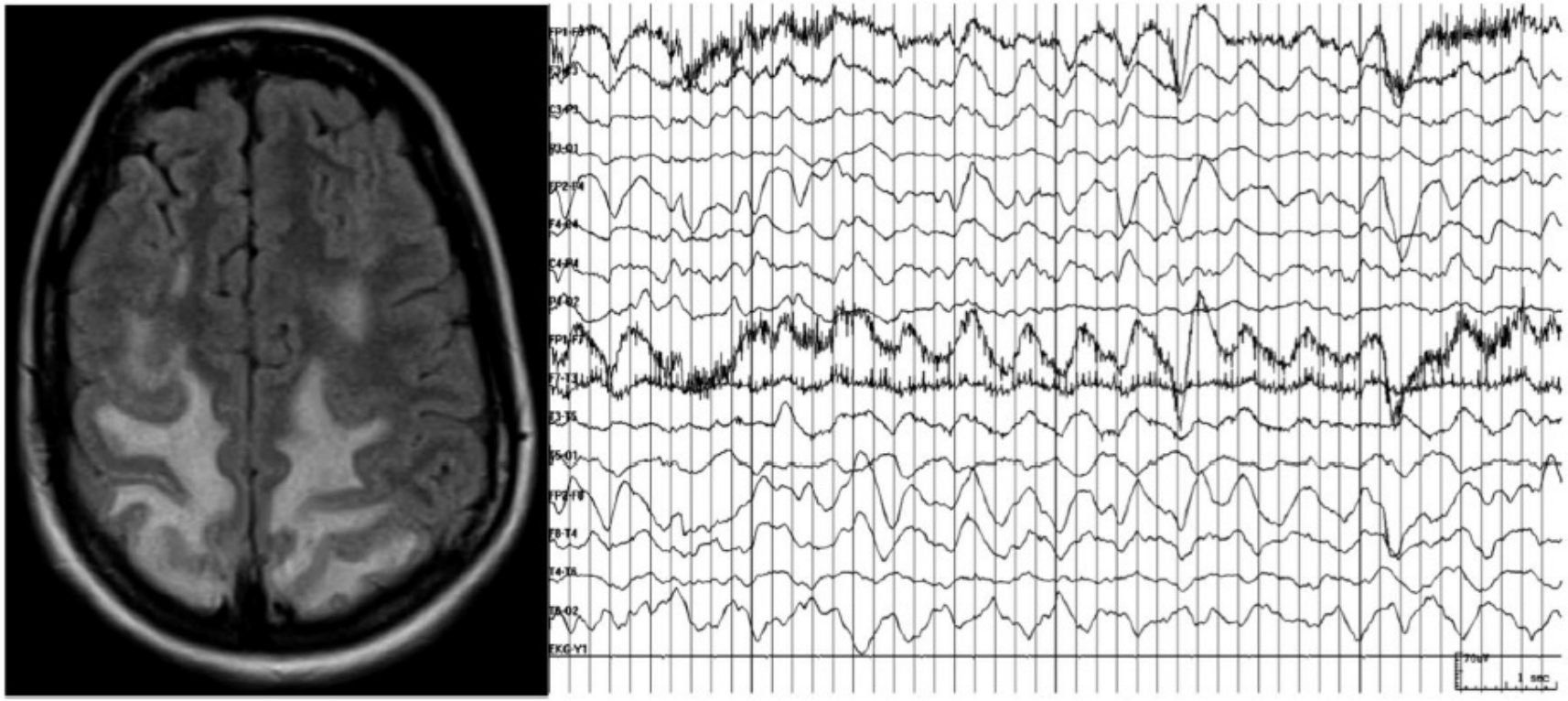


FIG. 4. Delta slowing in a patient with a posterior reversible encephalopathy syndrome. Axial brain MRI shows large and symmetric hyperintense subcortical and less cortical areas in the occipital, parietal, and frontal lobes of both hemispheres on the fluid attenuated inversion recovery sequences. The EEG reveals a generalized slowing of the background activity with frequencies in the delta (≤3 Hz) range and very few superimposed faster frequencies in the theta range (4 to 7 Hz). EEG calibration: 1 second per horizontal unit, 70 μV per vertical unit.

PRES

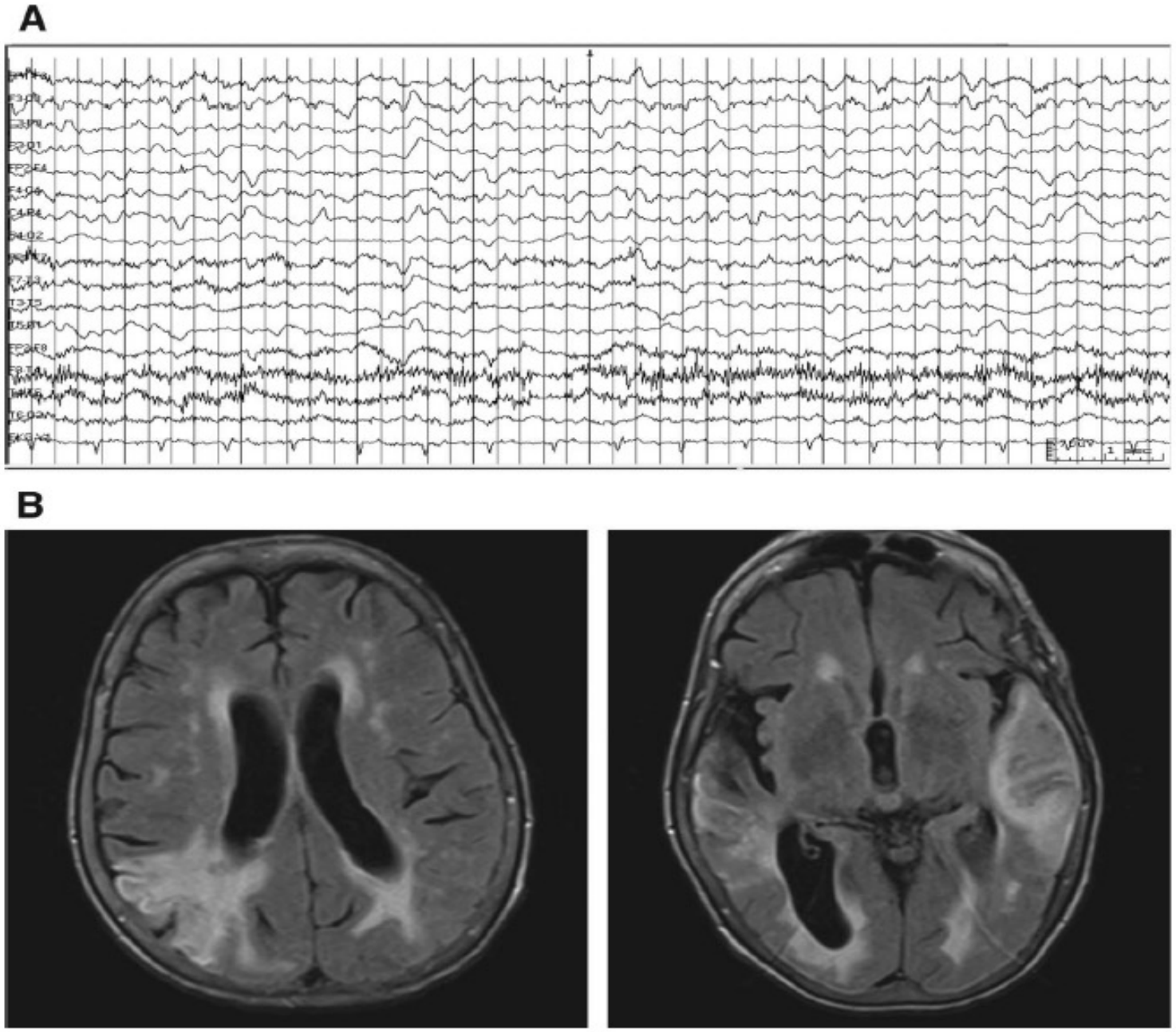


FIG. 9. A, EEG showing slow background activity and intrusion of polymorphic slow activity. B, MRI revealing periventricular white matter subcortical ischemic changes and bilateral cortical changes.

Stroke e crisi epilettiche

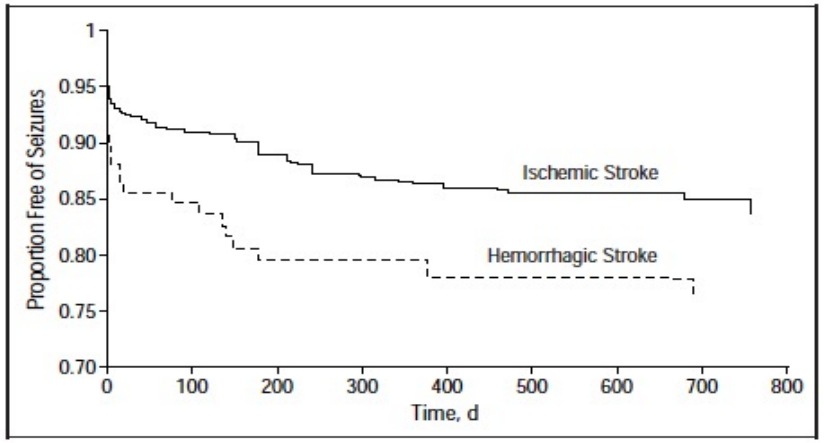


Figure 1. *Kaplan-Meier survival curves for patients with ischemic and hemorrhagic stroke showing the probability of remaining free of seizures after stroke. There was a significant difference (log-rank test) between the seizure event curves (P=.002).*

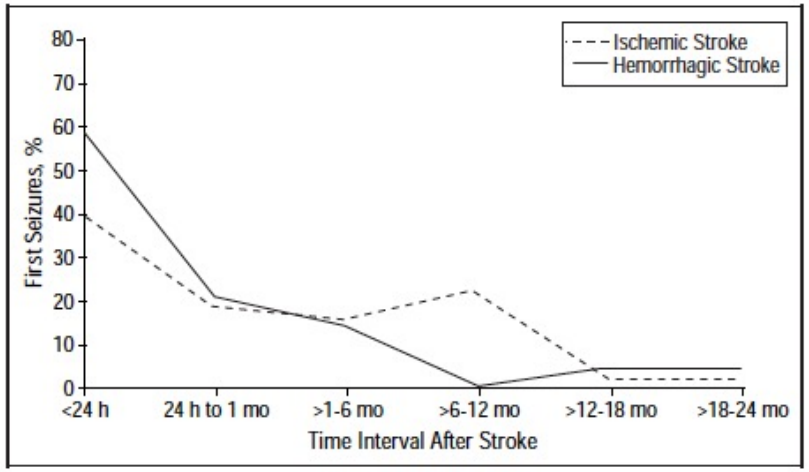


Figure 2. *Occurrence of the first seizure after stroke during set intervals. Most seizures occurred in the first 24 hours after stroke onset.*

Table 2. Cox Proportional Hazards Analysis of Clinical Risk Factors for Seizures After Ischemic Stroke*

	All First Seizures		Recurrent Seizures	
Variable	HR (95% CI)	*P*	HR (95% CI)	*P*
Age (per year of age)	0.99 (0.97-1.00)	.14	0.99 (0.97-1.02)	.64
Hemorrhagic infarction	1.14 (0.62-2.12)	.68	0.35 (0.08-1.54)	.17
High-risk embolic stroke	1.00 (0.67-1.50)	.99	0.68 (0.31-1.48)	.33
Infarct size				
Small	0.57 (0.22-1.50)	.25	0.31 (0.07-1.35)	.12
Medium	0.55 (0.19-1.60)	.27	0.42 (0.11-1.58)	.20
Large	0.84 (0.27-2.57)	.76	0.94 (0.25-3.57)	.93
Cortical location	2.09 (1.19-3.68)	.01	2.13 (0.60-7.53)	.24
Moderate disability	1.63 (0.97-2.75)	.07	3.62 (0.82-15.86)	.19
Severe disability	2.10 (1.16-3.82)	.02	2.63 (0.52-13.30)	.24
Late-onset seizures	. . .	. . .	12.37 (4.74-32.32)	.001

**HR indicates hazard ratio; CI, confidence interval.*

Table 3. Cox Proportional Hazards Analysis of Clinical Risk Factors for Seizures After Hemorrhagic Stroke*

	All First Seizures		Recurrent Seizures	
Variable	HR (95% CI)	*P*	HR (95% CI)	*P*
Age (per year of age)	1.00 (0.98-1.04)	.62	1.00 (0.95-1.10)	.89
Cisternal blood	1.15 (0.85-1.55)	.38	1.00 (0.78-1.36)	.83
Ventricular blood	1.00 (0.91-1.12)	.86	0.94 (0.78-1.13)	.49
Cortical location	3.16 (1.35-7.40)	.008	1.60 (0.35-7.17)	.55
Hemorrhage size				
Small	1.15 (0.40-3.25)	.80	2.00 (0.32-12.74)	.46
Medium	1.04 (0.35-3.13)	.94	1.60 (0.23-10.93)	.63
Large	0.78 (0.23-2.65)	.69	0.74 (0.05-9.88)	.82
Moderate disability	1.25 (0.44-3.46)	.68	. . .	. . .
Severe disability	0.51 (0.17-1.53)	.23	. . .	. . .
Late-onset seizures	. . .	. . .	3.38 (0.58-19.54)	.17

**HR indicates hazard ratio; CI, confidence interval. Neurological disability could not be used in the proportional hazards analysis of recurrent seizures because of limited patient numbers in this subset.*

Bladin et al., 2000 Arch Neurol

EEG Patterns and Epileptic Seizures in Acute Phase Stroke

O. Mecarelli[a] S. Pro[a] F. Randi[a] S. Dispenza[a] A. Correnti[b] P. Pulitano[a]
N. Vanacore[c] E. Vicenzini[a] D. Toni[b]

EEG revealed

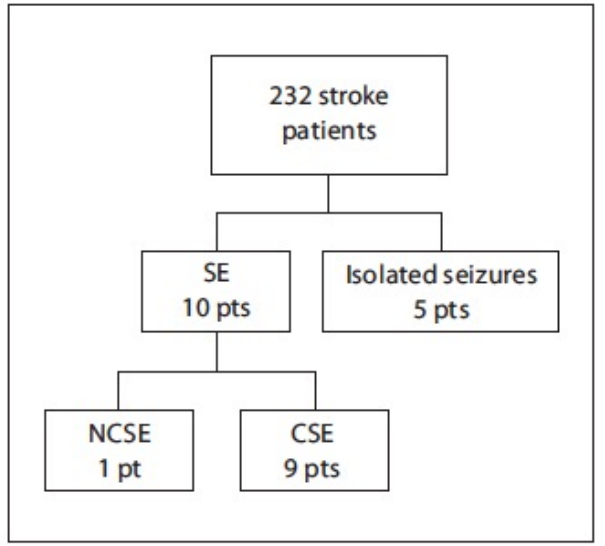
1. focal or diffuse slowing of background activity in 195 patients (84%),

--0/195 patients with either focal or diffuse slowing of background activity showed epileptic seizures.

2. Epileptiform focal abnormalities in 23 patients (10%)

-- LPDs in 14 patients (6%),

-- 3/23 patients with epileptiform focal abnormalities presented isolated partial motor seizures without secondary generalization.



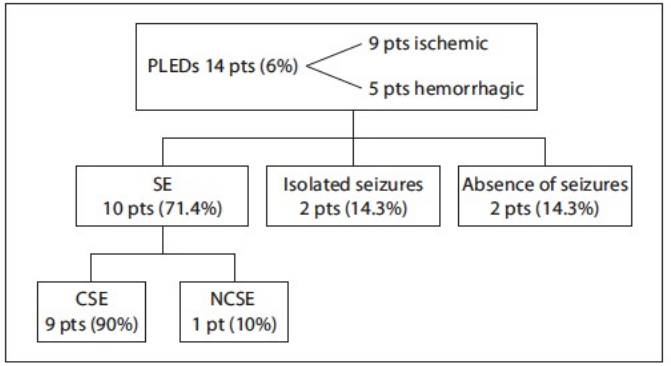
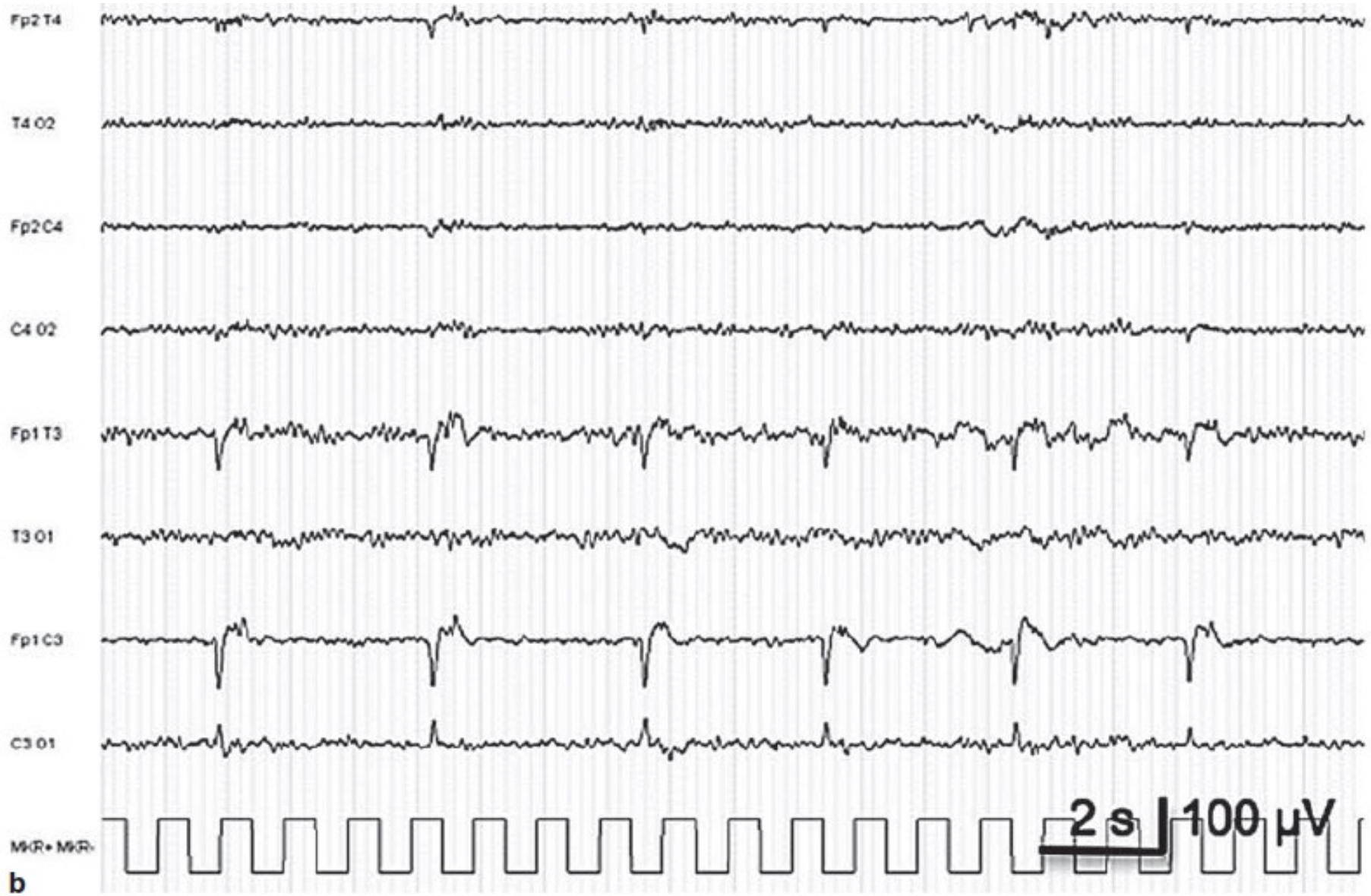


Fig. 1. Incidence of PLEDs and its clinical correlations. CSE = Convulsive status epilepticus; NCSE = non-convulsive status epilepticus.

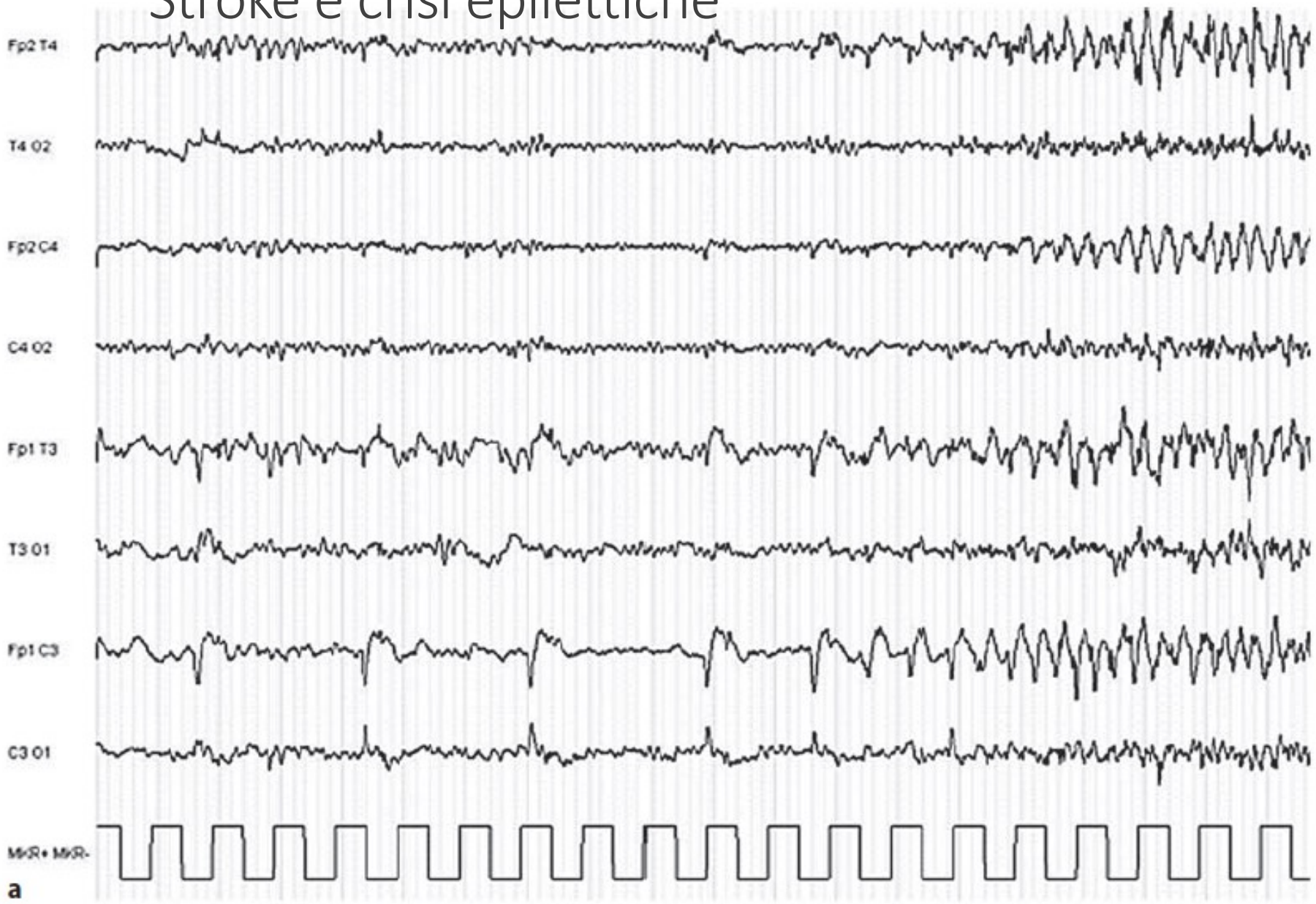


b

Stroke e crisi epilettiche

N=232 pts

EEG<24hh



Mecarelli et al., 2011

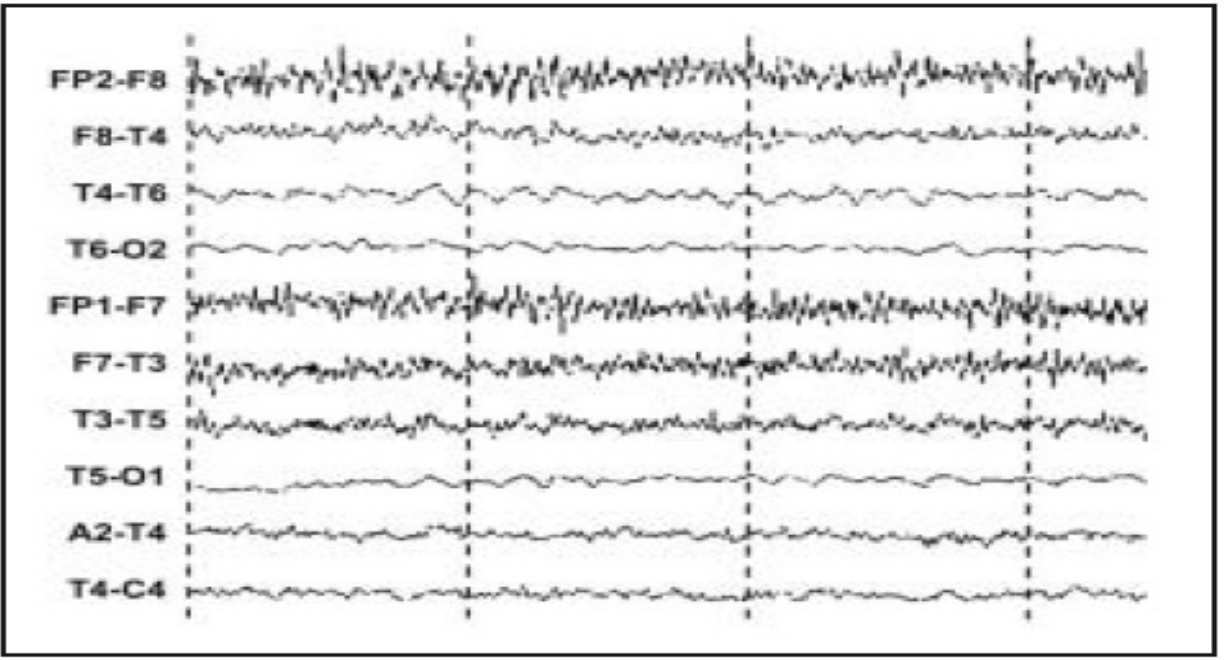


Figure 1. Electroencephalogram (EEG) obtained during the patient's waking state as patient responded appropriately to questions (sensitivity = 7 µV/mm, high-frequency filter = 35 cycles/second, low-frequency filter = 0.53 cycles/second). The EEG is characterized by theta frequency slowing and muscle artifact.

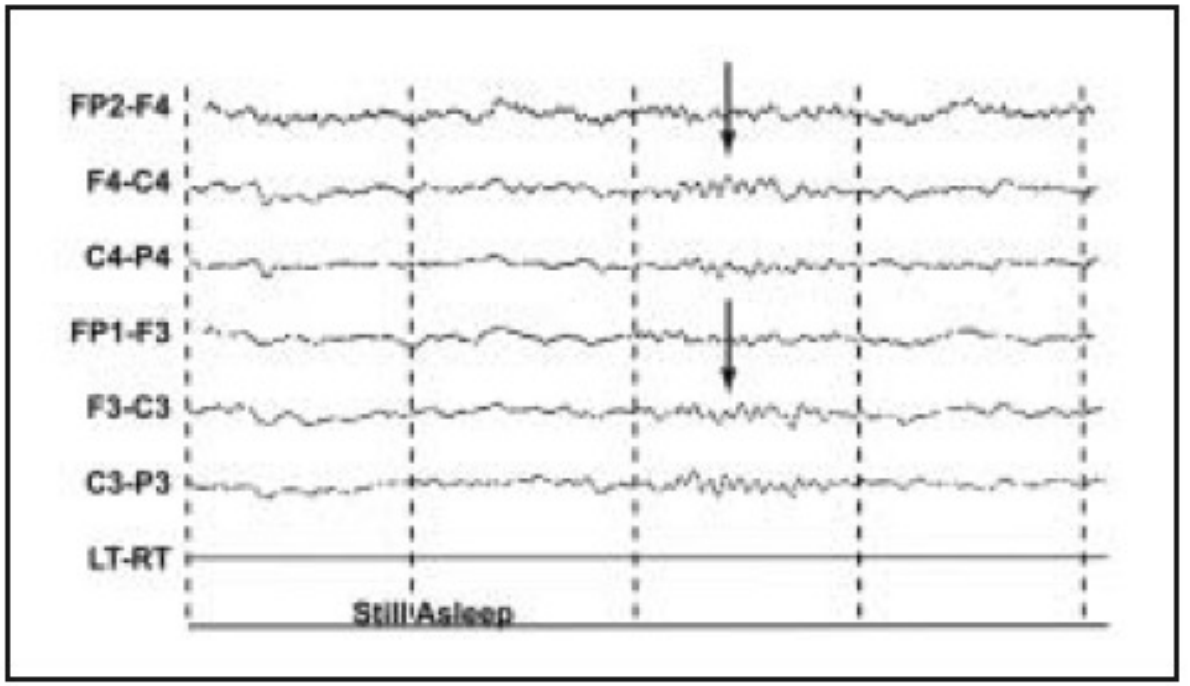
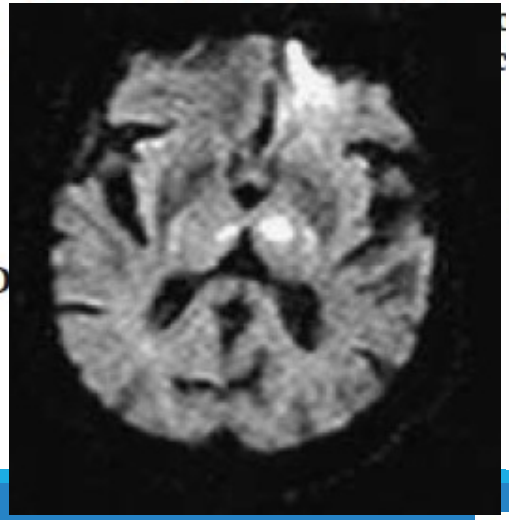
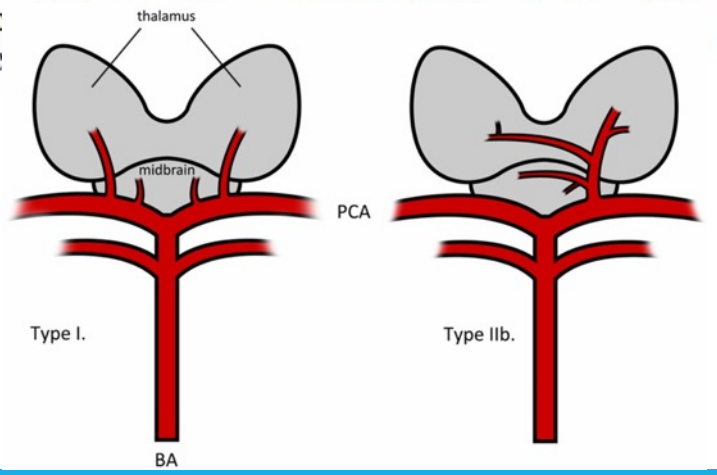


Figure 2. Electroencephalogram (EEG) obtained 9 minutes after Figure 1 EEG during the patient's episode of unresponsiveness. Note the 14-Hz sleep spindles prominent in the central channels (arrows) (sensitivity = 5 µV/mm, high-

Paroxysmal Sleep as a Presenting Symptom of Bilateral Paramedian Thalamic Infarctions

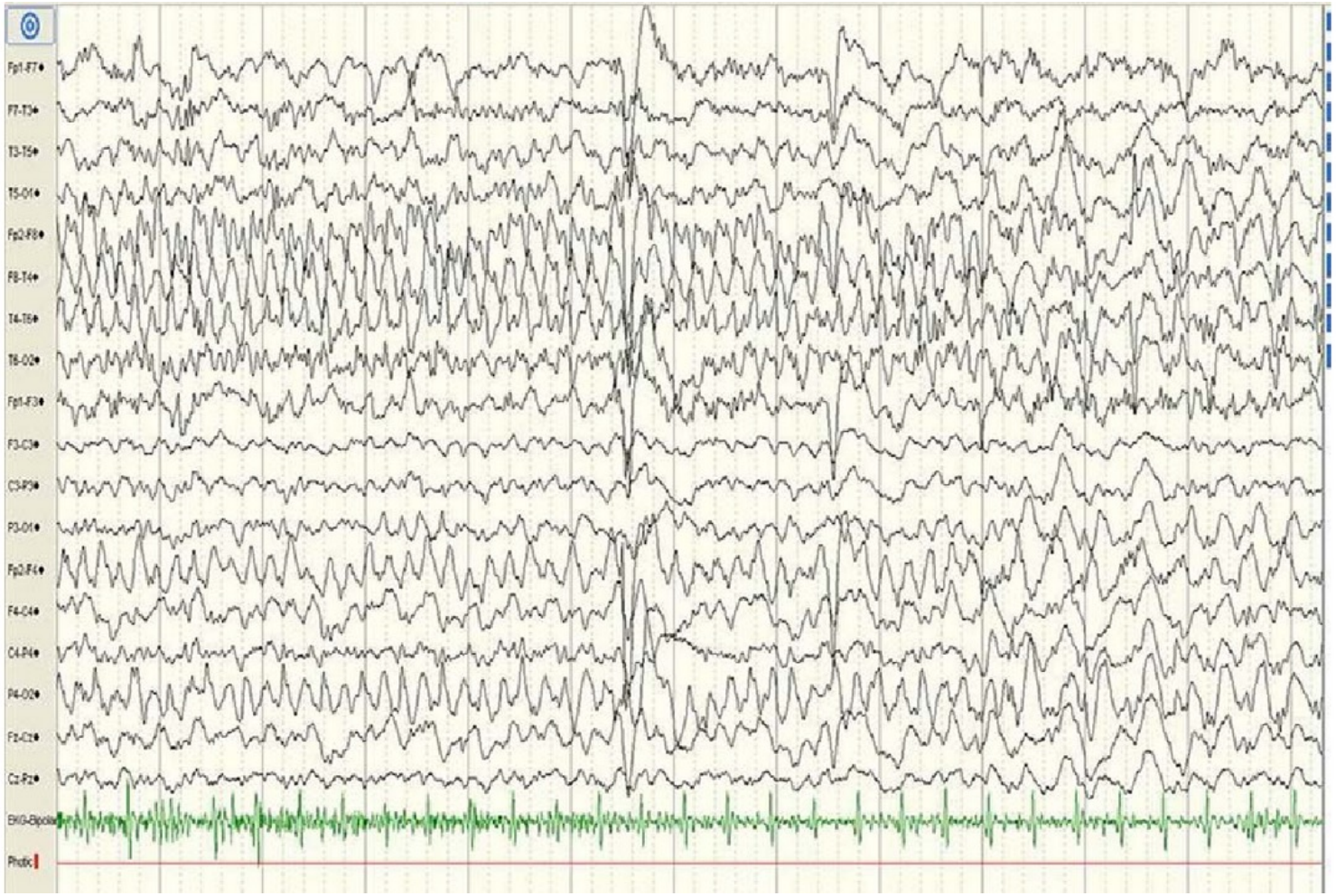
BRYAN BJORNSTAD, MD; SCOTT H. GOODMAN, MD; JOSEPH I. SIRVEN, MD; AND D

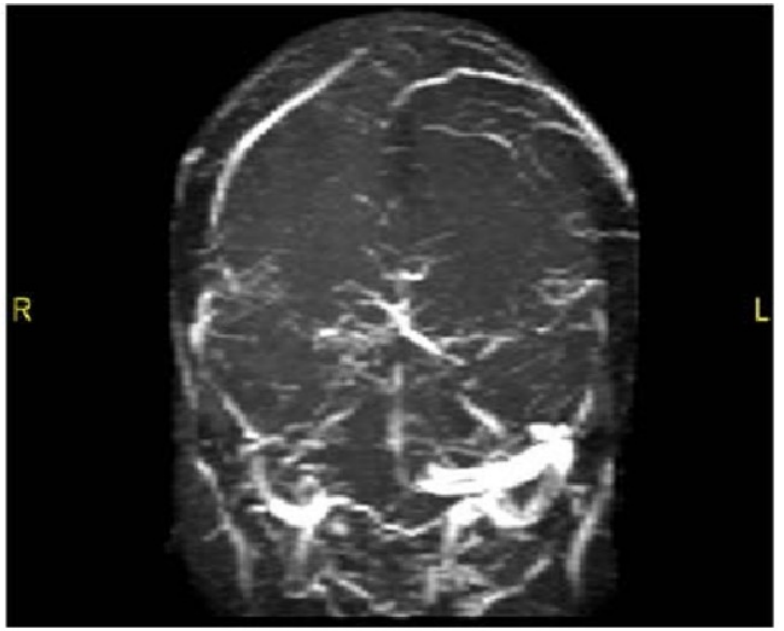




Mayo Clin Proc. 2003;78:347-349

Cerebral venous sinus thrombosis





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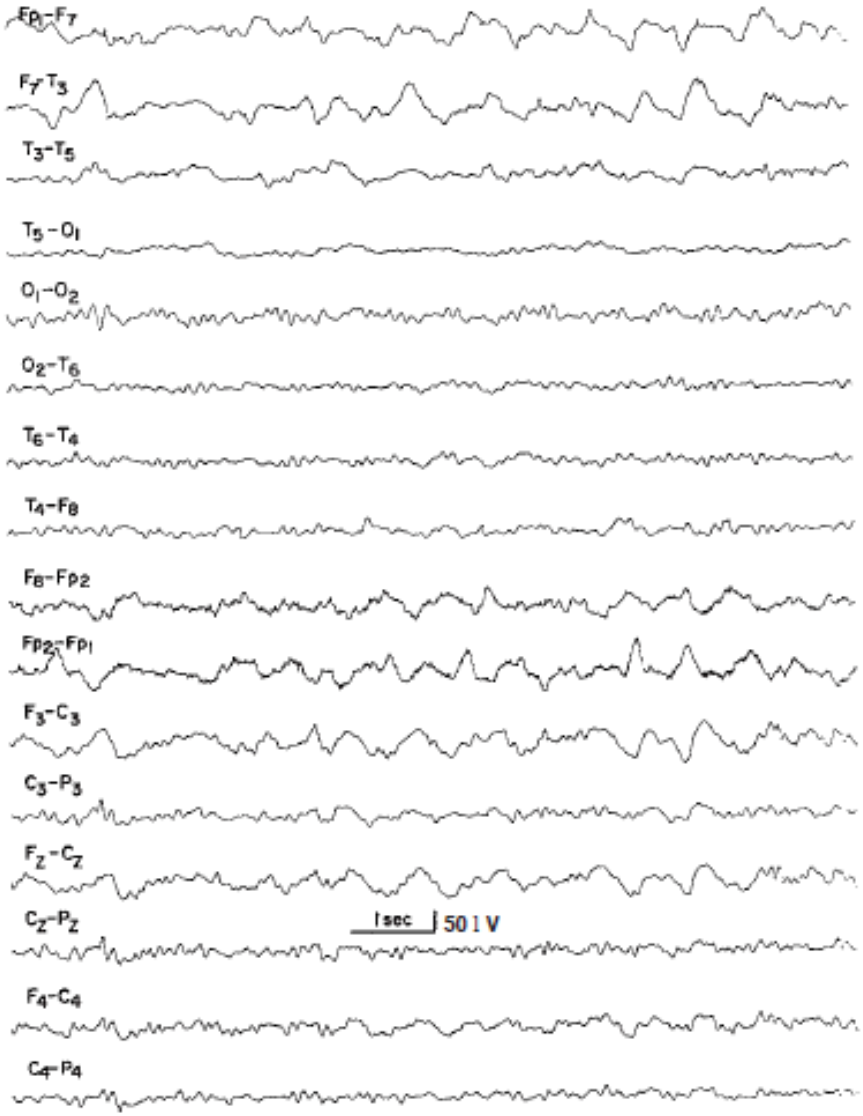


Figure 19.1 Age 57 years. Acute cerebrovascular event on the day before this record was obtained. There is marked left frontotemporal polymorphic delta activity. Also note alpha depression and loss of detail over left posterior quadrant. The right hemisphere and especially the right frontal area show some degree of delta activity.

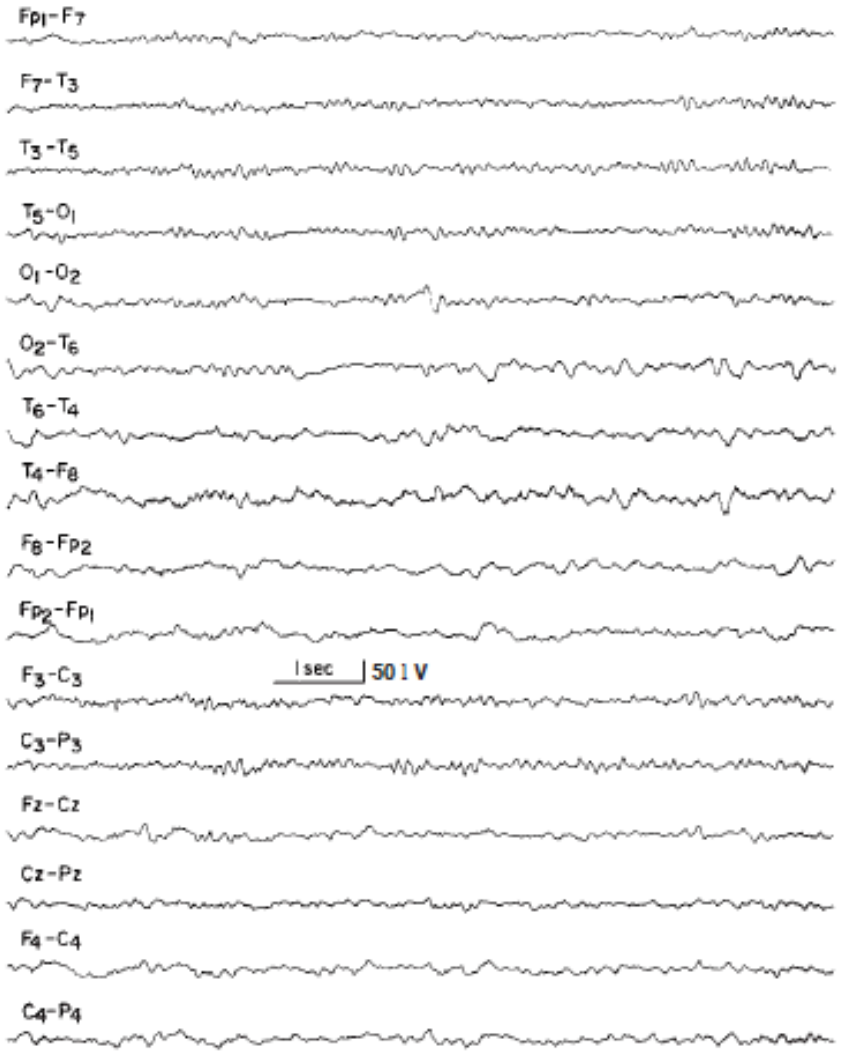
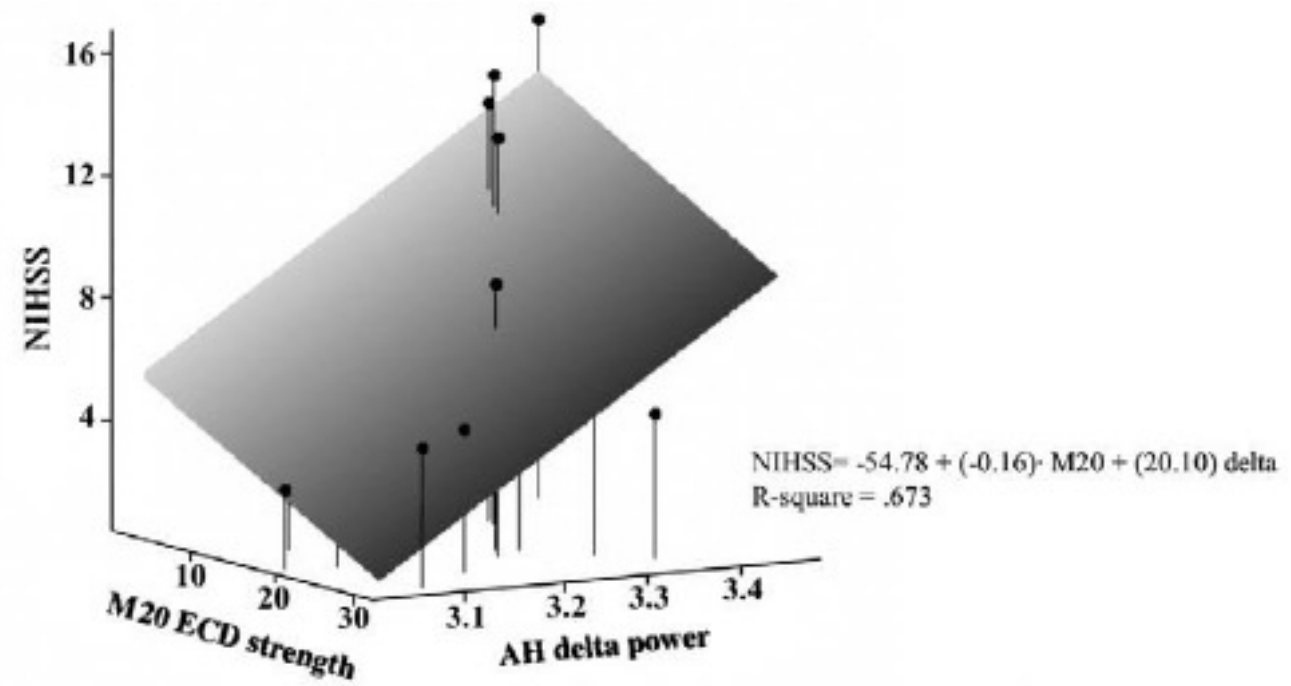
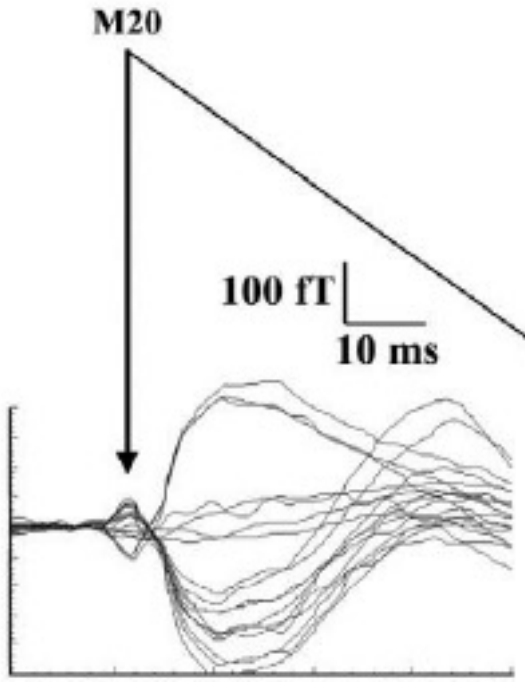
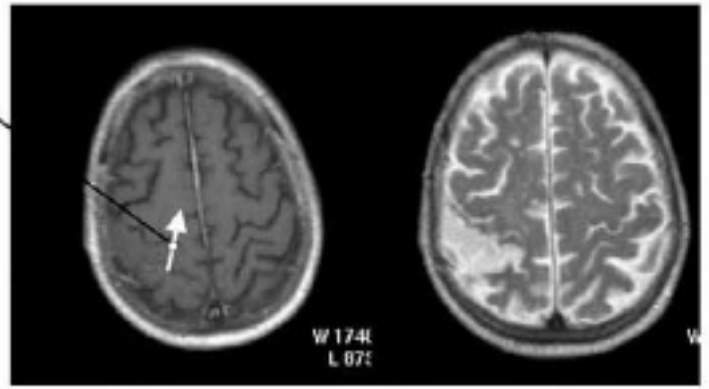
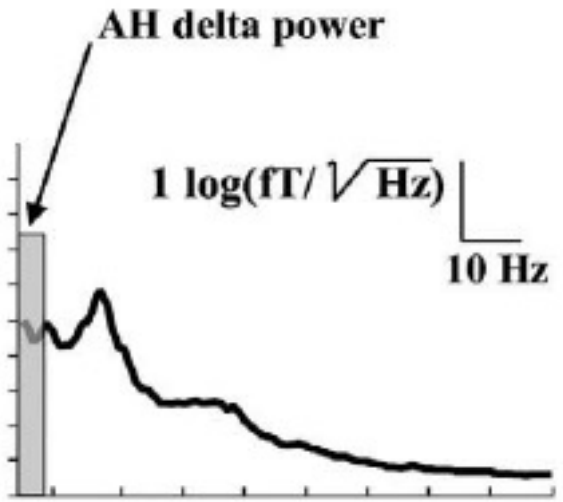


Figure 19.2 Recent acute cerebrovascular ischemia (3 days earlier) due to right middle cerebral artery thrombosis and good CT scan evidence of infarction in the corresponding territory. Acute left hemiplegia. Age 64 years. Patient awake; right-sided alpha diminished and a large zone of mixed 3 to 6 per second activity involving most of the right hemisphere.

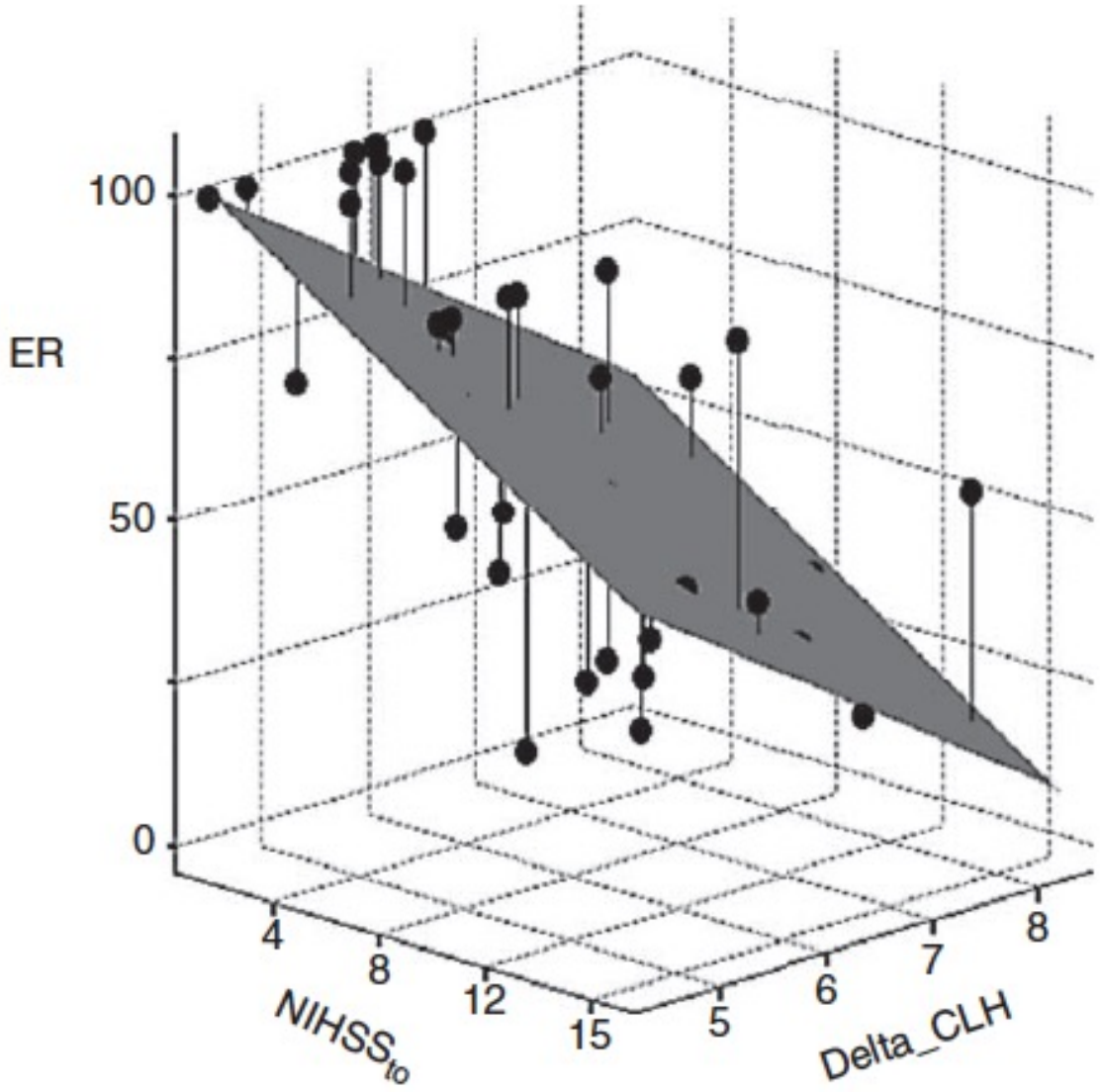






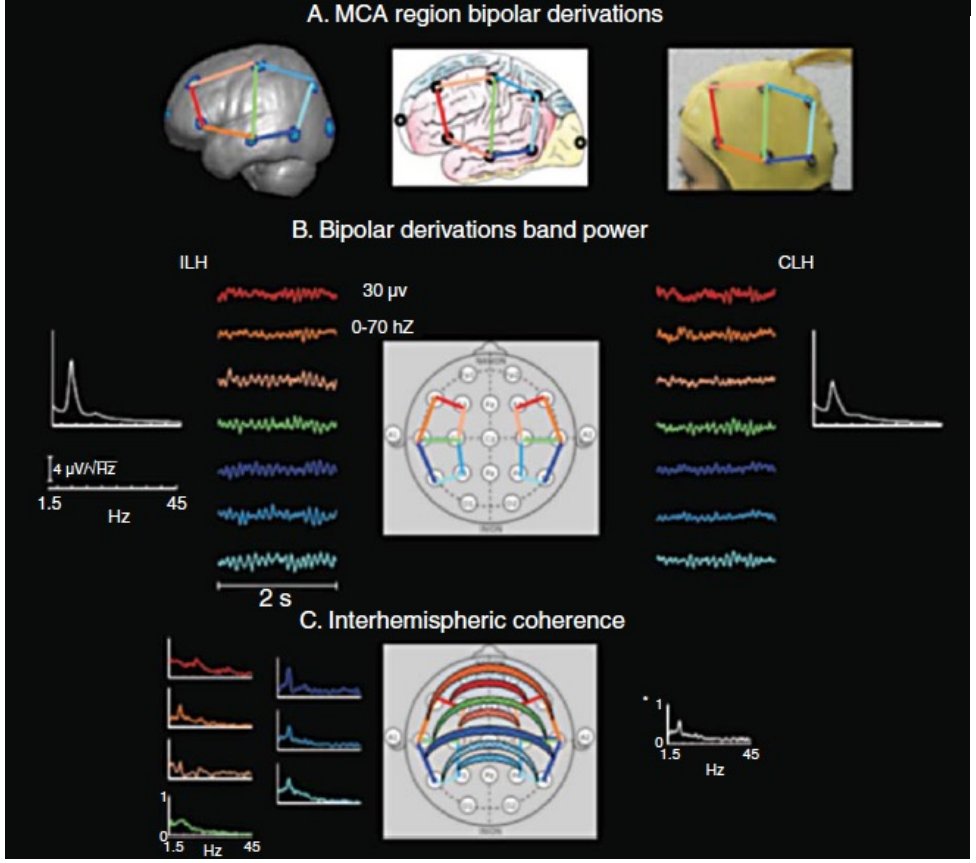


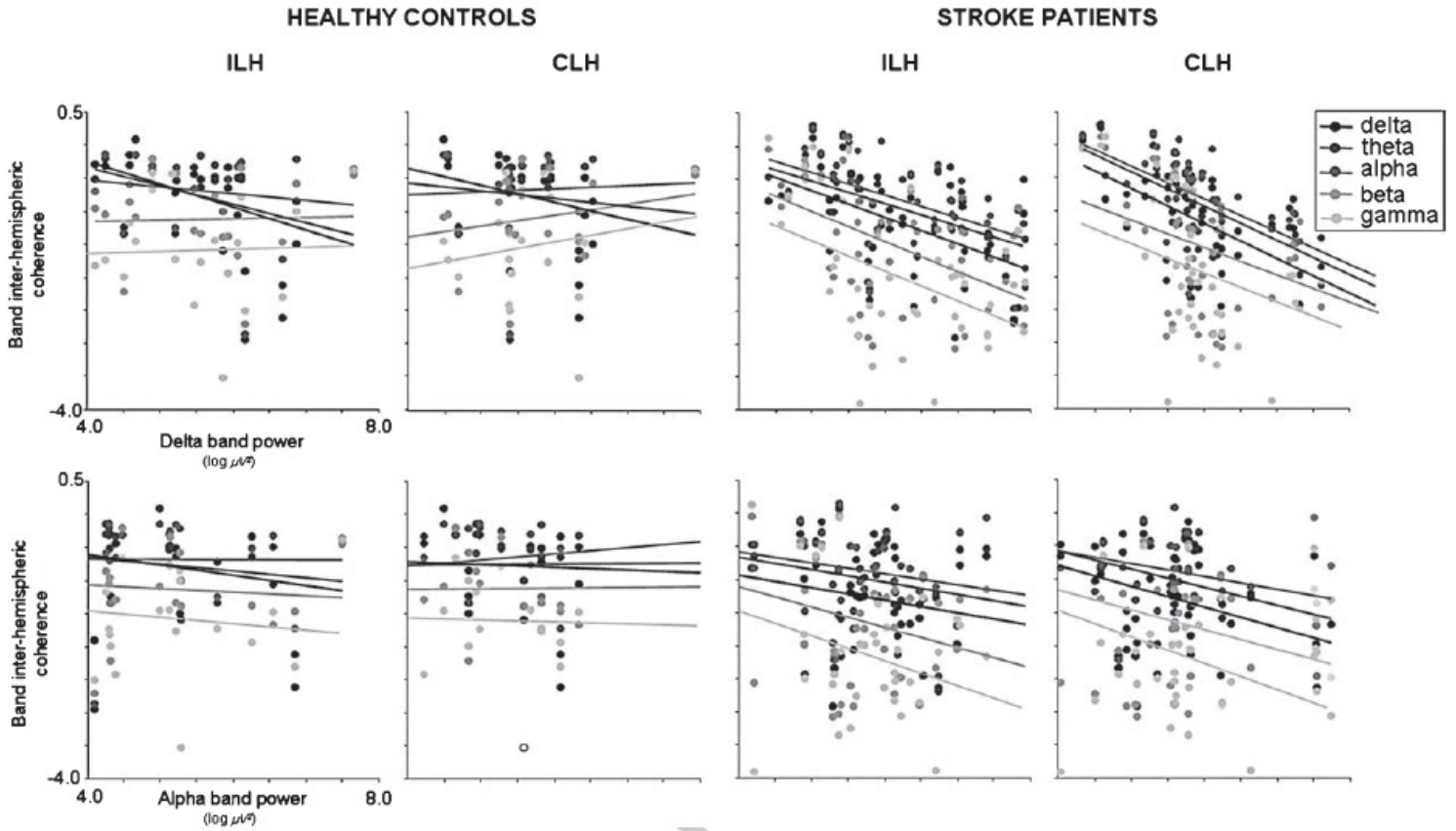
Assenza et al., 2009

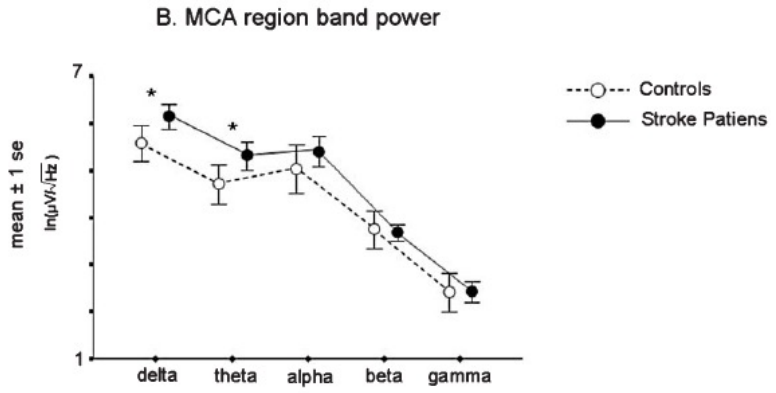


Effective recovery

Assenza et al., 2009



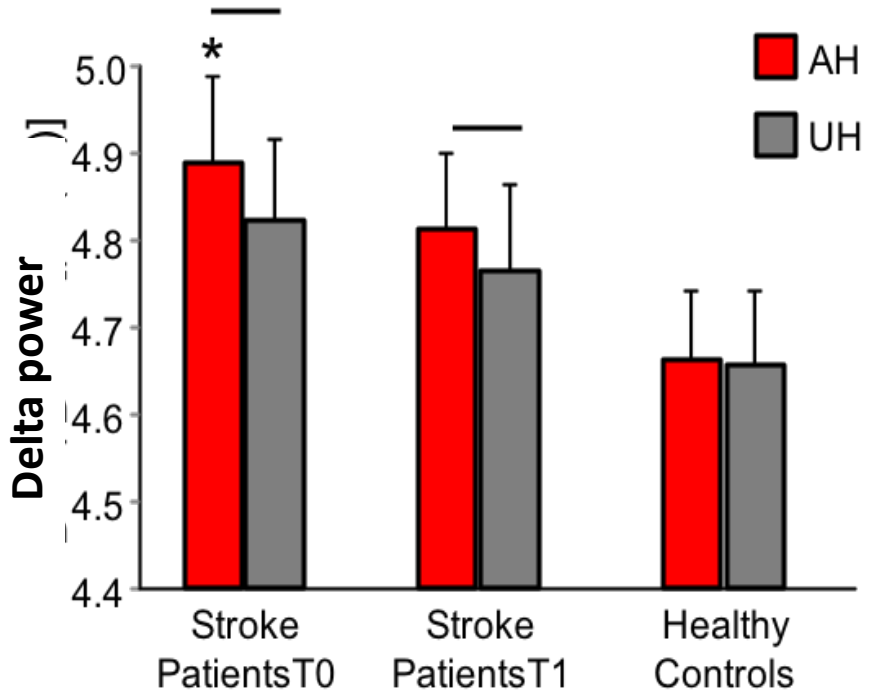




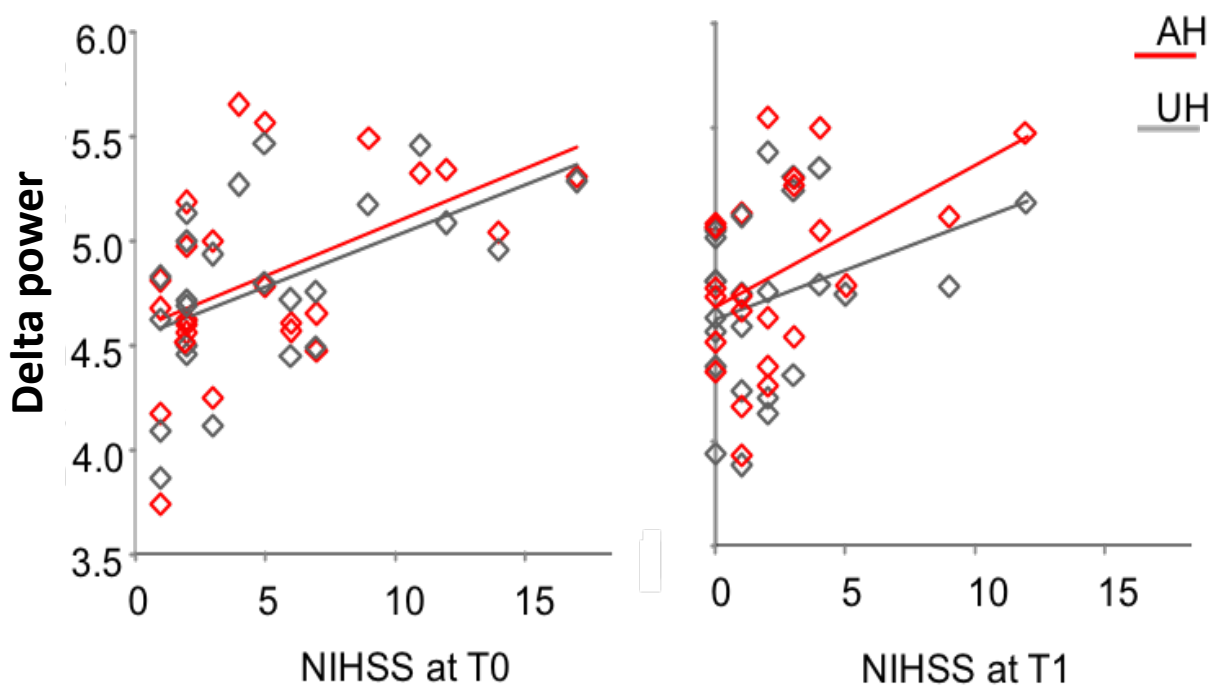
Assenza et al., 2013

CLH: contralesional hemisphere
ILH: psilesional hemisphere

A



B



AH: affected hemisphere
UH: unaffected hemisphere

T0= first week after stroke
T1:= 1 month after stroke

Zappasodi et al., NRR 2019

Traumi cranici

Traumi cranici

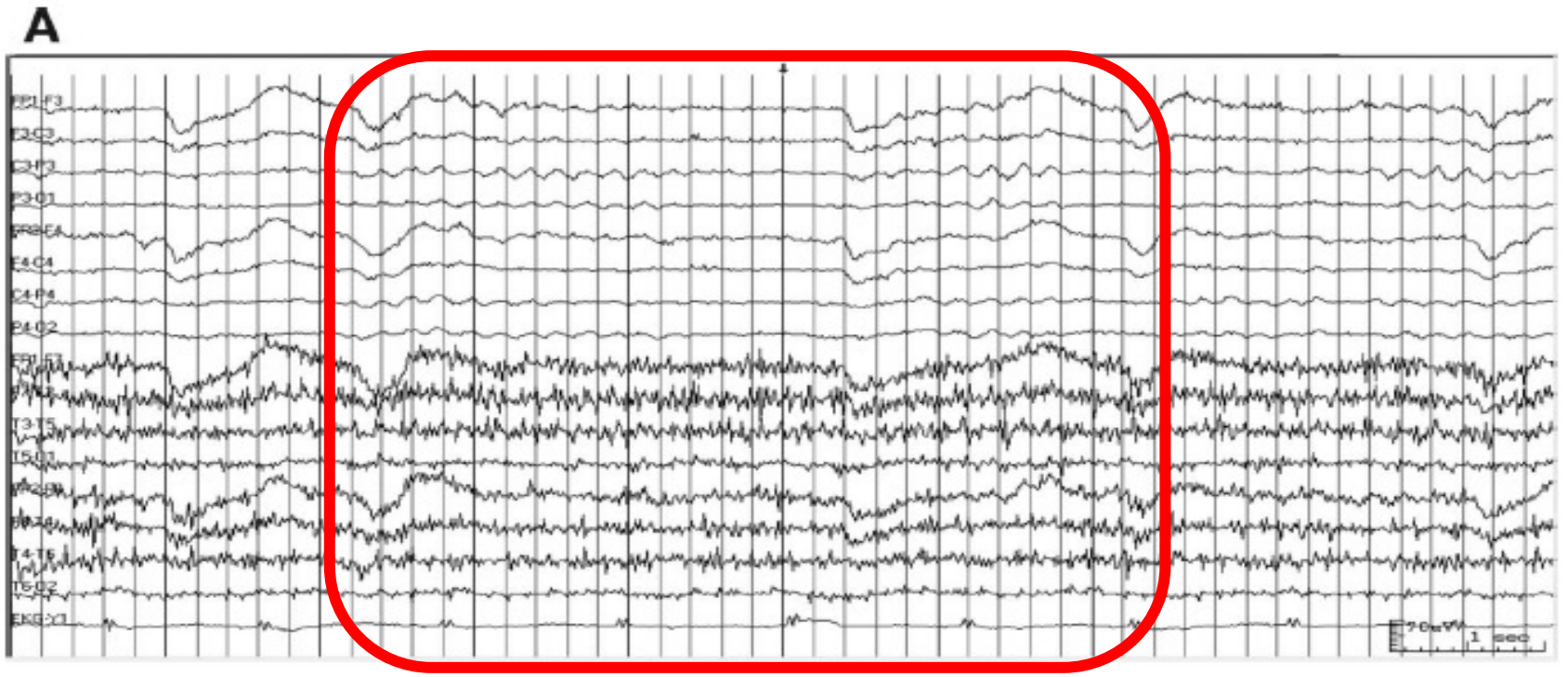


FIG. 5. A, EEG between eyeblinks showing a significant background slowing to 5 Hz without excess slow activity, indicating cortical rather than subcortical dysfunction. Note the preserved background reactivity, which mostly suggests a relatively good prognosis. B, MRI showing no significant subcortical white matter disease.

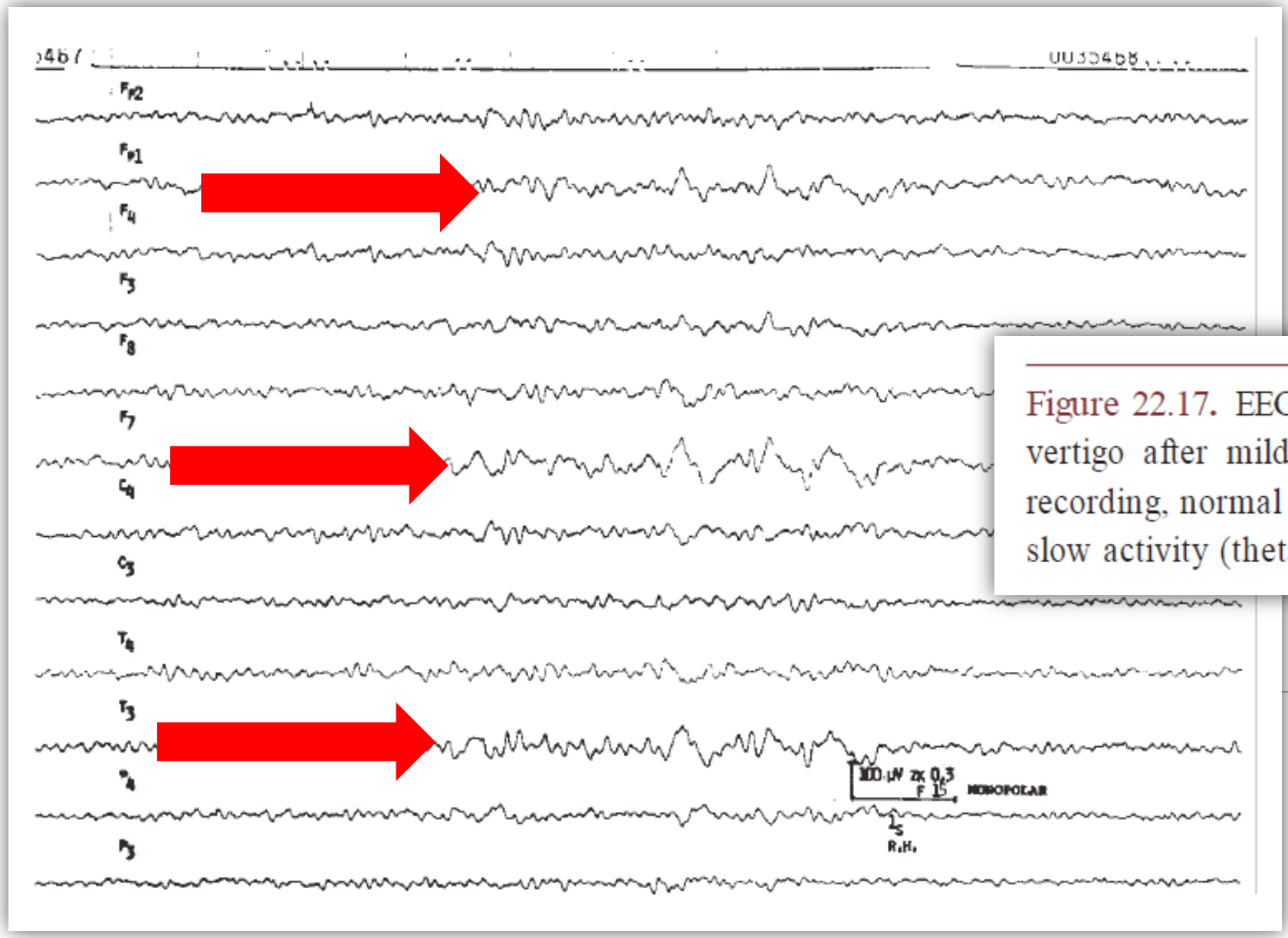
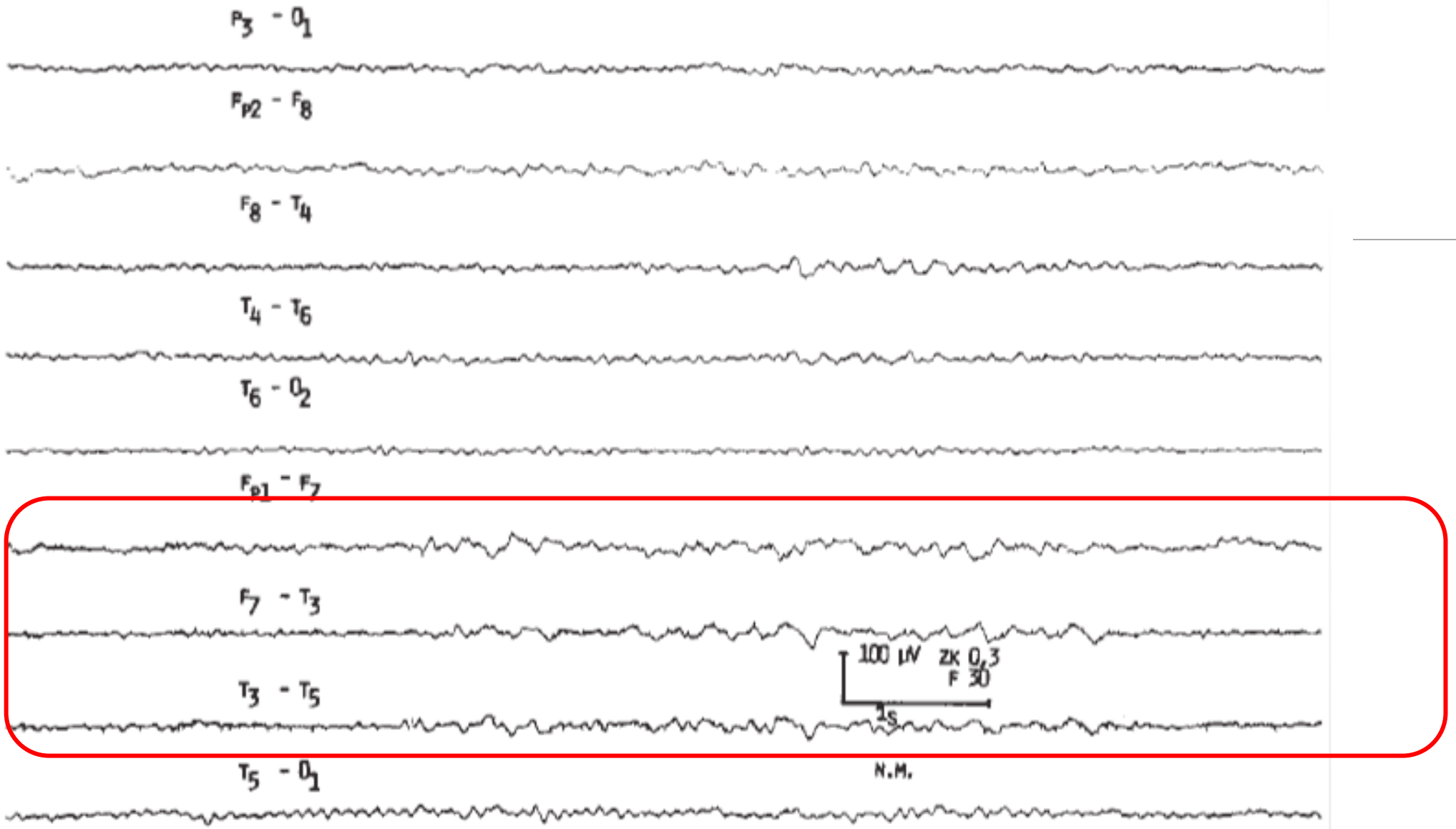


Figure 22.17. EEG from a 35-year-old man complaining of vertigo after mild cerebral contusion. At the time of EEG recording, normal neurologic status and CT scan. Intermittent slow activity (theta and delta) in left frontotemporal region.



EON & TC= Normale, vertigine soggettiva

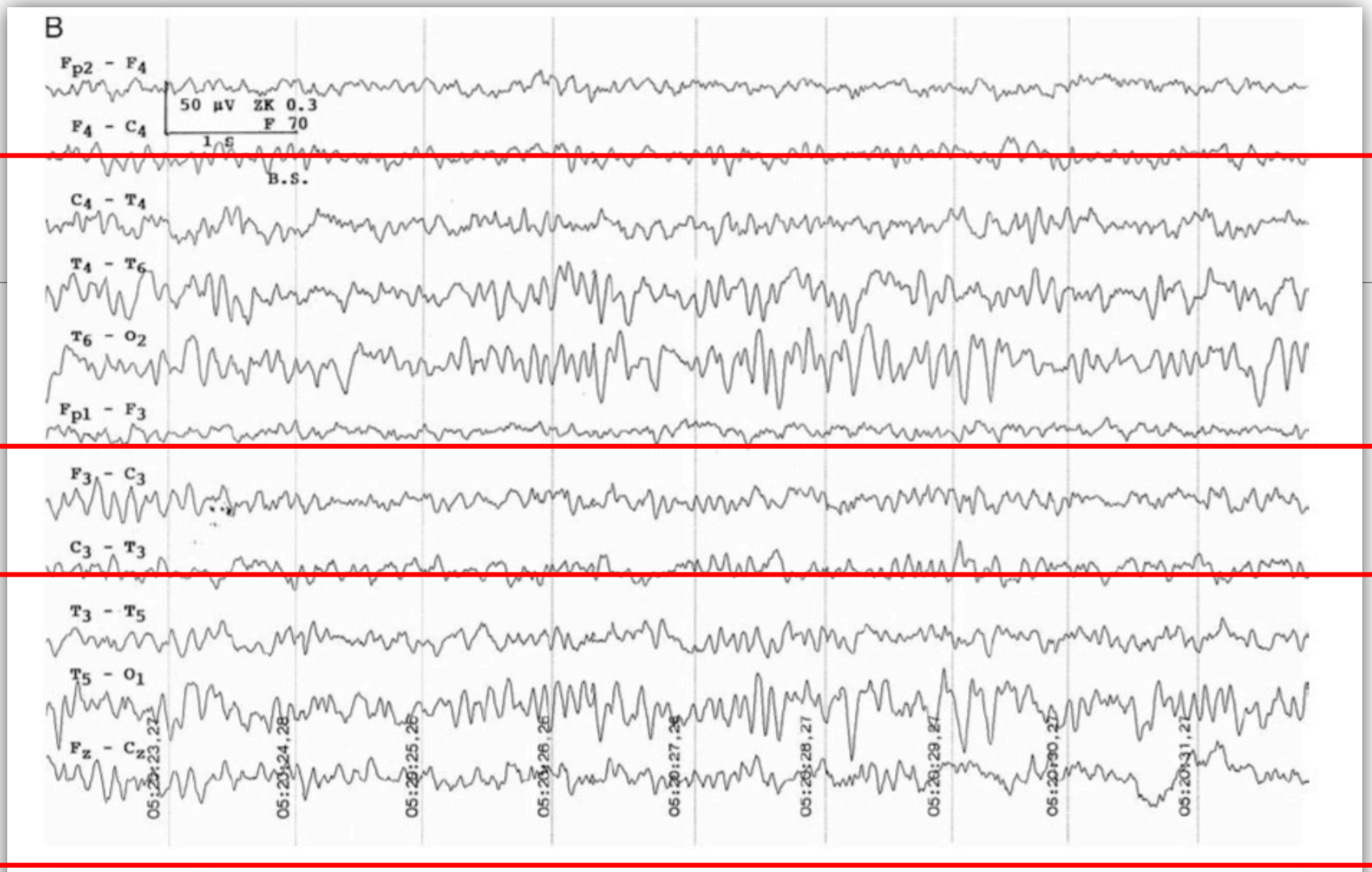
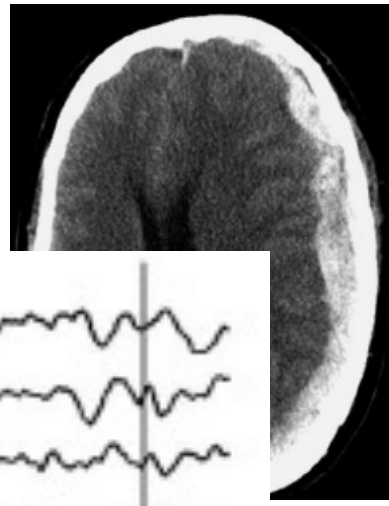
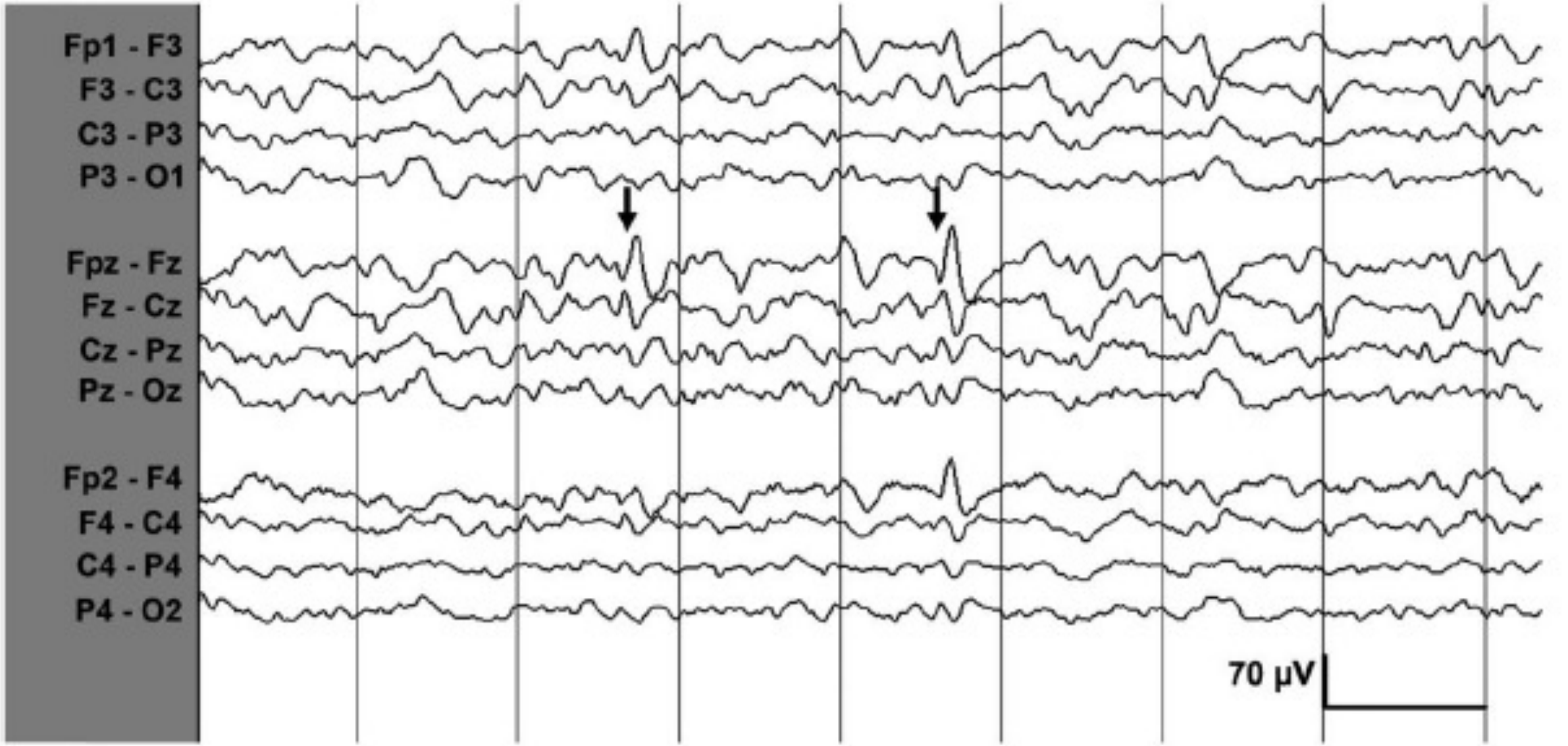


Figure 22.19 **A:** EEG from a 6-year-old girl who had a fall on the back of her head after an unexpected push while playing. She had a period of blurred vision, delayed answers to questions, disorientation, drowsiness, and stereotyped finger movements, which lasted about 1 hour. The CT and magnetic resonance imaging scans were normal. At the time of the EEG recording 2 days after the accident, the patient was alert with no neurologic or mental deficit. The EEG showed high-voltage occipital delta activity intermingled with sharp transients. **B:** EEG from the same patient 3 days later within the range of normal.

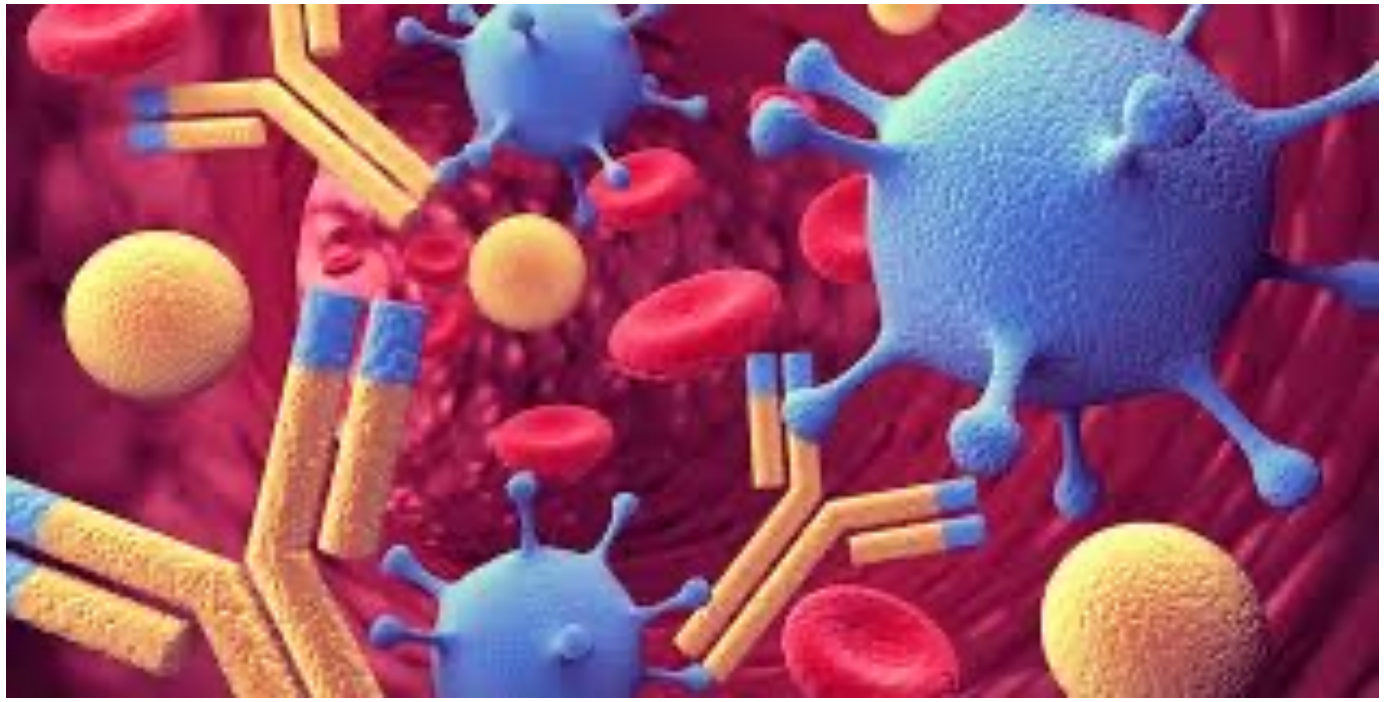
SUBDURAL HAEMATOMA



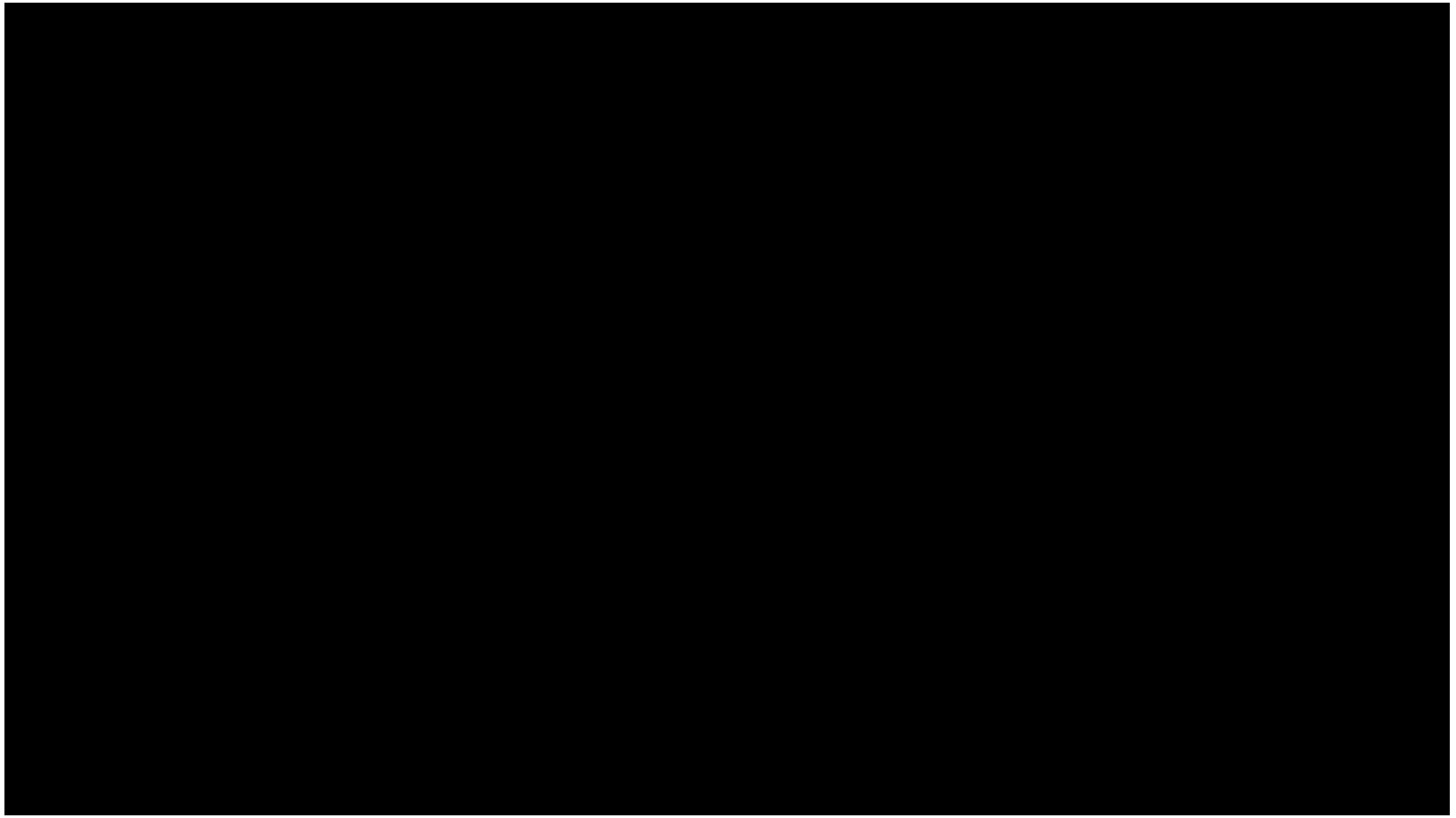


evacuated from the left side.

Encefalopatie autoimmuni



Anti-LGI encephalitis Facial-brachial seizures



Quali anomalie epilettiformi EEG ci aspettiamo in questa persona durante gli episodi?

1. LPD
2. GRDA
3. GPD
4. Crisi focale sulla corteccia motoria primaria controlaterale
5. Nessuna

Anti-LGI encephalitis Facial-brachial seizures

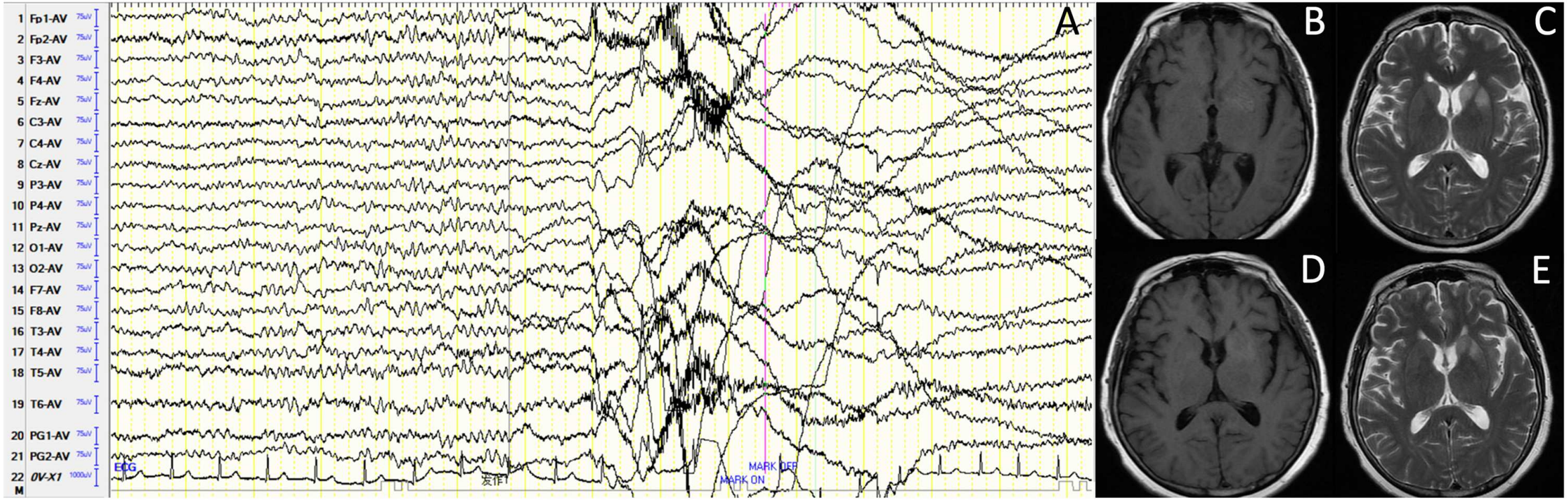
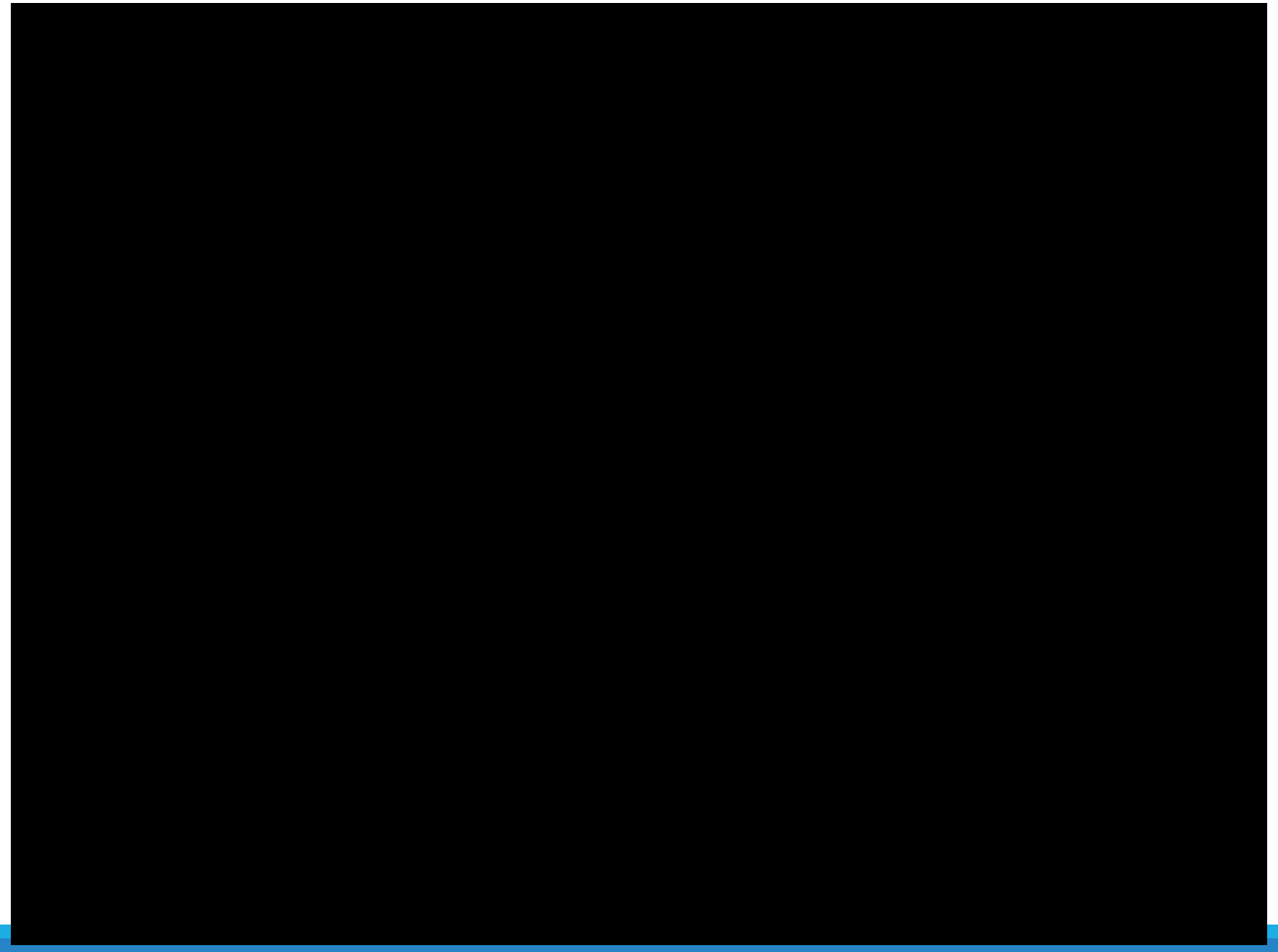
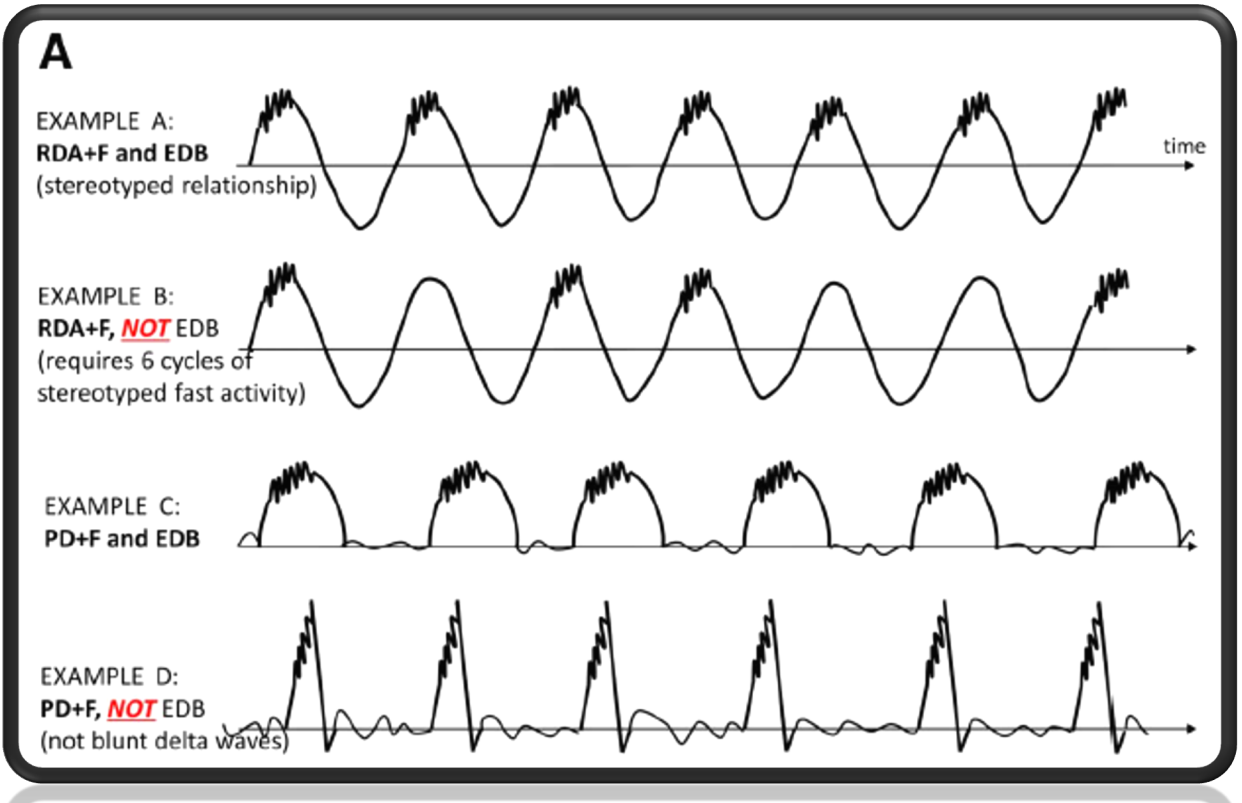


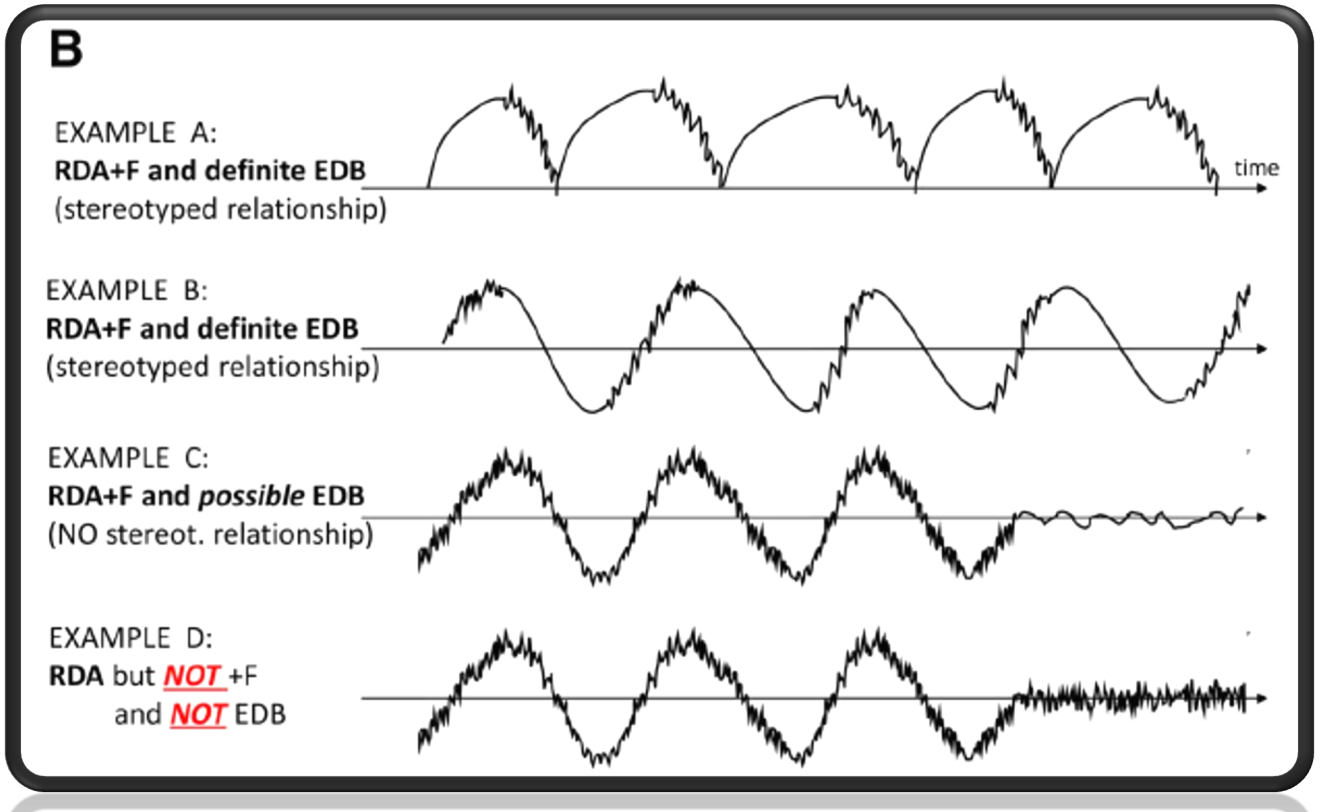
FIGURE 2 | Case 9, female, 63 years old, who was diagnosed with leucine-rich glioma-inactivated 1 protein (LGI1) antibody-associated autoimmune encephalitis (AE). **(A)** Ictal electroencephalogram (EEG) of faciobranchial dystonia seizure (FBDS) showed that 1 s before the clinical onset, the amplitude of all the leads suppressed, followed by artifact of movements, which continued for 5 s, and then recovered to background. **(B,C)** Nineteen days after onset and before immunotherapy, brain MRI showed high T1/T2 signal on the left basal ganglia (caudal nucleus and lenticular nucleus). **(D,E)** Thirty-three days after onset (10 days after immunotherapy), brain MRI showed significant improvement in the high T1/T2 signal on the left basal ganglia.

Encefalite anti-NMDAR

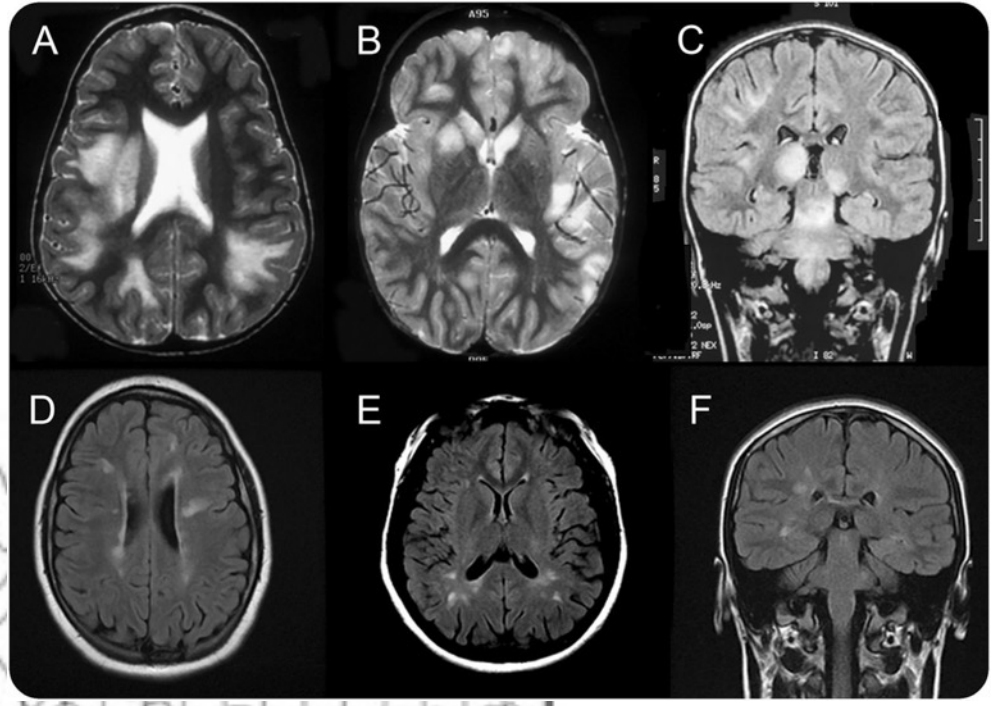


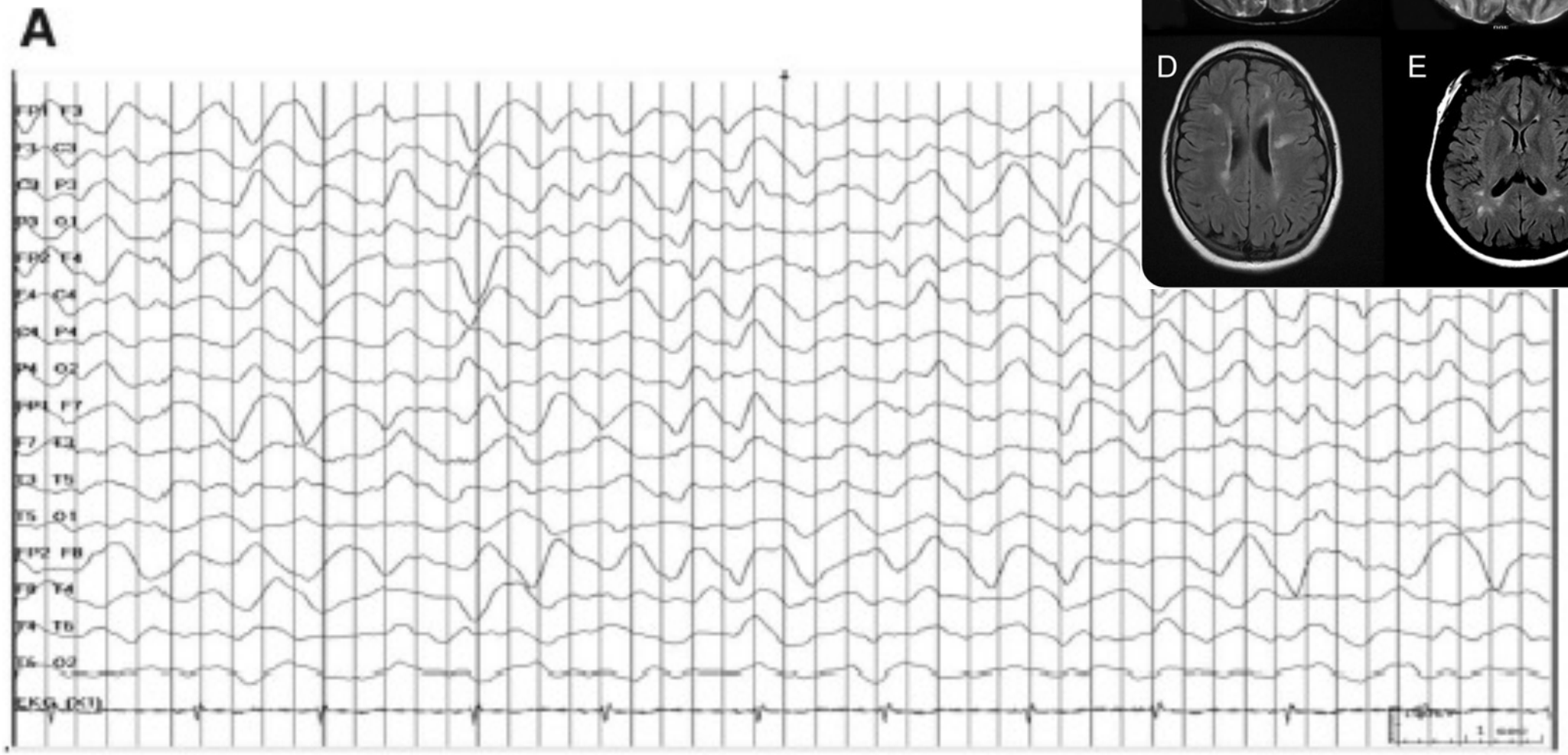
Encefalite anti-NMDAR





ADEM - Encefalomielite Acuta Disseminata



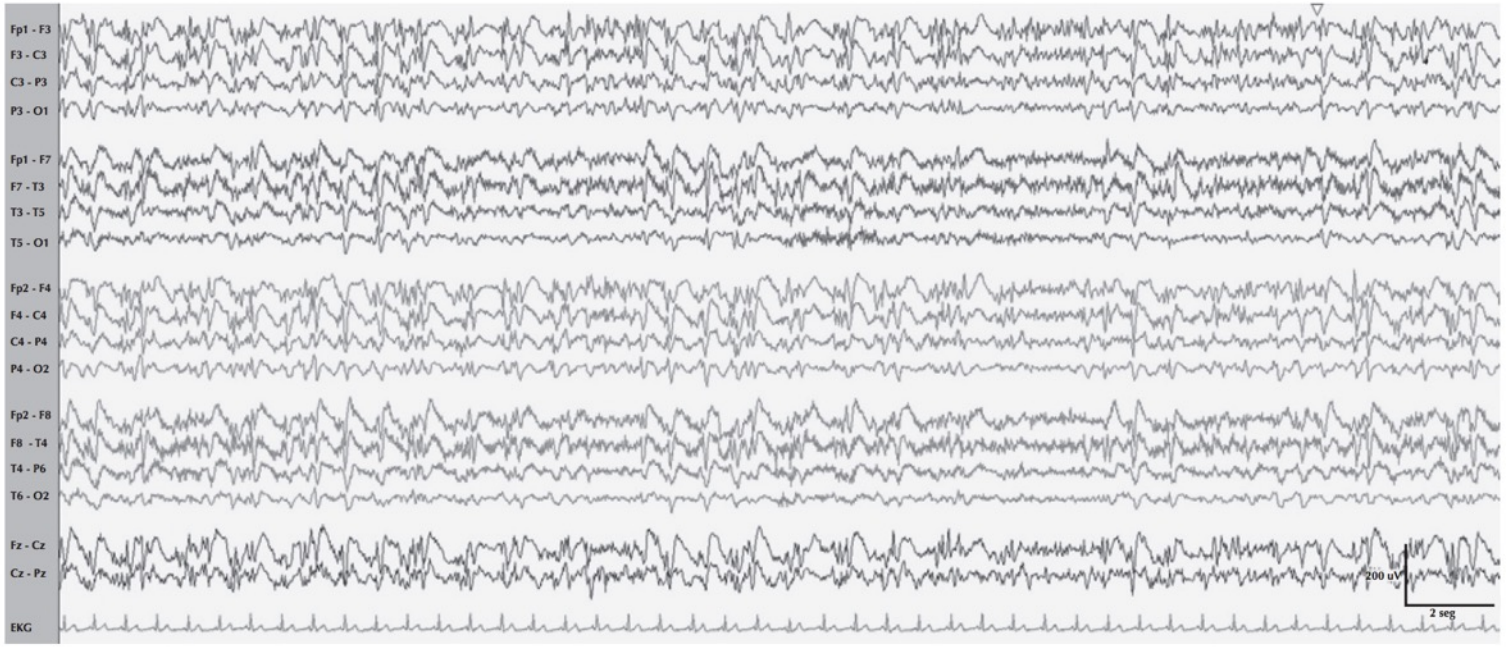


Encefalopatie tossiche

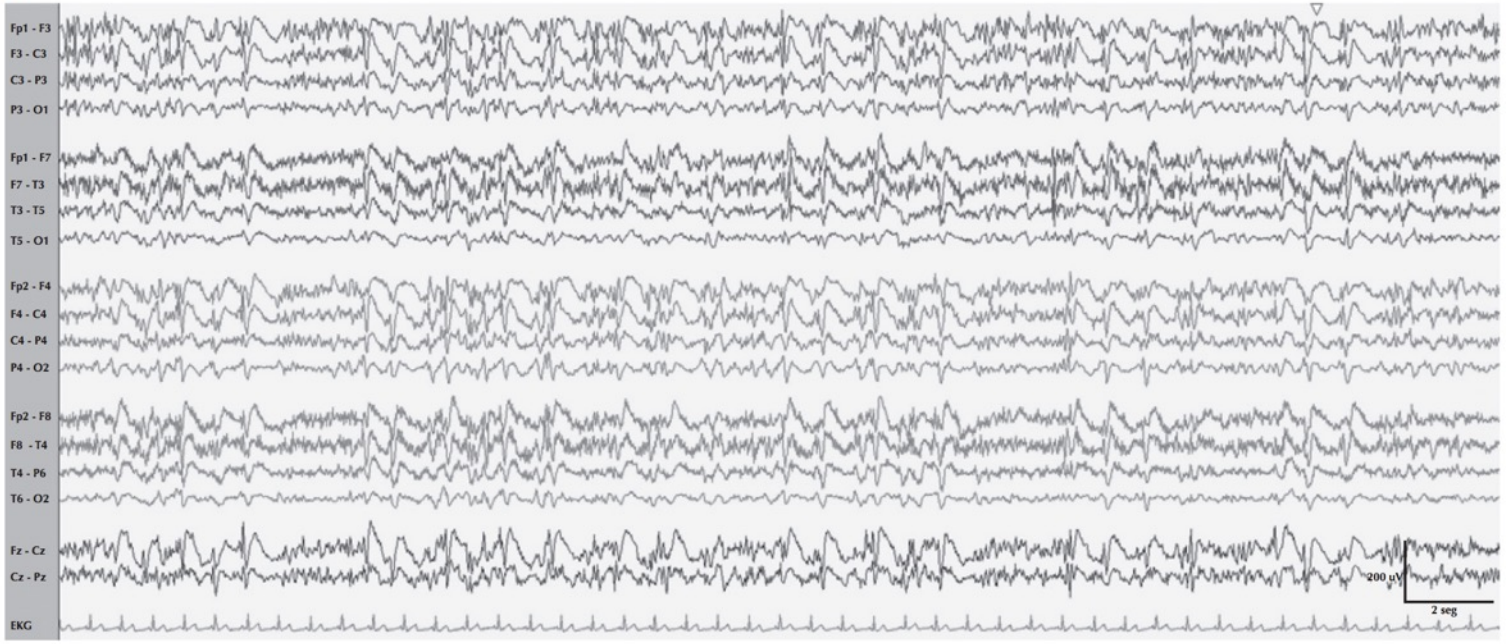
ENCEFALOPATIE TOSSICHE

De novo Absence Status of Late Onset (DNASLO)

tossicità da **Cefepime**

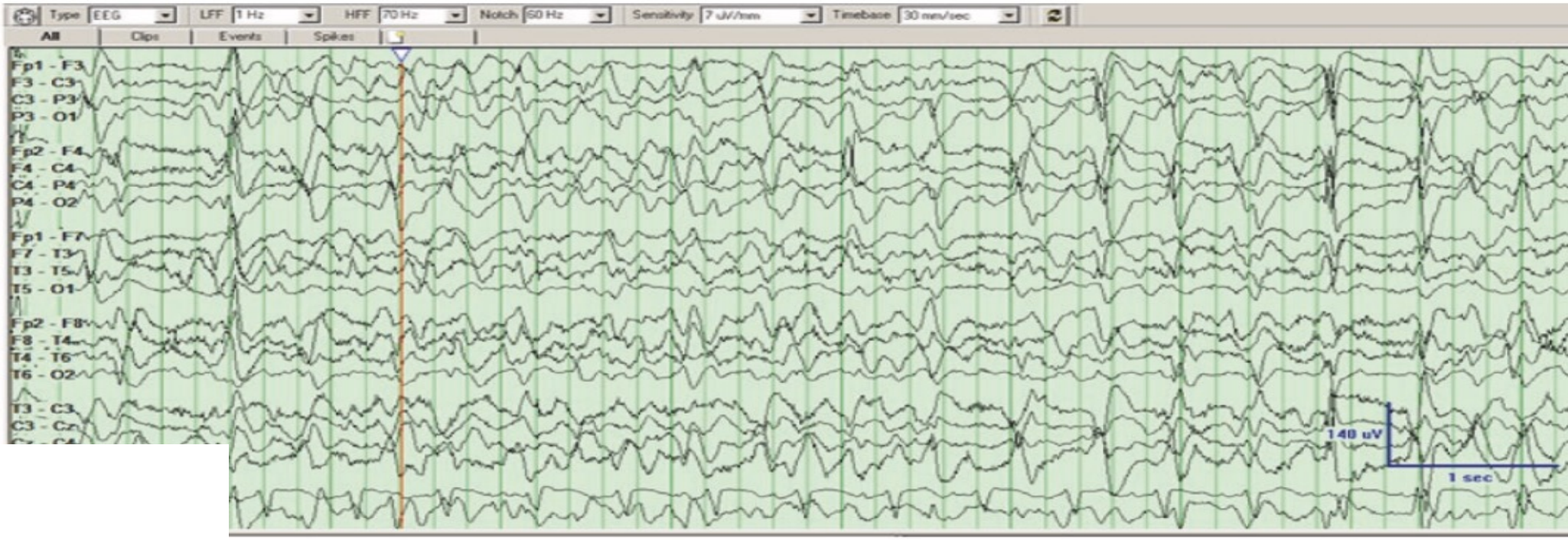


B

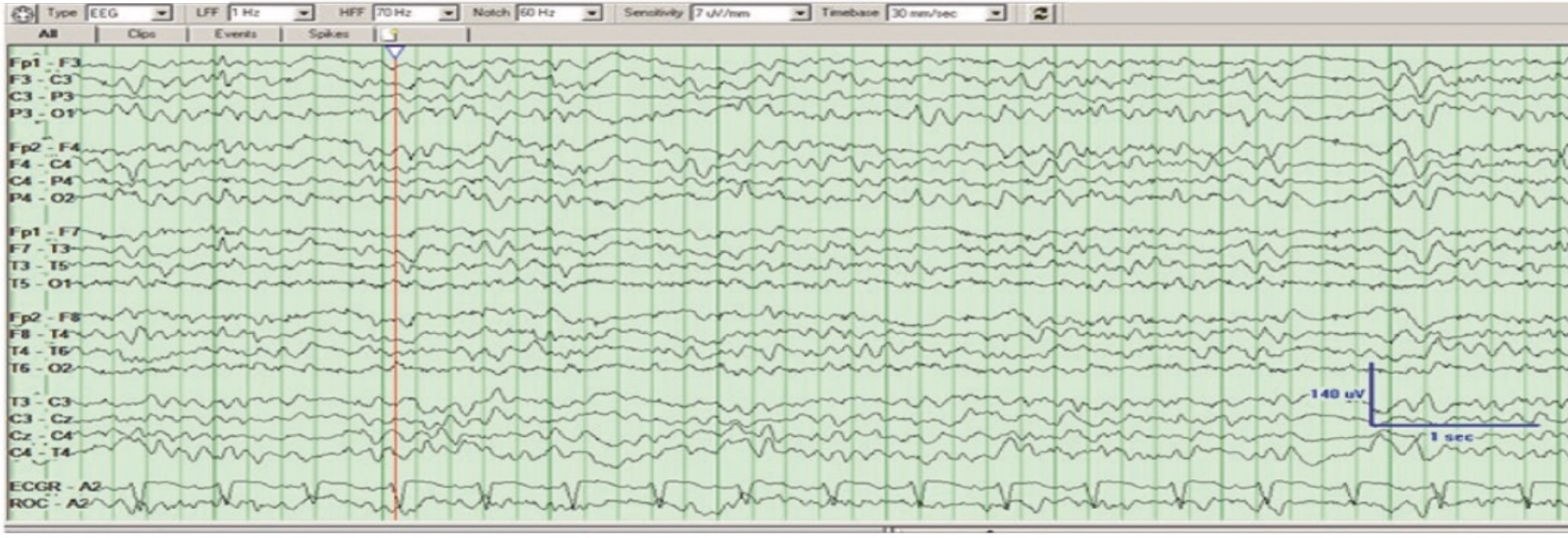


Clozapine

A



B



Benzodiazepine abuse

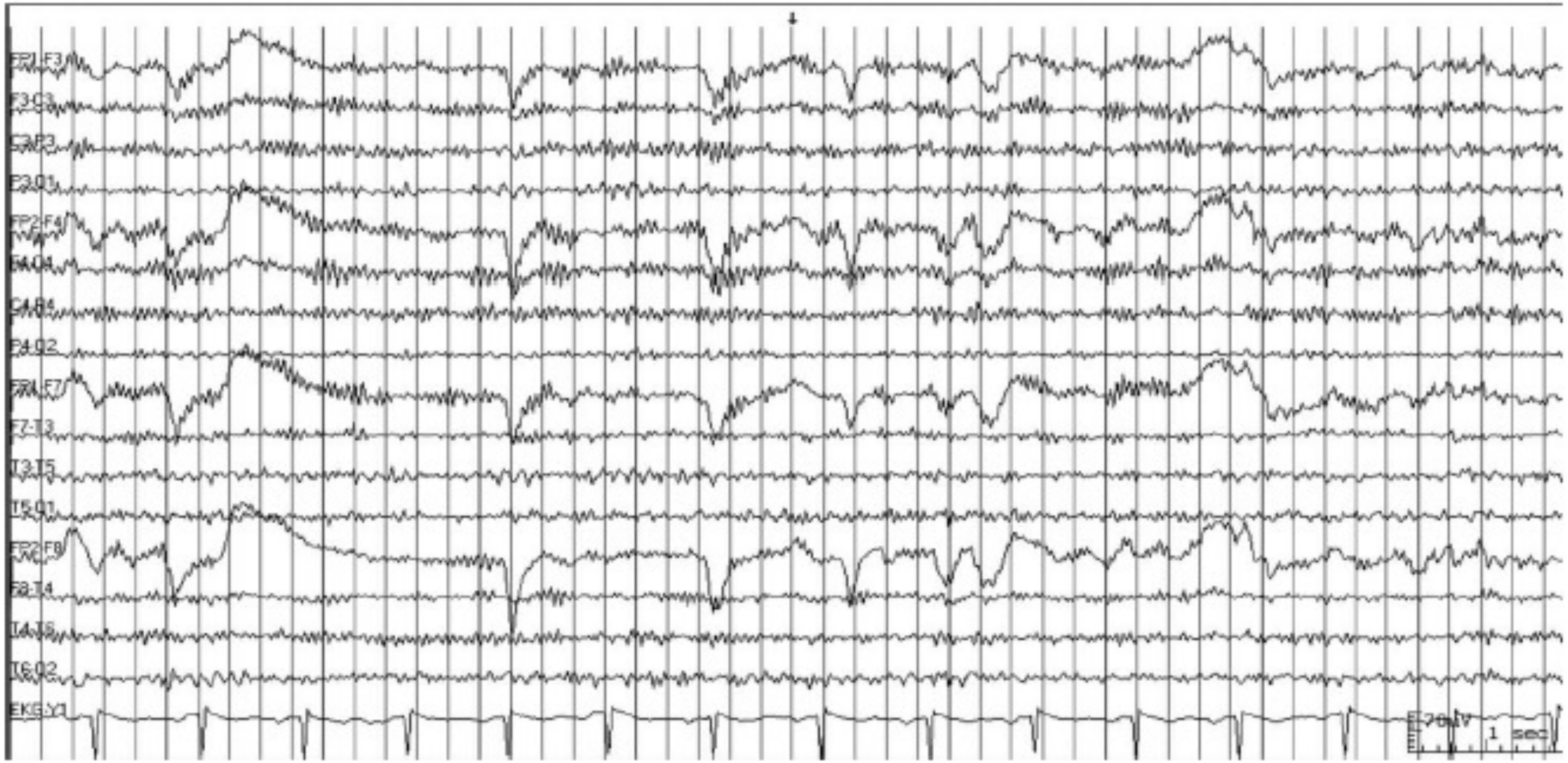
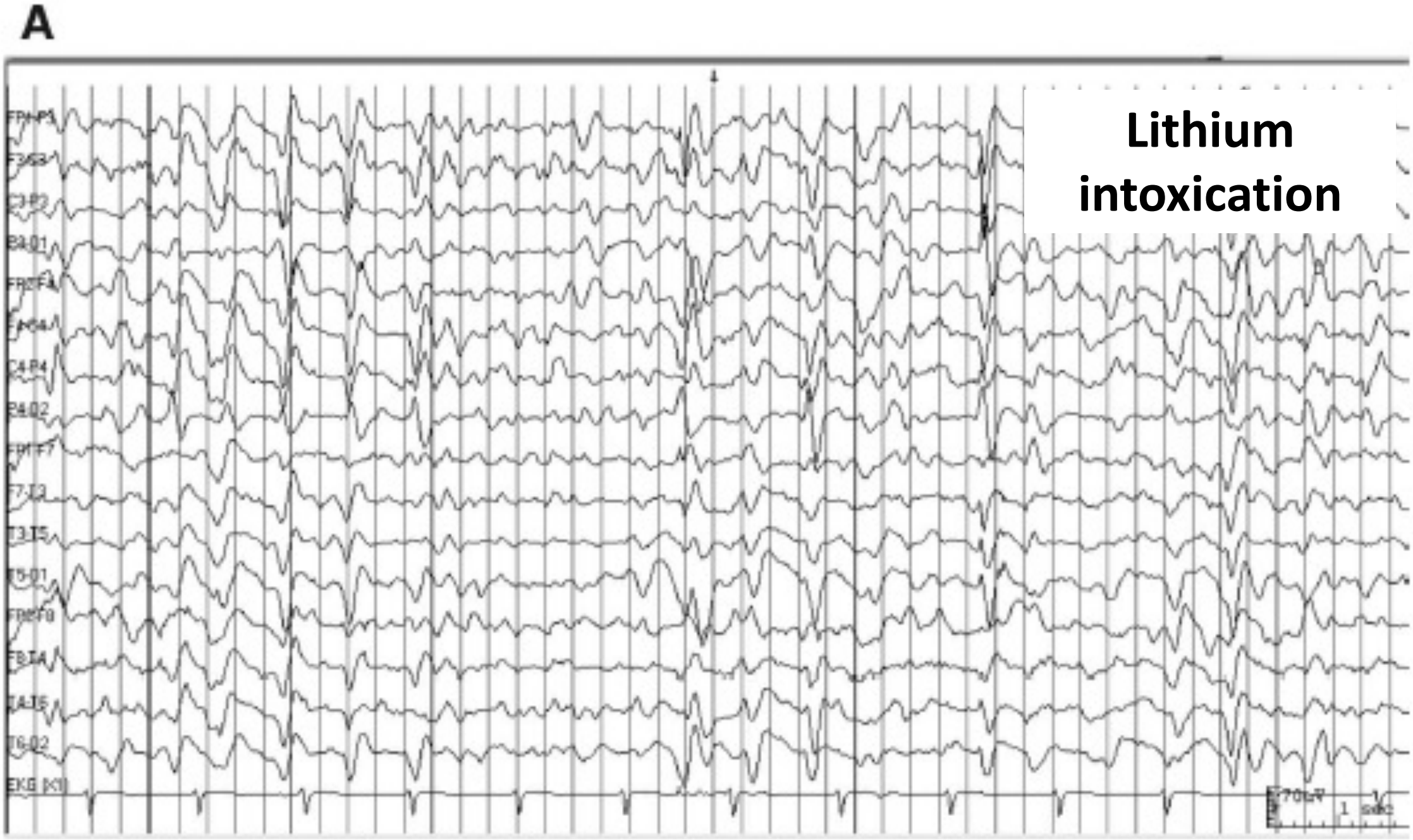


FIG. 1.
amoun
bursts,
regions


A
Lithium intoxication

Baclofen assumption

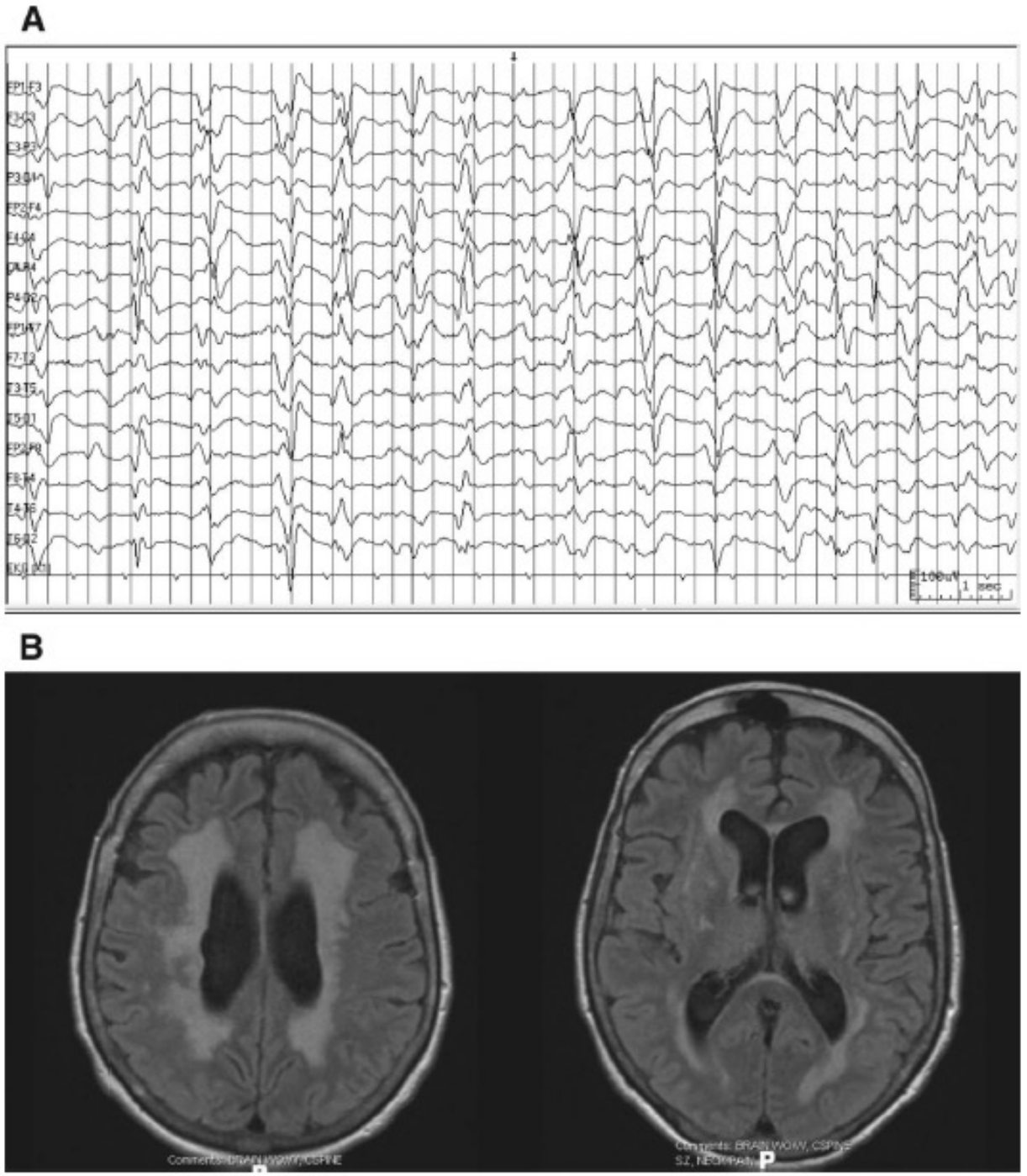


FIG. 14. A, Sharp irregular TWs with background activity in the θ range. B, MRI below showing marked cortical and subcortical (white matter) atrophies.

Tossicità da **Bupropione**

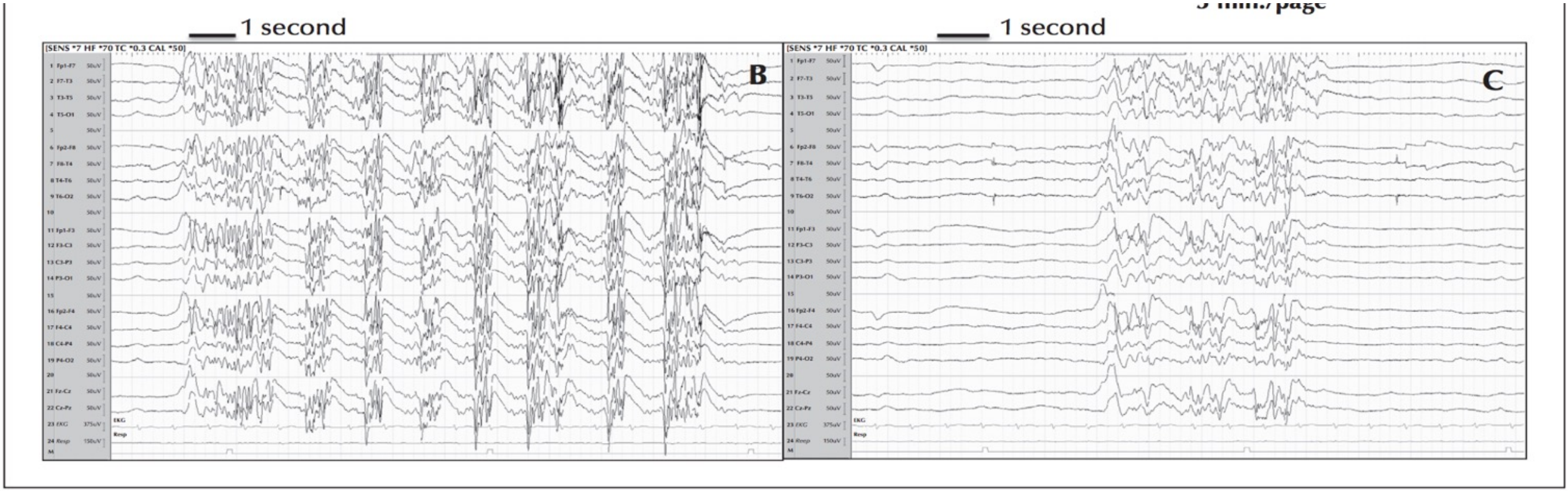
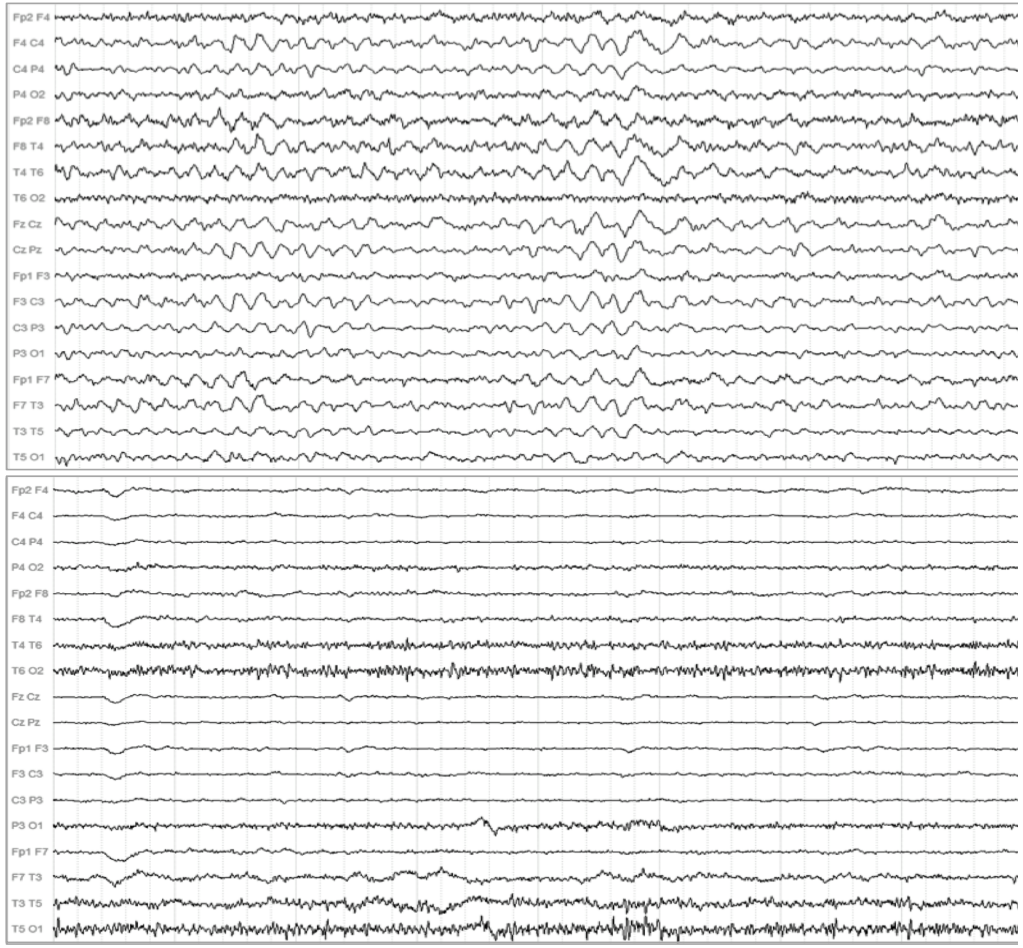
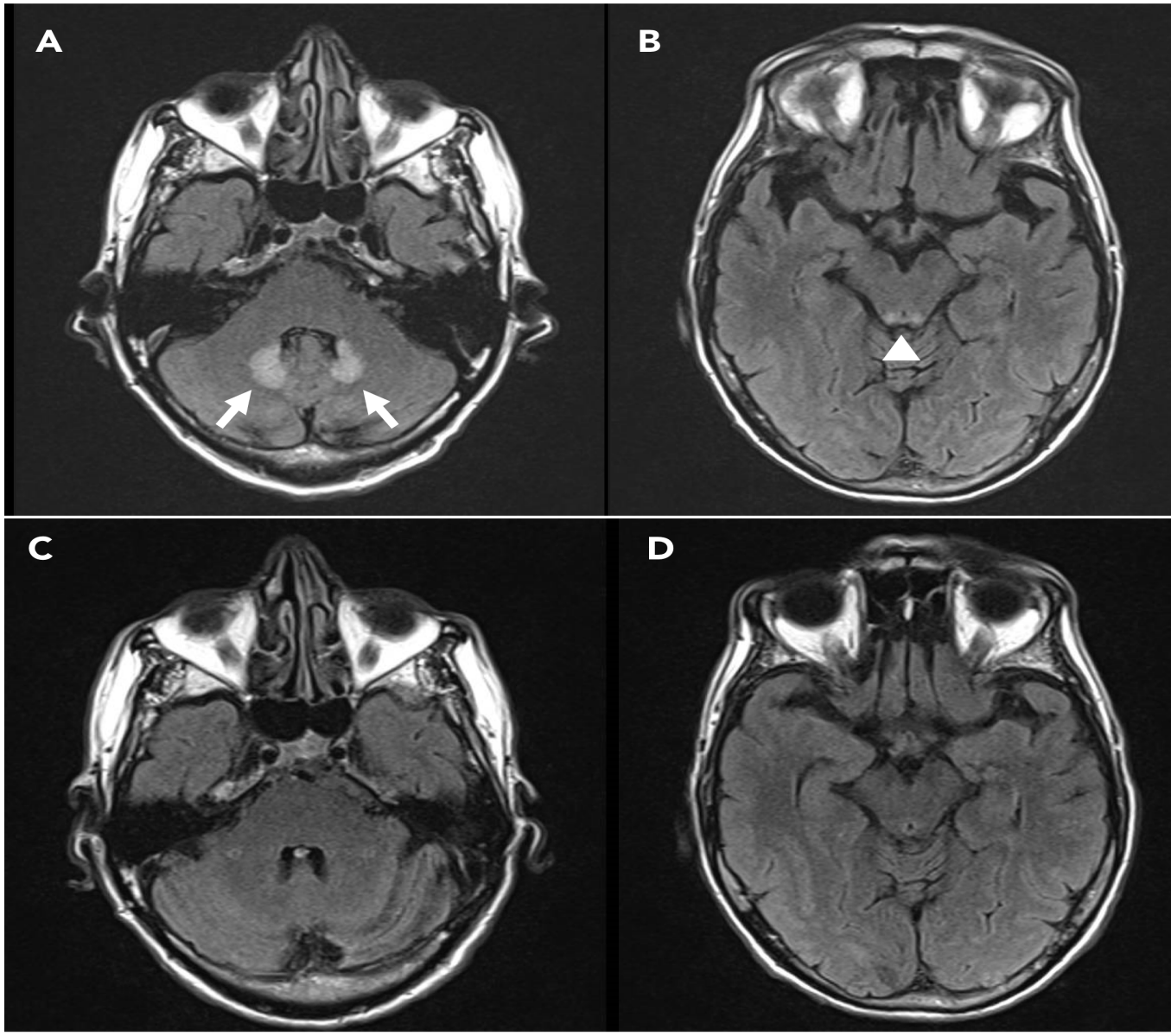


Figure 1. EEG during acute intoxication on a bipolar longitudinal montage on Day 1, when the patient was comatose and ventilated. (A) EEG with power spectrum showing a pattern of recurrent burst suppression at a scale of 5 minutes/page, at 50 μv. (B, C) EEG with a single group of generalized polyspike bursts with intermittent suppression at a scale of 15 seconds/page, at 50 μv, which were associated with periodic tonic upward gaze and neck extension.

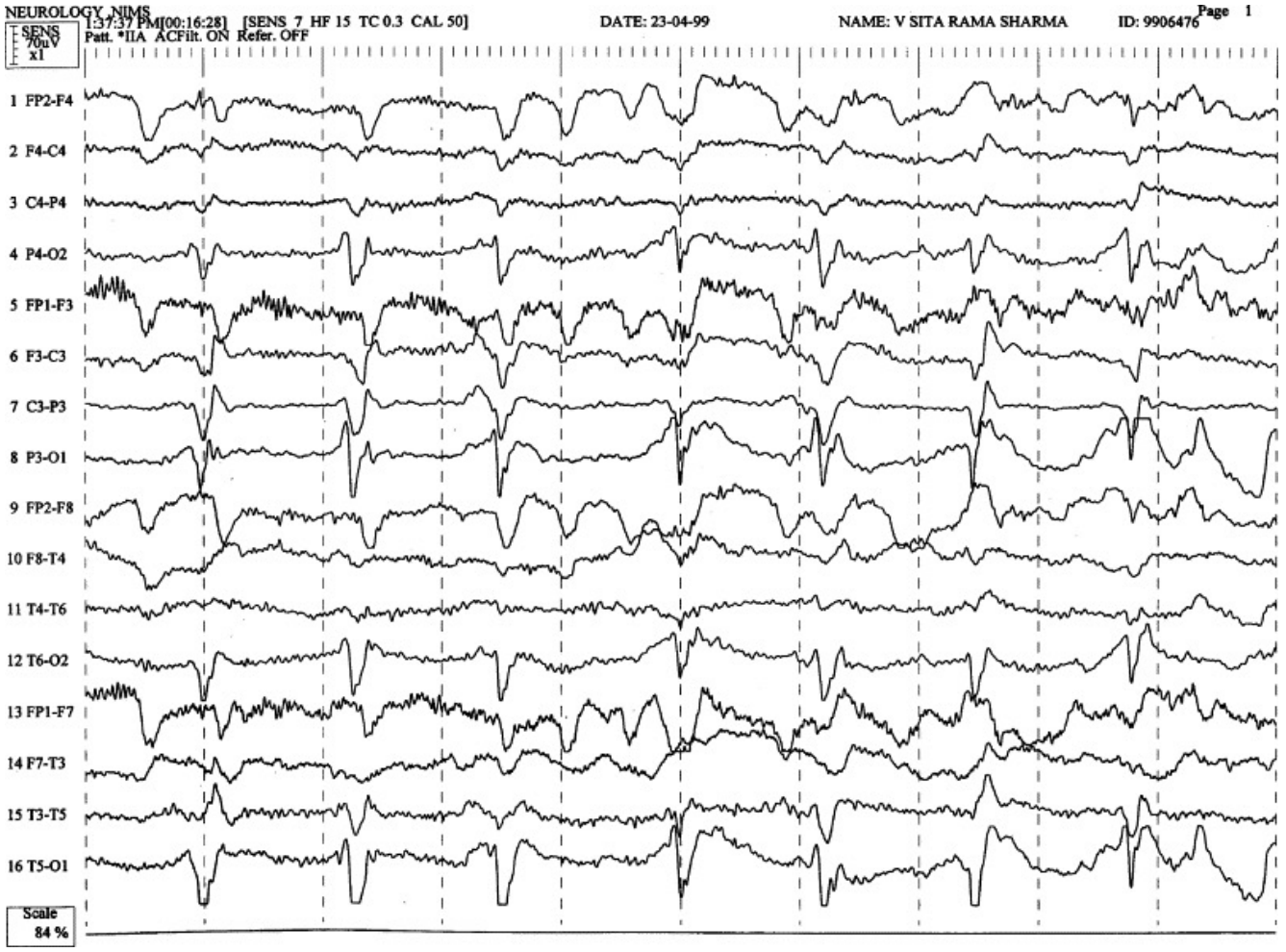
Tossicità da Metronidazolo



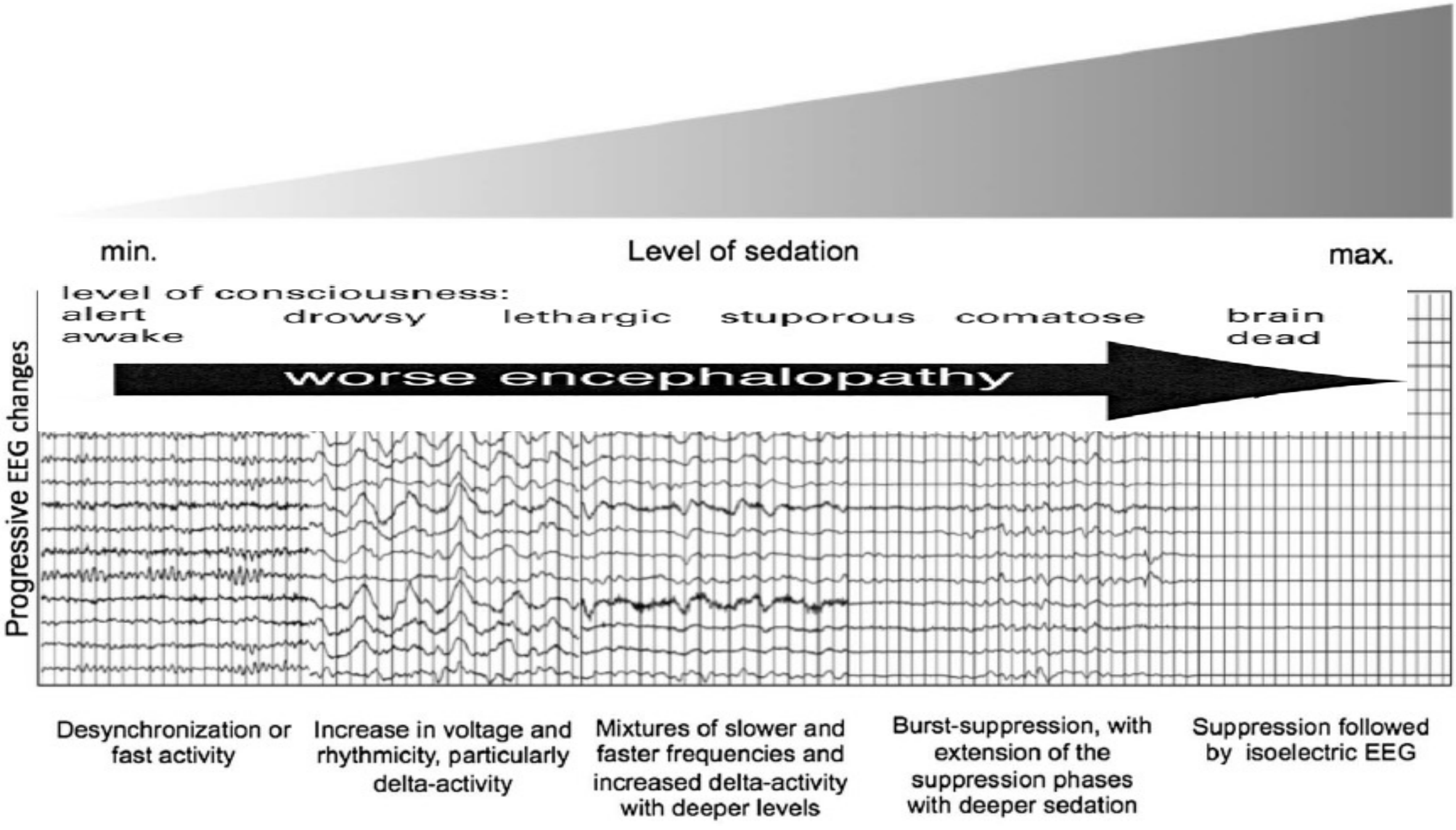


Alcohol

- Alcohol-increase in the alpha activity typically accompany alcohol consumption
- Beta activity substantially increased in withdrawal
- Delirium tremens- Excessive fast activity dominate the EEG tracing
- Delirium from other causes is associated with generalized slowing



Non trattiamo l’EEG ma i pazienti.
Il bello del gioco è cercare il correlato clinico, ma spesso non è affatto semplice.


min.
Level of sedation
max.
level of consciousness:
alert
awake
drowsy
lethargic
stuporous
comatose
brain
dead
worse encephalopathy
Progressive EEG changes
Desynchronization or fast activity
Increase in voltage and rhythmicity, particularly delta-activity
Mixtures of slower and faster frequencies and increased delta-activity with deeper levels
Burst-suppression, with extension of the suppression phases with deeper sedation
Suppression followed by isoelectric EEG

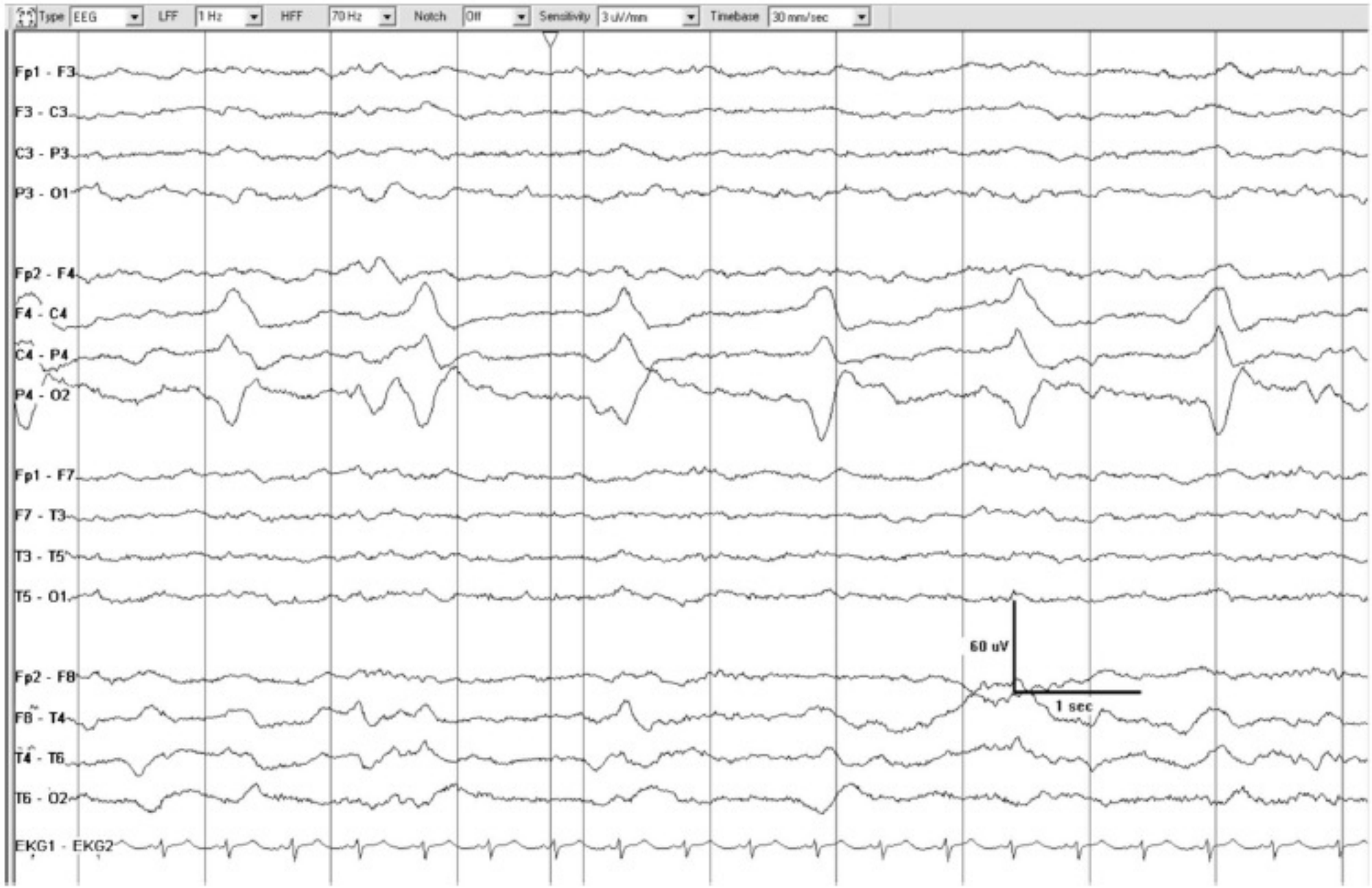


FIG. 1. LPDs: Sharply contoured lateralized periodic discharges. In this case, LPDs are unilateral.

MAIN TERM 1

Generalized (G)→ bilateral, bisynchronous and symmetric*

Frontally predominant**– anterior > posterior leads

Occipitally predominant**– posterior > anterior leads

Midline predominant**– midline > parasagittal leads

Lateralized (L)→ unilateral or bilateral and synchronous but asymmetric

Unilateral or **Bilateral Asymmetric** (purely unilateral versus bilaterally and synchronous but consistently more prominent on one side)

Hemispheric or predominantly involving one lobe (**frontal, parietal, temporal, or occipital**)

Bilateral Independent (BI)→ two simultaneous and asynchronous lateralized patterns, one in each hemisphere

Multifocal (Mf)→ three or more asynchronous lateralized patterns, at least one in each hemisphere

Symmetric or **Asymmetric** (bilaterally and asynchronous symmetrically versus consistently more prominent on one side)

Hemispheric or predominantly involving one lobe (**frontal, parietal, temporal, or occipital**)

MAIN TERM 2

Periodic Discharges (PD)→ at least 6cycles of discharges with a uniform morphology & duration with a quantifiable and regular or near regular inter-discharge interval

Discharges are waveforms with ≤3 phases or any waveform lasting ≤0.5 seconds regardless of number of phases.

The recurrence of discharges in PD have to occur at regular or near regular intervals (i.e., the period must vary by <50% from one cycle to the next cycle in the majority of cycle pairs).

Rhythmic Delta Activity (RDA)→ at least 6 cycles of a waveform ≤4Hz with uniform morphology and duration without an interval

Similarly to PD, the duration of one cycle of an RDA must vary by <50% from one cycle to the next cycle in the majority of cycle pairs.

! *If a pattern meets criteria for PD and RDA simultaneously, reader should interpret as PD+R rather than RDA+S.*

Spike-and-wave or Sharp-and-wave (SW)→ polyspike, spike or sharp wave followed by a slow wave in a consistent, regularly repeating and alternating pattern; no interval between SW complexes

MODIFIERS

Prevalence → percent of record occupied by *each* pattern

Continuous – ≥ 90%
Abundant – 50 – 89%
Frequent – 10 – 49%
Occasional – 1-9%
Rare – < 1%

Frequency → specify typical rate and ranges (minimum, maximum) of discharges per second

Amplitude → typical amplitude ***

Absolute (*for RDA, PD, and SW*)
Very low – < 20 microvolts
Low – 20 – 49 microvolts
Medium – 50 – 199 microvolts
High – ≥ 200 microvolts

Relative *(for PDs only)*
≤2 or >2

Polarity → of the phase of highest amplitude in the typical discharge §

Positive
Negative
Dipole, horizontal/tangential
Unclear
This modifier only applies to PD and SW as it refers to discharges

Evolution → specify the change in behavior over time for frequency, location and morphology §§

Evolving – unequivocal & sequential changes in:
--*frequency* – ≥ 2 consecutive changes in same direction by at least 0.5Hz sustained over 3 cycles
--*morphology* – ≥ 2 consecutive changes into different morphology
--*location* – spreading into or out of ≥ 2 standard 10-20 electrodes

Fluctuating – ≥3 changes in frequency, location or morphology, no more than one minute apart

Static – changes in pattern not qualifying as evolving or fluctuating

Specify the minimum and maximum frequency and the extent of spreading (none, unilateral, or bilateral)

Plus → accompanying ictal-appearing feature

+F –superimposed theta or faster activity. *PD or RDA only*
+R – superimposed rhythmic activity. *PD only*
+S – superimposed sharp waves, spikes, or sharply contoured waveform. *RDA only*

Plus features may co-exist as +FR or +FS

Duration → specify typical as well as longest duration of pattern (if not continuous)

Very long – ≥ 1 hour
Long – 5 – 59 minutes
Intermediate – 1 – 4.9 minutes
Brief – 10 – 59 seconds
Very Brief - < 10 seconds

Number of phases → total number of baseline crossings of a typical discharge plus one §§§

1, 2, 3 or ≥4
This modifier only applies to PD and SW as it refers to discharges

Sharpness → specify for the phase of highest amplitude AND the sharpest phase of a typical discharge, if different

Spiky – < 70 milliseconds
Sharp – 70-200 milliseconds
Sharply contoured – theta or delta waves with a steep slope to one side of wave and/or pointy appearance a inflection point; > 200 **milliseconds**
Blunt – smooth or sinusoidal waveform
This modifier only applies to PD and SW as it refers to discharges

Stimulus-Induced (SI) → consistently triggered by alerting stimulus (internal or external)

Stimulus-Induced – induced by stimulus (ok to occur at times without clear stimulation as it may represent internal alerting stimulus)
Spontaneous – NEVER clearly induced by stimulation

MINOR MODIFIERS

Quasi- → computational analysis demonstrating 25-50% variation in cycle length

Lag → consistent delay >100 milliseconds anterior-posteriorly

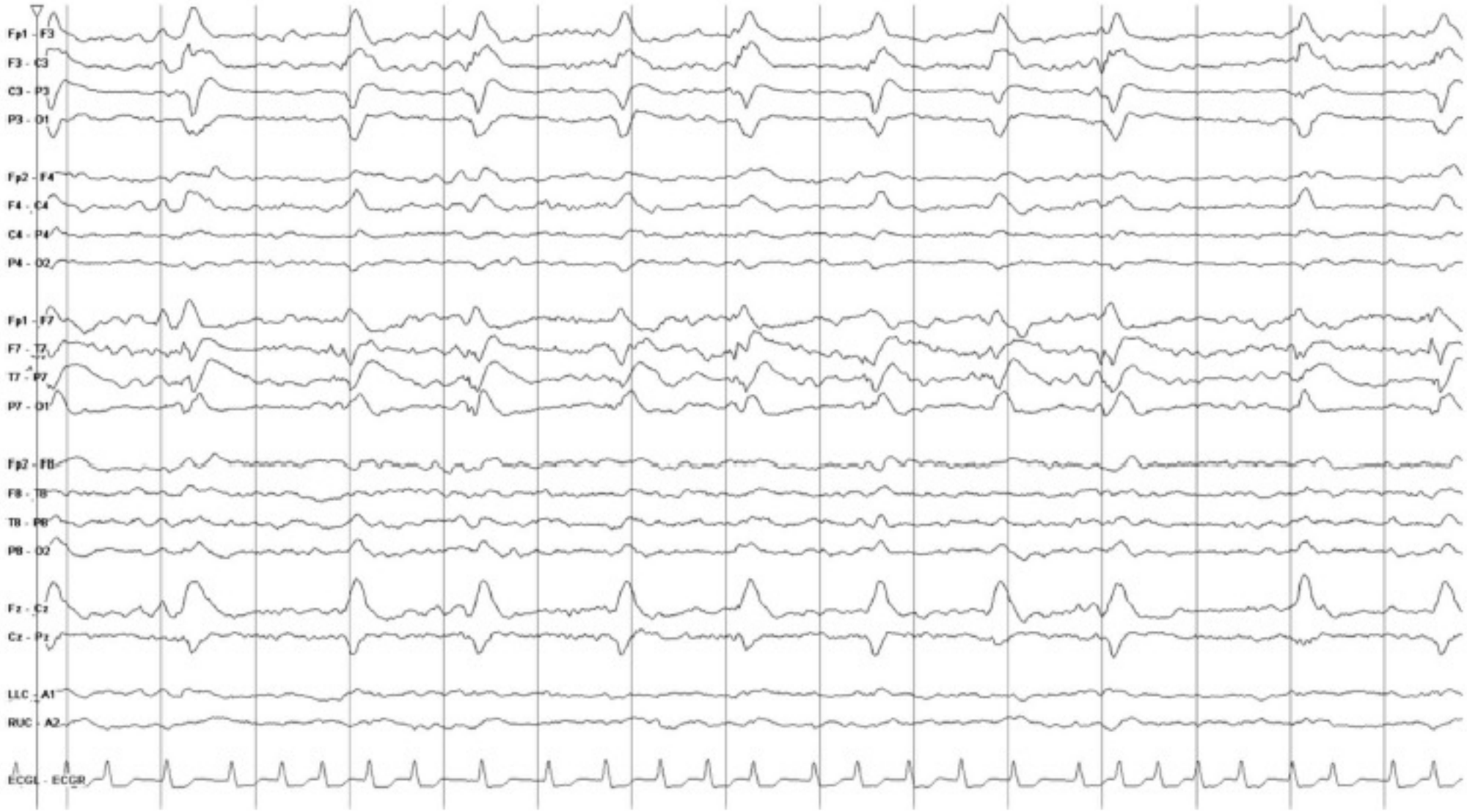


FIG. 2. LPDs: Sharply contoured lateralized periodic discharges. In this case, PDs are bilateral asymmetric.

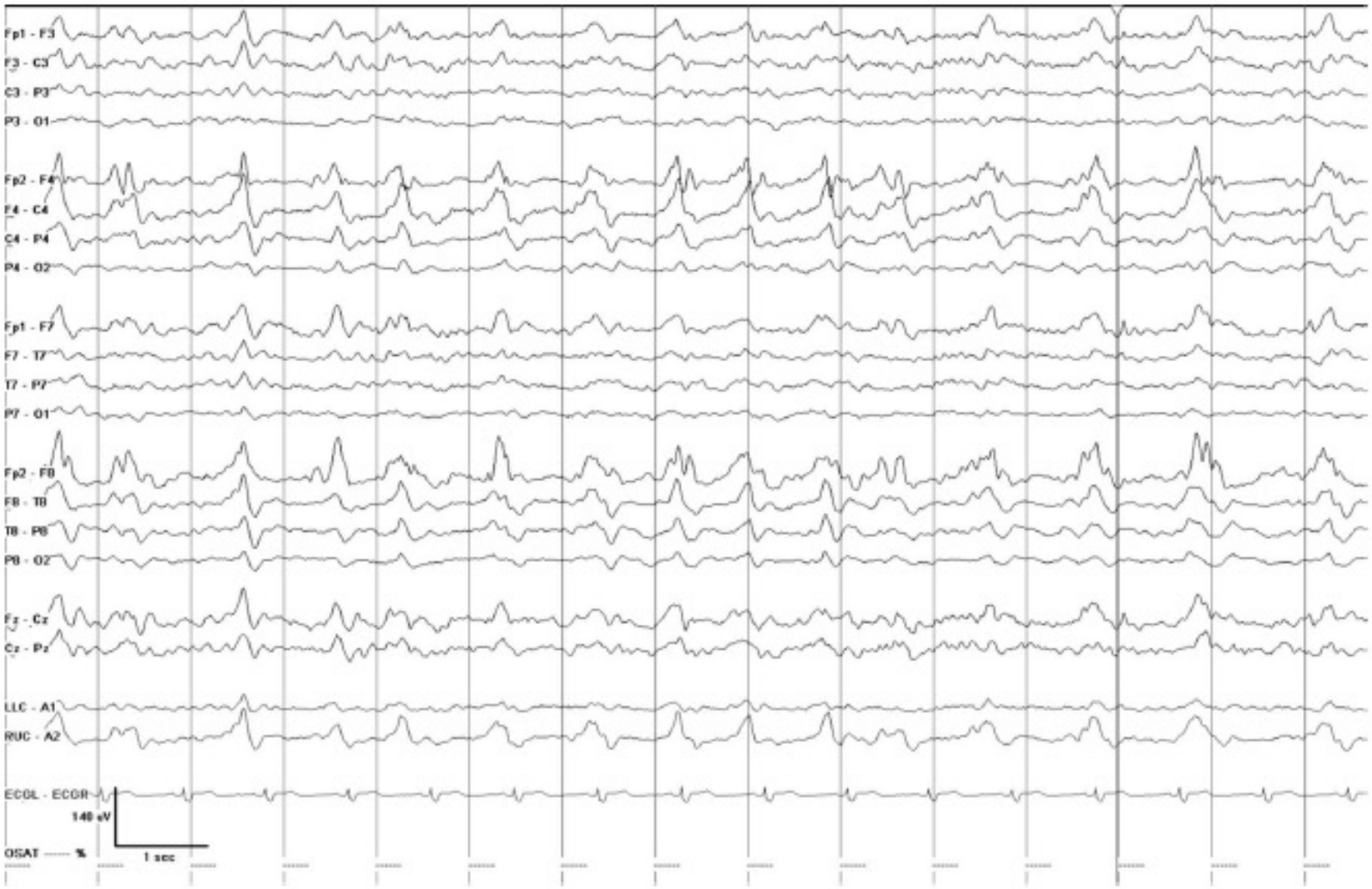


FIG. 3. LPDs: Sharply contoured lateralized periodic discharges. In this case, PDs are bilateral asymmetric. Although some discharges are on the border of sharp, most are sharply contoured.

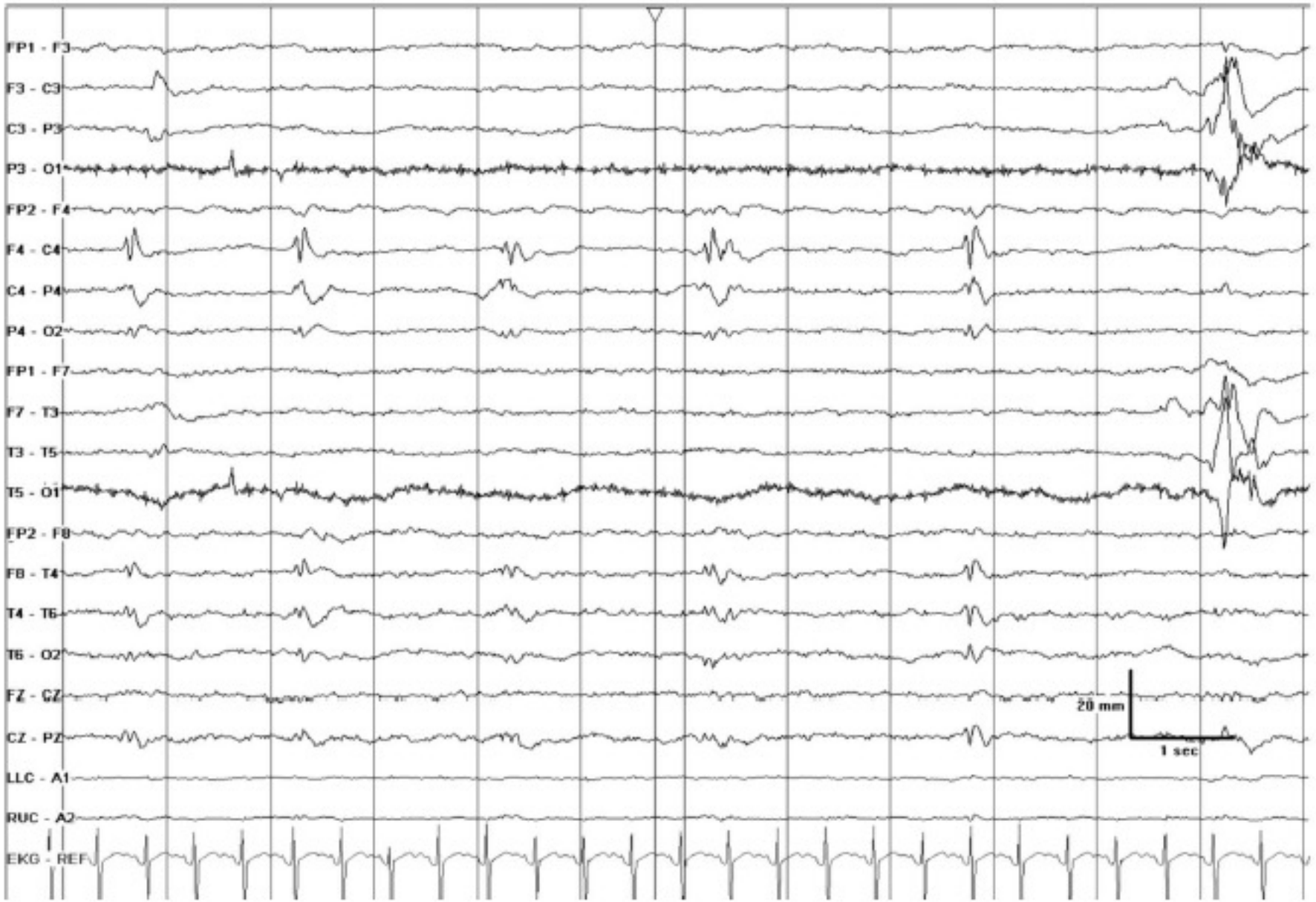


FIG. 4. LPDs: 0.5 per second spiky lateralized periodic discharges.

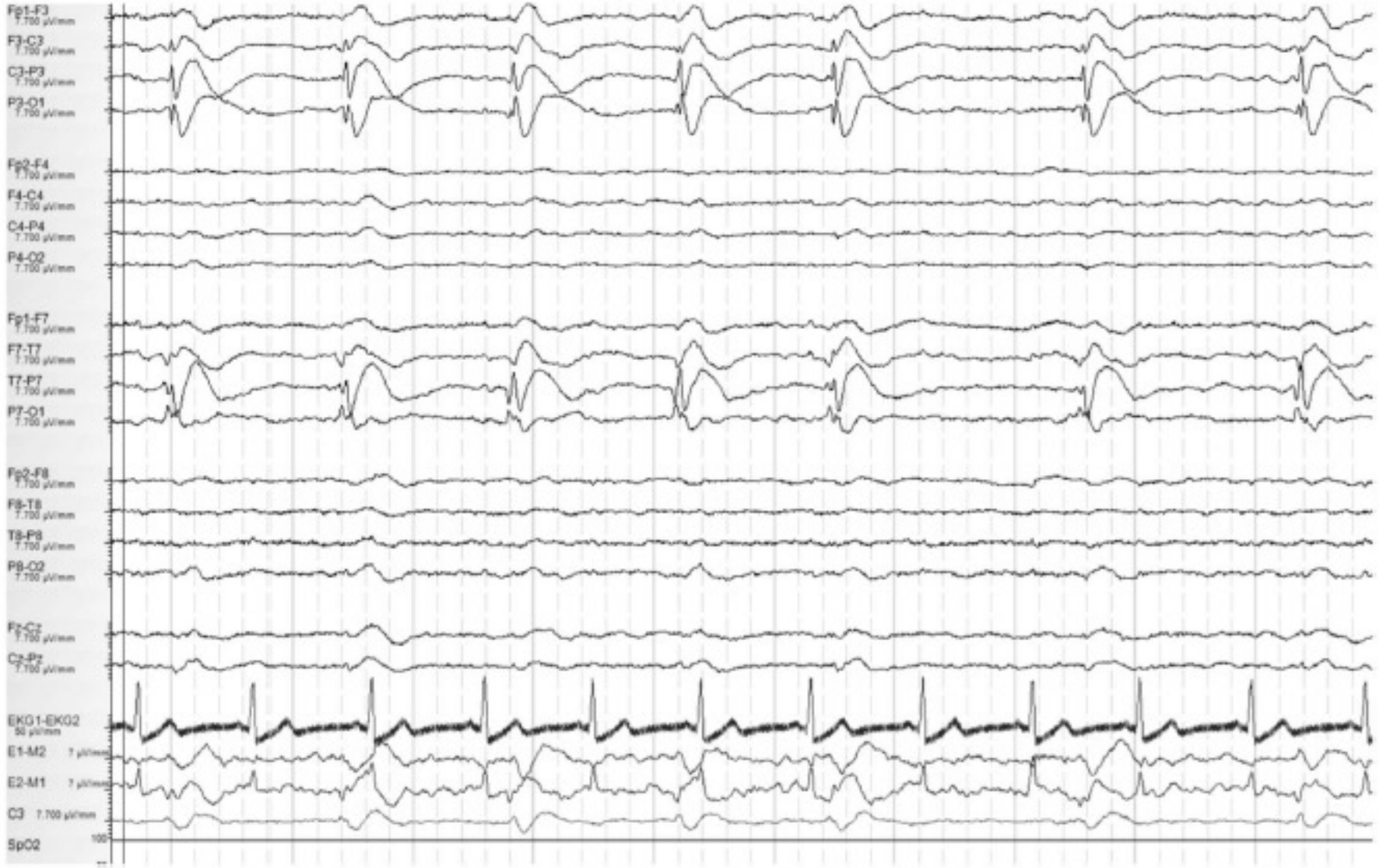


FIG. 5. LPDs: 0.5-1 per second spiky lateralized periodic discharges. Despite their spike-and-wave morphology, the discharges are periodic (as there is a quantifiable inter-discharge interval between consecutive waveforms and recurrence of the waveform at nearly regular intervals).

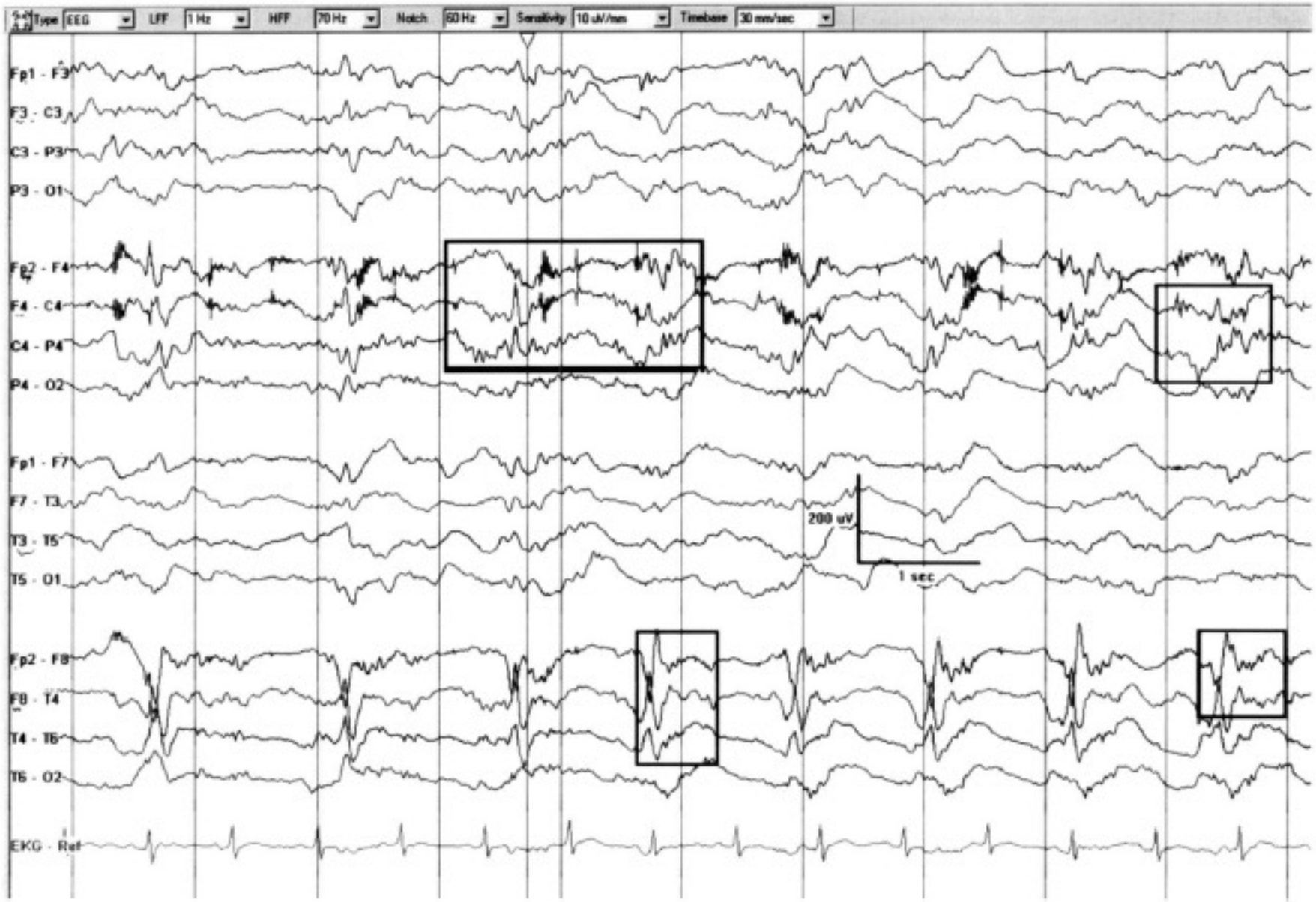


FIG. 6. LPDs+F: 0.5 to 1 per second spiky LPDs with superimposed burst of low amplitude fast activity (highlighted in boxes).

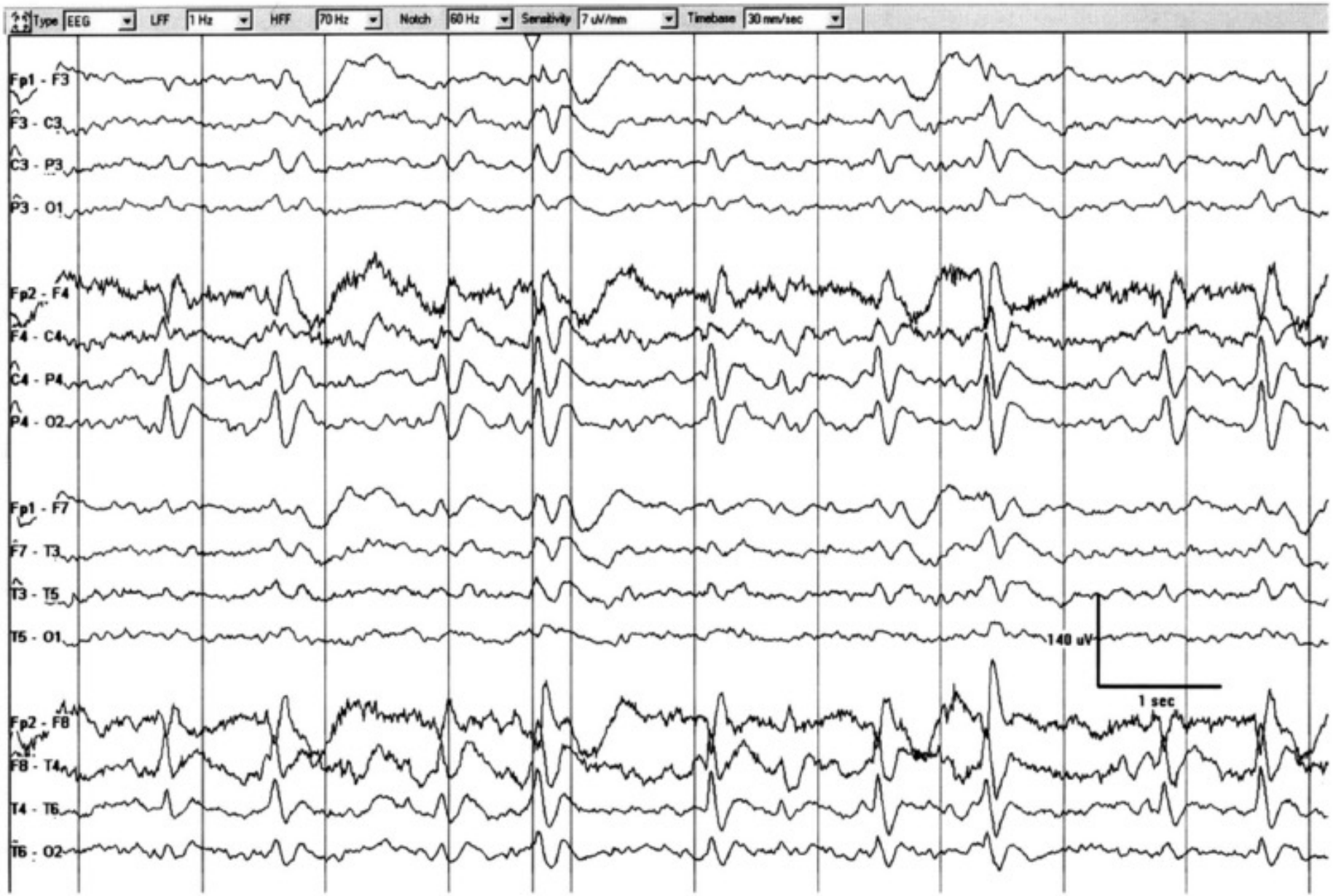


FIG. 7. LPDs+R: Irregular (in morphology and repetition rate) 0.5-1 per second quasi-periodic discharges with superimposed quasi-rhythmic delta activity in the right hemisphere with occasional spread to the left. Less "stable" pattern and more ictal-appearing than LPDs alone; compare with Figure 1.

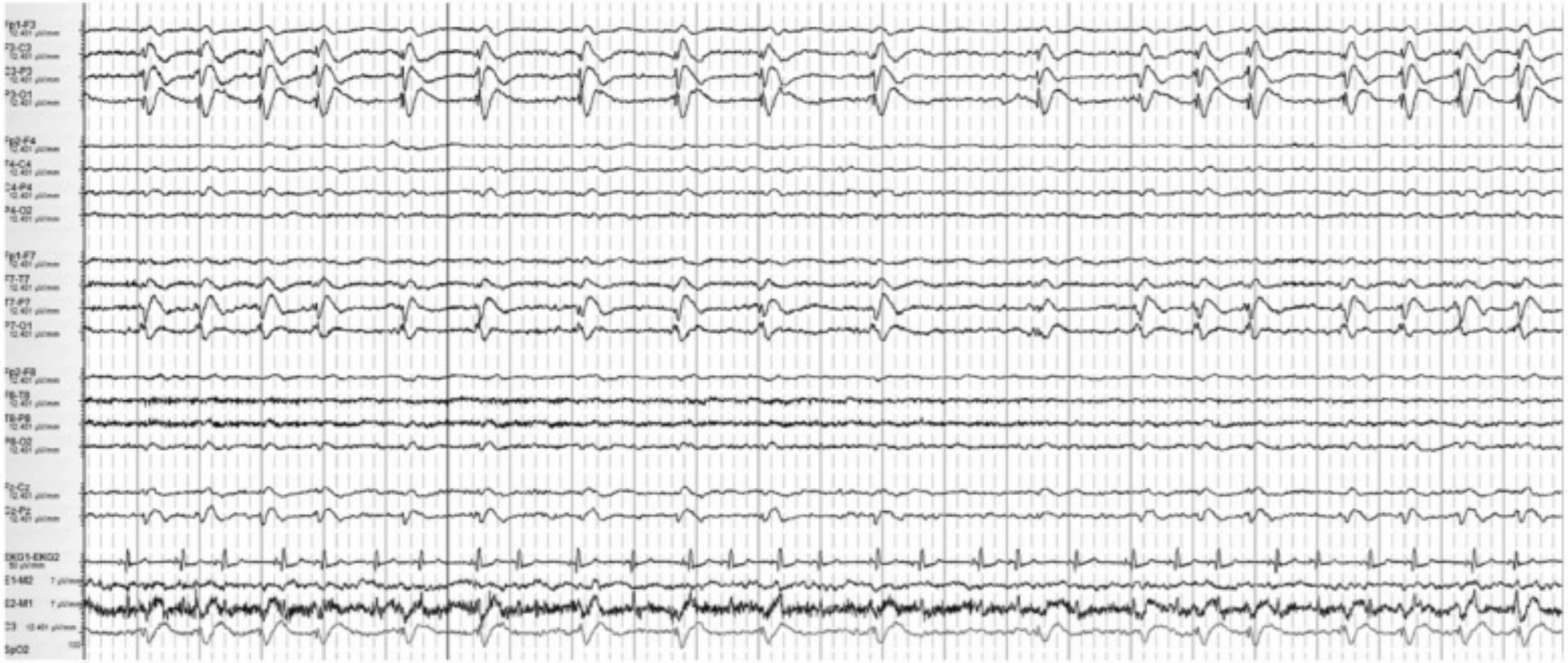


FIG. 8. Fluctuating LPDs: Lateralized periodic discharges that fluctuate in frequency between 0.5 and 1 per second.

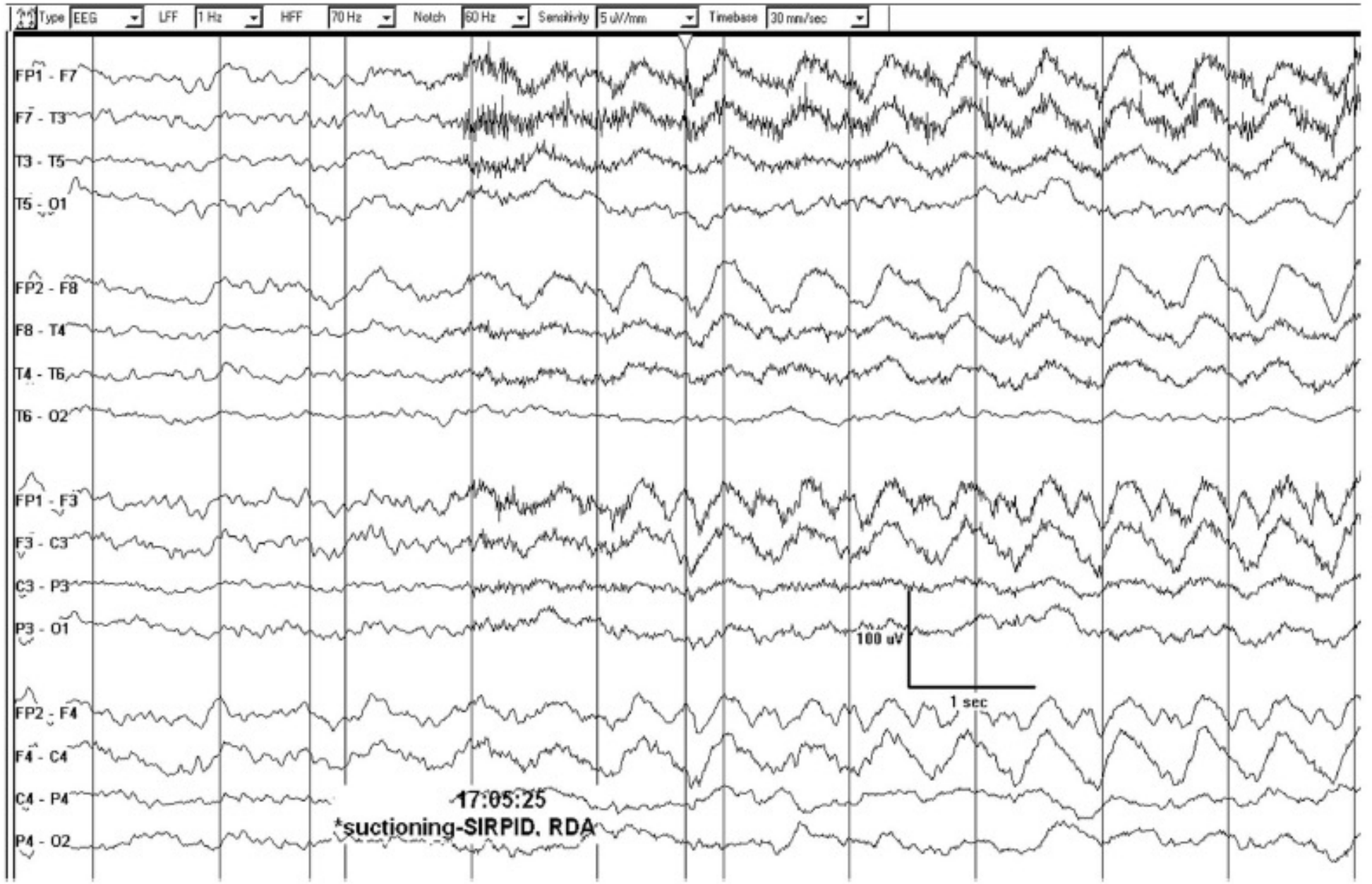


FIG. 15. SI-GRDA: Stimulus-induced generalized rhythmic delta activity, frontally predominant. In this case, the pattern was elicited by suctioning the patient.

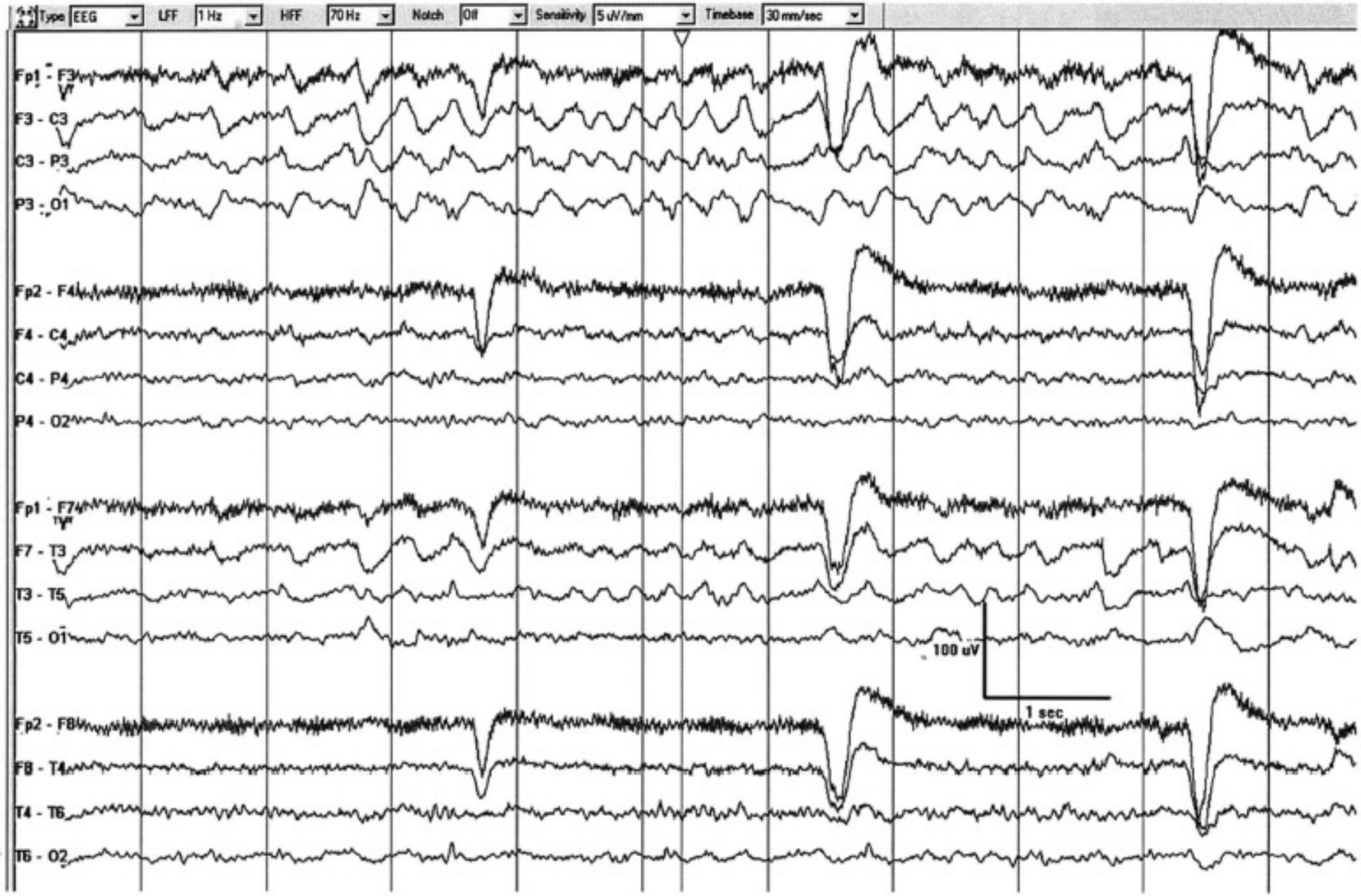


FIG. 16. Evolving LRDA: Lateralized rhythmic delta activity that evolves in morphology and frequency. It begins as low voltage sharply contoured 1.5 Hz delta in the left parasagittal region, evolves to 3 Hz rhythmic delta, then again slows.

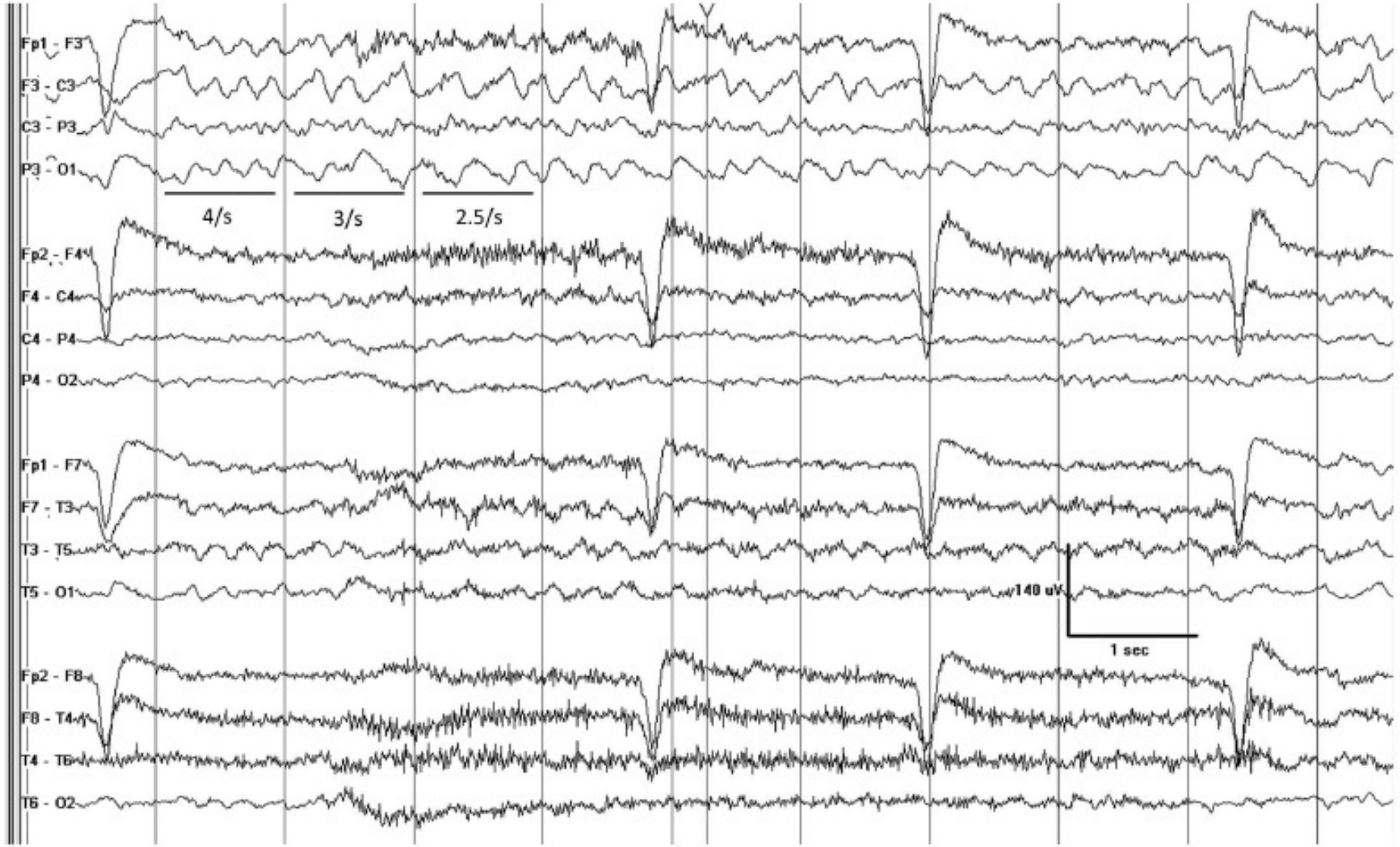


FIG. 17. Evolving LRDA: Lateralized rhythmic delta activity that evolves in frequency and morphology from a 4 per second blunt RDA to a 2.5 per second sharply contoured RDA.