5th Congress of the European Academy of Neurology

Oslo, Norway, June 29 - July 2, 2019

Teaching Course 5

Refractory status epilepticus: What to do and how dangerous is it to the brain? (Level 2)

What is the role for EEG in RSE?

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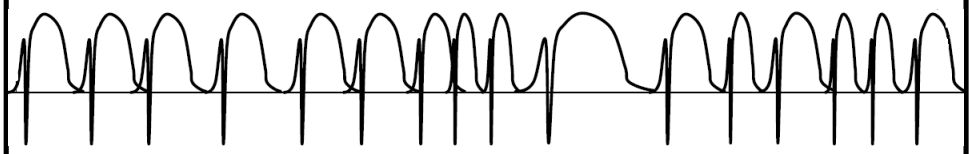
What is the role of EEG in Refractory & Super-Refractory Status Epilepticus?



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Disclosure

- No conflict of interest related to this topic.

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Outline

- Diagnosis & classification
- Monitoring of therapeutic effect (anesthetics)
- Monitoring of brain function during withdrawal of anesthetics / after SE

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SPECIAL REPORT

A definition and classification of status epilepticus – Report of the ILAE Task Force on Classification of Status Epilepticus

*†‡Eugen Trinka, §Hannah Cock, ¶Dale Hesdorffer, #Andrea O. Rossetti, **Ingrid E. Scheffer, ††Shlomo Shinnar, ‡‡Simon Shorvon, and §§Daniel H. Lowenstein

Epilepsia, **(*):1–9, 2015
doi: 10.1111/epi.13121

Eugen Trinka is professor and chairman of Department of Neurology, Paracelsus Medical University Salzburg Austria.

SUMMARY

The Commission on Classification and Terminology and the Commission on Epidemiology of the International League Against Epilepsy (ILAE) have charged a Task Force to revise concepts, definition, and classification of status epilepticus (SE). The proposed new definition of SE is as follows: *Status epilepticus is a condition resulting either from the failure of the mechanisms responsible for seizure termination or from the initiation of mechanisms, which lead to abnormally, prolonged seizures (after time point t_1). It is a condition, which can have long-term consequences (after time point t_2), including neuronal death, neuronal injury, and alteration of neuronal networks, depending on the type and duration of seizures.* This definition is conceptual, with two operational dimensions: the first is the length of the seizure and the time point (t_1) beyond which the seizure should be regarded as "continuous seizure activity." The second time point (t_2) is the time of ongoing seizure activity after which there is a risk of long-term consequences. In the case of convulsive (tonic–clonic) SE, both time points (t_1 at 5 min and t_2 at 30 min) are based on animal experiments and clinical research. This evidence is incomplete, and there is furthermore considerable variation, so these time points should be considered as the best estimates currently available. Data are not yet available for other forms of SE, but as knowledge and understanding increase, time points can be defined for specific forms of SE based on scientific evidence and incorporated into the definition, without changing the underlying concepts. A *new diagnostic classification system* of SE is proposed, which will provide a framework for clinical diagnosis, investigation, and therapeutic approaches for each patient. There are four axes: (1) semiology; (2) etiology; (3) electroencephalography (EEG) correlates; and (4) age. Axis 1 (semiology) lists different forms of SE divided into those with prominent motor systems, those without prominent motor systems, and currently indeterminate conditions (such as acute confusional states with epileptiform EEG patterns). Axis 2 (etiology) is divided into subcategories of known and unknown causes. Axis 3 (EEG correlates) adopts the latest recommendations by consensus panels to use the following descriptors for the EEG: name of pattern, morphology, location, time-related features, modulation, and effect of intervention. Finally, axis 4 divides age groups into neonatal, infancy, childhood, adolescent and adulthood, and elderly.

KEY WORDS: Status epilepticus, Seizure, Definition, Classification, Seizure duration.

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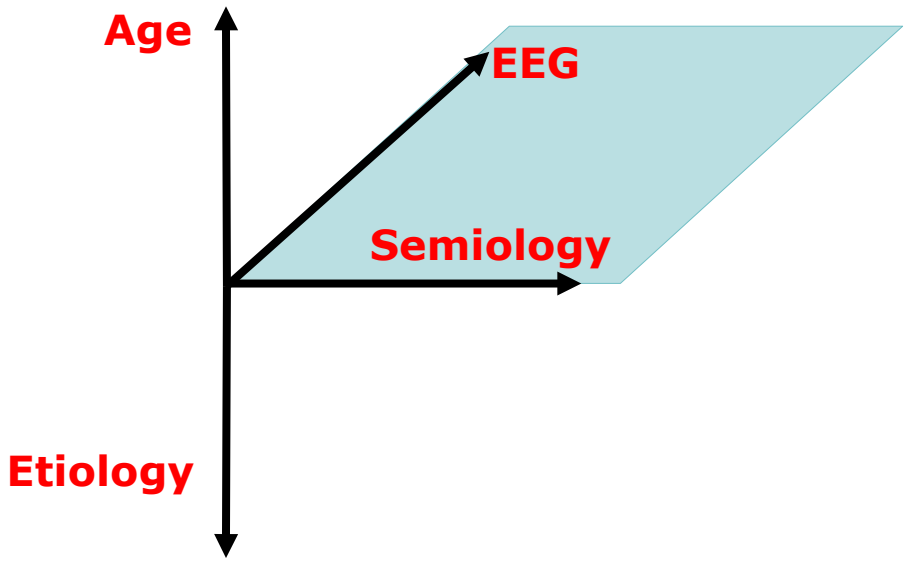

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(A) With prominent motor symptoms

A.1 Convulsive SE (CSE, synonym: tonic–clonic SE)
- A.1.a. Generalized convulsive
- A.1.b. Focal onset evolving into bilateral convulsive SE
- A.1.c. Unknown whether focal or generalized

A.2 Myoclonic SE (prominent epileptic myoclonic jerks)
- A.2.a. With coma
- A.2.b. Without coma

A.3 Focal motor
- A.3.a. Repeated focal motor seizures (Jacksonian)
- A.3.b. Epilepsia partialis continua (EPC)
- A.3.c. Adversive status
- A.3.d. Oculoclonic status
- A.3.e. Ictal paresis (i.e., focal inhibitory SE)

A.4 Tonic status

A.5 Hyperkinetic SE

(B) Without prominent motor symptoms (i.e., nonconvulsive SE, NCSE)

B.1 NCSE with coma (including so-called "subtle" SE)

B.2 NCSE without coma
- B.2.a. Generalized
  - B.2.a.a Typical absence status
  - B.2.a.b Atypical absence status
  - B.2.a.c Myoclonic absence status
- B.2.b. Focal
  - B.2.b.a Without impairment of consciousness (aura continua, with autonomic, sensory, visual, olfactory, gustatory, emotional/ psychic/experiential, or auditory symptoms)
  - B.2.b.b Aphasic status
  - B.2.b.c With impaired consciousness
- B.2.c Unknown whether focal or generalized
  - B.2.c.a Autonomic SE

Can be diagnosed without EEG

EEG is needed for diagnosis

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- **Wide variety of ictal EEG-patterns**

- **Wide variety of SE EEG-patterns**

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What EEG features will help diagnosing NCSE?

- No single-criterion.

- Set of combination – several criteria,
- that cover broadly the ictal EEG-patterns (→ sensitivity),
- yet not catching too many false positives (→ specificity).

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Several attempts to develop EEG-criteria for NCSE:

An assessment of nonconvulsive seizures in the intensive care unit using continuous EEG monitoring:

An investigation of variables associated with mortality

G. Bryan Young, MD; Kenneth G. Jordan, MD; and Gordon S. Doig, MSc, DVM

NEUROLOGY 1996;47:83–89

***Table 1** Criteria for seizure*

Guideline: To qualify at least *one* of primary criteria 1–3 *and one or more* of secondary criteria, with discharges >10 seconds

J Clin Neurophysiol. 2005 Apr;22(2):79-91.

Which EEG patterns warrant treatment in the critically ill? Reviewing the evidence for treatment of periodic epileptiform discharges and related patterns.

Chong DJ[1], Hirsch LJ.

EEG criteria for nonconvulsive status epilepticus

Peter W. Kaplan

Epilepsia, **48**(Suppl. 8):39–41, 2007

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Epilepsia, 54(Suppl. 6):28–29, 2013
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STATUS EPILEPTICUS 2013

Unified EEG terminology and criteria for nonconvulsive status epilepticus

*†Sándor Beniczky, ‡Lawrence J. Hirsch, §Peter W. Kaplan, ¶Ronit Pressler,
**Gerhard Bauer, †††‡‡‡Harald Aurlien, ††††Jan C. Brøgger, and §§Eugen Trinka

Table 1. Working clinical criteria for nonconvulsive status epilepticus

Patients without known epileptic encephalopathy
EDs > 2.5 Hz, *or*
EDs ≤ 2.5 Hz or rhythmic delta/theta activity (>0.5 Hz) AND one of the following:
EEG and clinical improvement after IV AED[a], *or*
Subtle clinical ictal phenomena during the EEG patterns mentioned above, *or*
Typical spatiotemporal evolution[b]
Patients with known epileptic encephalopathy
Increase in prominence or frequency of the features mentioned above, when compared to baseline **with** observable change in clinical state
Improvement of clinical and EEG[a] features with IV AEDs

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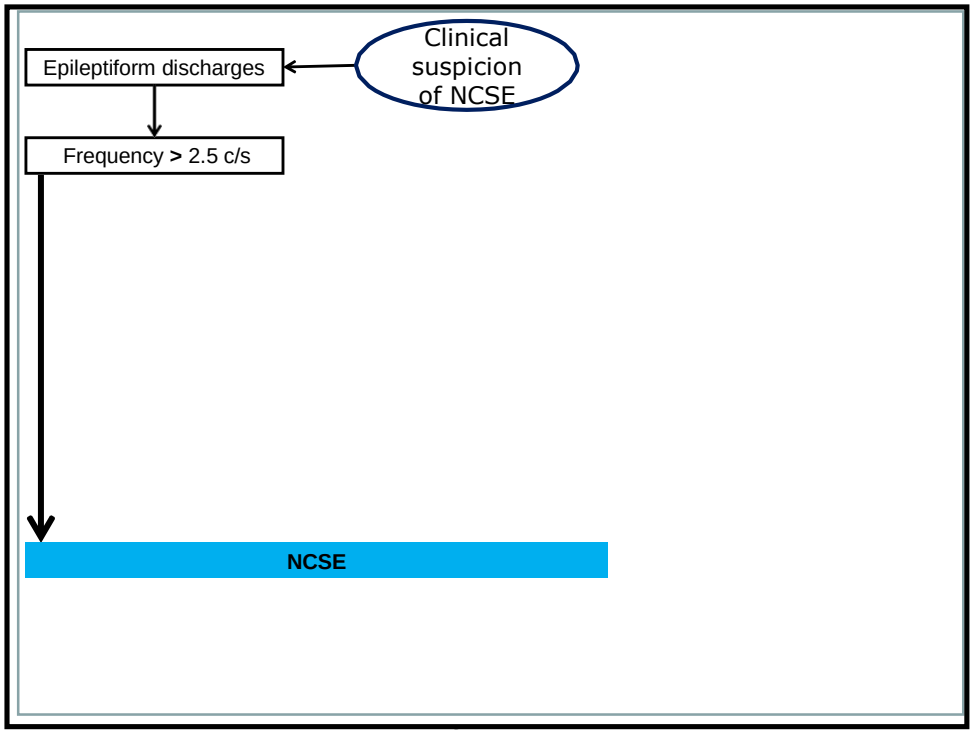
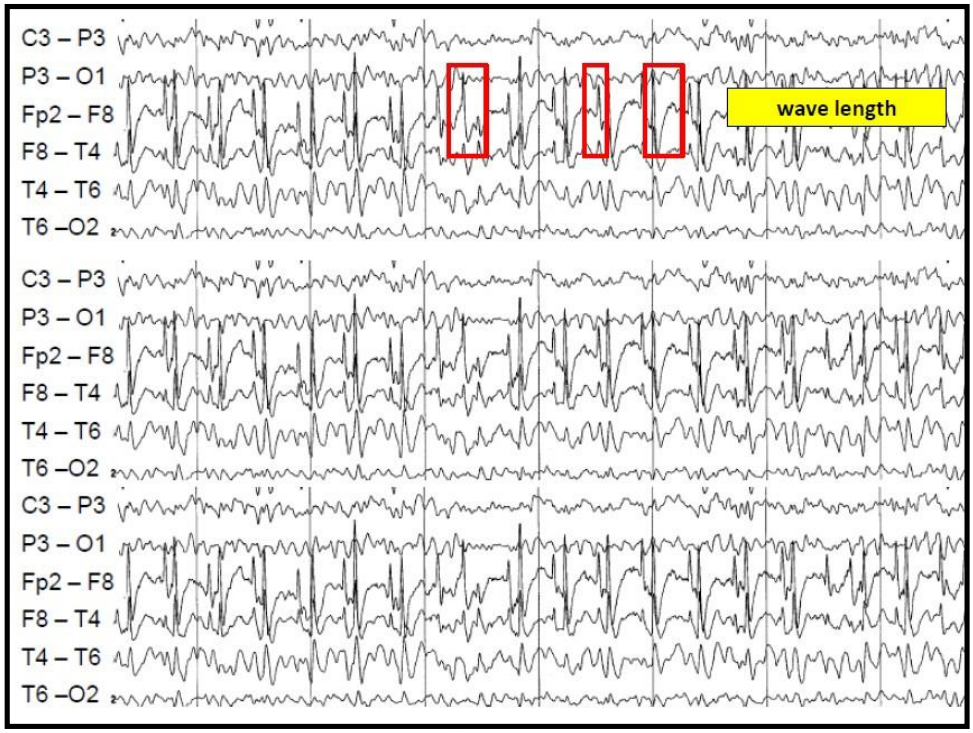
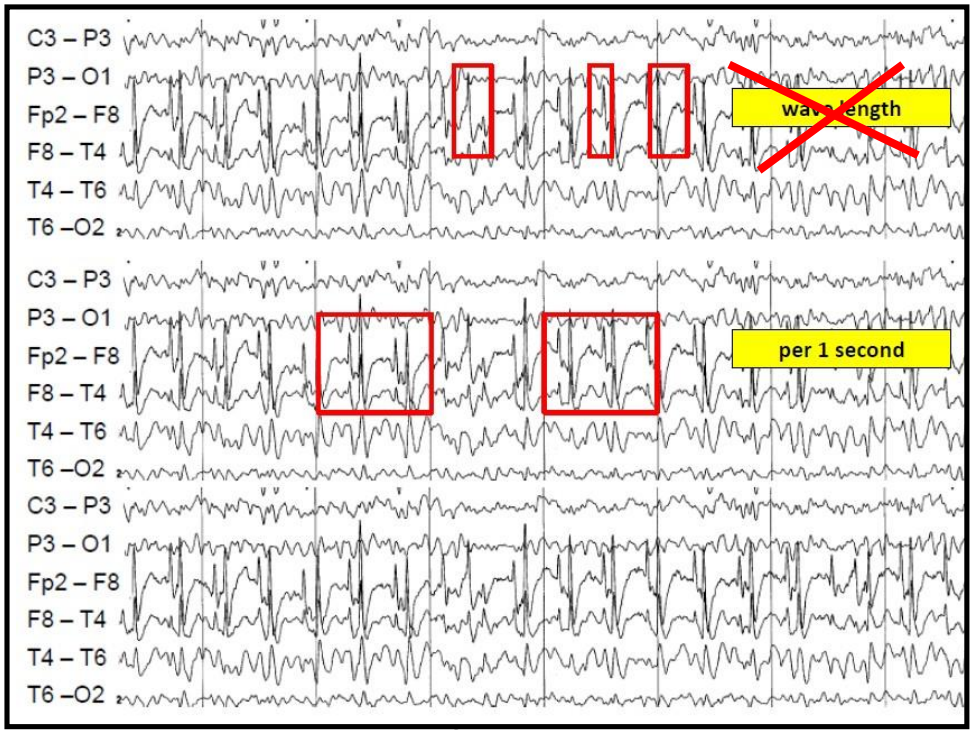
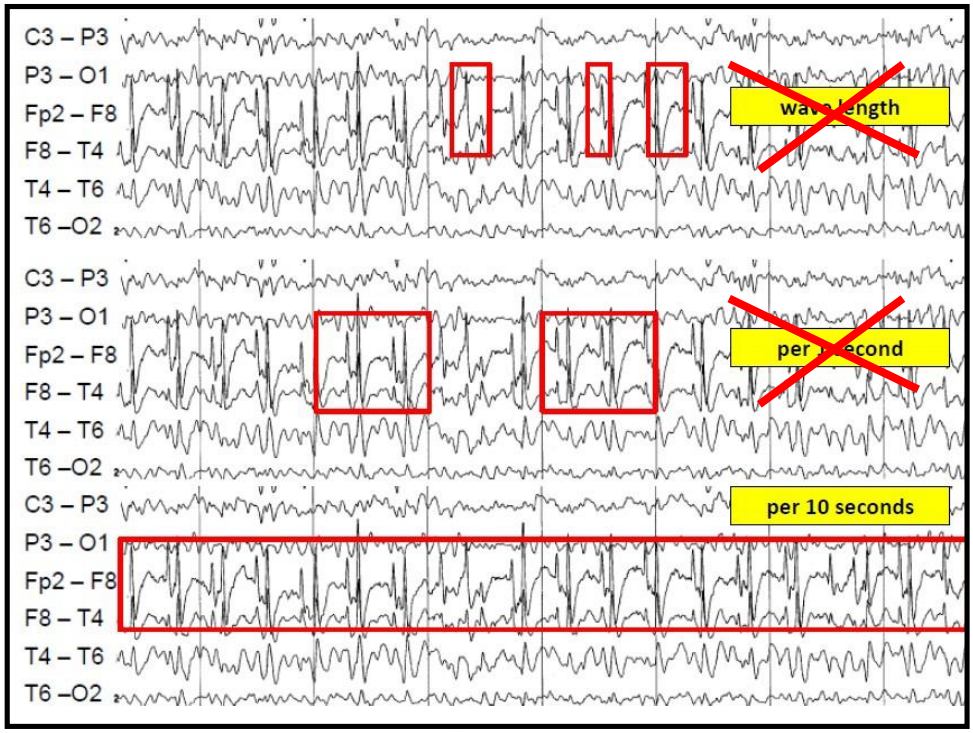
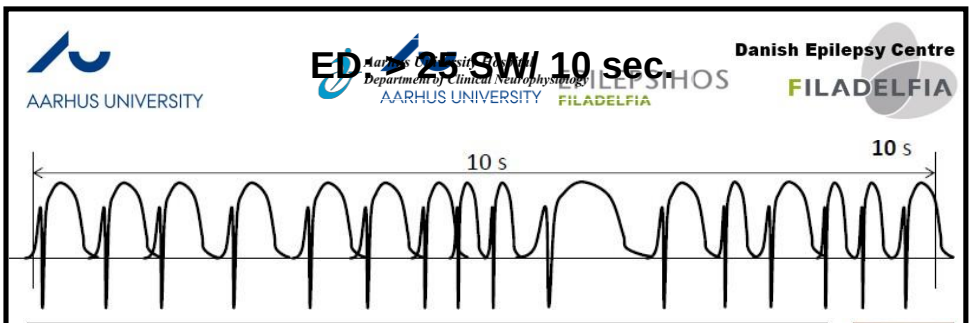



C3 – P3
P3 – O1
Fp2 – F8
F8 – T4
T4 – T6
T6 –O2
wave length


C3 – P3
P3 – O1
Fp2 – F8
F8 – T4
T4 – T6
T6 –O2
per 1 second



ED > 25 SW/ 10 sec.



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

Brief Communication

Salzburg Consensus Criteria for Non-Convulsive Status Epilepticus – approach to clinical application

M. Leitinger [a,d], S. Beniczky [b,c], A. Rohracher [a,d], E. Gardella [b], G. Kalss [a,d], E. Qerama [c], J. Höfler [a,d], A. Hess Lindberg-Larsen [c], G. Kuchukhidze [a,d], J. Dobesberger [a,d], P.B. Langthaler [a,d], E. Trinka [a,d,*]

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[c] Department of Clinical Neurophysiology, Aarhus University Hospital, Denmark
[d] Centre for Cognitive Neuroscience, Salzburg, Austria

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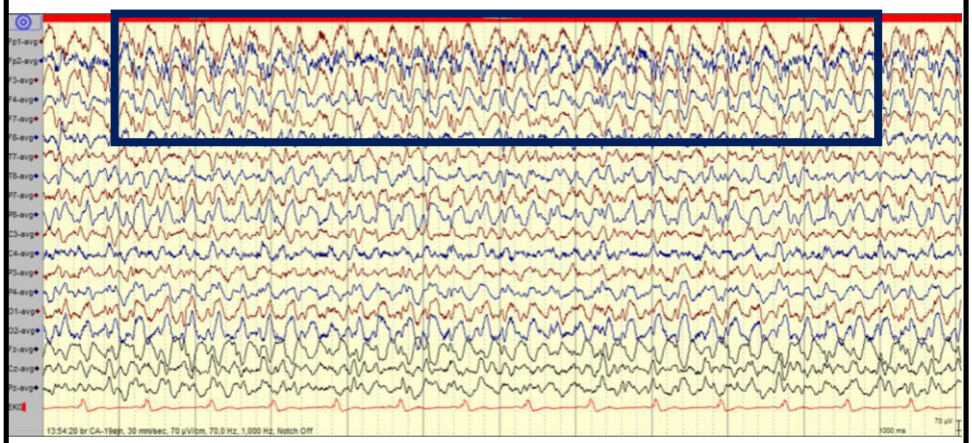
31 SW / 10 seconds



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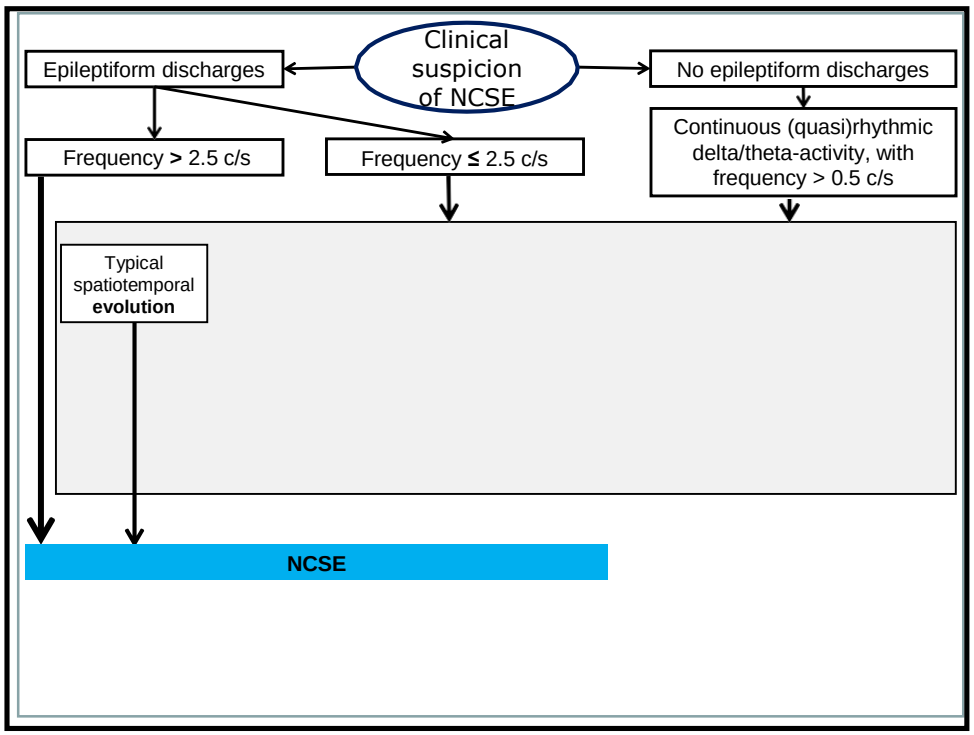

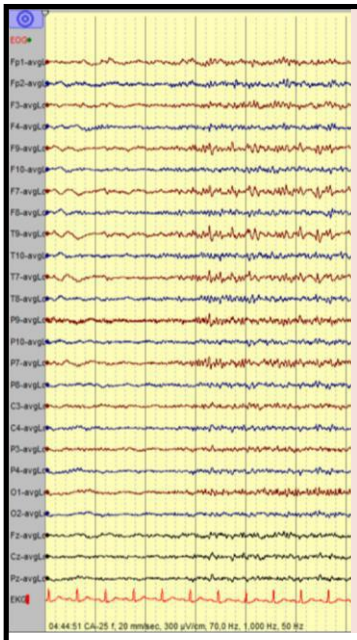

Typical spatiotemporal evolution

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]

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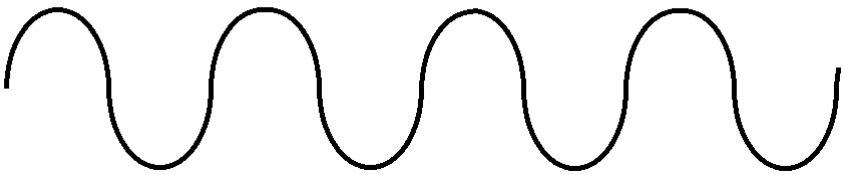
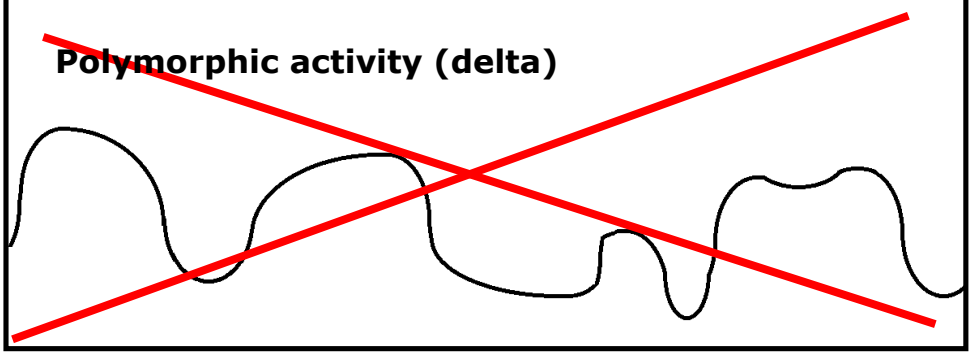
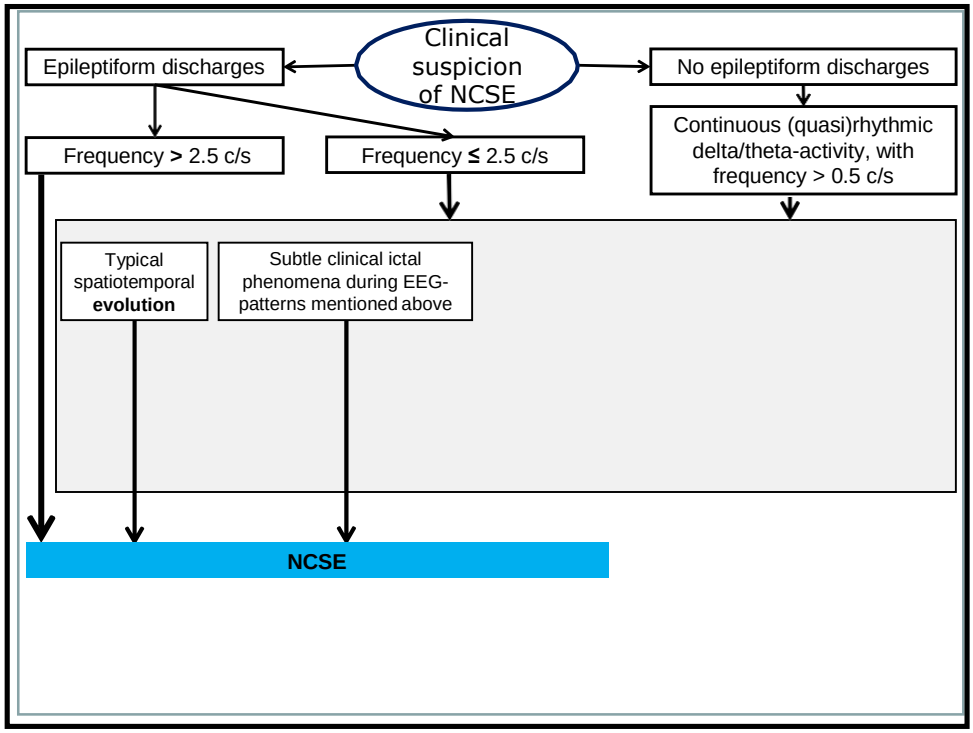
(Quasi)Rhythmic activity



Polymorphic activity (delta)





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Semiology of Subtle Seizures

- Discrete phenomena like:
  - twitches of the eyelids, face, jaw, extremities or the trunk
  - head and/or eye deviation
  - peculiar automatisms.

- They occur when the patient experiences such a degree of encephalopathy that an electromechanical dissociation occurs, so that in spite of continuous ictal activity in the brain, only subtle motor phenomena are generated.

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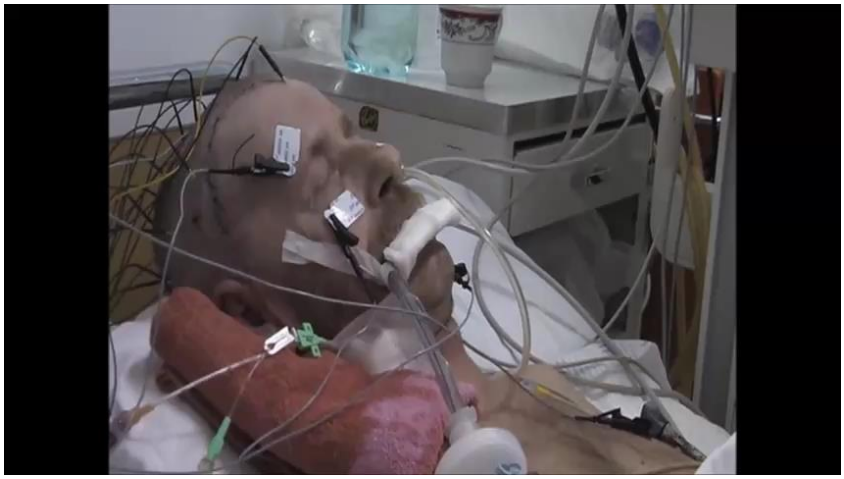
Video – subtle seizures



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Subtle seizure phenomena	NCSE (n=14)			Coma without NCSE (n=46)		
	Number of patients	Body part	Occurrence	Number of patients	Body part	Occurrence
Myoclonus	10 (71%)	Tongue: 2 Perioral: 2 Face: 2 UL: 6 LL: 4	Almost continuous: 3 Sporadic: 2 In clusters: 5 (4-30; 20)*	19 (41%)	Eyelid: 1 Face: 1 UL: 11 LL: 7 Axial: 3	Almost continuous: 5 Sporadic: 8 In clusters: 6 (4-120; 9)*
Tonic muscle activation	3 (21%)	UL: 1 LL: 3	Duration: 1-10 s (mean: 5 s)	19 (41%)	Face: 1 UL: 16 LL: 12 Axial: 1	Duration: 1-30s (mean: 4 s)
Automa-tisms	2 (14%)	Oro-facial:1 UL: 1	Almost continuous: 1 Sporadic: 1	8 (17%)	Oro-facial: 4 UL: 3 LL: 2	Almost continuous: 2 Sporadic: 6
Eye-deviation	2 (14%)		Almost continuous: 1 Sporadic: 1	4 (9%)		Almost continuous: 1 Sporadic: 3

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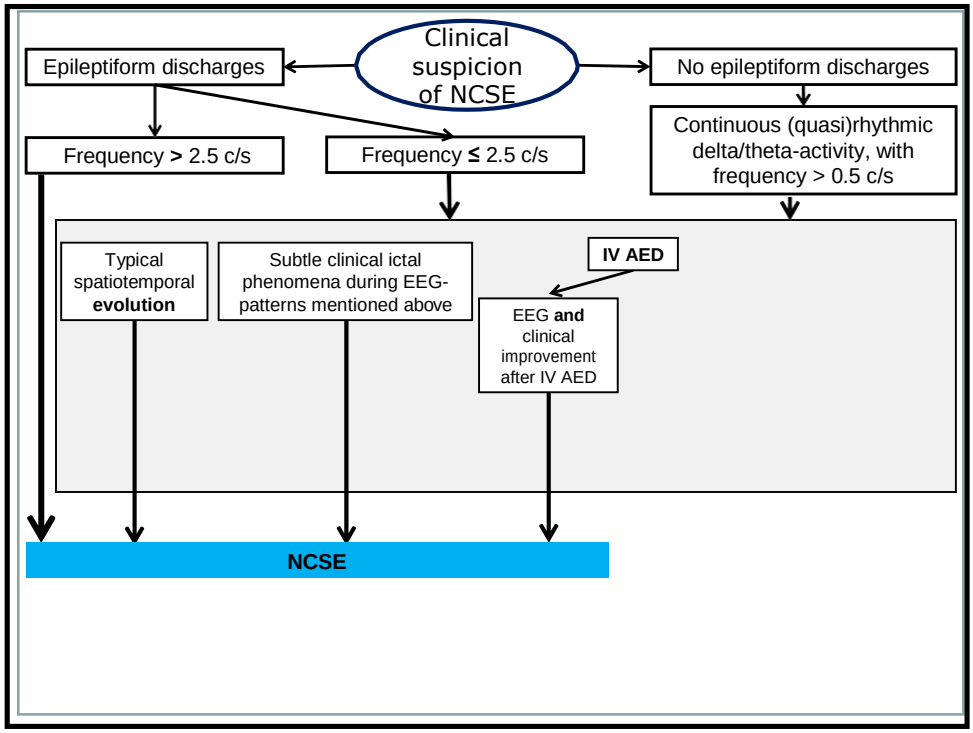
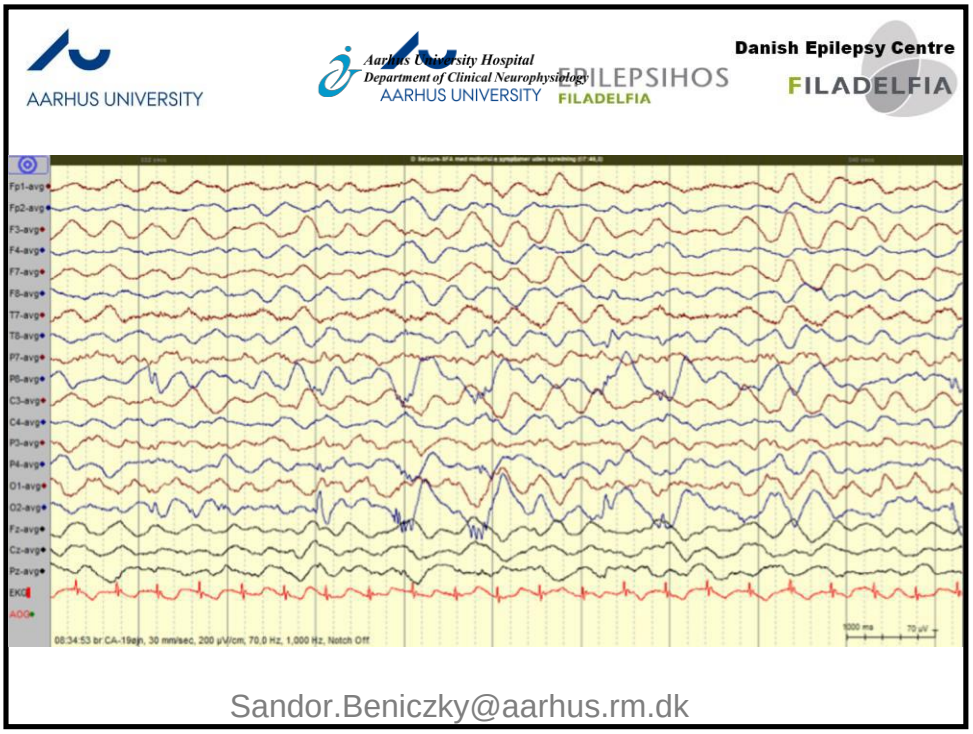

Clinical suspicion of NCSE
Epileptiform discharges
No epileptiform discharges
Frequency > 2.5 c/s
Frequency ≤ 2.5 c/s
Continuous (quasi)rhythmic delta/theta-activity, with frequency > 0.5 c/s
Typical spatiotemporal evolution
Subtle clinical ictal phenomena during EEG-patterns mentioned above
IV AED
EEG and clinical improvement after IV AED
NCSE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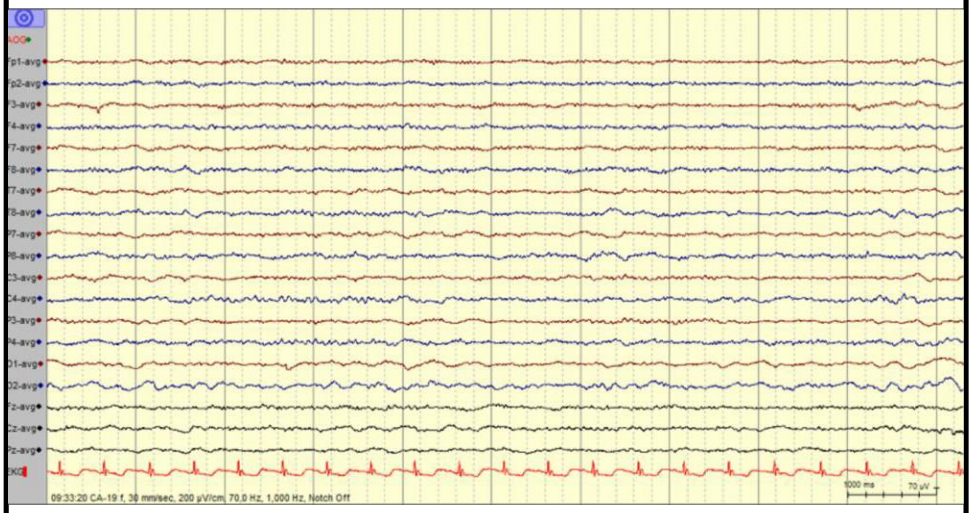

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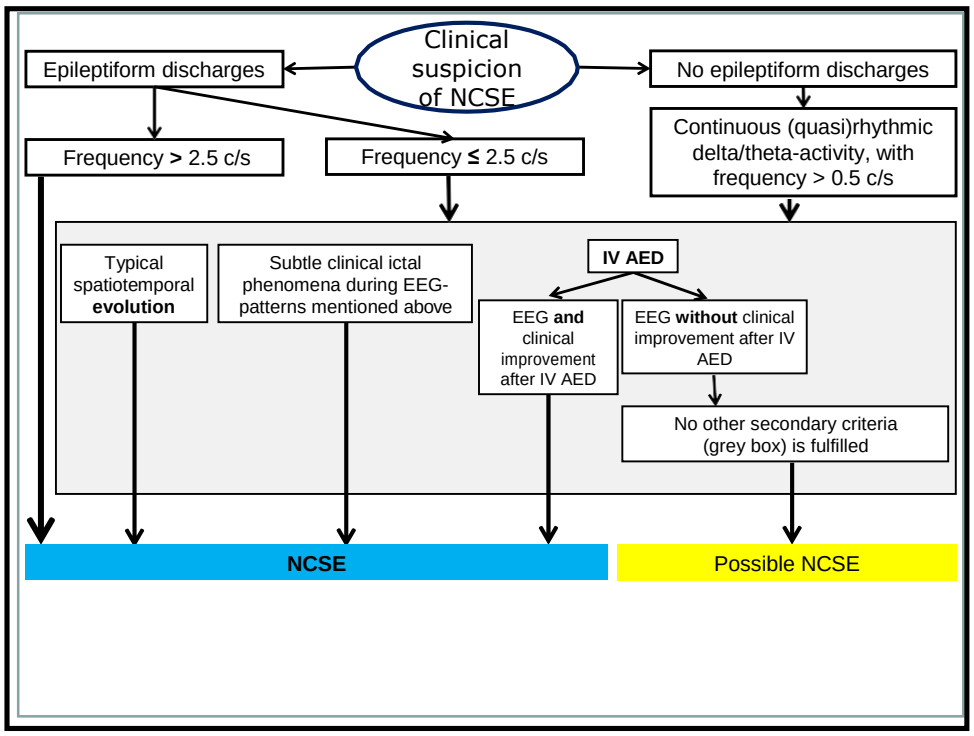

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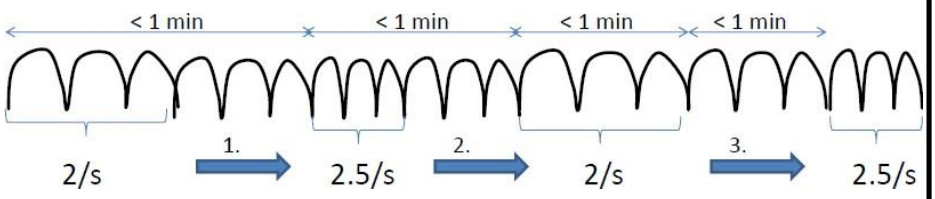
Fluctuation without definite evolution
Three or more changes, not more than 1 min apart, in frequency (by at least 0·5 per s) or three or more changes in location (by at least one standard interelectrode distance), but not qualifying as evolving.[9]



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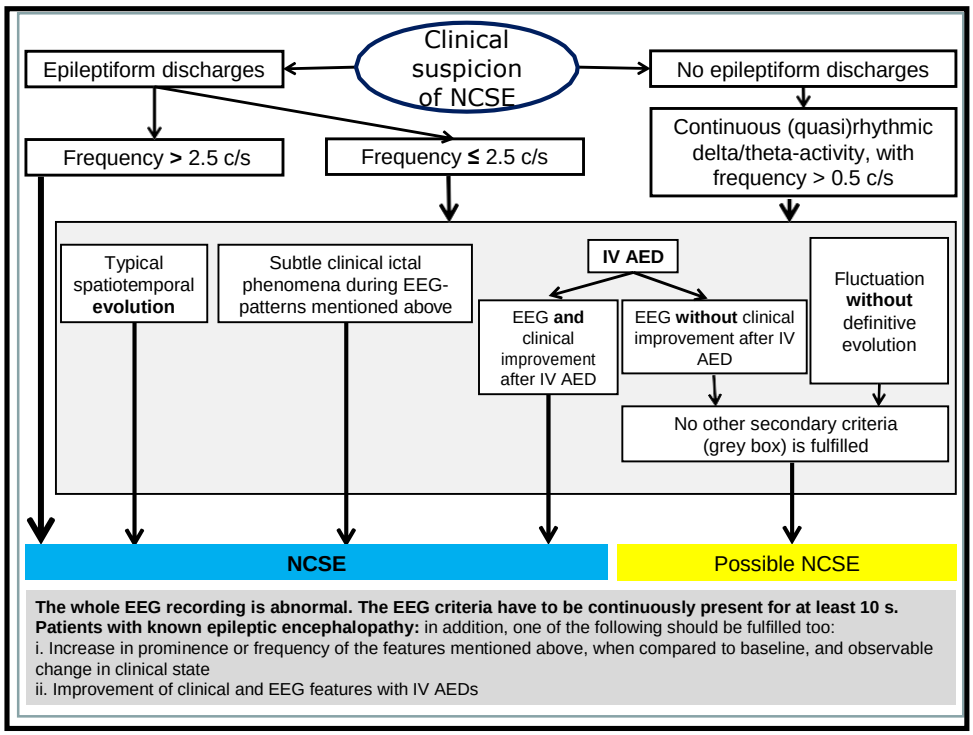

The whole EEG recording is abnormal. The EEG criteria have to be continuously present for at least 10 s.
Patients with known epileptic encephalopathy: in addition, one of the following should be fulfilled too:
i. Increase in prominence or frequency of the features mentioned above, when compared to baseline, and observable change in clinical state
ii. Improvement of clinical and EEG features with IV AEDs

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- **How accurate is this?**
- **Does it work in all the different types of NCSE patients?**
- **None of the NCSE-criteria have been clinically validated before.**

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Diagnostic accuracy study on the EEG criteria for NCSE

- Intrinsic limitation: lack of a proper ”gold standard”
- In such cases gold standard derived from consensus decision inferred from multimodal data:
  - all clinical data
  - para-clinical data:
    - EEG readings (not assessed using Salzburg criteria)
    - laboratory data
    - neuroimaging data
  - therapeutic response
  - follow-up & final outcome.

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Study design

- STARD criteria
- Three centers:
  - *Danish Epilepsy Centre*
  - *Aarhus University Hospital*
  - *Paracelsus Medical University, Salzburg*
- Blinded evaluation of the EEGs
- independently by two experts.
- Consecutive patients:
  - Validation group: clinical suspicion of NCSE
  - Control group: abnormal EEG but no clinical suspicion of NCSE

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Inter-rater agreement for the Salzburg criteria:

	All patients	Validation group	Control group
Salzburg criteria	0·87 (0·81–0·92)	0·81 (0·71–0·89)	0·94 (0·87–0·98)

Table 2: Inter-rater agreement (κ [95% CI])

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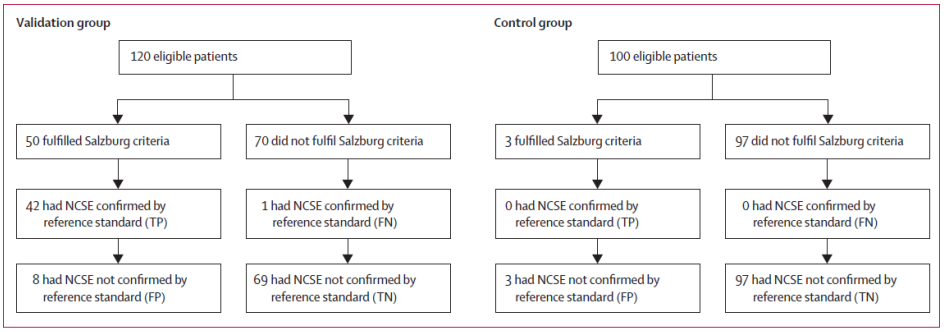

Figure 2: Study profile
FN=false negative. FP=false positive. TN=true negative. TP=true positive.

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Diagnostic accuracy of the Salzburg criteria:

Patients (n)	Sensitivity (%)	Specificity (%)	Accuracy (%)
120	97·7	89·6	92·5

- Sliding window: 10 seconds
- Positives = NCSE + Possible NCSE

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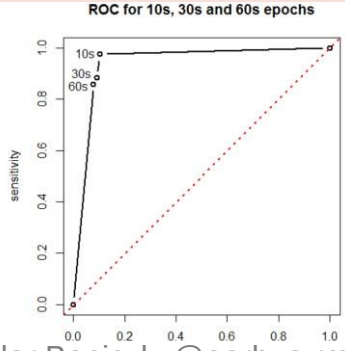
Patients (n)	Time-epoch	Sensitivity (%)	Specificity (%)	Accuracy (%)
120	10 s	97·7	89·6	92·5
120	30 s	88·4	90·9	90·0
120	60 s	86·0	92·2	90·0



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Possible NCSE → Negative (n=19)

	Patients (n)	Sensitivity (%)	Specificity (%)	Accuracy (%)
All patients	120	97·7	89·6	92·5
Possible NCSE considered negative	120	79·1	97·4	90·8

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	Patients (n)	Time-epoch	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)
Coma							
Non-coma	88	10 s	96·7	87·9	80·6	98·1	90·9
Coma	32	10 s	100	94·7	92·9	100	96·9
Hypoxic							
Non-hypoxic	105	10 s	97·2	88·4	81·4	98·4	91·4
Post-hypoxic	15	10 s	100	100	100	100	100
Epilepsy							
Pre-existing epilepsy	45	10 s	95·7	81·8	84·6	94·7	88·9
Without pre-existing epilepsy	75	10 s	100	92·7	83·3	100	94·7
Epileptic encephalopathy							
Epileptic encephalopathy	6	10 s	75·0	100	100	66·7	83·3
Without epileptic encephalopathy	114	10 s	100	89·3	83·0	100	93·0
Age							
Age <10 years	10	10 s	100	100	100	100	100
Age ≥10 years	110	10 s	97·1	89·3	81·0	98·5	91·8

Data are n (%), unless otherwise stated. No significant differences between subgroups. PPV=positive predictive value. NPV=negative predictive value.

Table 3: Diagnostic accuracy for the various subgroups and disorders in the validation cohort

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Diagnostic accuracy of the Salzburg EEG criteria for non-convulsive status epilepticus: a retrospective study

Lancet Neurol 2016; 15: 1054–62

Markus Leitinger, Eugen Trinka, Elena Gardella, Alexandra Rohracher, Gudrun Kalss, Erisela Qerama, Julia Höfler, Alexander Hess, Georg Zimmermann, Giorgi Kuchukhidze, Judith Dobesberger, Patrick B Langthaler, Sándor Beniczky

- The Salzburg criteria for NCSE:
  - have high diagnostic accuracy
  - excellent inter-rater agreement
  - suitable for implementation in clinical practice.

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Next (2019?) edition of ACNS nomenclature

- Will include electrographic seizures and non-convulsive status epilepticus
- All-in-one paper ☺
- Largely based on Salzburg criteria- though with some minor modification

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- **Non-convulsive electrographic seizure (NCSz)**
  - ≥ 10s of:
  - 1. EDs ≥ 2.5 Hz (≥ 25 discharges /10s), or
  - 2. Evolving pattern, or
  - 3. Patterns with:
    - a. time-locked subtle seizure manifestations
    - b. EEG and clinical improvement with an IV-AEDs

  (Only EEG improvement = possible NCSz /NCSE)

- **Non-convulsive status epilepticus (NCSE)**
  - > 10 minutes *or*
  - a total duration of >50% of any 60-minutes period

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- **Diagnostic dichotomy: SE = yes / no (maybe)**

- **Pathophysiologic process: ictal-interictal continuum**
  - → Patterns that indicate significantly higher seizure-risk
  - LPDs: the highest association with seizures
    - regardless of frequency
    - association was greater when the Plus modifier was present
  - LRDA & GPDs were associated with seizures when:
    - Frequency ≥ 1.5 Hz, or
    - Plus modifier was present
  - Increased prevalence / frequency = increased seizure-risk

CEEGs from 4772 critically ill patients
Rodriguez Ruiz et al, JAMA Neurol 2017

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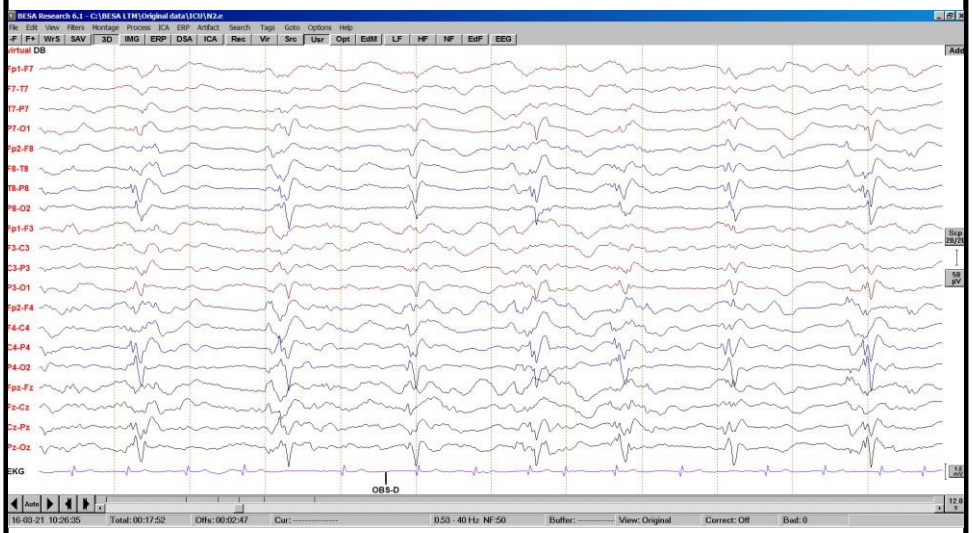

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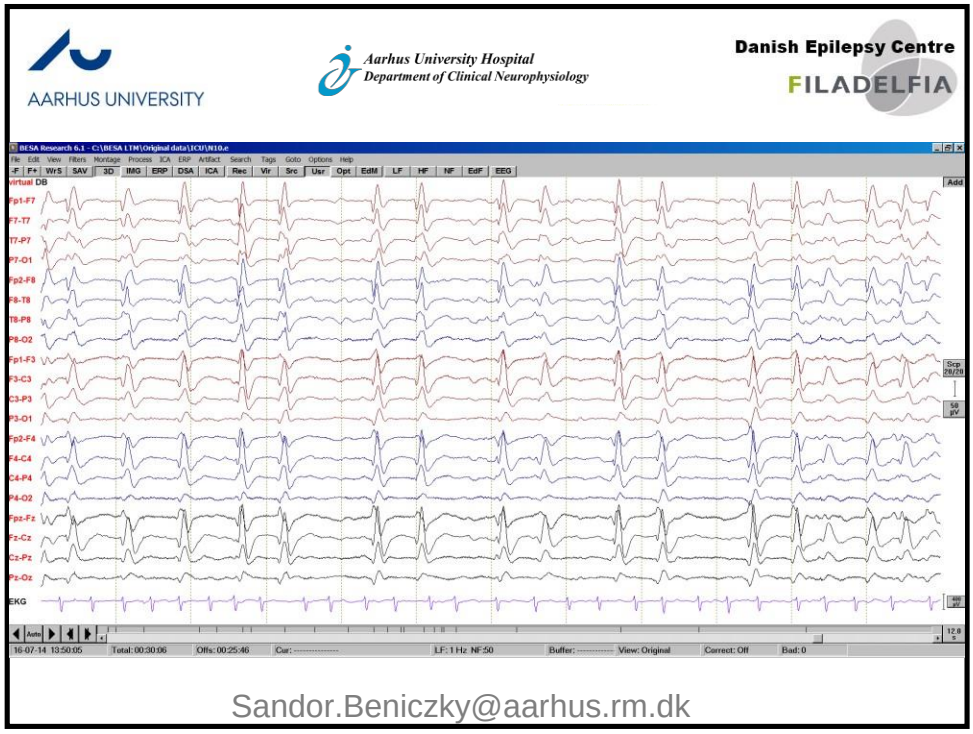
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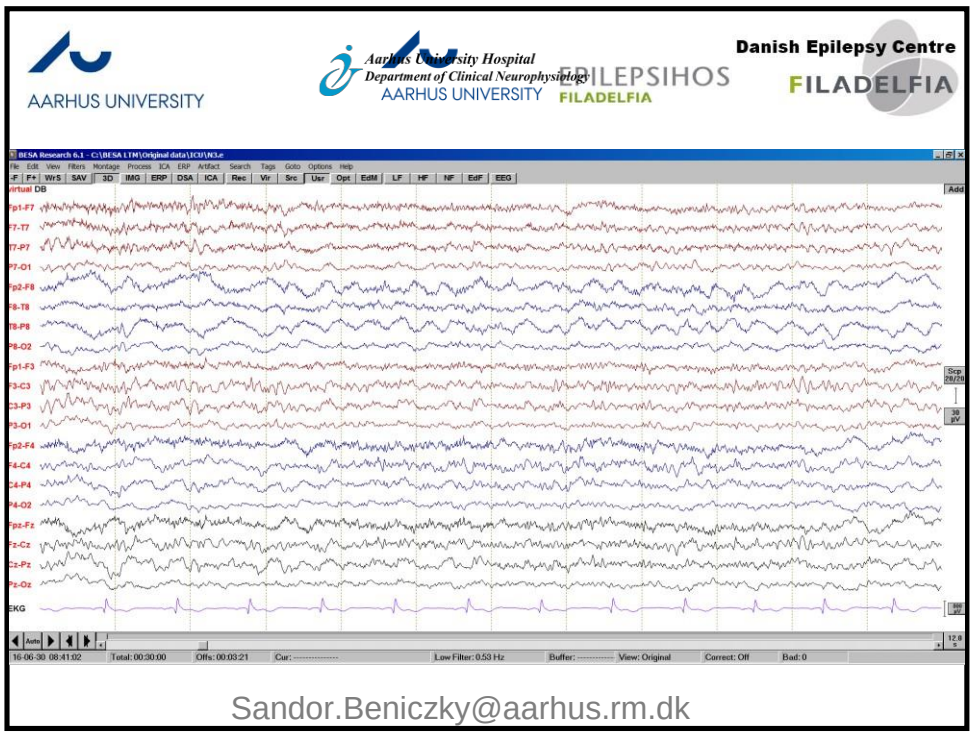
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Monitoring of therapeutic effect: Anesthetics / therapeutic coma

Induction ↓

- Increased β power
- Increased Δ power
- Declining β
- Suppressions with
  increasing/decreasing duration
- Bursts with
  decreasing/increasing duration
- Isoelectric EEG

↑ Withdrawal

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Monitoring of therapeutic effect: Anesthetics / therapeutic coma

- Seizure suppression
- Burst-suppression?
  - Bursts (up to 5s) + suppression (<10 μV; 8-12 s)
- Suppression (Isoelectric EEG)

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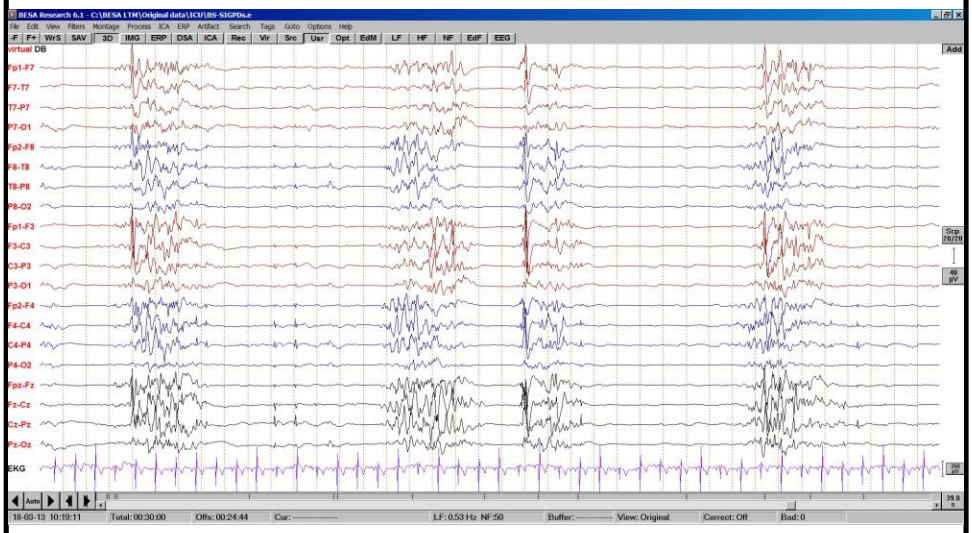

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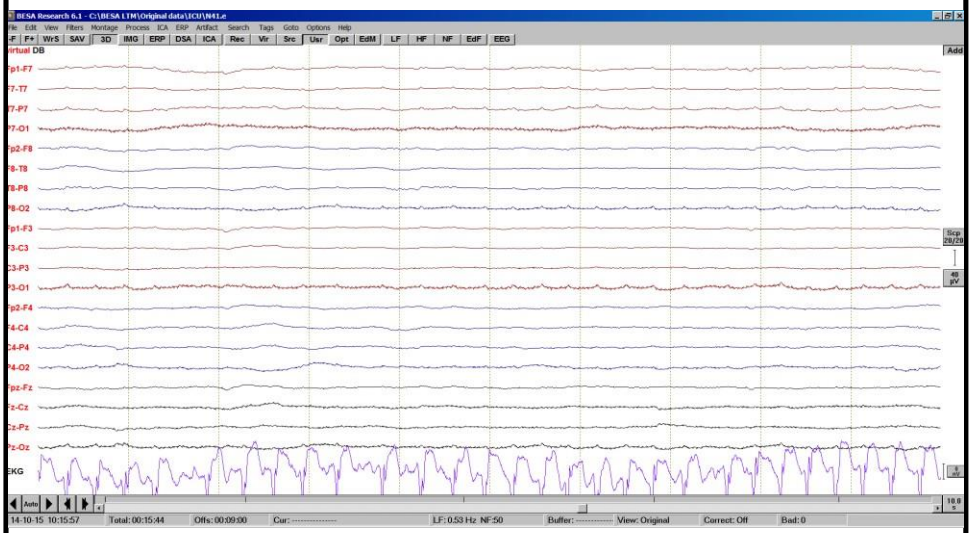

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Monitoring of brain function during withdrawal of anesthetics / after SE

- Do seizures / SE return?
- Emergence of EEG patterns indicating increased seizure-risk?

- CAVEAT: Paradoxical effect of drug-withdrawal
  - Anesthetic wean → hyperexcitability
  - Successful wean despite emergence of Ictal–Interictal EEG patterns during the weaning (Alvin et al., Neurocrit Care 2018)

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Summary

- Diagnosis & classification
  - NCSE: EEG is a must!
  - Dynamics evolution CSE←→NCSE
  - Salzburg criteria / IC-II continuum
- Monitoring of therapeutic effect (anesthetics)
  - Seizure suppression!
  - Burst-suppression / isoelectric EEG?
- Monitoring during withdrawal of anesthetics
  - Caveat: paradoxical effect

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