EEG in patients with disturbed consciousness

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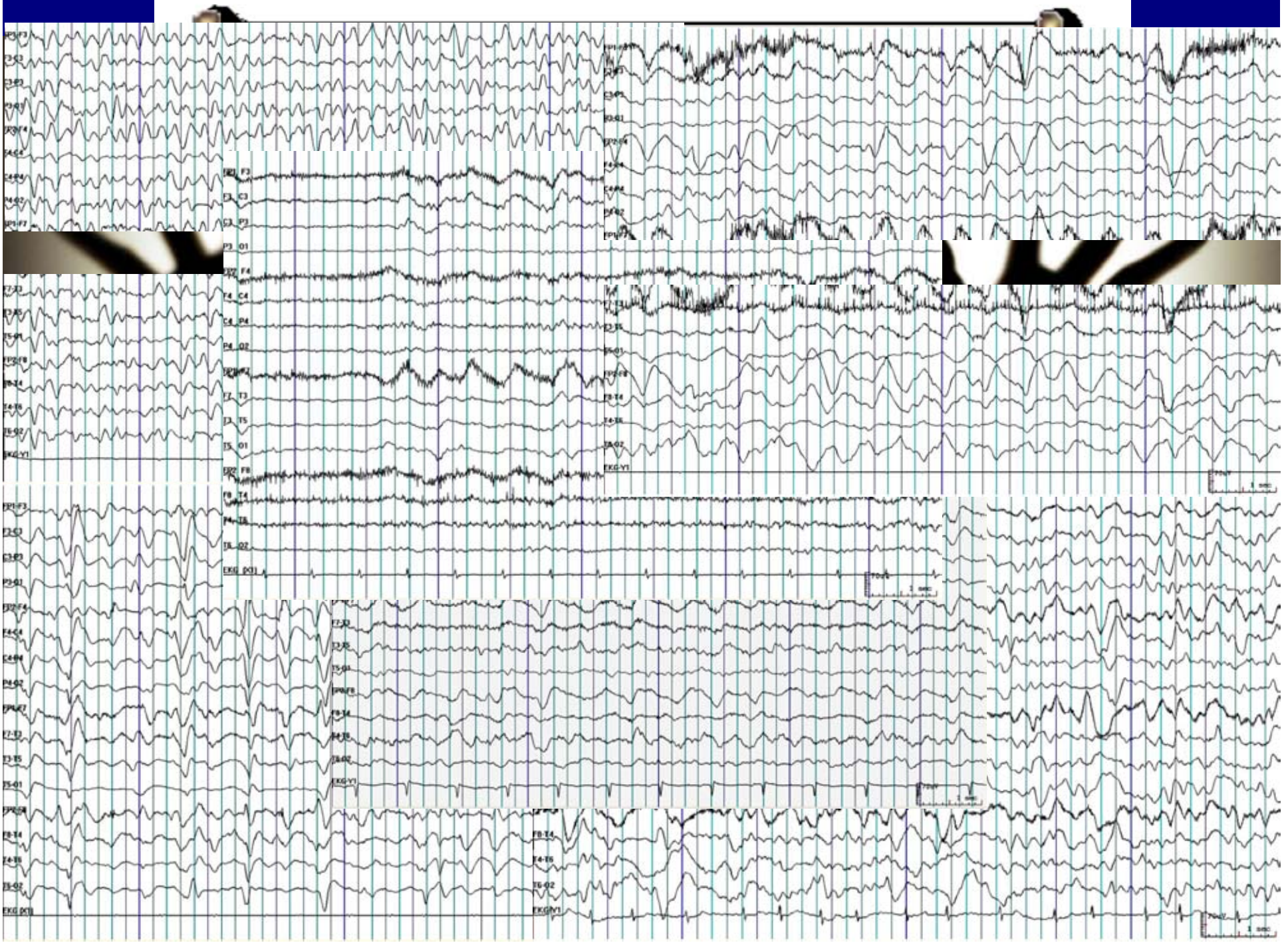
Disclosures and COI

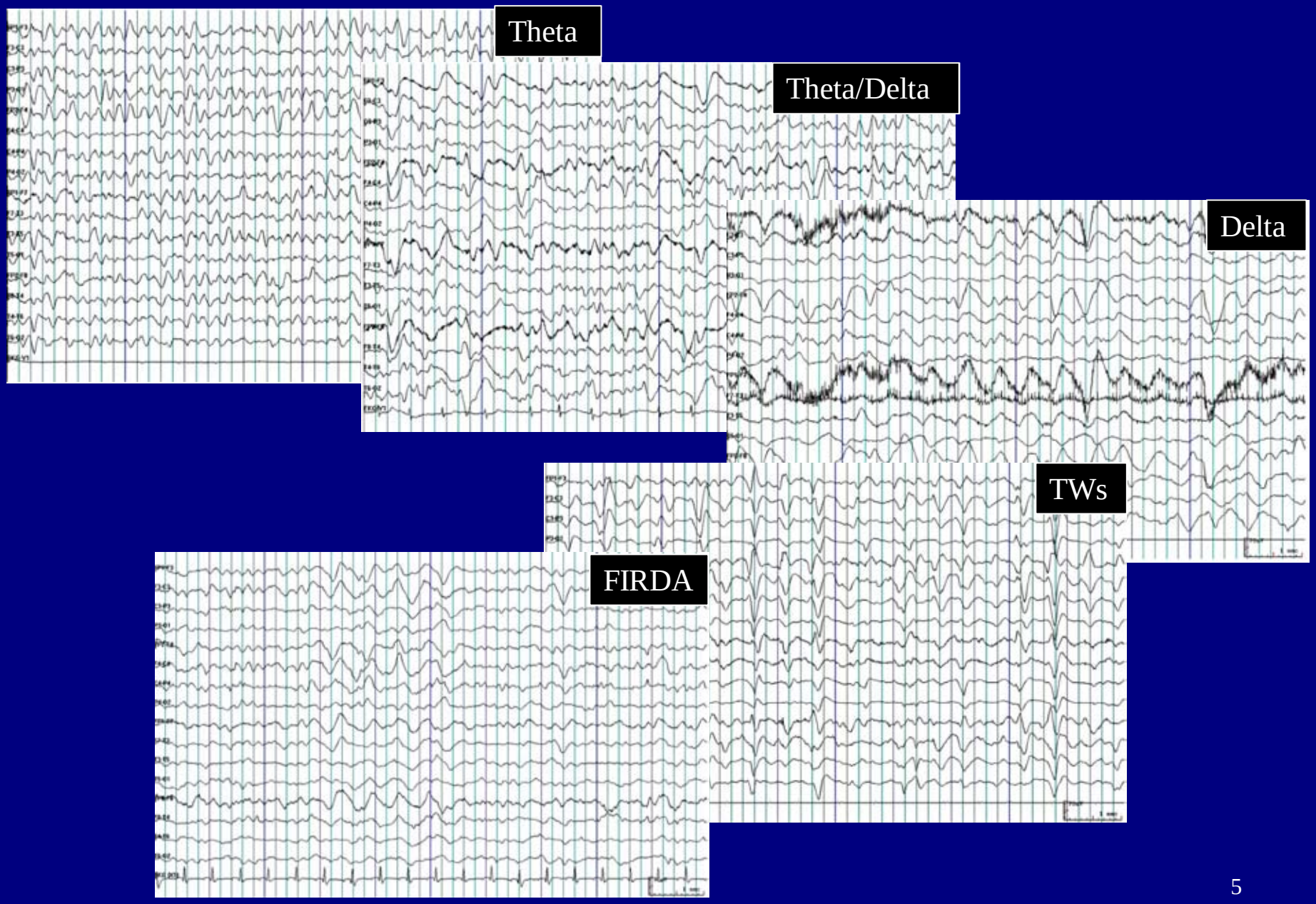
- Textbooks on EEG and clinical neurophysiology



Learning Objectives

- Learn EEG patterns in encephalopathies and confusional states
- Know conditions associated with certain EEG patterns
- Know evolution of EEG with severity of encephalopathy
- Understand use of EEG in prognosis after CRA/anoxia

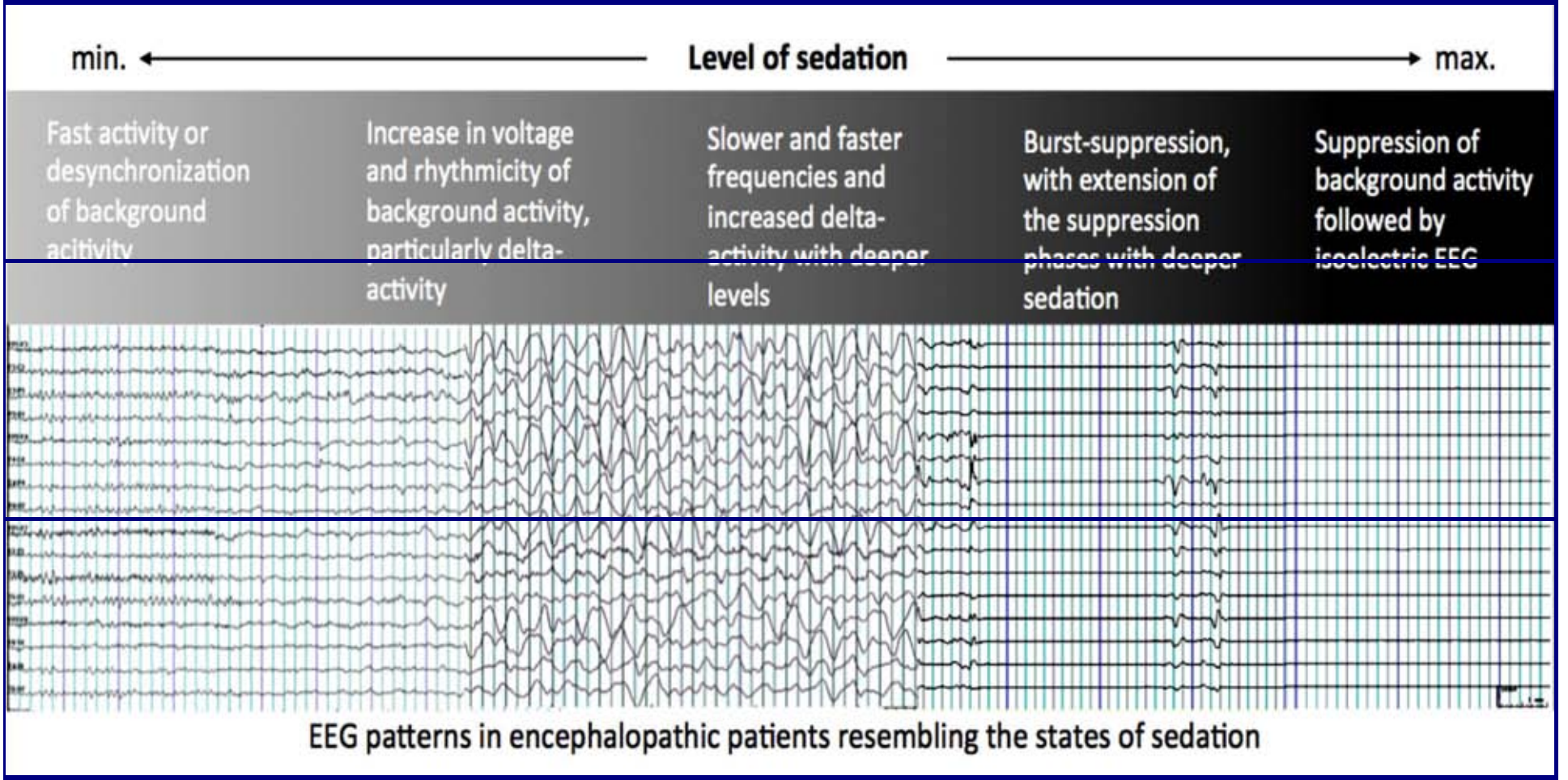
The predefined EEG patterns



The EEG in Acute Encephalopathies

- Many EEG patterns are non-specific for etiology and prognosis
- EEG can suggest a toxic/metabolic cause for encephalopathy
- Certain patterns provide prognosis in anoxic encephalopathy

- Mild: slowing of the posterior dominant alpha rhythm
- Moderate: Alpha replaced by diffuse theta. EEG is reactive
- Moderately Severe: high voltage delta activity with loss of fast frequencies. EEG less variable. Decreased reactivity.
- Severe: low voltage delta activity; minimal variability and reactivity. EEG becomes isoelectric and unreactive.



EEG patterns in encephalopathic patients resembling the states of sedation

Stockard & Bickford. The neurophysiology of anesthesia.
In: The neurophysiology of anesthesia. Amsterdam: Excerpta Medica, 1975;3-46.

Sutter R, Kaplan P W. J Clin Neurophysiol 2013

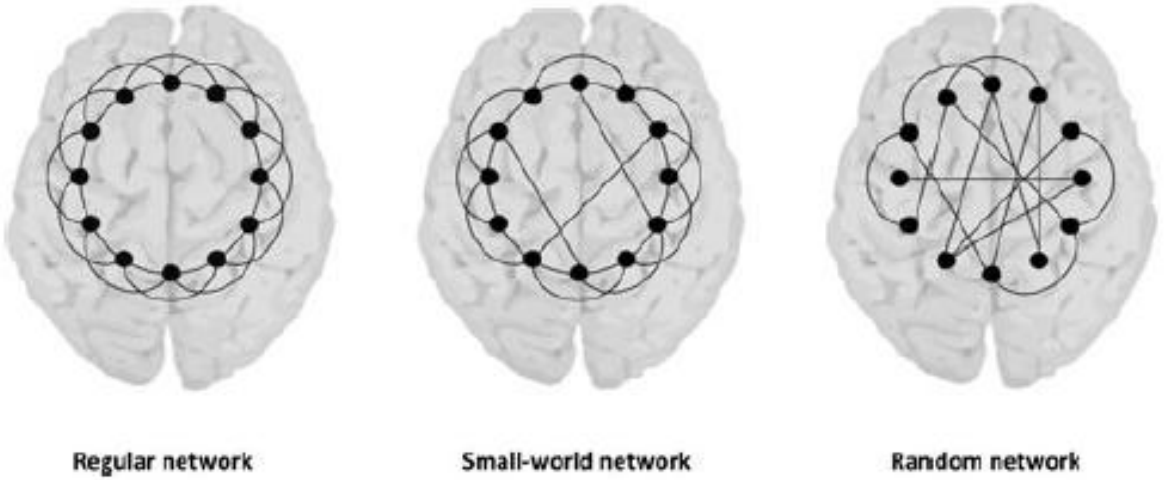
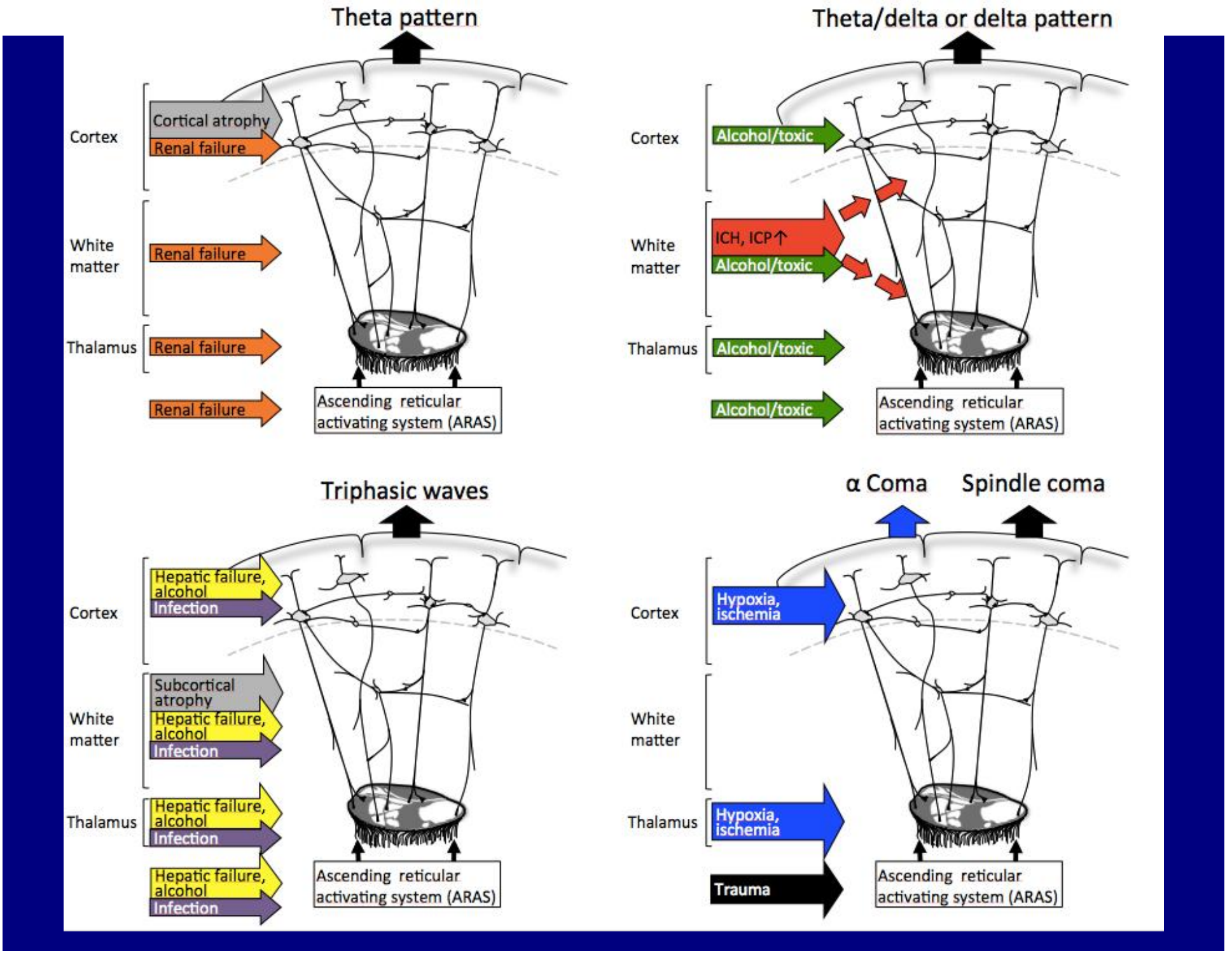
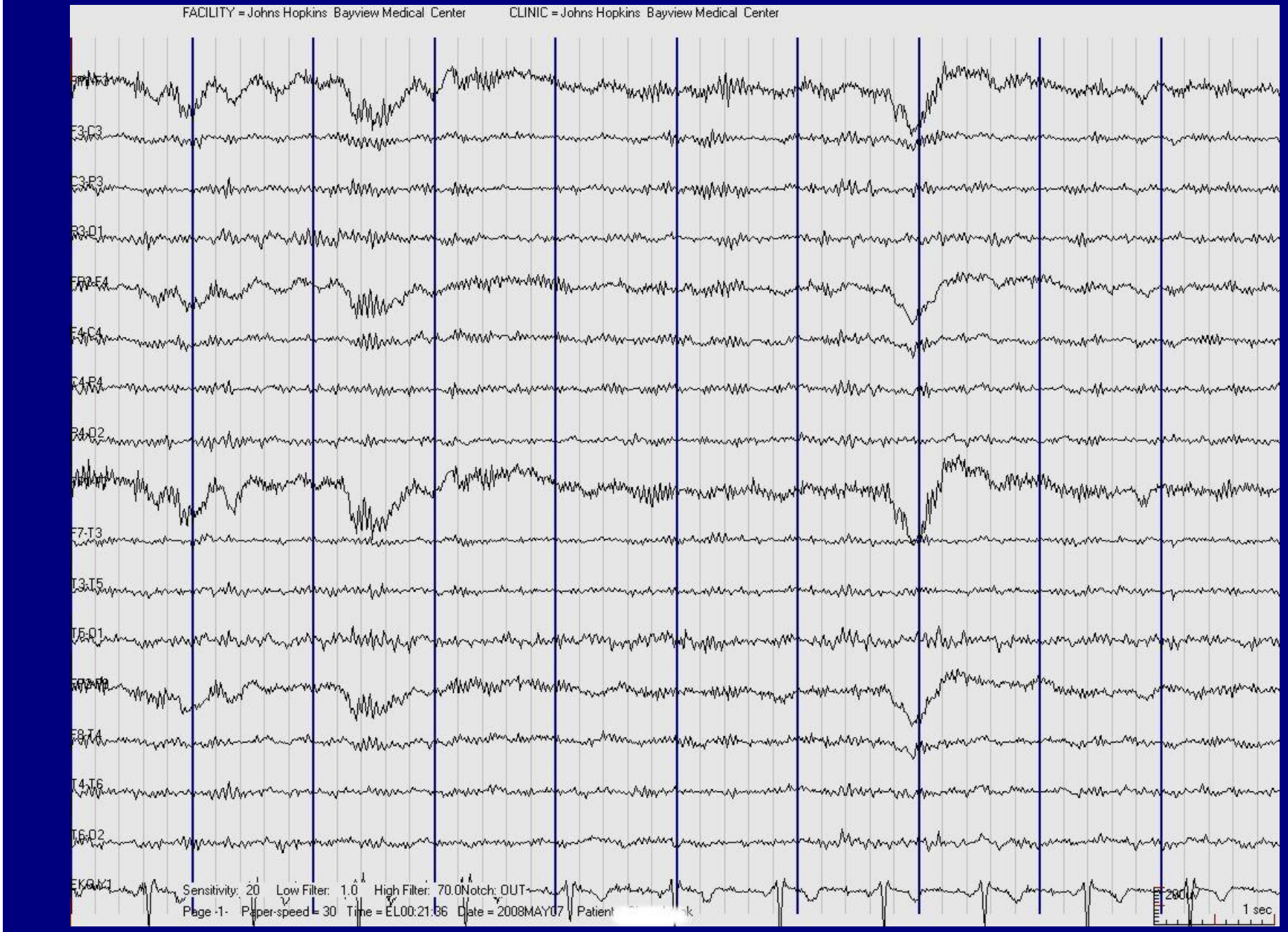


Figure 2.

Three basic network topologies: regular, small-world, and random. A regular network (left figure) has a characteristic high average clustering coefficient (on average, there is a high connection probability of nearest neighboring nodes) and a long average path length (on average, many connections must be traversed to travel from one node to another) resulting in a good local connectedness. The opposite is true for a random network (right figure) (low average clustering coefficient and short average path length). In a small-world network configuration (middle figure), the advantages of a regular and random network are combined into an efficient network topology.

Epilepsia © ILAE


Theta pattern
Cortex
Cortical atrophy
Renal failure
White matter
Renal failure
Thalamus
Renal failure
Renal failure
Ascending reticular activating system (ARAS)
Theta/delta or delta pattern
Cortex
Alcohol/toxic
White matter
ICH, ICP↑
Alcohol/toxic
Thalamus
Alcohol/toxic
Alcohol/toxic
Ascending reticular activating system (ARAS)
Triphasic waves
Cortex
Hepatic failure, alcohol
Infection
White matter
Subcortical atrophy
Hepatic failure, alcohol
Infection
Thalamus
Hepatic failure, alcohol
Infection
Hepatic failure, alcohol
Infection
Ascending reticular activating system (ARAS)
α Coma
Spindle coma
Cortex
Hypoxia, ischemia
White matter
Thalamus
Hypoxia, ischemia
Trauma
Ascending reticular activating system (ARAS)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C3-P3
P3-O1
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T5-O1
F8-T4
T4-T6
T6-O2
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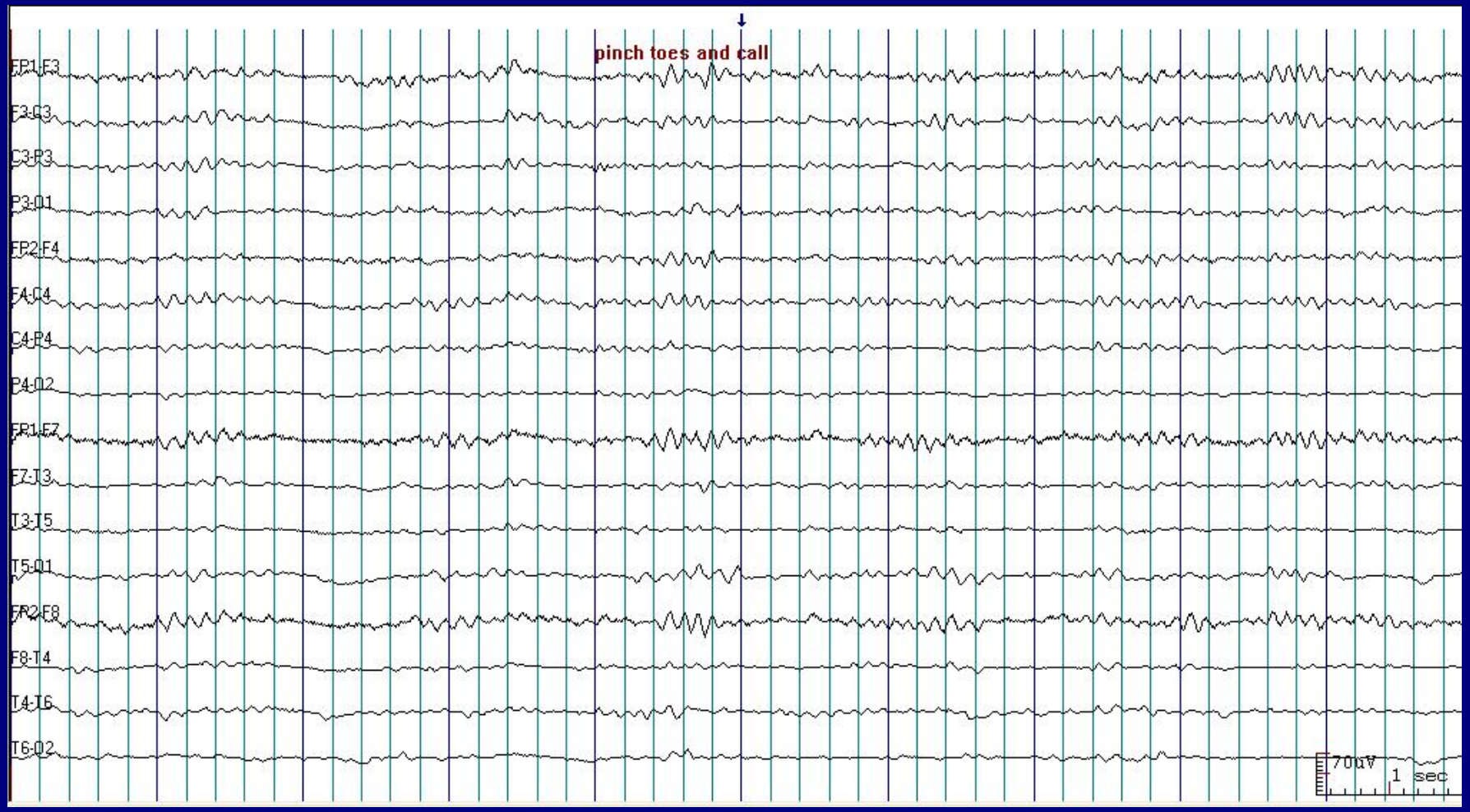
1 sec

Beta patterns

- Consists of beta frequency patterns
- Unlike beta drowsy patterns, it is:
  - More diffusely distributed
  - Anteriorly predominant; up to 35-40 Hz; some spindling
  - May be invariable and unreactive; patient unarousable (beta coma > 30 uV)
- Most common causes:
- Sedative withdrawal states
- Minimal brain dysfunction, dyslexia
- Brainstem infarction (Otomo E. Beta activity in the electroencephalogram in cases of coma due to acute brainstem lesion. J Neurol Neurosurg Psychiatry 1966;29:383-390)
- Drug intoxication usually with benzodiazepines, chloral hydrate or barbiturates


pinch toes and call
FP1-F3
F3-C3
C3-P3
P3-O1
FP2-F4
F4-C4
C4-P4
P4-O2
FP1-F7
F7-T3
T3-T5
T5-O1
FP2-F8
F8-T4
T4-T6
T6-O2
70uV
1 sec

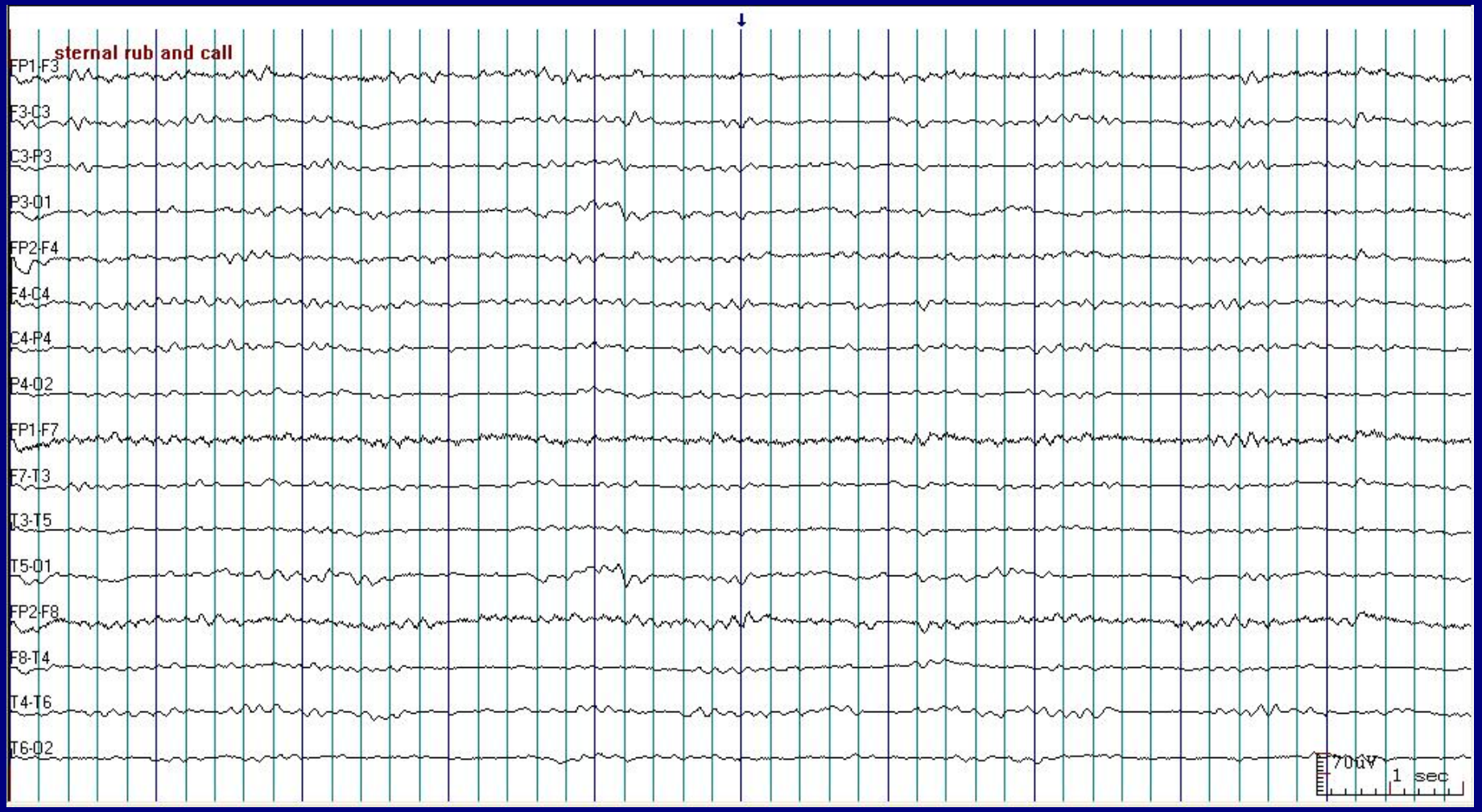
Alpha Coma

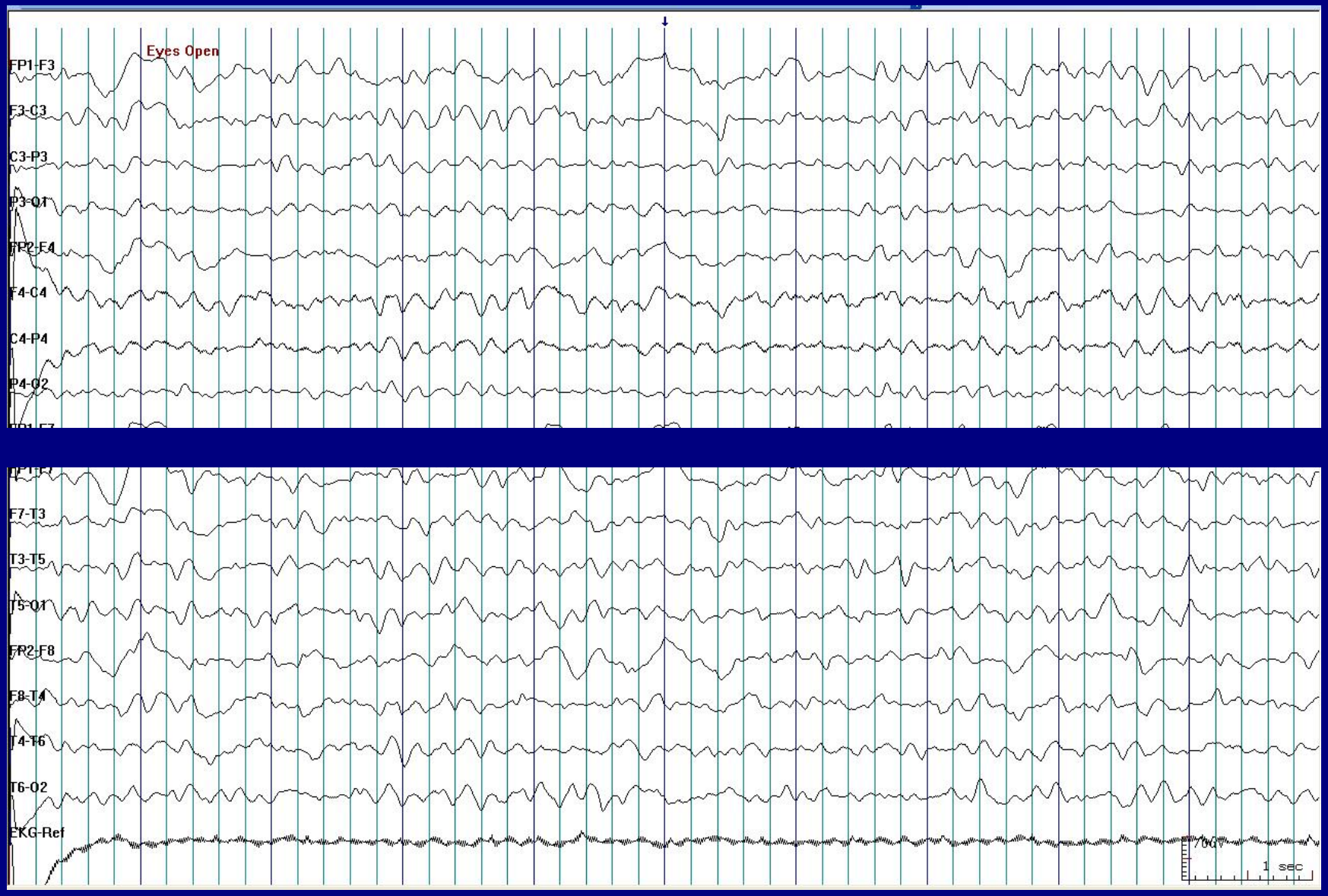
- First described by Loeb and Poggio (1953) in a patient with brainstem hemorrhage.
- Consists of alpha frequency activity
- Unlike waking alpha rhythm, it is:
  - Diffusely distributed
  - Anteriorly dominant
  - May be invariable and unreactive
- Most common causes:
- Anoxia (Alving et al 1979 and Austin et al 1988)
- Brainstem infarction (Grindal et al 1977)
- Traumatic brain injury (Chatrian et al, 1963)
- Drug intoxication (Carroll and Mastaglia 1979) usually with benzodiazepines or tricyclic antidepressants

Alpha Coma: Outcome?

- Overall mortality for the group was 256/335 (76%)
- Etiology predicted outcome (% mortality)
  - Anoxia: 88%
  - Brainstem infarct 90%
  - Drug intoxication 8%

Kaplan et al. Clin Neurophys 1999


sternal rub and call
FP1-F3
F3-C3
C3-P3
P3-O1
FP2-F4
F4-C4
C4-P4
P4-O2
FP1-F7
F7-T3
T3-T5
T5-O1
FP2-F8
F8-T4
T4-T6
T6-O2
70uV
1 sec

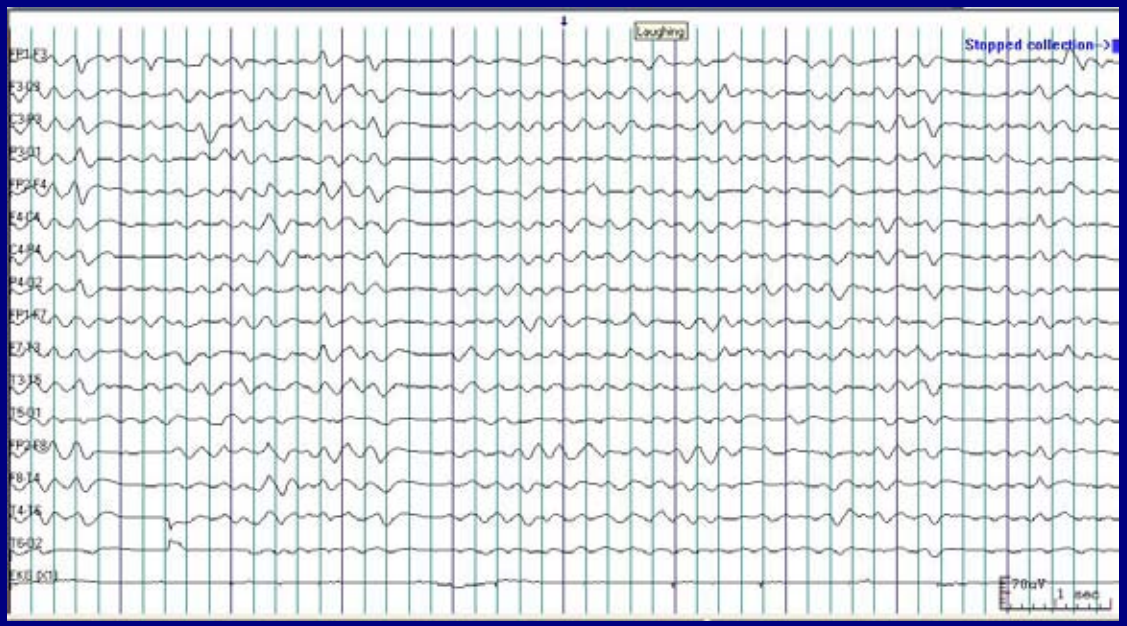

Eyes Open
FP1-F3
F3-C3
C3-P3
P3-O1
FP2-F4
F4-C4
C4-P4
P4-O2
F7-T3
T3-T5
T5-O1
FP2-F8
F8-T4
T4-T6
T6-O2
EKG-Ref
1 sec

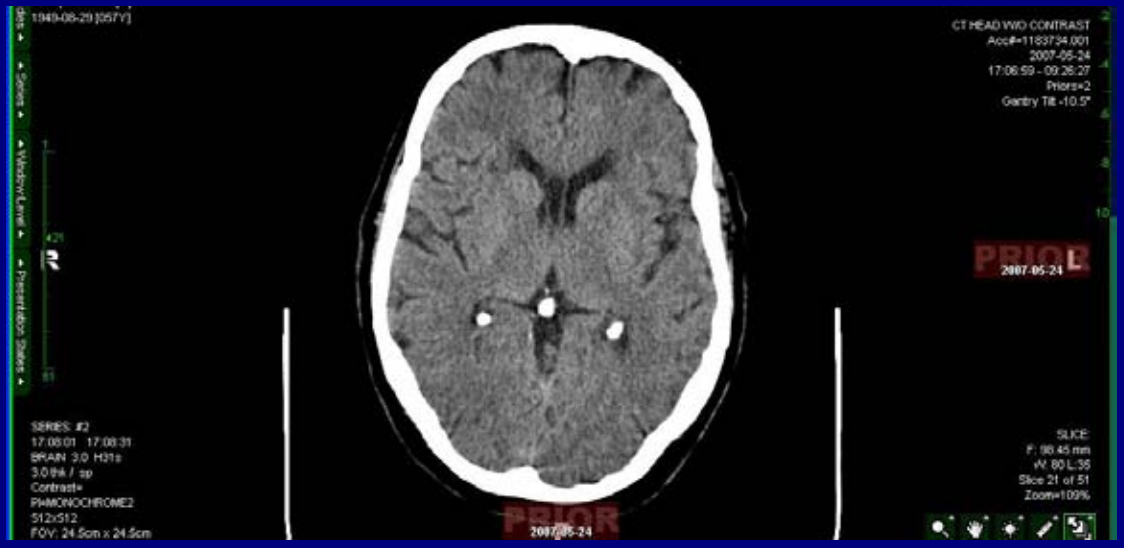
Theta encephalopathy

- Mild diffuse cortical dysfunction, elderly patients with multifactorial causes: toxic, metabolic, infectious and cerebral atrophy
- Confusion; intact brainstem
- Static encephalopathies,
- Reversible metabolic causes – good prognosis; atrophy - static

Gloor P et al., Brain. 1968; Silverman D. EEG Clin Neuro. 1963; Sutter, Stevens, Kaplan J Neurol 2013)

60-year-old, confused man after profound hypoglycemia from **acute** insulin excess





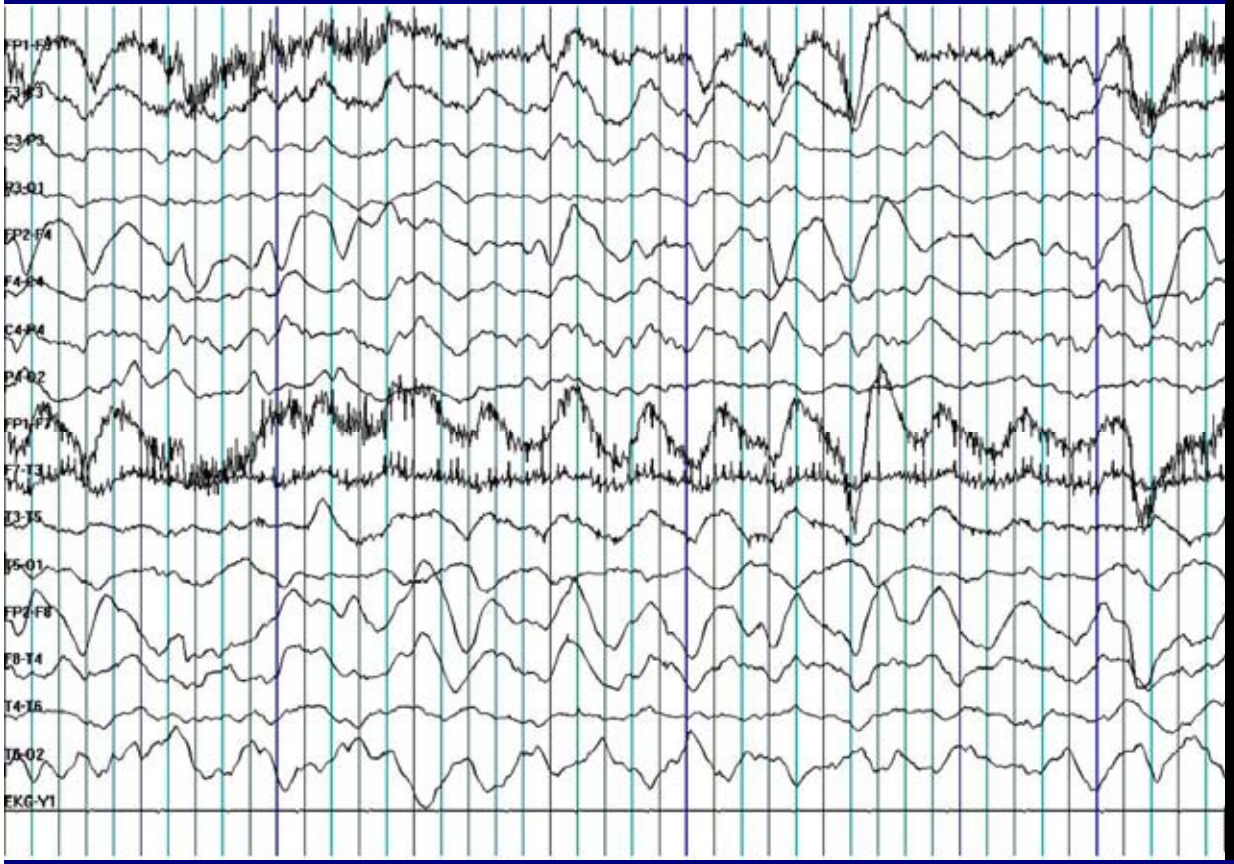
Polymorphic Delta Activity

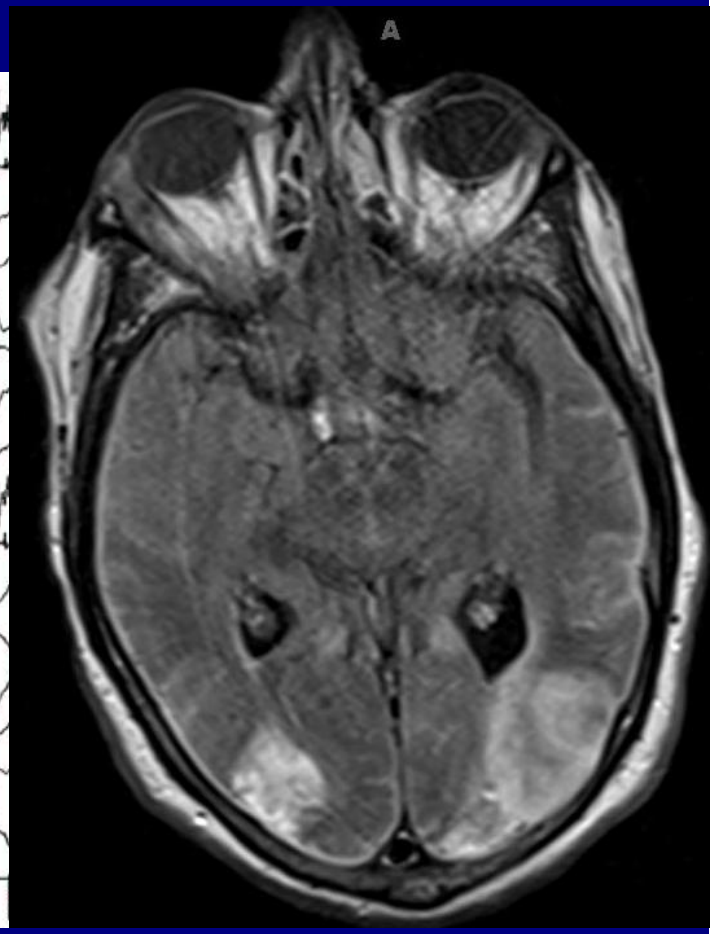
- Polymorphic delta activity (PDA): suggests dysfunction of the subcortical white matter
- Lesions which partially deafferent the subcortical white matter cause PDA
- PDA is generated by pyramidal neurons in layers II, III and V
- Caused by deep midline dysfunction near corpus callosum, brainstem, raised ICP, trauma, PRES, ICH, malarial encephalitis, severe metabolic failures
- Guarded prognosis as many causes are poorly reversible. PRES, narcosis – better prognosis

Sutter, Stevens, Kaplan J Neurol 2013

Structural correlates of encephalopathic patterns in EEG

Delta and PRES

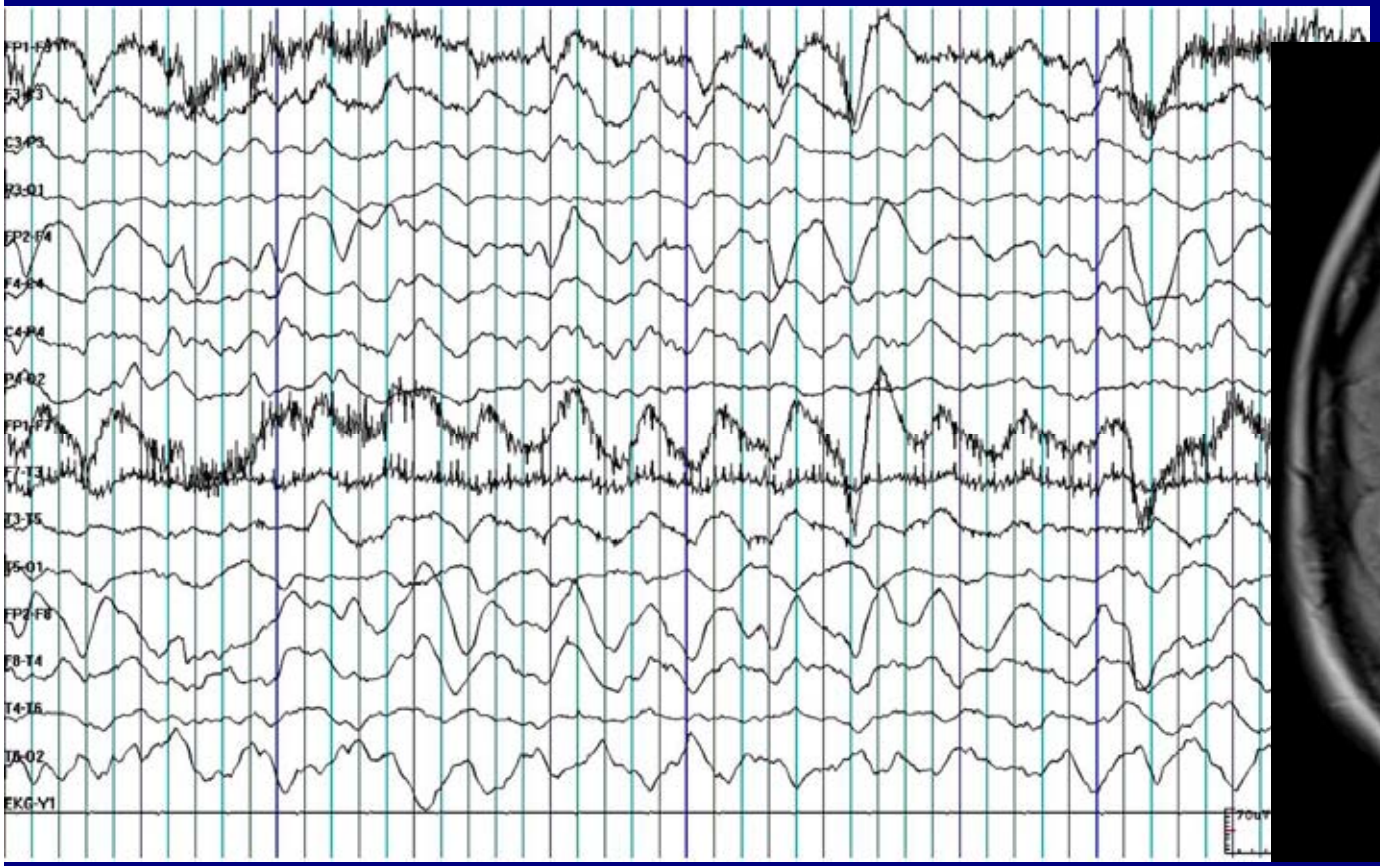


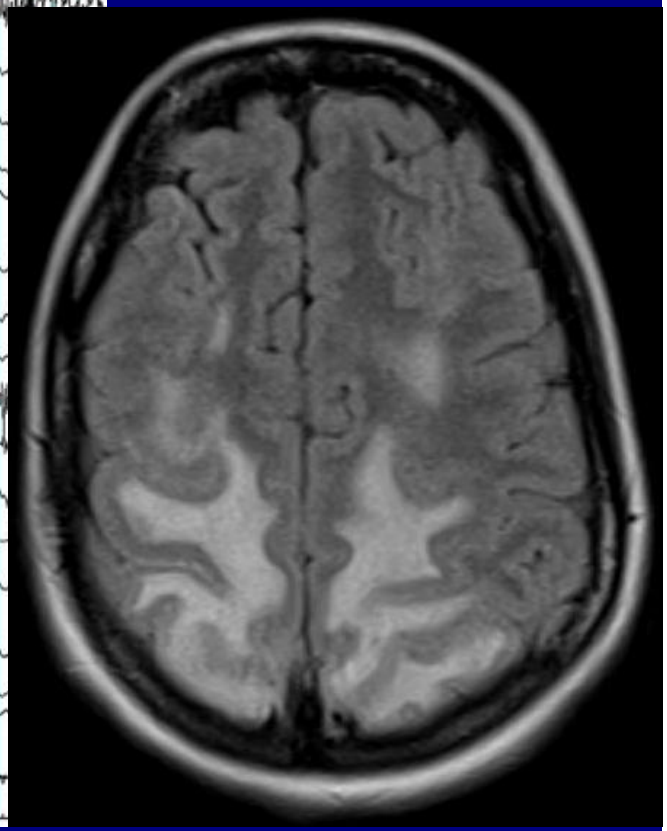


Structural correlates of encephalopathic patterns in EEG

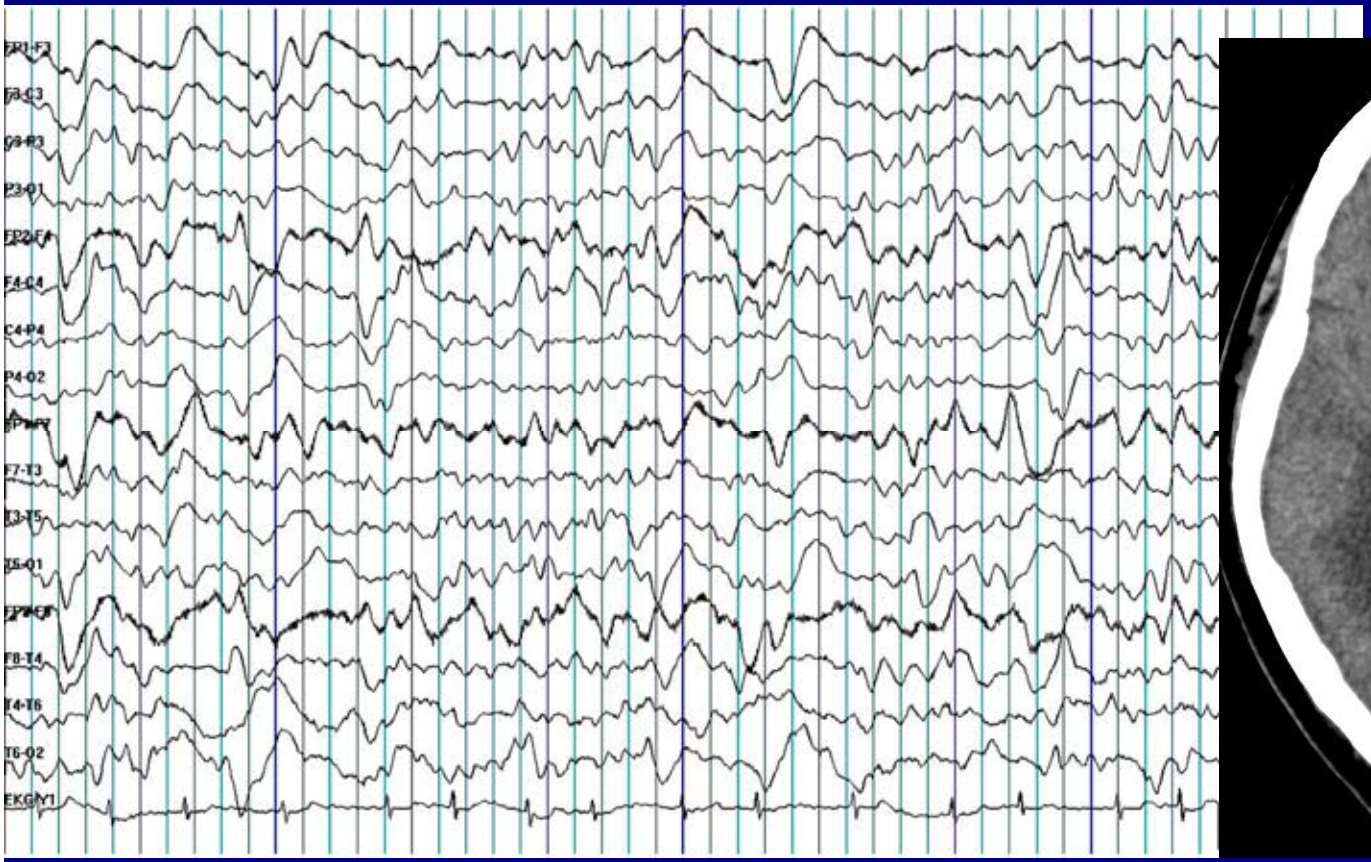
	Crude			Adjusted*		
Abnormalities / EEG pattern	**OR**	**95% CI**	**p-value**	**OR**	**95% CI**	**p-value**
PRES						
Delta	7.4	1.18 – 46.8	**0.033**	4.9	0.65 – 37.1	0.124

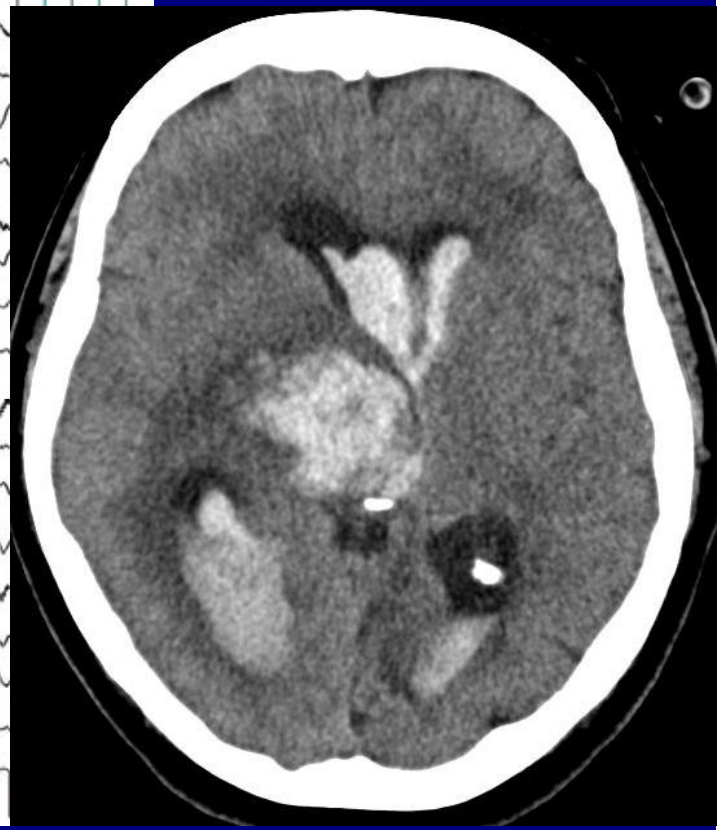
Delta and PRES



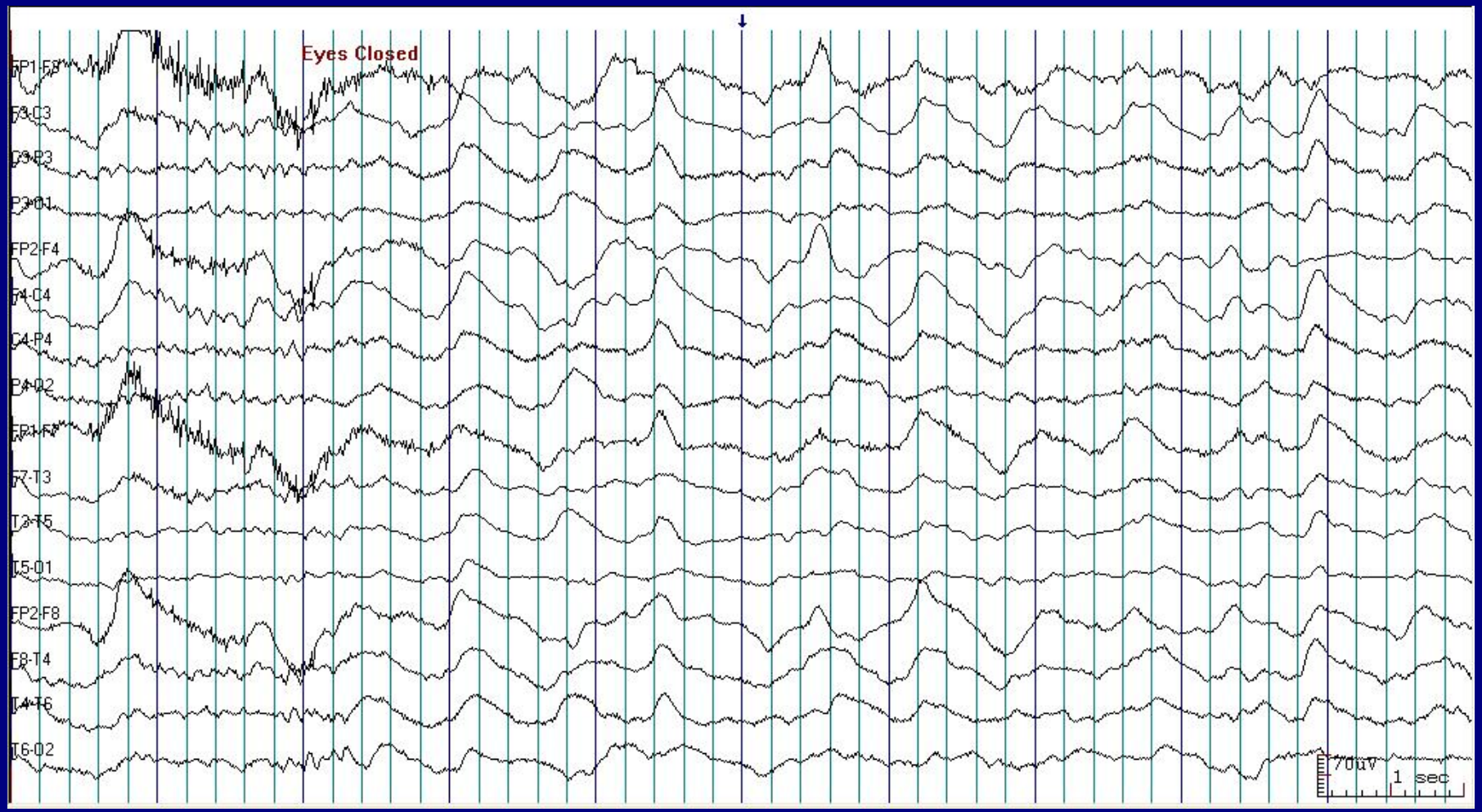


Theta-delta and ICH

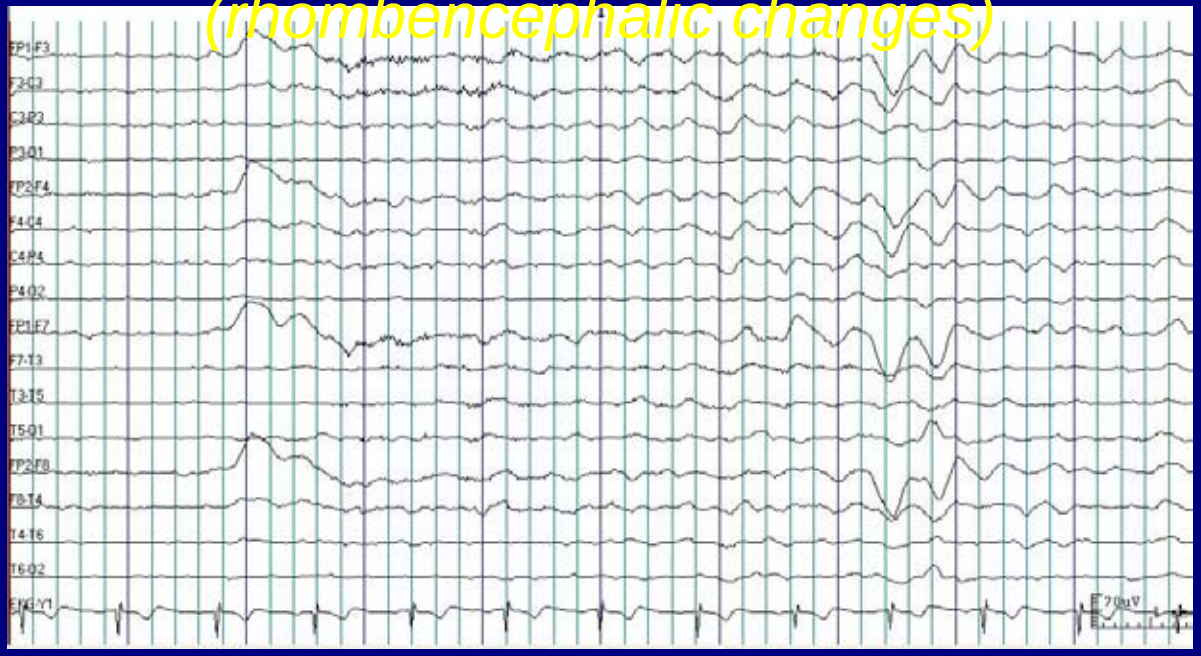


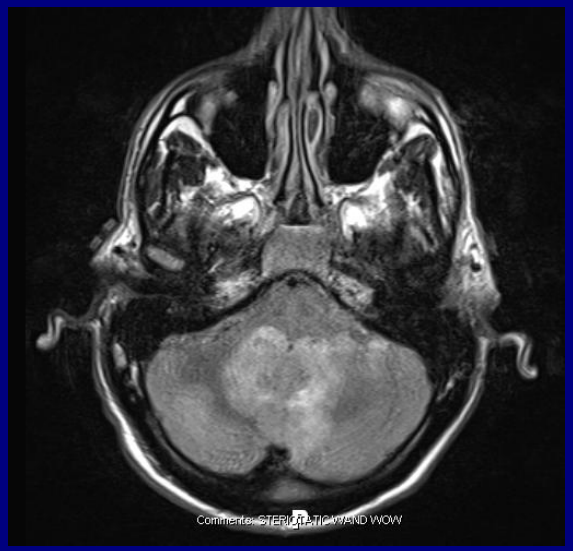


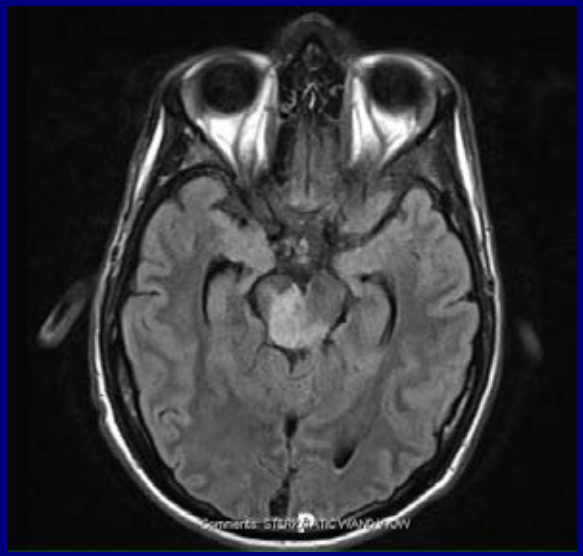
Metabolic encephalopathy – CO2 Narcosis [pCO2 - 107 mmHg]



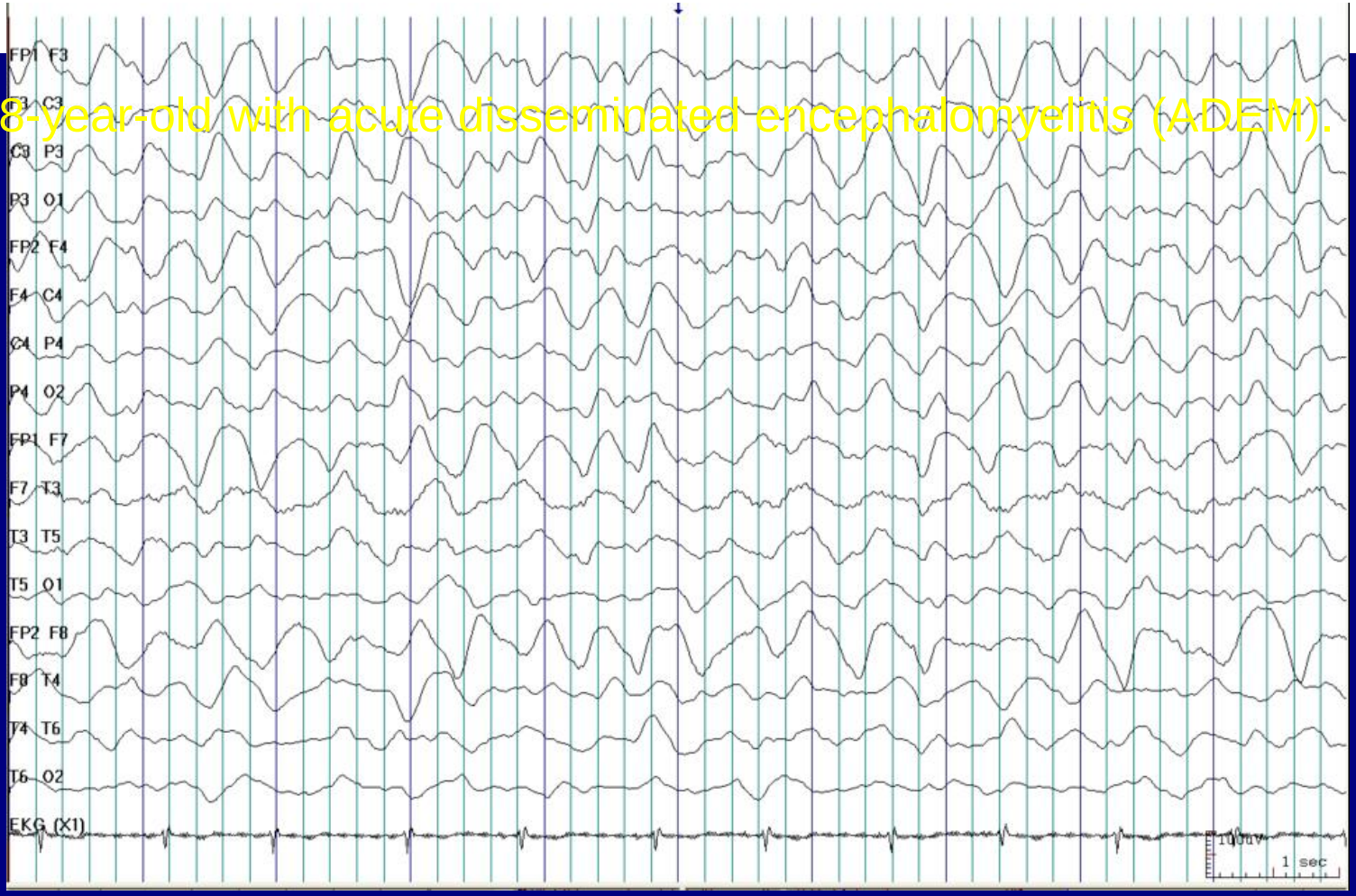
Diffuse slow activity (theta-delta) /MRI *midline/posterior fossa (rhombencephalic changes)*

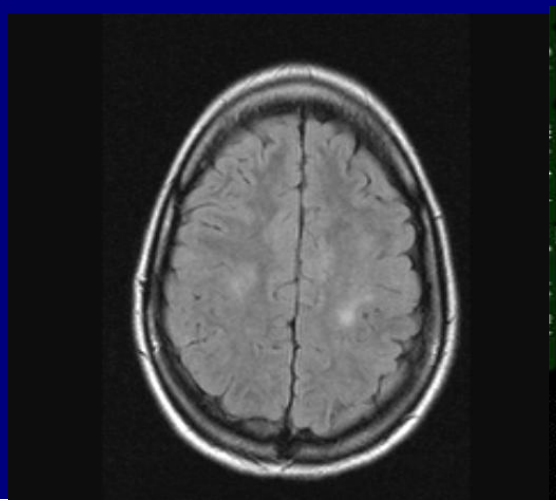


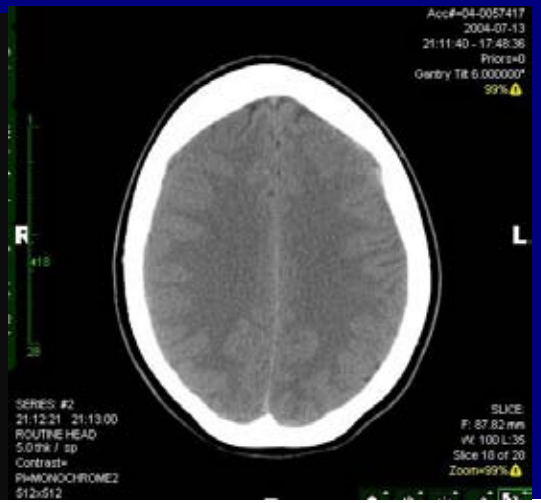




18-year-old with acute disseminated encephalomyelitis (ADEM).

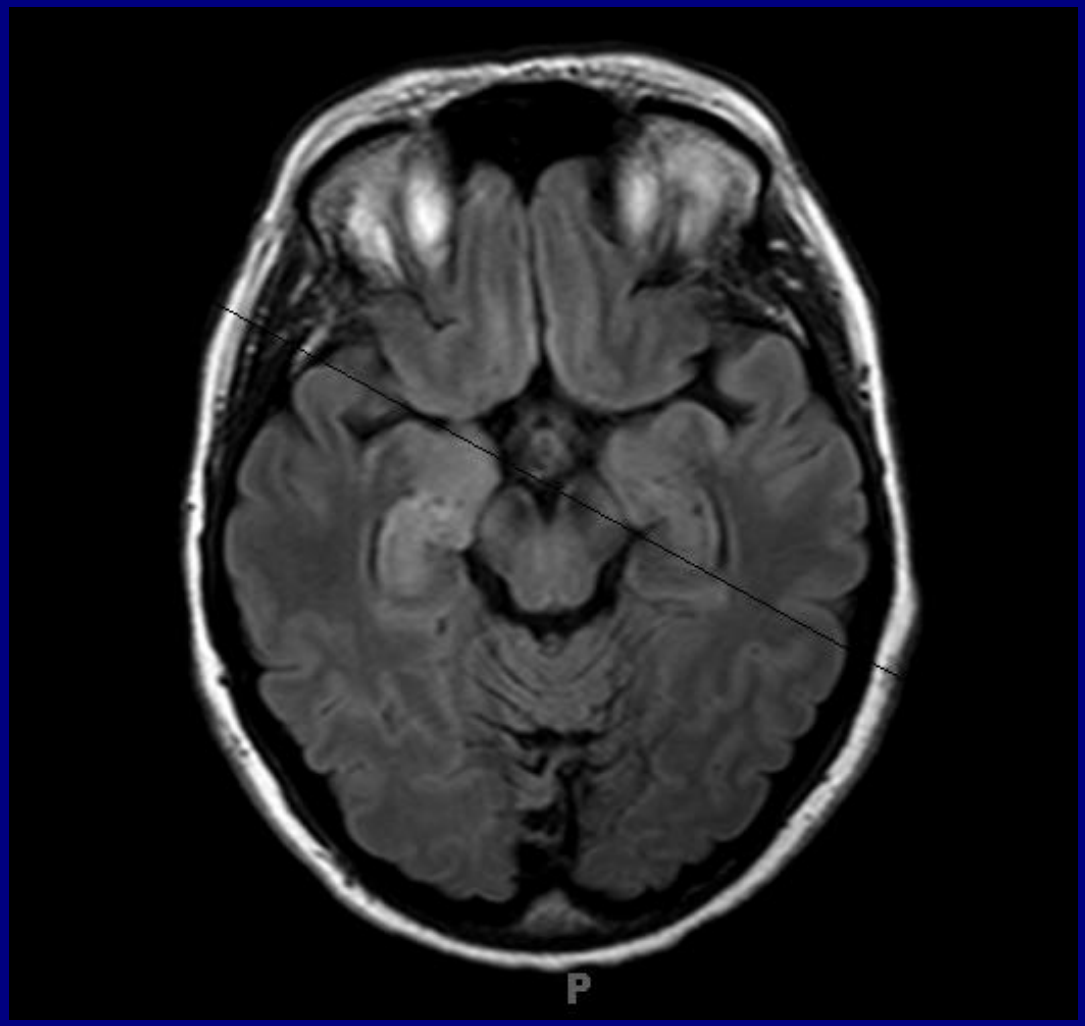






Continuous Rhythmic Delta Activity (RDA)

- Deep-seated epileptic foci
- Limbic encephalitis
- Limbic status epilepticus
- Types of toxic-metabolic encephalopathies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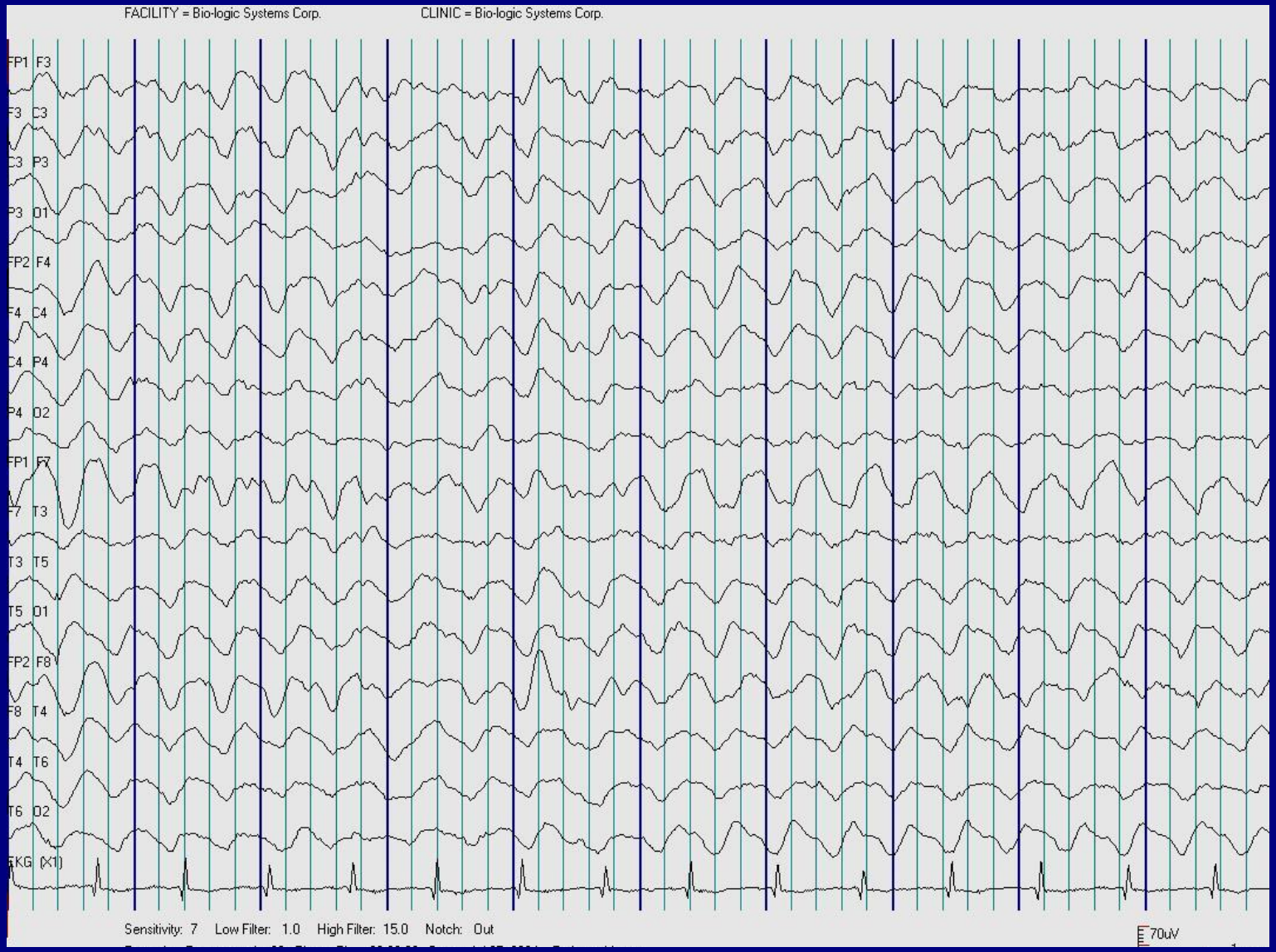
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CLINIC = Bio-logic Systems Corp.
FP1 F3
F3 C3
C3 P3
P3 O1
FP2 F4
F4 C4
C4 P4
P4 O2
FP1 F7
F7 T3
T3 T5
T5 O1
FP2 F8
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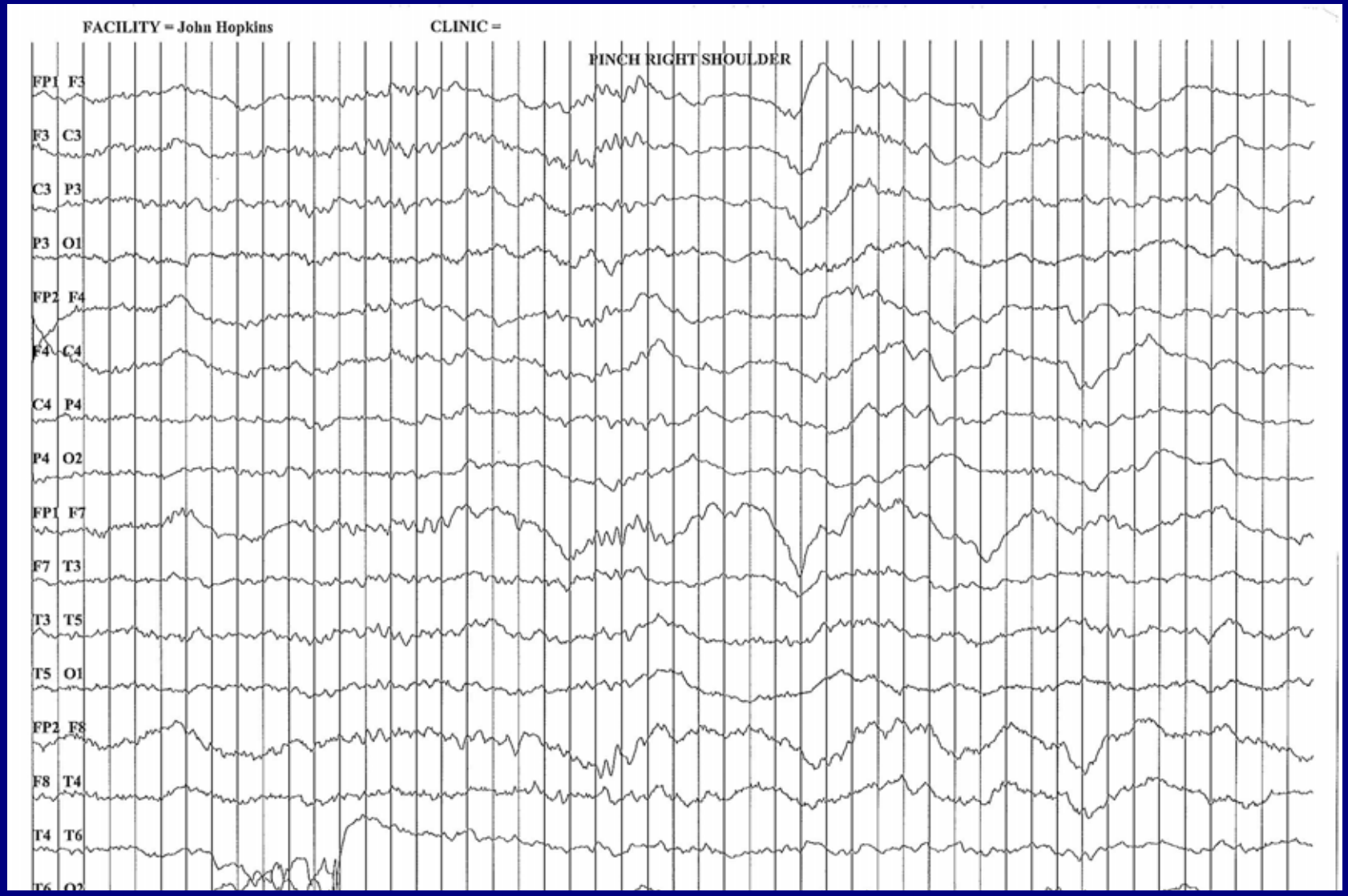
T4 T6
T6 O2
EKG (X1)
Sensitivity: 7 Low Filter: 1.0 High Filter: 15.0 Notch: Out
70uV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PINCH RIGHT SHOULDER
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F4 C4
C4 P4
P4 O2
FP1 F7
F7 T3
T3 T5
T5 O1
FP2 F8
F8 T4
T4 T6
T6 O2

Spindle Coma

- First described by Jasper and Van Buren (1953) in a patient neoplasia of the midbrain near the 3rd ventricle
- Consists of sleep-like activity with 9-14 Hz spindles
- Activity may occur on routine or prolonged EEG recordings, may be reactive to noxious stimuli, but not with awakening
- Traumatic brain injury Anoxia (Chatrian et al, 1963)
- Cerebral and brain stem strokes (Britt, 1980)
- Drug intoxication (Rumpl; Britt; Melo)
- Hypoxia (Rumpl; Britt; Melo)
- Meta-analysis (Kaplan et al. 2000)

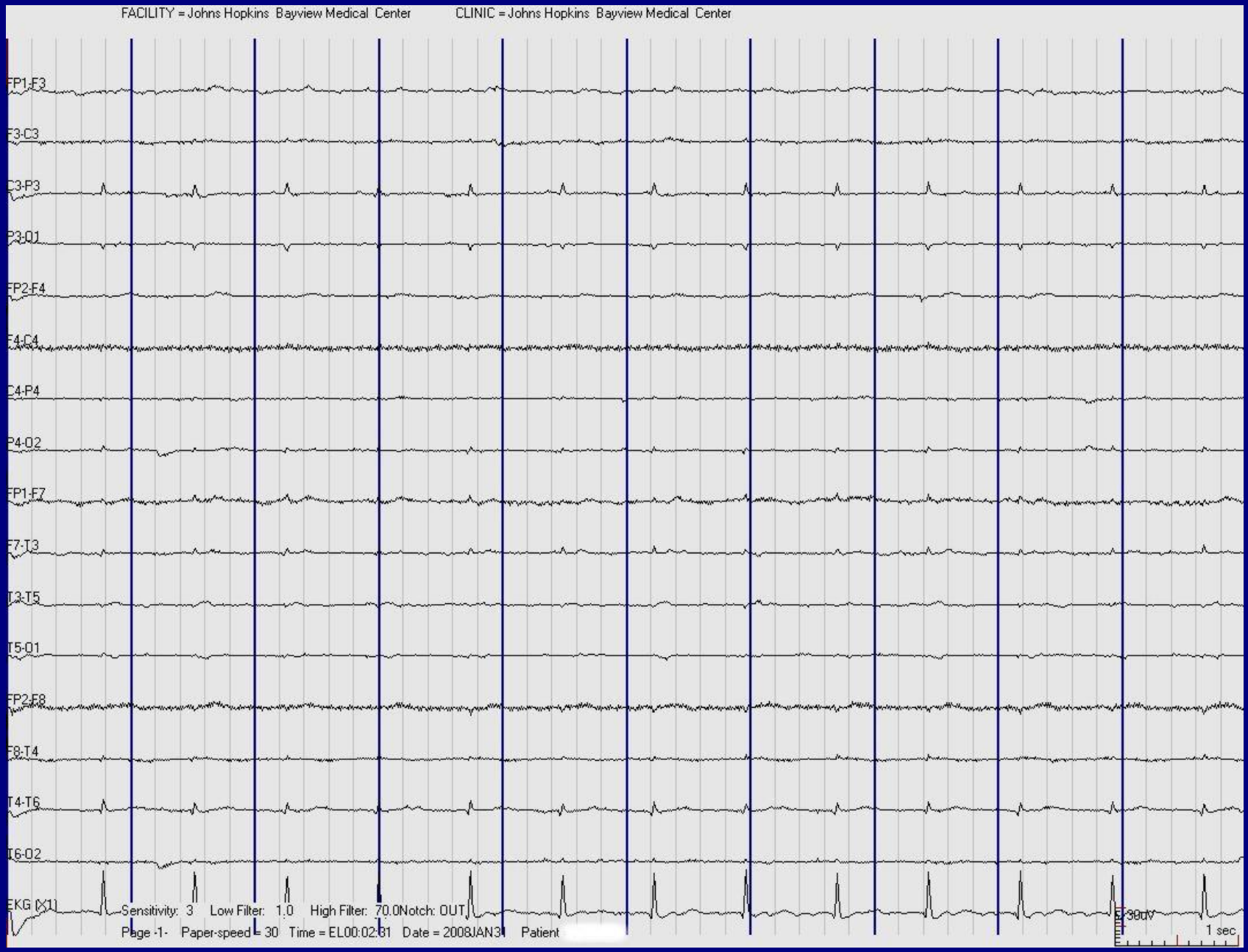
Spindle Coma: What Features Predict Outcome?

- Overall mortality for the group was 56/242 (23%)
- Etiology predicted outcome (% mortality)
  - Structural/brainstem path 73%
  - Hypoxia 33%
  - Trauma 15% Good outcome
  - Drugs/seizures/enceph 0% Good outcome

Kaplan et al, Clin Neurophys 2000

Low Voltage Severely Slow Unreactive and Electrocerebral Inactivity (ECI)

- Low voltage : (<20uV) delta activity
- ECI: no cerebral activity at instrumental sensitivities of 2uV/mm > 30 minutes
- No variability or reactivity
- Causes: anoxia, severe metabolic and ischemia
- After anoxia: prognosis - 0% return to consciousness (Zandbergen et al. Lancet)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FP1-F7
F7-T3
T3-T5
T5-O1
FP2-F8
F8-T4
T4-T6
T6-O2
EKG (X1)
Sensitivity: 3 Low Filter: 1.0 High Filter: 70.0Notch: OUT
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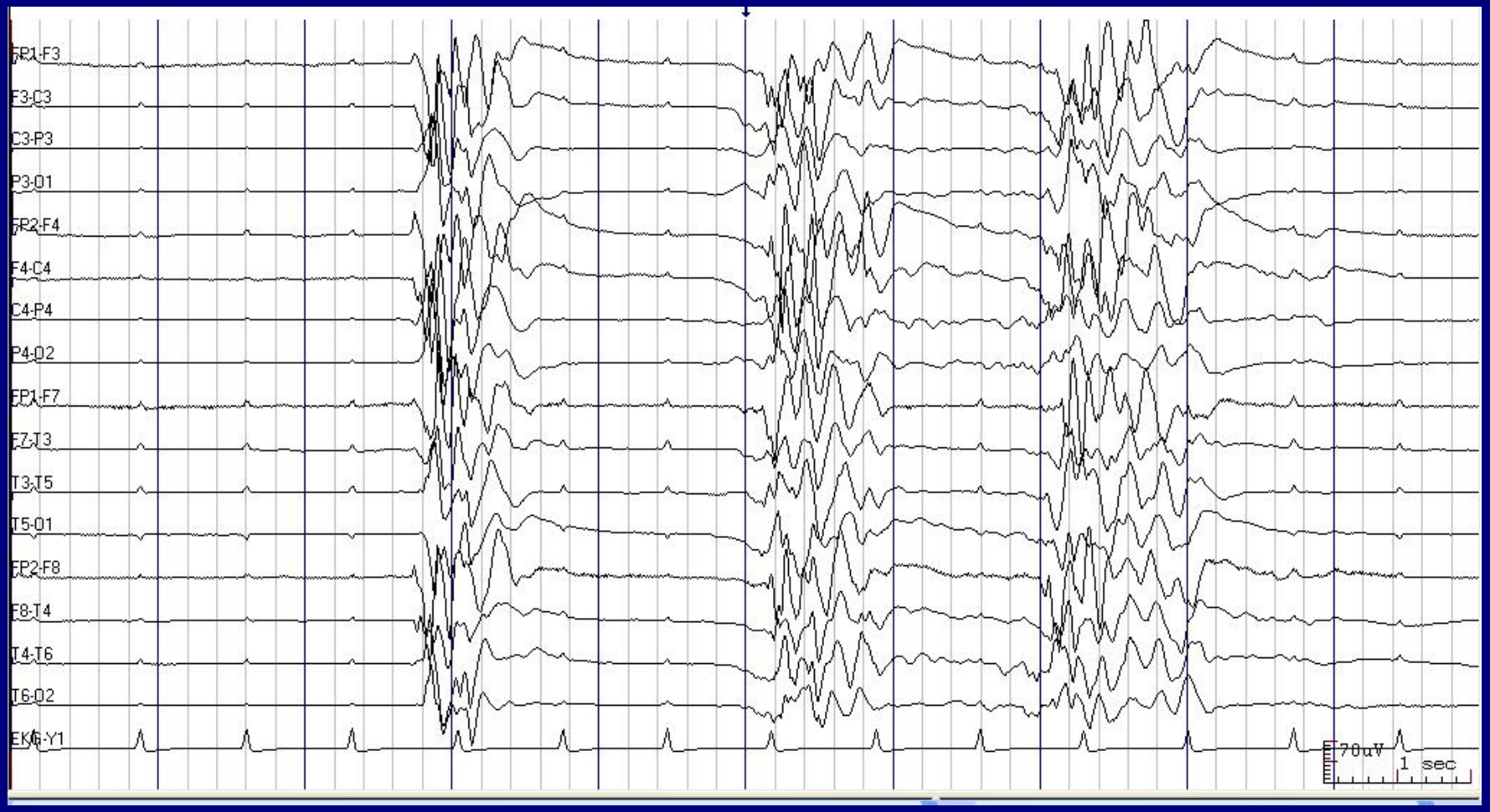
30uV
1 sec

Burst Suppression

- Consists of generalized bilaterally synchronous high-voltage theta/delta waves often with spikes and sharp waves
- In sedative (barbiturate or benzodiazepine intoxication) without anoxia, it has excellent prognosis
- After cardiac arrest and without hypothermia, prognosis is 0% for return to consciousness


FP1-F3
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F4-C4
C4-P4
P4-O2
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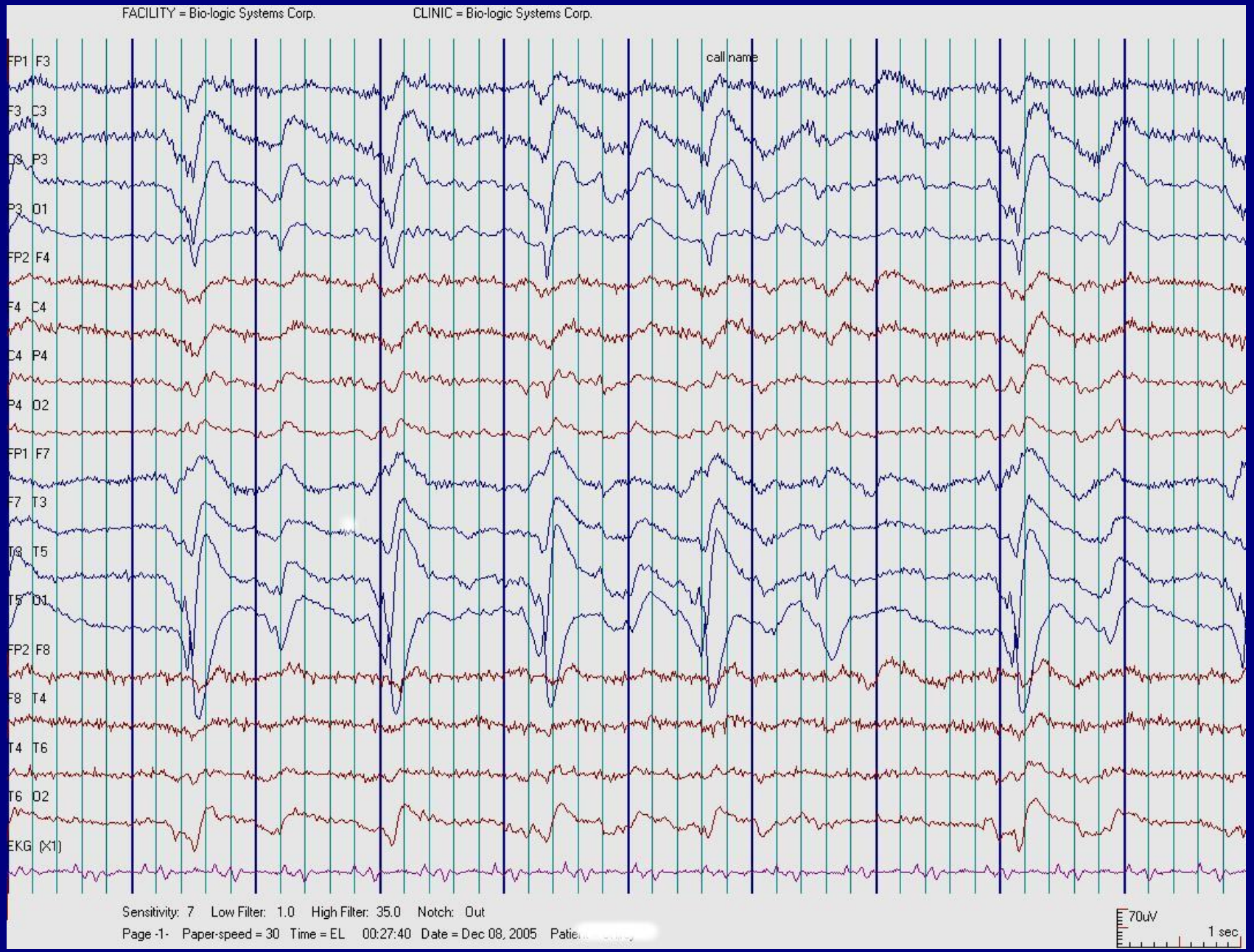
F7-T3
T3-T5
T5-O1
FP2-F8
F8-T4
T4-T6
T6-O2
EKG-Y1
70uV
1 sec

Burst Suppression

- EEG attenuations between bursts occur usual instrumental sensitivities for ≥1 second at least every 20 seconds (Synek 1988; Young 1997)
- In anoxic coma, it usually has an abysmal prognosis
- In sedative (barbiturate or benzodiazepine intoxication) without anoxia, it has excellent prognosis

Periodic EEG Activity

- Periodic lateralized epileptiform discharges (PLEDs). Structural (stroke, ICH, abscess). Moderate prognosis (Chatrian et al. EEG Clin Neurophys 1964)
- Bilateral independent periodic lateralized epileptiform discharges (BiPLEDs). Encephalitis, anoxia, metastases. Guarded prognosis (de la Paz, Brenner Arch Neurol 1981)
- Bilaterally synchronous periodic epileptiform discharges (GPEDs). Anoxia. Abysmal prognosis (Husain et al. JCNP 1999)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FP2 F8
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T6 O2
EKG (X1)
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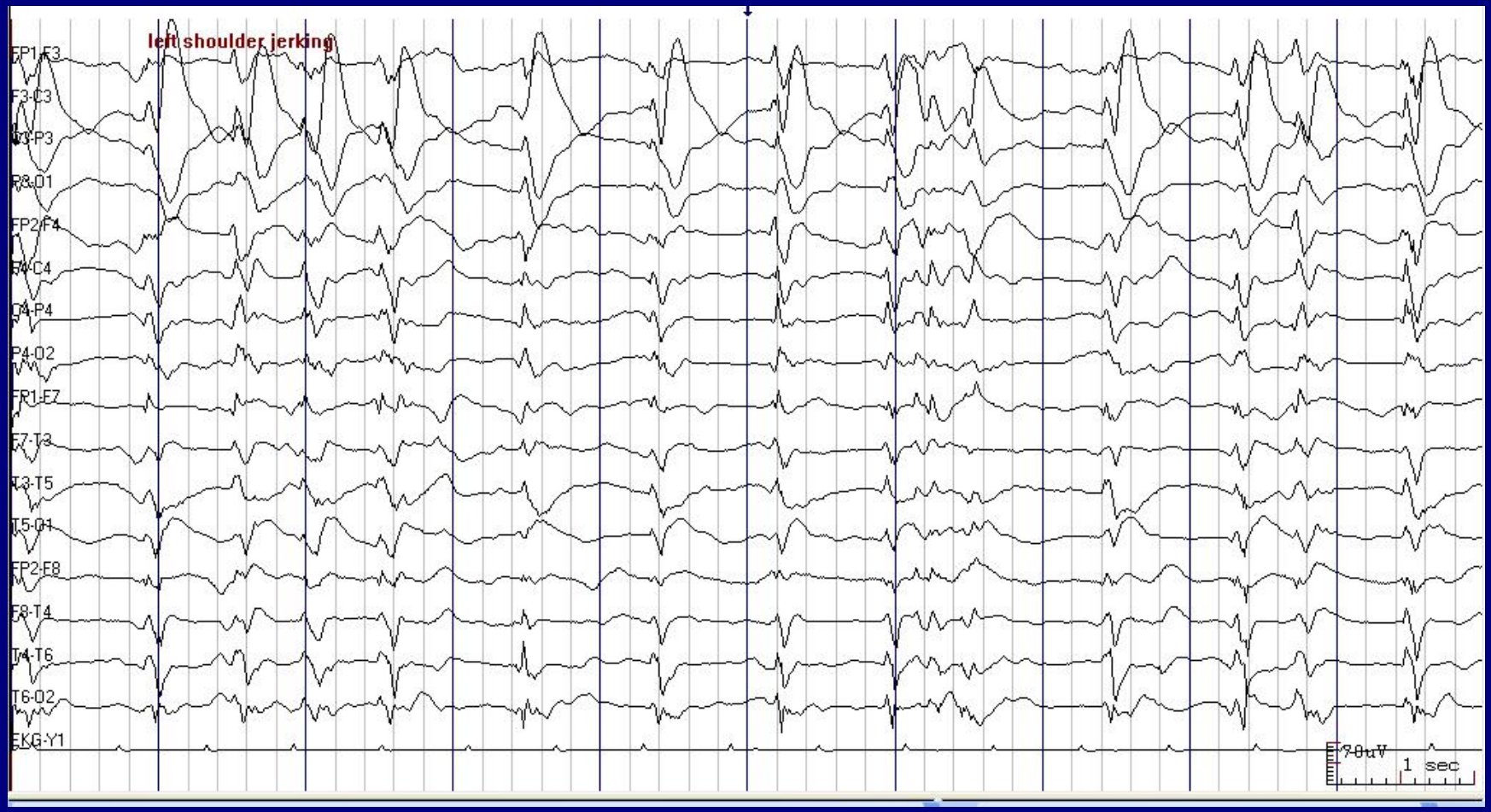
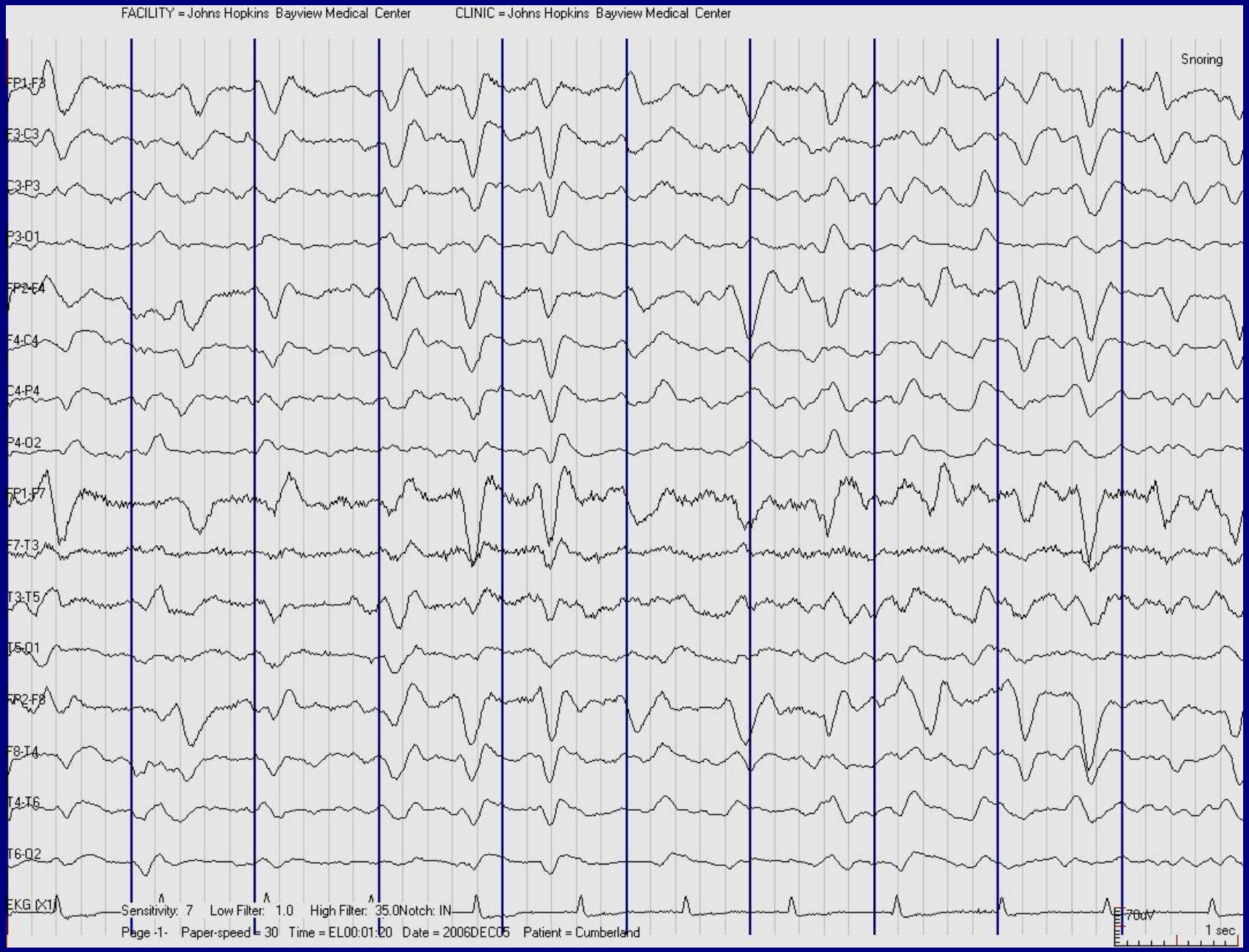
1 sec


left shoulder jerking
FP1-F3
F3-C3
C3-P3
P3-O1
FP2-F4
F4-C4
C4-P4
P4-O2
FP1-F7
F7-T3
T3-T5
T5-O1
FP2-F8
F8-T4
T4-T6
T6-O2
EKG-Y1
70uV
1 sec

Hepatic Encephalopathy: Triphasic Waves

- First described as “blunt spike-wave activity” by Foley et al (1950)
- Bickford and Butt (1955) used the term “triphasic waves”
- Three phases, largest phase having surface positivity. Preceded and followed by smaller amplitude negative waves. Repeat at 2-3Hz
- Usually anteriorly predominant, but diffuse.
- Reversible causes better prognosis. 30% mortality.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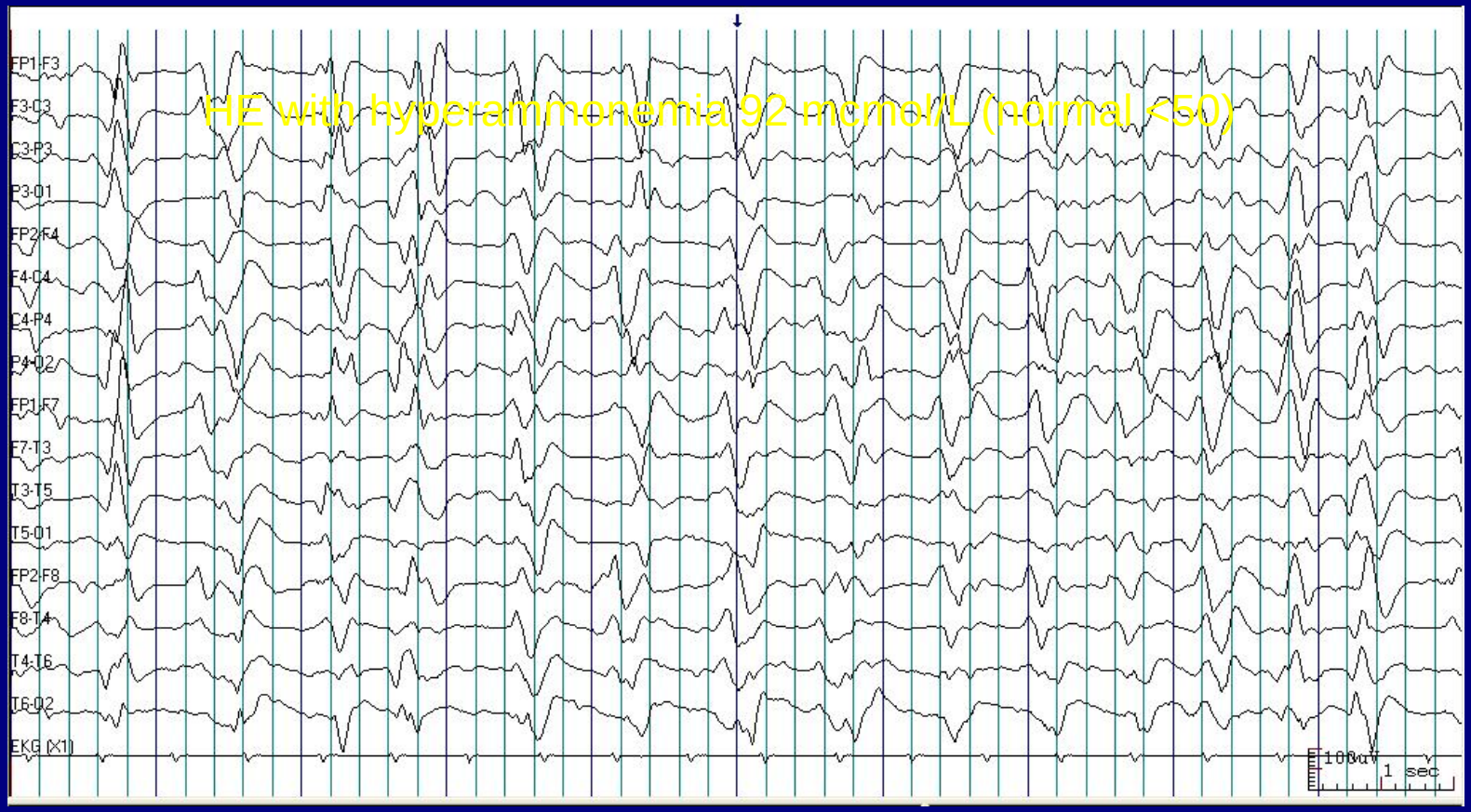
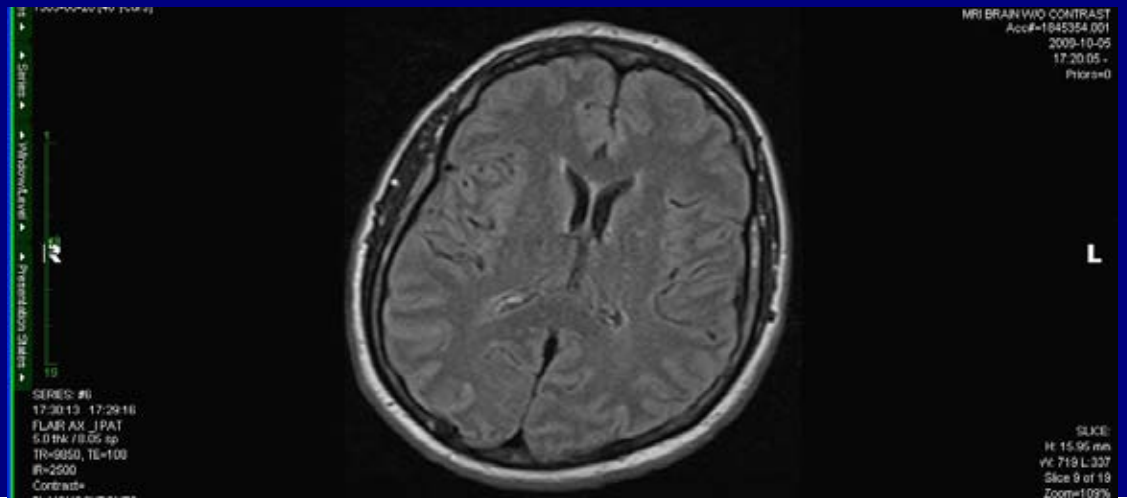
Snoring
FP1-F3
F3-C3
C3-P3
P3-O1
FP2-F4
F4-C4
C4-P4
P4-O2
FP1-F7
F7-T3
T3-T5
T5-O1
FP2-F8
F8-T4
T4-T6
T6-O2
EKG (X1)
Sensitivity: 7 Low Filter: 1.0 High Filter: 35.0Notch: IN
Page -1- Paper-speed = 30 Time = EL00:01:20 Date = 2006DEC05 Patient = Cumberland
70uV
1 sec

Triphasic Waves: Specificity

- Typical vs. Atypical triphasic wvaes
- These waves are non-specific and are also commonly seen in renal failure (Fisch and Klass 1988; Sundaram and Blume 1987).
- They have also been described in CJD, anoxia and less commonly hyponatremia and L-dopa induced encephalopathy

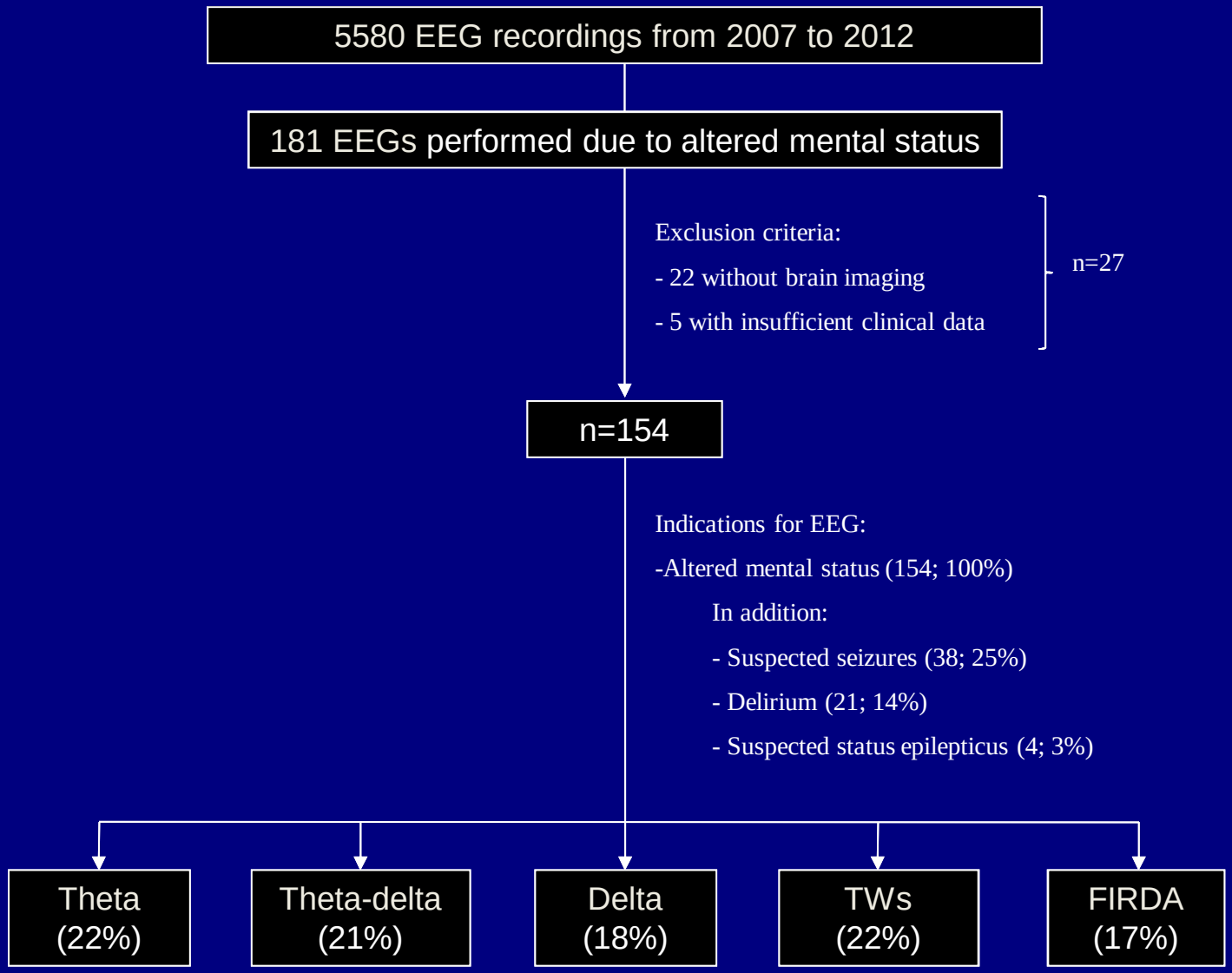

HE with hyperammonemia 92 mcmol/L (normal <50)
FP1-F3
F3-C3
C3-P3
P3-O1
FP2-F4
F4-C4
C4-P4
P4-O2
FP1-F7
F7-T3
T3-T5
T5-O1
FP2-F8
F8-T4
T4-T6
T6-O2
EKG (X1)
100uV
1 sec

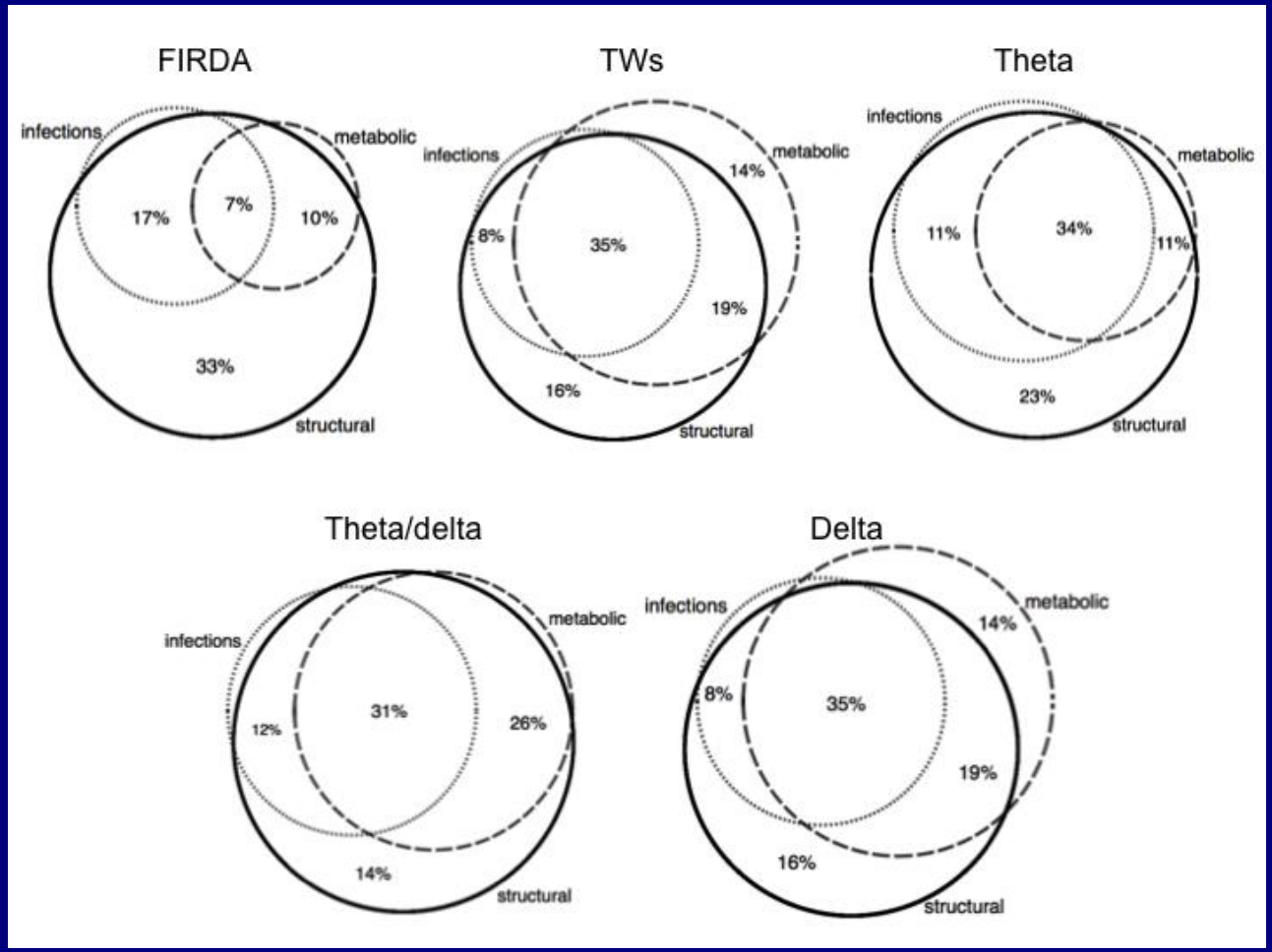

MRI BRAIN W/O CONTRAST
L
R

Clinical predispositions for triphasic waves (TWs)

- Without subcortical disease/ atrophy
  - Hepatic encephalopathy, hyperammonemia
  - Uremia, other marked electrolyte abnormalities
  - Anoxia
  - Toxins/drugs (e.g. lithium, baclofen)
- With white matter disease or diffuse
  - Mild infections (e.g. UTI, atrophy URTI)
  - Lesser degrees of electrolyte imbalance
  - Toxins

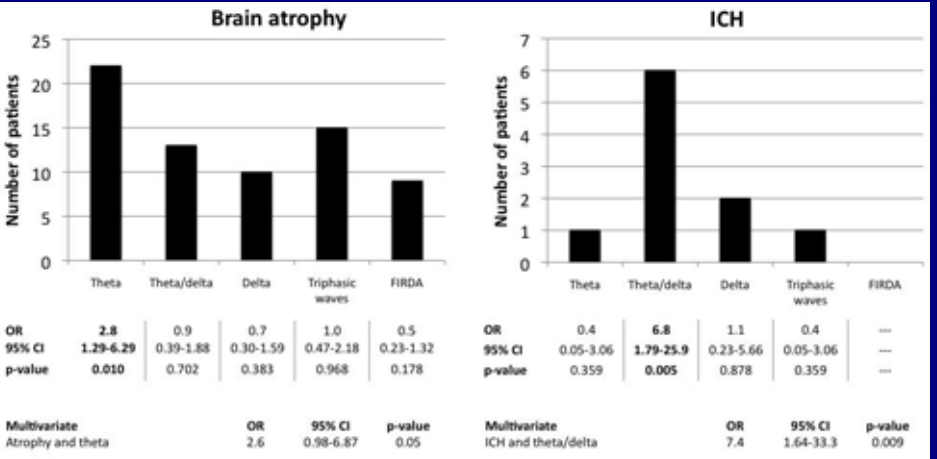
EEG Patterns in encephalopathy (Sutter, Stevens, Kaplan J Neurol 2013)




FIRDA
infections
metabolic
17%
7%
10%
33%
structural
TWs
infections
metabolic
14%
8%
35%
19%
16%
structural
Theta
infections
metabolic
11%
34%
11%
23%
structural
Theta/delta
infections
metabolic
12%
31%
26%
14%
structural
Delta
infections
metabolic
14%
8%
35%
19%
16%
structural

Structural abnormalities, encephalopathy and EEG patterns

Sutter, Stevens, Kaplan J Neurol 2013

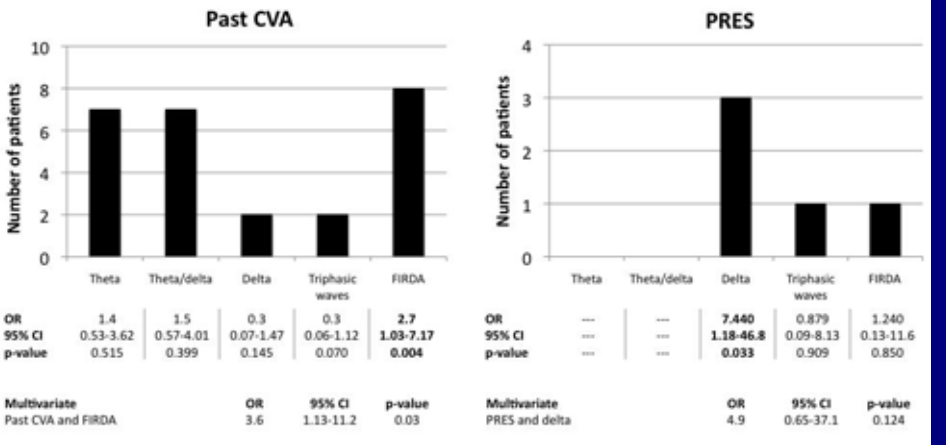


	Theta	Theta/delta	Delta	Triphasic waves	FIRDA
OR	**2.8**	0.9	0.7	1.0	0.5
95% CI	**1.29-6.29**	0.39-1.88	0.30-1.59	0.47-2.18	0.23-1.32
p-value	**0.010**	0.702	0.383	0.968	0.178

Multivariate	OR	95% CI	p-value
Atrophy and theta	2.6	0.98-6.87	0.05

	Theta	Theta/delta	Delta	Triphasic waves	FIRDA
OR	0.4	**6.8**	1.1	0.4	---
95% CI	0.05-3.06	**1.79-25.9**	0.23-5.66	0.05-3.06	---
p-value	0.359	**0.005**	0.878	0.359	---

Multivariate	OR	95% CI	p-value
ICH and theta/delta	7.4	1.64-33.3	0.009

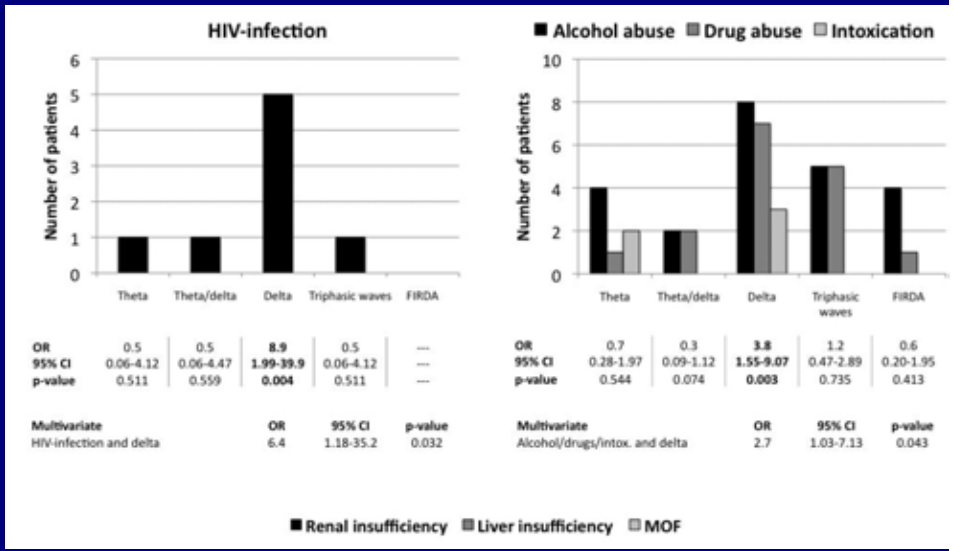


	Theta	Theta/delta	Delta	Triphasic waves	FIRDA
OR	1.4	1.5	0.3	0.3	**2.7**
95% CI	0.53-3.62	0.57-4.01	0.07-1.47	0.06-1.12	**1.03-7.17**
p-value	0.515	0.399	0.145	0.070	**0.004**

Multivariate	OR	95% CI	p-value
Past CVA and FIRDA	3.6	1.13-11.2	0.03

	Theta	Theta/delta	Delta	Triphasic waves	FIRDA
OR	---	---	**7.440**	0.879	1.240
95% CI	---	---	**1.18-46.8**	0.09-8.13	0.13-11.6
p-value	---	---	**0.033**	0.909	0.850

Multivariate	OR	95% CI	p-value
PRES and delta	4.9	0.65-37.1	0.124

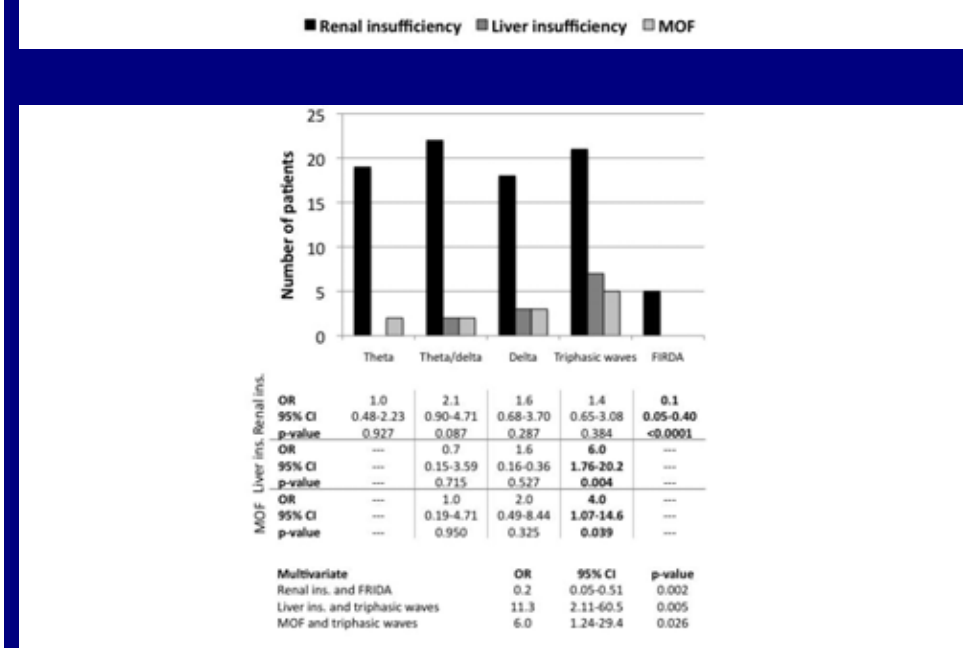


	Theta	Theta/delta	Delta	Triphasic waves	FIRDA
OR	0.5	0.5	**8.9**	0.5	---
95% CI	0.06-4.12	0.06-4.47	**1.99-39.9**	0.06-4.12	---
p-value	0.511	0.559	**0.004**	0.511	---

Multivariate	OR	95% CI	p-value
HIV-infection and delta	6.4	1.18-35.2	0.032

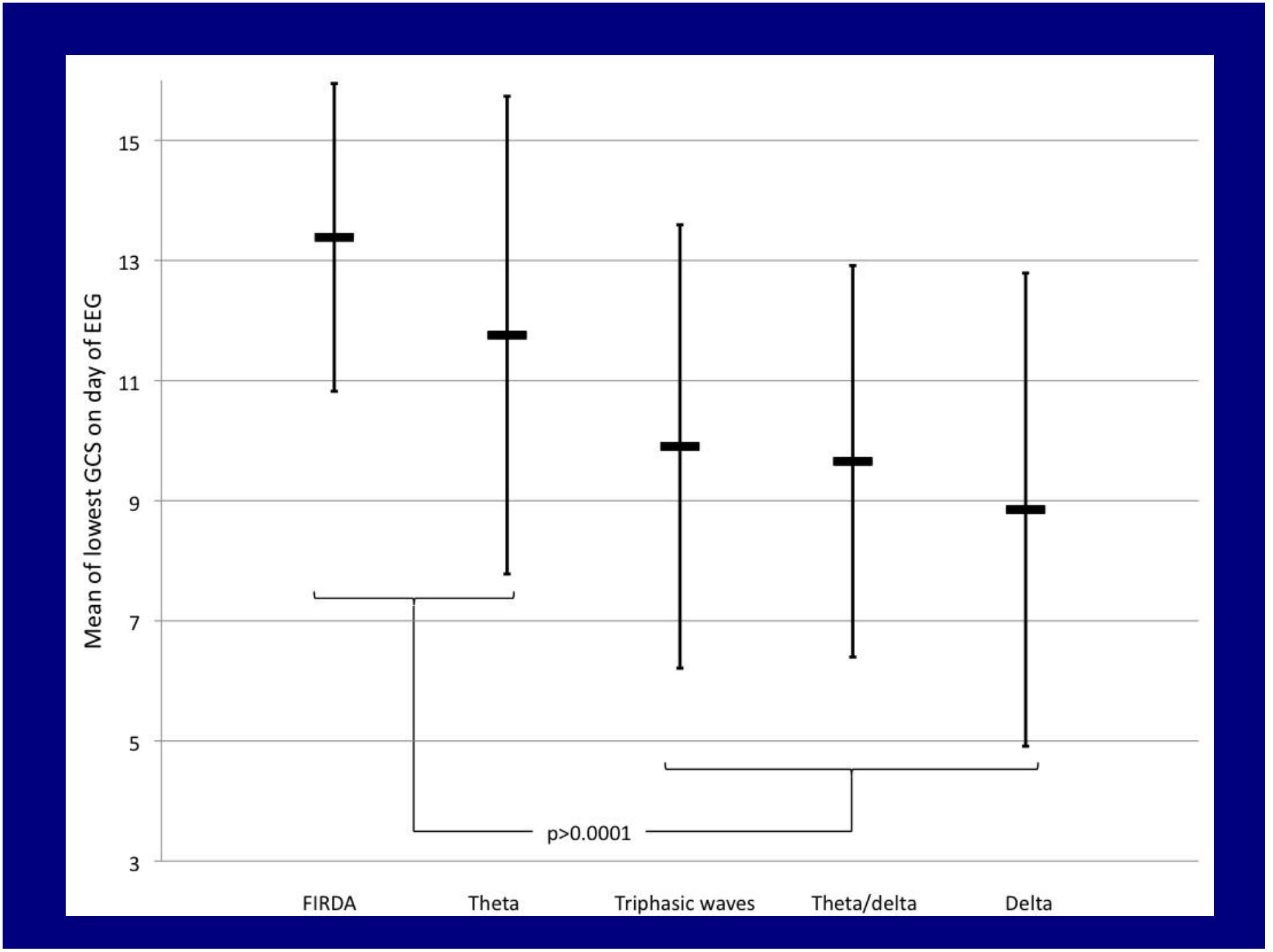
	Theta	Theta/delta	Delta	Triphasic waves	FIRDA
OR	0.7	0.3	**3.8**	1.2	0.6
95% CI	0.28-1.97	0.09-1.12	**1.55-9.07**	0.47-2.89	0.20-1.95
p-value	0.544	0.074	**0.003**	0.735	0.413

Multivariate	OR	95% CI	p-value
Alcohol/drugs/intox. and delta	2.7	1.03-7.13	0.043



	Theta	Theta/delta	Delta	Triphasic waves	FIRDA
Renal ins. OR	1.0	2.1	1.6	1.4	**0.1**
95% CI	0.48-2.23	0.90-4.71	0.68-3.70	0.65-3.08	**0.05-0.40**
p-value	0.927	0.087	0.287	0.384	**<0.0001**
Liver ins. OR	---	0.7	1.6	**6.0**	---
95% CI	---	0.15-3.59	0.16-0.36	**1.76-20.2**	---
p-value	---	0.715	0.527	**0.004**	---
MOF OR	---	1.0	2.0	**4.0**	---
95% CI	---	0.19-4.71	0.49-8.44	**1.07-14.6**	---
p-value	---	0.950	0.325	**0.039**	---

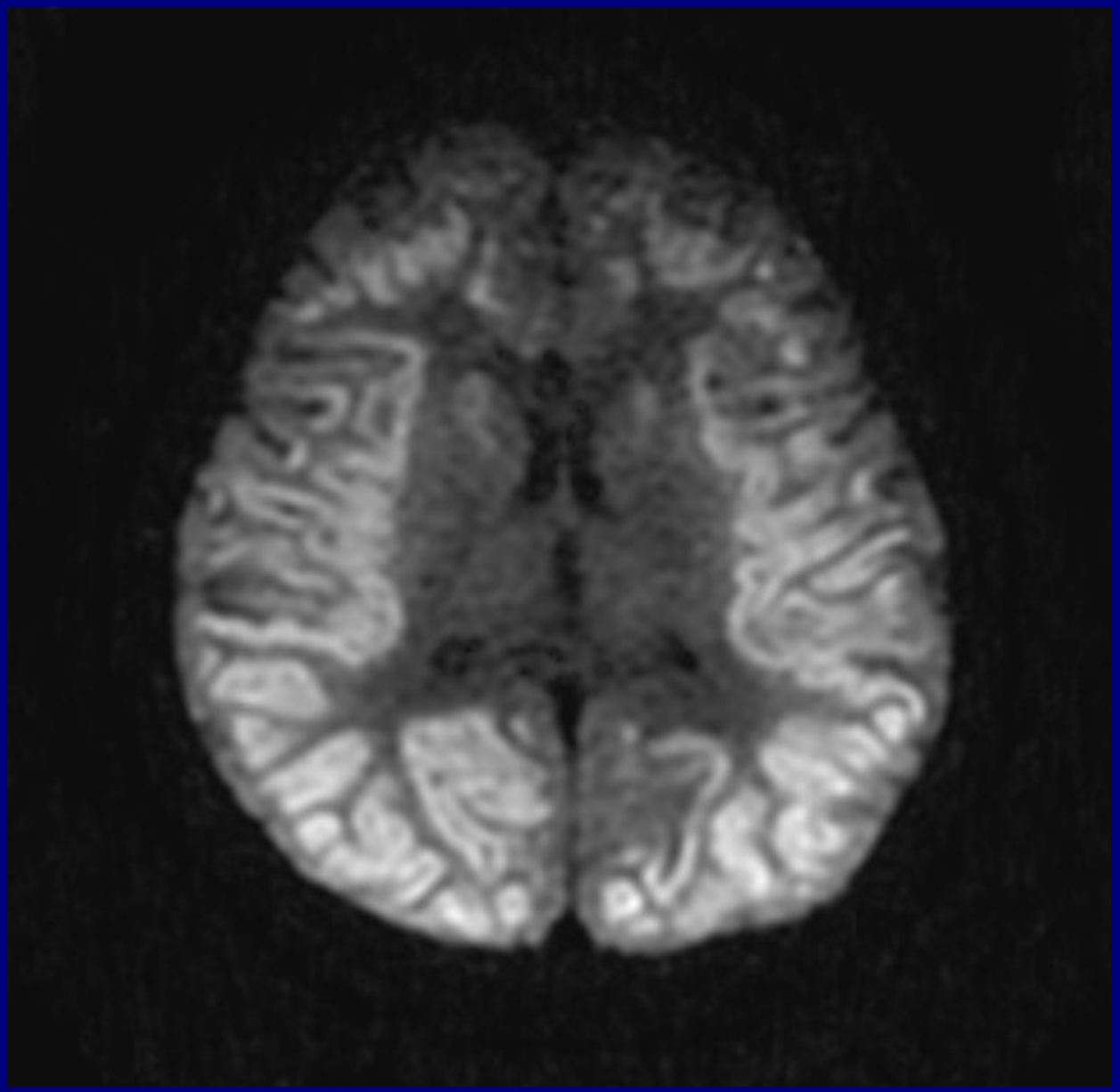
Multivariate	OR	95% CI	p-value
Renal ins. and FRIDA	0.2	0.05-0.51	0.002
Liver ins. and triphasic waves	11.3	2.11-60.5	0.005
MOF and triphasic waves	6.0	1.24-29.4	0.026


Mean of lowest GCS on day of EEG
15
13
11
9
7
5
3
p>0.0001
FIRDA
Theta
Triphasic waves
Theta/delta
Delta

Categorical and continuous short-term outcomes

Discharge destination	n	%	OR	95% CI	p-value*
Back home					
Theta	14	41	1.7	0.73 - 3.88	0.226
Theta/delta	7	22	0.4	0.17 - 1.09	0.075
Delta	6	21	0.2	0.07 - 0.65	**0.007**
Triphasic waves	12	35	1.1	0.48 - 2.54	0.817
FIRDA	18	69	4.4	1.72 -11.40	**0.002**
Death					
Theta	2	6	0.2	0.05 - 1.17	0.077
Theta/delta	7	22	2.7	0.90 - 7.92	0.078
Delta	0	0	--	--	--
Triphasic waves	10	29	4.5	1.57 - 12.70	**0.005**
FIRDA	0	0	--	--	--
GOS (continuous)	**Mean**	**SD**	**ß-coefficient**		**p-value****
Theta	3.5	±1.1	0.2		0.382
Theta/delta	3.0	±1.4	-0.5		**0.040**
Delta	3.7	±0.9	-0.02		0.955
Triphasic waves	2.9	±1.4	-0.5		**0.018**
FIRDA	4.4	±0.8	1.0		**<0.0001**
GOS (categorical)	**n**	**%**	**OR**	**95% CI**	**p-value***
GOS > 3					
Theta	16	47	1.2	0.52 - 2.69	0.694
Theta/delta	10	31	0.4	0.17 - 0.93	**0.033**
Delta	15	54	0.9	0.35 - 2.14	0.748
Triphasic waves	14	41	0.8	0.34 - 1.74	0.530
FIRDA	21	81	4.8	1.63 - 13.90	**0.004**
GOS 1 - 3					
Theta	17	50	0.8	0.37 - 1.93	0.694
Theta/delta	22	69	2.5	1.08 - 5.98	**0.033**
Delta	12	43	1.2	0.47 - 2.89	0.748
Triphasic waves	20	59	1.3	0.58 - 2.92	0.530
FIRDA	5	19	0.2	0.07 - 0.61	**0.004**

*Multivariate logistic regression model adjusted for age
**Multivariate linear regression model adjusted for age



Classification System for Coma

- Grade I: regular alpha, some theta
- Grade II: predominant theta
- Grade III: widespread delta (reactive or non-reactive), or spindle coma
- Grade IV: burst-suppression pattern; alpha coma pattern, theta coma pattern or delta (<20uV).
- Grade V: ECI (<2uV)

EEG patterns predictive of poor outcome in anoxia:

Grades 4 to 5

- Low-voltage unreactive & invariable EEG
- Burst Suppression
- Unreactive alpha coma
- ECI

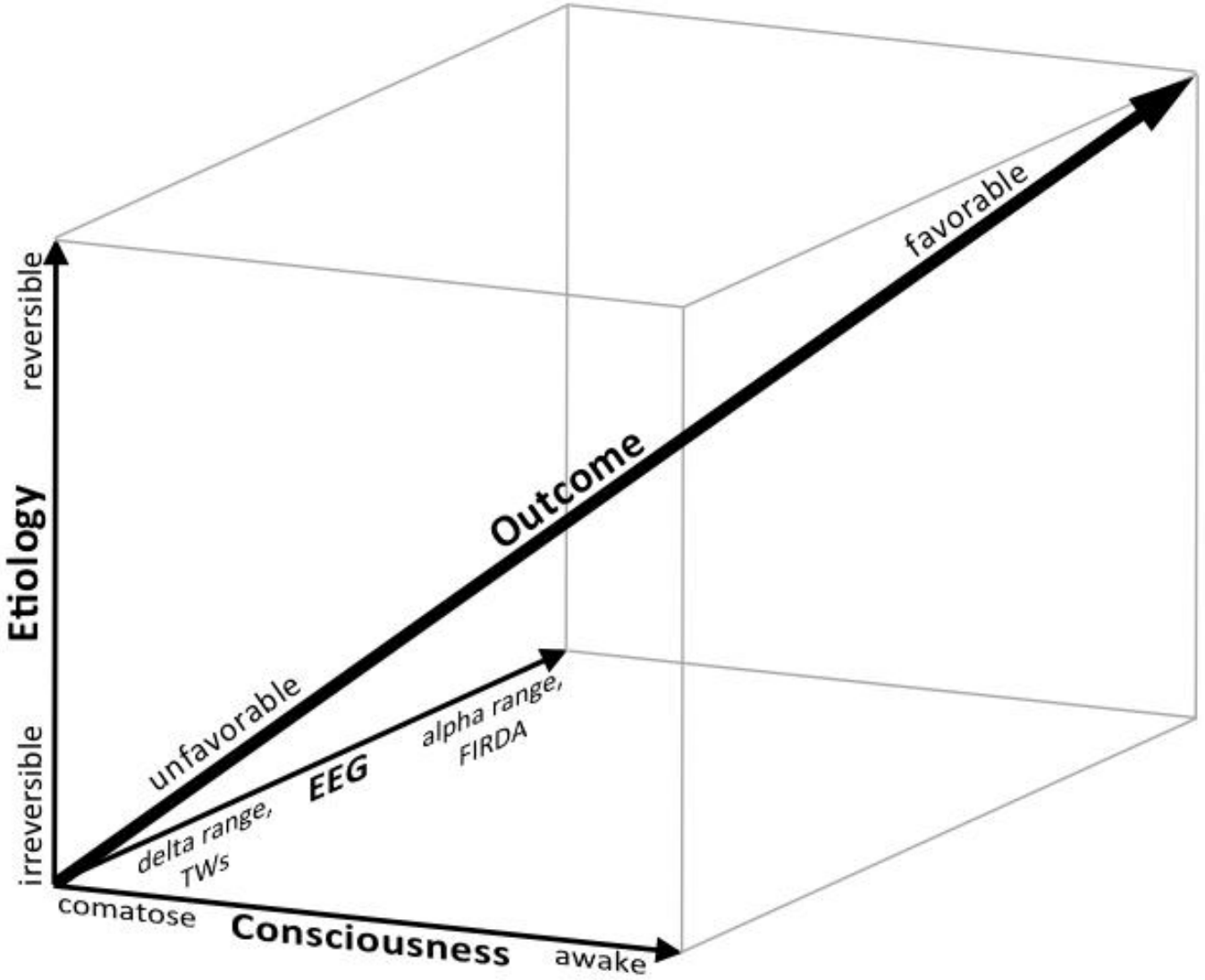
Synek 1988

EEG Grading for Anoxic Coma after CRA

Good Outcome

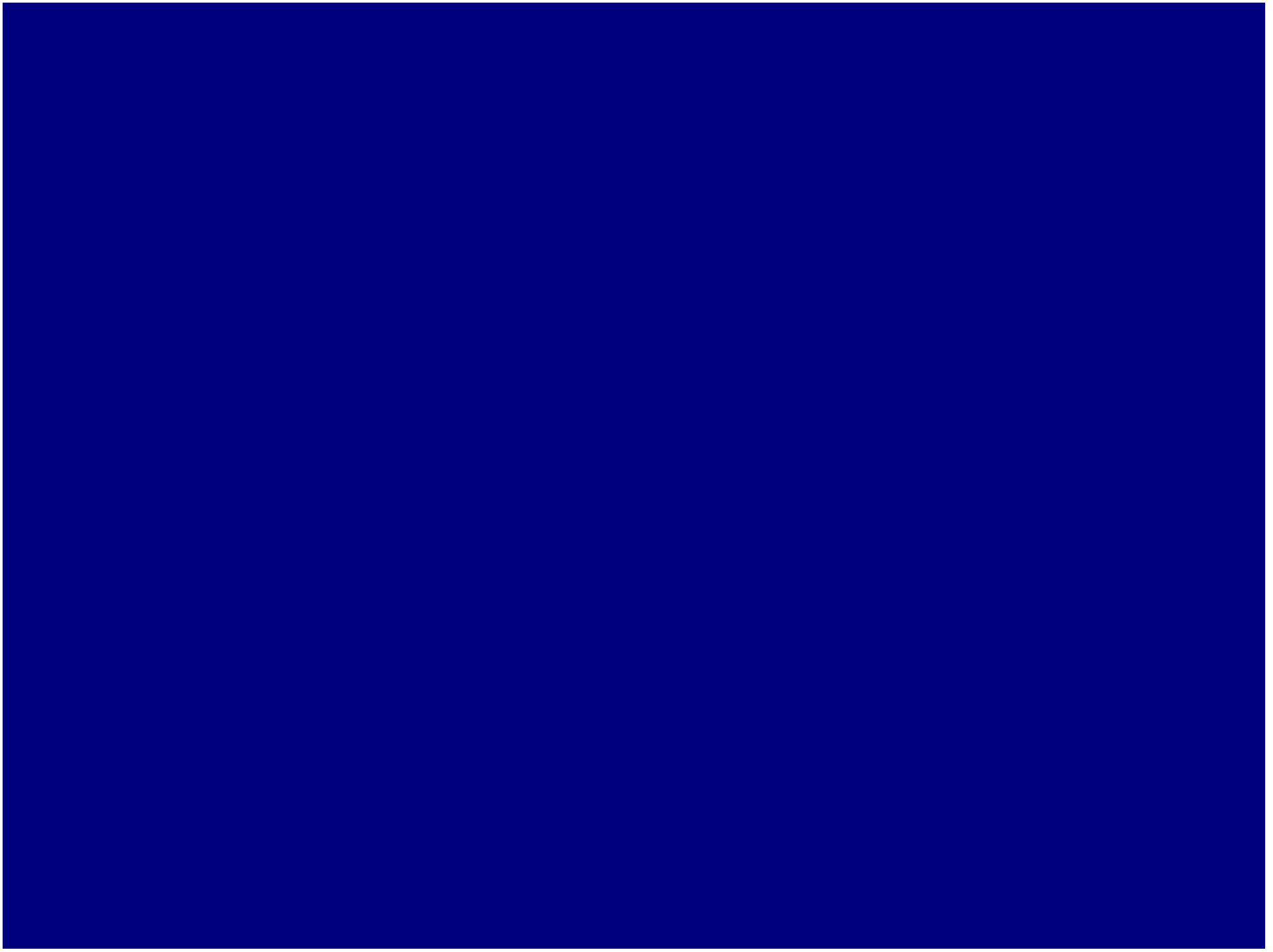
Grade 1	48/61	(79%)
Grade 2	45/88	(51%)
Grade 3	11/43	(26%)
Grade 4	0/138	(0%)
Grade 5	0/70	(0%)

Bassetti, Scollo-Lavizarri Euro Neurol 1987

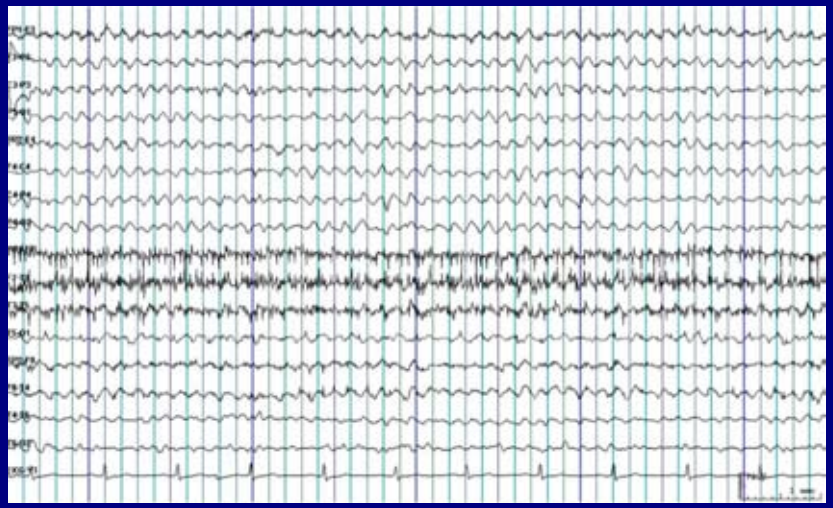

reversible
Etiology
irreversible
unfavorable
Outcome
favorable
delta range,
TWs
EEG
alpha range,
FIRDA
comatose
Consciousness
awake

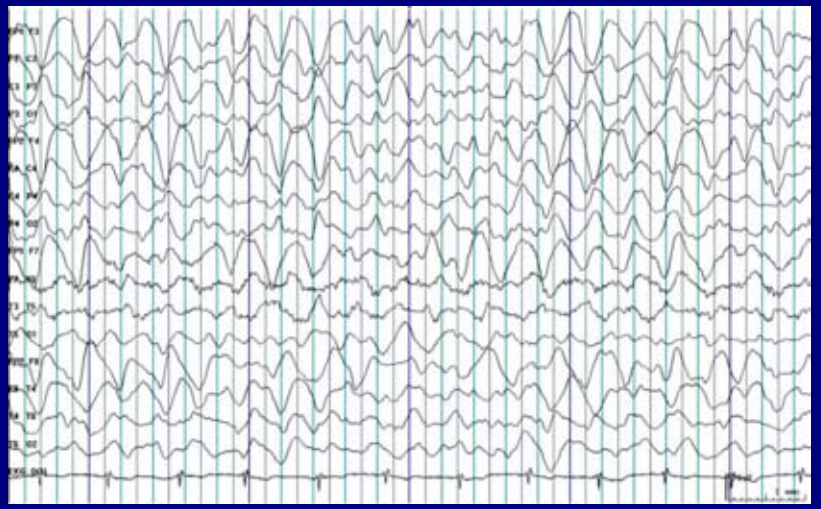
Summary

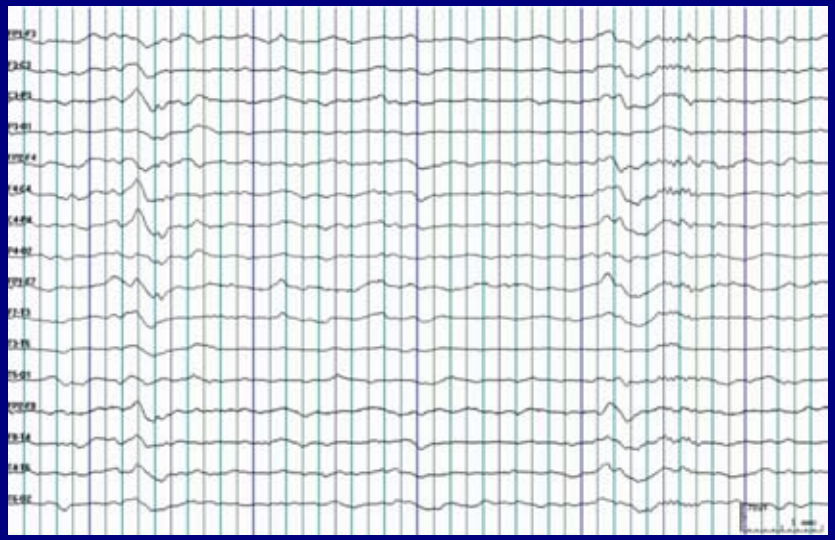
- Most EEG patterns are non-specific
- Certain EEG patterns may suggest an etiology.
- EEG helps in determining degree of cerebral compromise
- EEG can monitor effects of treatment
- EEG can uncover occult epileptiform activity as a cause of encephalopathy

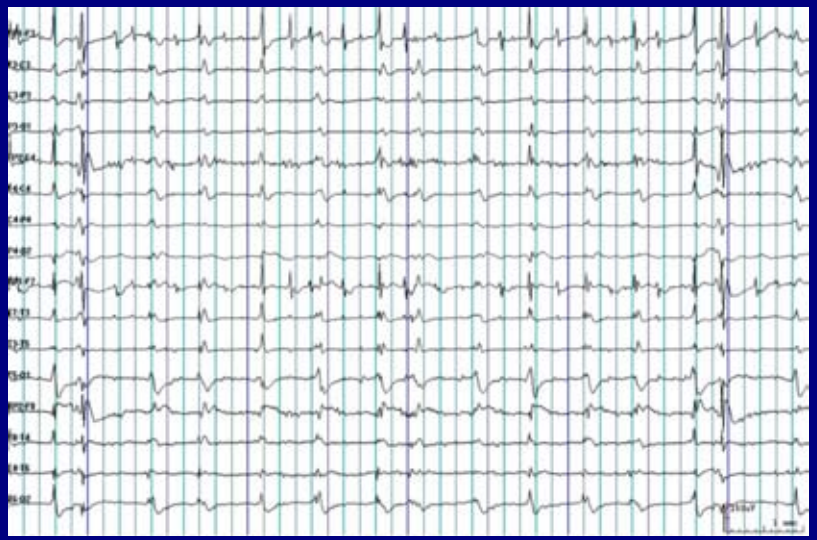


Which of the following EEG patterns suggest the best prognosis after cardiac arrest, not treated by hypothermia:





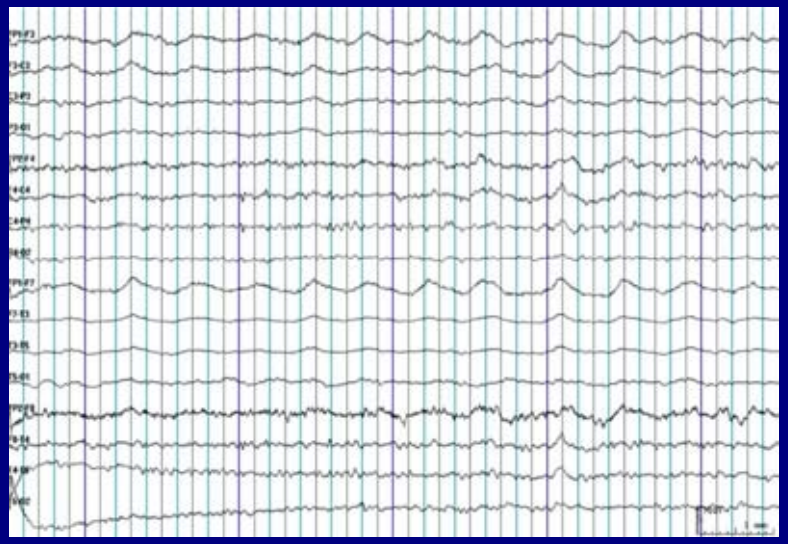


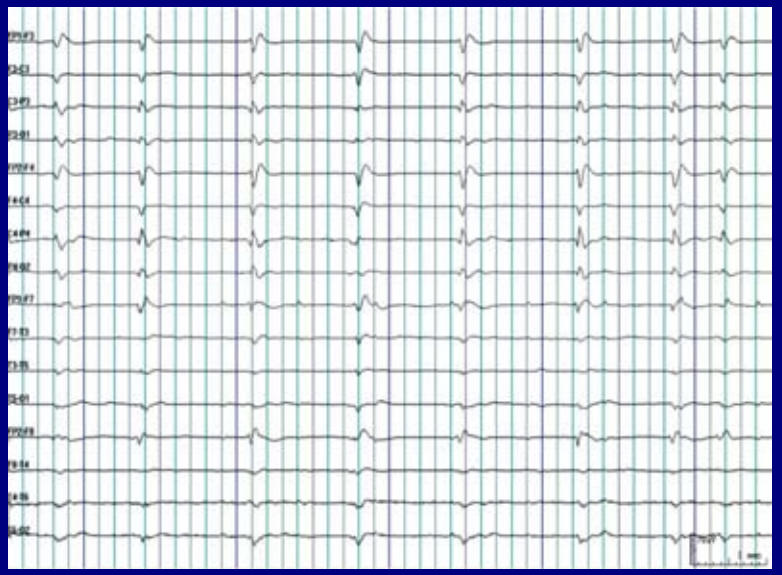


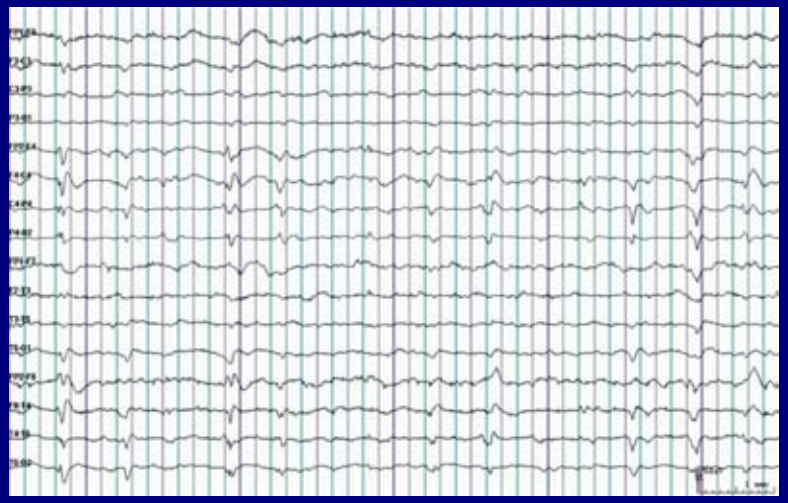
Which of the following is a well-recognized cause of triphasic waves?

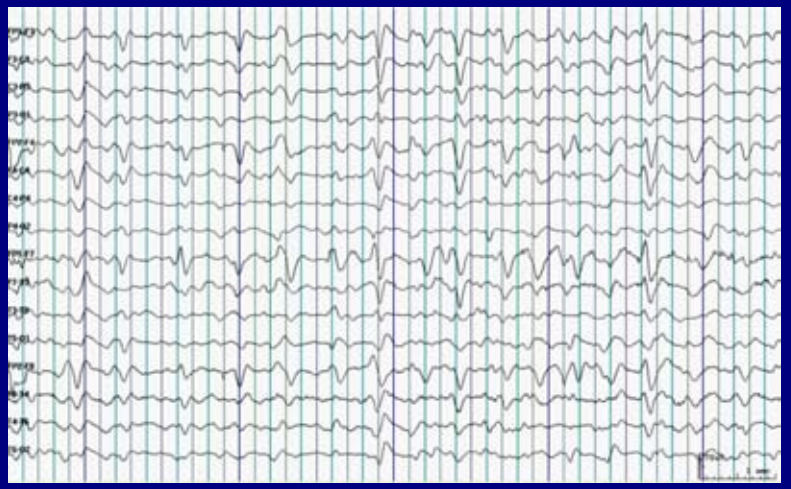
- Hypocalcemia
- Hypernatremia
- Baclofen toxicity
- Alprazolam toxicity

Which of the following is most likely to represent a seizure pattern?









During hypothermia after cardiac arrest:

- A suppressed EEG heralds a poor prognosis
- Alpha patterns suggest recovery
- A reactive background heralds good prognosis
- No prognostication can be under from EEG at this time.

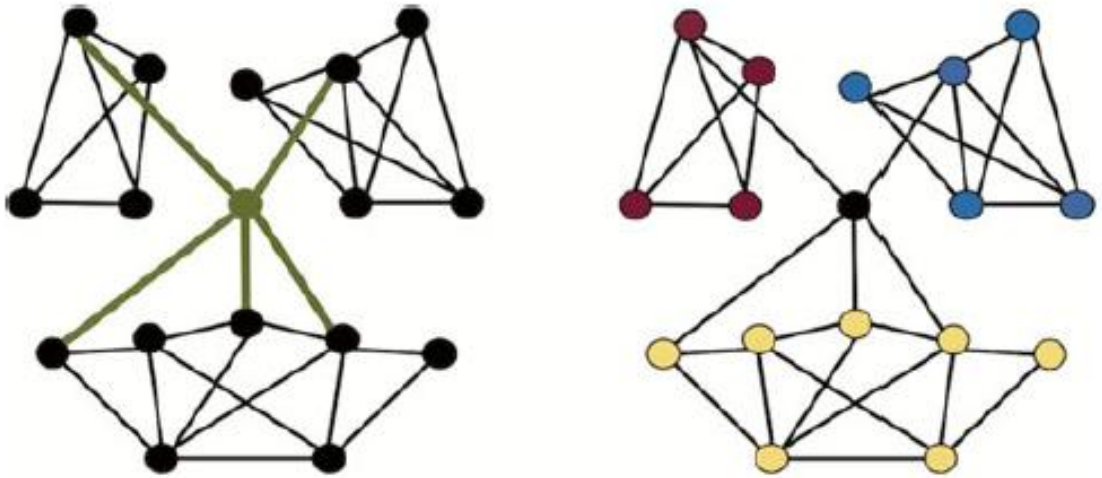


Figure 3.

Two schematic illustrations of networks. In the left figure, an example of hub node is highlighted in green: it has a central position in the network, has many connections with other nodes, and connects different modules. Several network measures exist to quantify this hub node. The simplest form is "degree," which simply counts the number of connections of a node. "Strength" also takes into account the weight of the connections (in this figure, all connections have the same weight). More advanced calculations include betweenness centrality and eigenvector centrality. Betweenness centrality takes into account the fraction of all shortest path length that needs to pass through a specific node. Eigenvector centrality determines the importance of a node on the basis of the number and weight of connections to other nodes and how those other nodes are connected. In the right figure, three groups of nodes (red, yellow, and blue) represent so-called modules. Modules are groups of nodes that are more strongly connected with each other than with nodes from different modules.

Epilepsia © ILAE

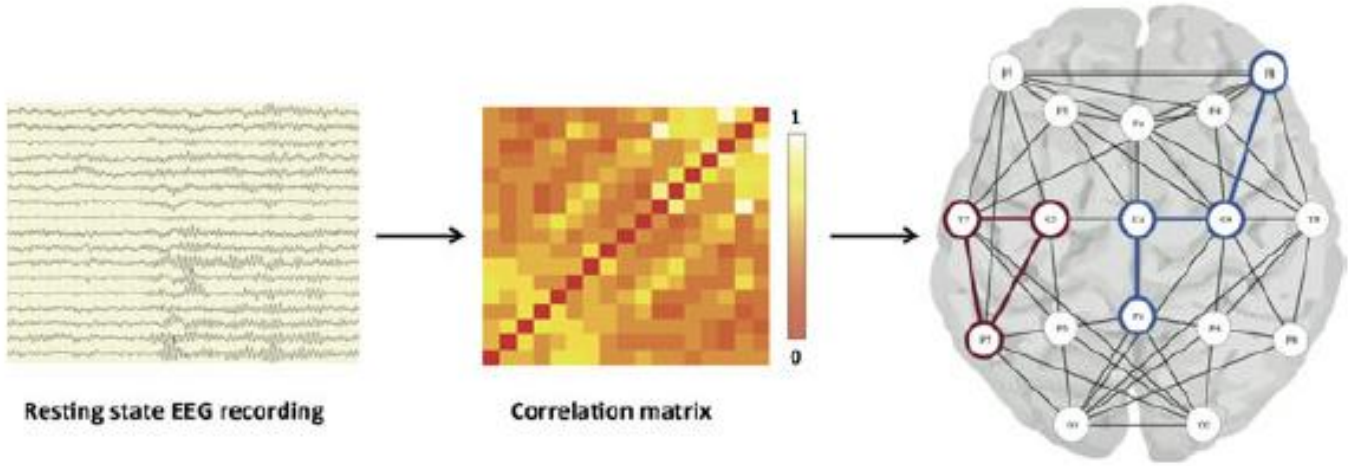
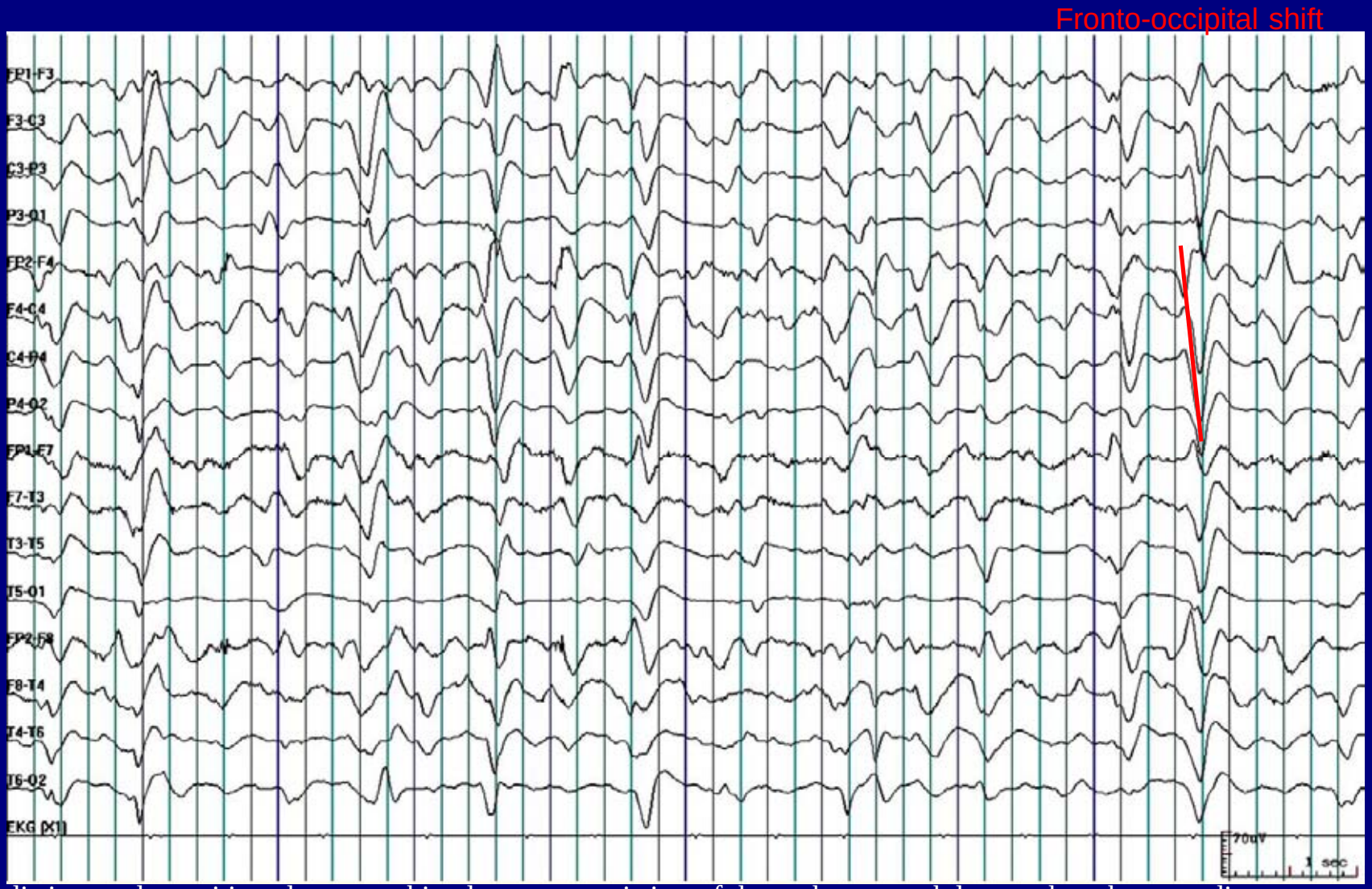


Figure 1.

Illustration of a network based on EEG data (average network based on children with idiopathic epilepsy [n = 25]; unpublished data). After selecting resting state EEG epochs, we computed a correlation matrix based on the linear and nonlinear interdependencies between two channels (in this case we used the synchronization likelihood [SL]). In the matrix, light-colored boxes indicate a high correlation. The diagonal was set on 0. After constructing a correlation matrix, various software programs allow the construction of visual graphs (figure on the right). For illustration purposes, we applied a threshold (k = 4) to obtain a binary network. Two elementary building blocks of the network are highlighted: clustering coefficient (in red) and shortest path length (in blue). Recording specifications: broadband frequency (0.5 45 Hz). Electrodes Fp1, Fp2, A1, and A2 were left out of the network due to myogenic and (eye) movement artifacts.

Epilepsia © ILAE

Triphasic waves



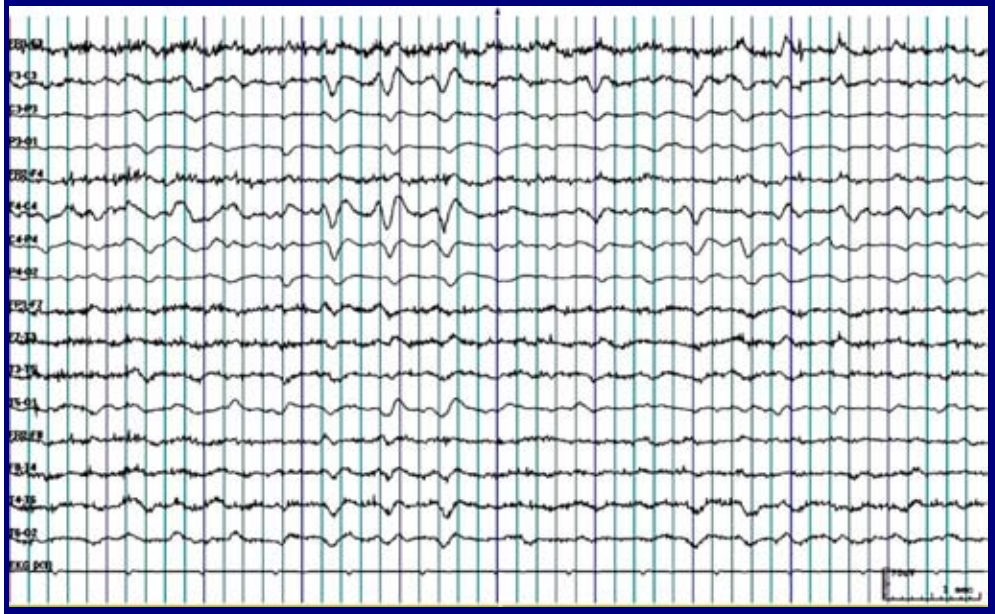
distinct and repetitive electrographic elements consisting of three phases, each longer than the preceding one: a surface positive high-amplitude (greater than 70 µV) wave (wave 2) is preceded and followed by negative waves with smaller amplitude (waves 1 and 3)

Clinical predispositions for triphasic waves (TWs)

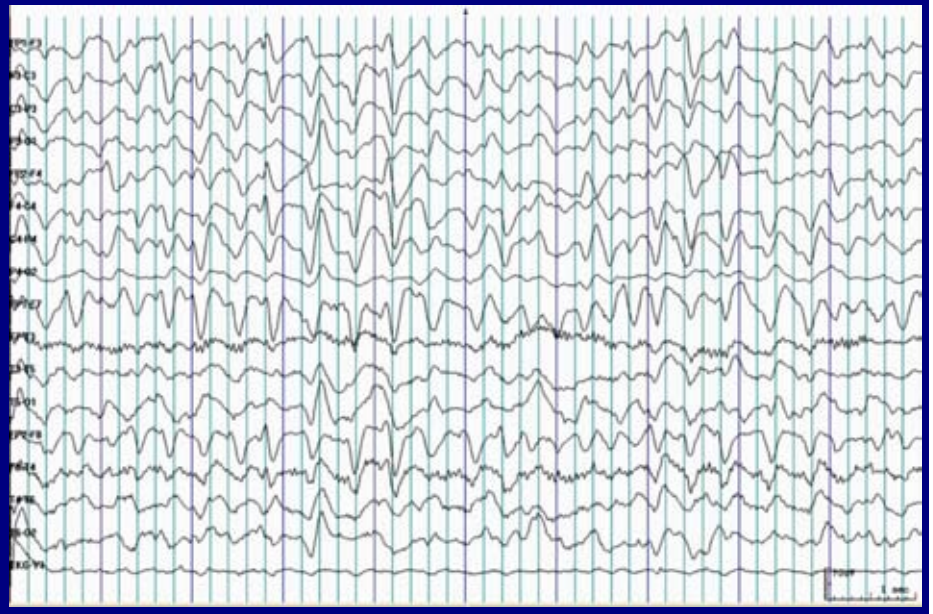
- Without white matter disease/subcortical atrophy
  - Hepatic encephalopathy, hyperammonemia
  - Uremia, other marked electrolyte abnormalities
  - Anoxia
  - Toxins/medications (e.g. lithium, baclofen)
- With white matter disease/subcortical or diffuse
  - Mild infections (e.g. UTI, atrophy URTI)
  - Lesser electrolyte imbalance; Toxins

Our hypothesis

Specific EEG characteristics in encephalopathic patients with TWs are predictive of short-term outcome.



Better?



Worse?

Sutter R, Stevens RD, Kaplan PW. 2013; submitted

Critical interventions / critical care

	n	%
Critical care	108	70.13
Neurocritical care units (NCCU)	58	37.66
Medical intensive care units (MICU)	32	20.78
Cardiac care units (CCU)	15	9.74
Surgical intensive care units (SICU)	3	1.95

	n	%
I.V. anesthetics/benzodiazepins 24 prior or during EEG	20	12.99
Renal replacement	26	16.88
Mechanically ventilated	39	25.32
CPR	5	3.25

à No significant differences between the five groups

Sutter R, Stevens RD, Kaplan PW. J Neurol. 2013.

Multivariable analyzes for death in patients with TWs

	Univariable			Multivariable*		
Death	**OR**	**95% CI**	**p-value***	**OR**	**95%CI**	**p-value***
Age	1.01	0.98-1.05	0.461			
Renal insufficiency	3.18	1.12-8.99	**0.029**			
Lowest GCS on day of EEG	0.81	0.70-0.95	**0.010**			
Mechanical ventilation	4.72	1.70-13.1	**0.003**	6.47	1.98-21.12	**0.002**
No reaction of EEG background activity	4.34	1.50-12.6	**0.007**	3.73	1.08-12.80	**0.037**

No other EEG characteristics of TWs were associated with death

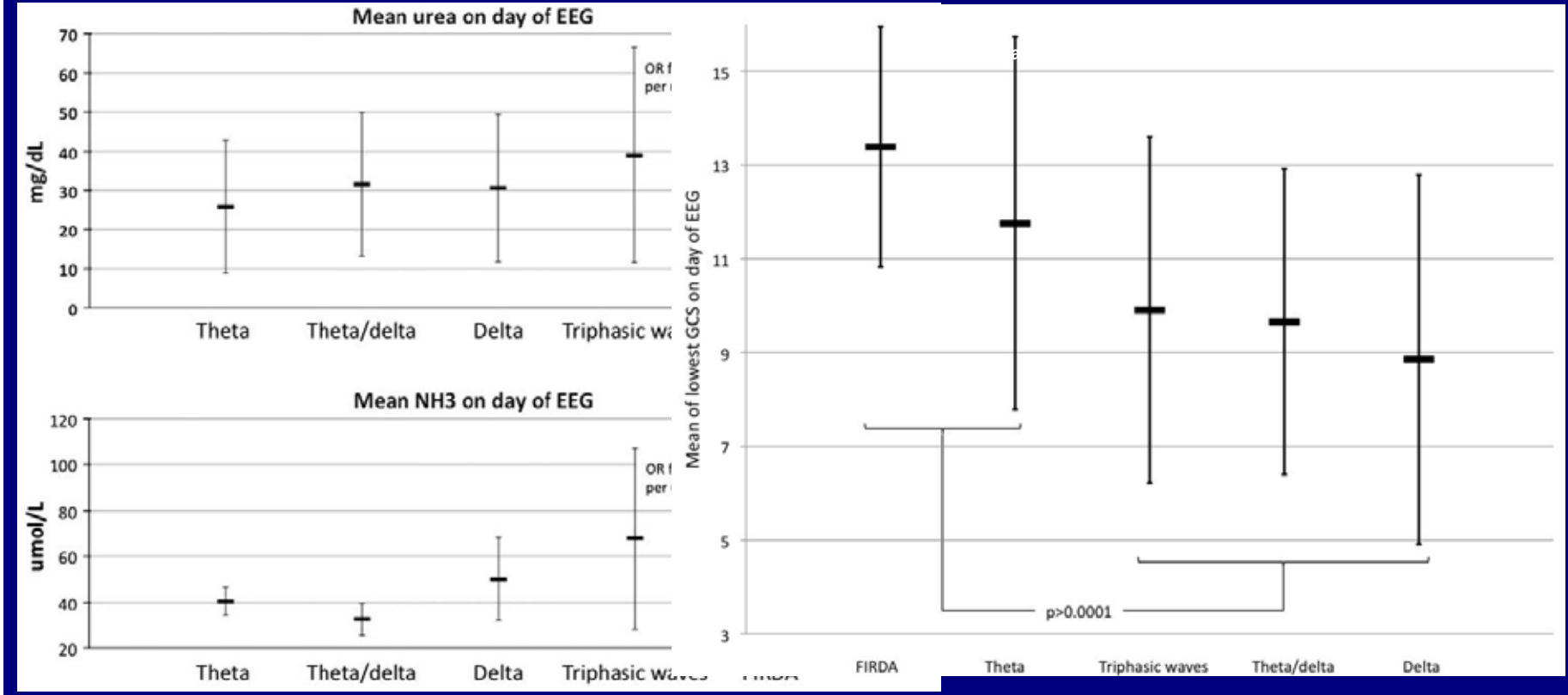
Sutter R, Stevens RD, Kaplan PW. 2013; submitted

Univariable analysis of EEG characteristics with outcome in patients with TWs

	Survivors (n=84)		Non-survivors (n=21)		
Background activity	**n**	**%**	**n**	**%**	**p-value***
Alpha	0	0	0	0	0.747
Alpha/theta	12	14	2	10	
Theta	24	29	6	29	
Theta/delta	42	50	13	62	
Delta	6	7	0	0	
% of TWs§					
0 - 10%	6	7	2	10	0.092
11 - 20%	9	11	1	5	
21 - 30%	15	18	1	5	
31 - 40%	14	17	9	43	
> 40%	40	48	8	38	
Arousal / reaction on stimulation (3 missing values)					
Frequency of TWs					
Increased	40	48	13	62	0.250
Decreased	32	38	4	19	
None	12	14	4	19	
Frequency of EEG background activity					
Increased	63	75	9	43	**0.013** ←
Decreased	8	10	3	14	
None	13	15	9	43	

Sutter R, Stevens RD, Kaplan PW. 2013; submitted

Non-structural correlates of encephalopathic patterns in EEG



Sutter R, Stevens RD, Kaplan PW. J Neurol. 2012; epub ahead of print.