IRCCS FONDAZIONE STELLA MARIS

Tuscany Region PhD Programme in Neurosciences

STATO EPILETTICO NON CONVULSIVO

INQUADRAMENTO E GESTIONE TERAPEUTICA

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No conflicts of interest to declare

DEFINITION OF STATUS EPILEPTICUS

International League Against Epilepsy

HISTORICAL CONTEXT

"seizure that persists for a sufficient length of time or is repeated frequently enough to produce a fixed and enduring condition" (ILAE, 1970)

"seizure that persists for a sufficient length of time or is repeated frequently enough that recovery between attacks does not occur" (ILAE, 1981)

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, 56(10):1515–1523, 2015
doi: 10.1111/epi.13121

SE 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 that 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.

Seizure xxx (2016) xxx–xxx

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Seizure

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

25 years of advances in definition, classification and treatment of status epilepticus☆

Eugen Trinka[a,b,c,*], Reetta Kälviäinen[d,e]

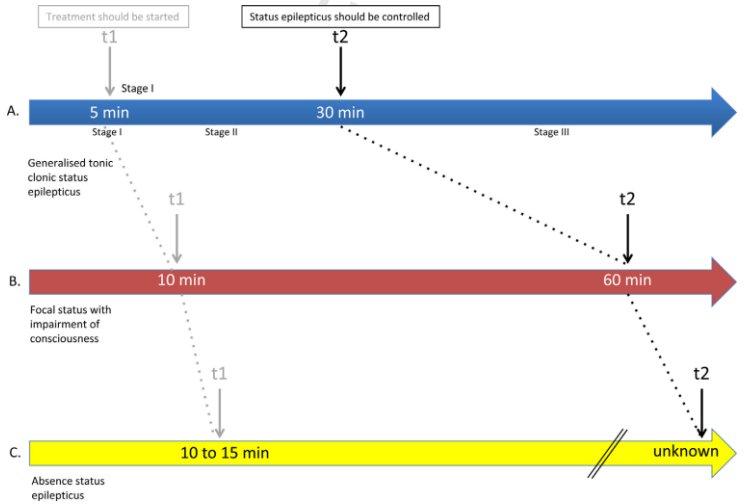

Fig. 1. Operational dimensions with t1 indicating the time that emergency treatment of SE should be started and t2 denoting the time at which long term consequences may be expected. Time (t_1), when a seizure is likely to be prolonged leading to continuous seizure activity. Time (t2), when a seizure may cause long-term consequences (including neuronal injury, neuronal death, alteration of neuronal networks and functional deficits). For generalized tonic clonic status the stages have been added (stage I 5–10 min; stage II 10–30 min; stage III 30–30 min) [13].

For classification of SE we propose the following four axes:

1 Semiology
2 Etiology
3 EEG correlates
4 Age

Table 2. Axis I: Classification of status epilepticus (SE)

(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

NCSE

Table 4. Etiology of status epilepticus

Known (i.e., symptomatic)
- Acute (e.g., stroke, intoxication, malaria, encephalitis, etc.)
- Remote (e.g., posttraumatic, postencephalitic, poststroke, etc.)
- Progressive (e.g., brain tumor, Lafora's disease and other PMEs, dementias)
- SE in defined electroclinical syndromes

Unknown (i.e., cryptogenic)

Table 3. Currently indeterminate conditions (or "boundary syndromes")

Epileptic encephalopathies
Coma with non evolving epileptiform EEG pattern[a]
Behavioral disturbance (e.g., psychosis) in patients with epilepsy
Acute confusional states, (e.g., delirium) with epileptiform EEG patterns

[a]Lateralized and generalized periodic discharges with monotonous appearance are not considered as evolving EEG patterns.[26,27]

Epilepsia, 56(10):1515–1523, 2015
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NON CONVULSIVE STATUS EPILEPTICUS

NCSE is a highly heterogeneous clinical condition and broadly defined as a paroxysmal change in behavior and/or mental processes from baseline without convulsive activity, associated with continuous paroxysmal activity or electrographic discharges on the electroencephalogram

Meierkord H, Holtkamp M. Non-convulsive status epilepticus in adults: clinical forms and treatment. *Lancet Neurol.* 2007;6:329-339.
Maganti R, Gerber P, Drees C, Chung S. Nonconvulsive status epilepticus. *Epilepsy Behav.* 2008;12:572-586.

NCSE can present various and subtle symptoms, which include two semiological spectrums

(a) Negative Symptoms: anorexia, aphasia/mutism amnesia, catatonia, coma, confusion, lethargy and staring;

(b) Positive Symptoms: agitation/aggression, automatisms, blinking, delirium, delusions, echolalia, facial twitching, laughter, nausea/vomiting, nystagmus/eye deviation, perseveration, psychosis and tremulousness

Jirsch J, Hirsch LJ. Nonconvulsive seizures: developing a rational approach to the diagnosis and management in the critically ill population. *Clin Neurophysiol.* 2007;118:1660-1670.

Neurology® Clinical Practice

Epidemiology, diagnosis, and management of nonconvulsive status epilepticus

Opening Pandora's box

Raoul Sutter, MD
Stephan Rüegg, MD
Peter W. Kaplan, MB, BS, FRCP

Neurology: Clinical Practice ❙ December 2012

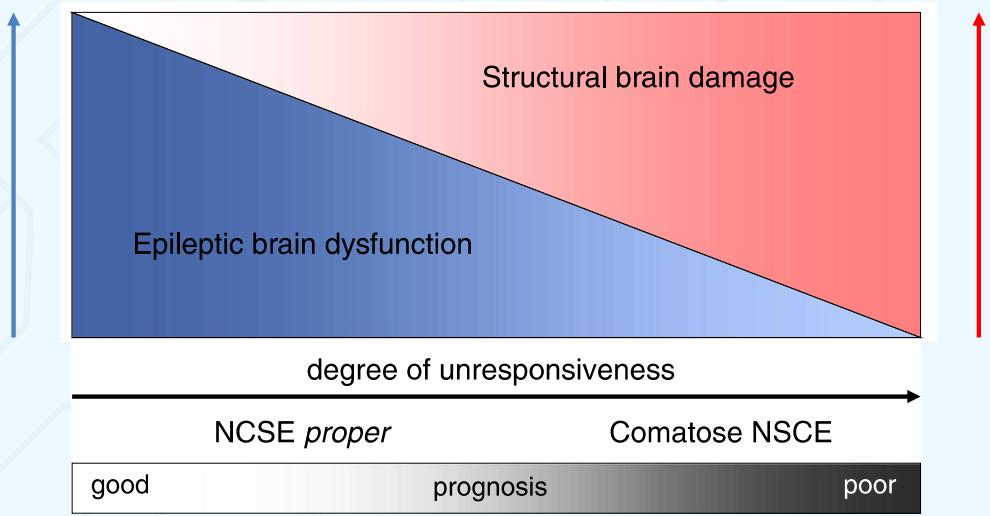
Therapeutic approaches to NCSE are diverse and controversial, stemming from the different prognoses of the subtypes of NCSE. Prospective, randomized trials regarding treatment of NCSE are lacking.

AS in IGE
Late AS de novo
Atypical AS

Focal SE with impaired consciousness,
Aura continua,
Status aphasicus

Acute symptomatic focal SE +/- EPC
Subtle SE

Coma with GPD
Coma with LPD



E. Trinka, M. Leitinger / Epilepsy & Behavior 49 (2015) 203–222

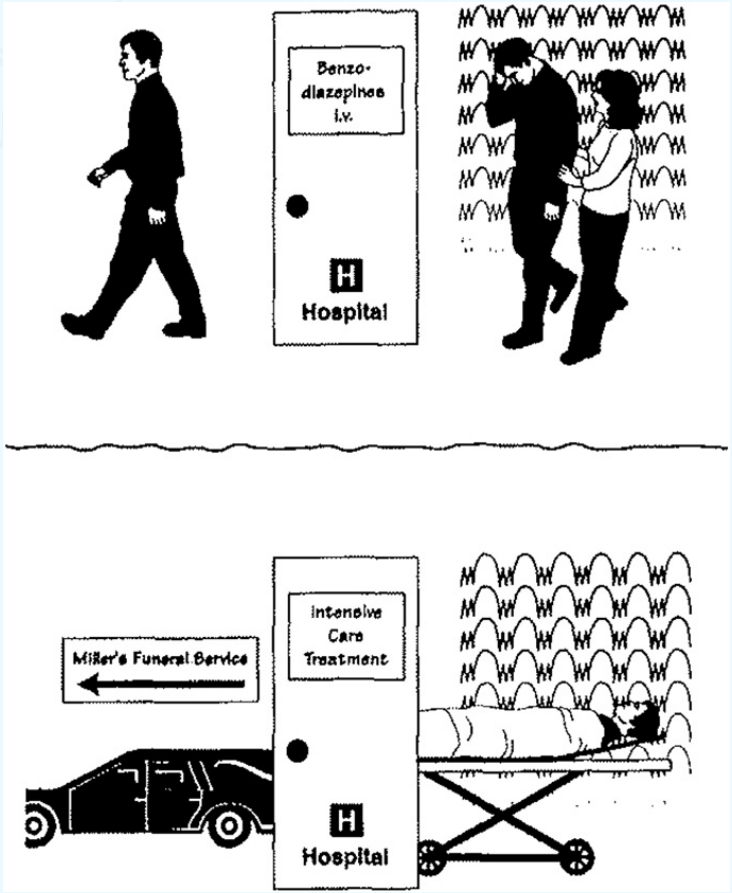


Fig. 1. Cartoon depicting the similarities of EEG patterns in absence status and generalized periodic discharges in the comatose patients (from 22, Fig. 3).

Epilepsy: mimics, borderland and chameleons

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Abstract
Epilepsy mimics such as syncope and psychogenic attacks, can present like epilepsy, and can be erroneously managed as epilepsy. There are also several conditions at the borderland that closely relate to epilepsy yet are probably separate from it, eg, migralepsy and parasomnia. Finally, there are times when epileptic seizures resemble one of the epilepsy mimics. This is epilepsy in disguise—the epilepsy chameleons. Seizures with typically unusual manifestations, such as occipital or parietal lobe seizures, or those occurring in situations where another cause seems more likely, eg, in a person with alcoholism, may well be overlooked as epilepsy and initially escape diagnosis. This review explores the mimics of adult epilepsy, the epilepsy borderland, and focuses particularly on epilepsy chameleons.

Introduction

The wide differential diagnosis of episodic altered consciousness presents a major diagnostic problem.[1] Epilepsy mimics, notably syncope and psychogenic attacks, can present like epilepsy, and may be managed erroneously as epilepsy (often for the long term), yet clearly are distinct from it. Furthermore, there are several conditions closely relating to epilepsy that are probably separate from epilepsy, for example, migralepsy and parasomnia: these are at the borderland of epilepsy. Finally, there are situations when epileptic seizures resemble one of the epilepsy mimics. This is epilepsy in disguise—an epilepsy chameleon.[2] Figure 1 shows the diagrammatic relationship between epilepsy, its borderland, its mimics and its chameleons. Note that epilepsy mimics are part of other conditions' chameleons list, for example, of transient ischaemic attacks, dementia, multiple sclerosis. Similarly, all epilepsy chameleons appear on the lists of other conditions' mimics (figure 2).

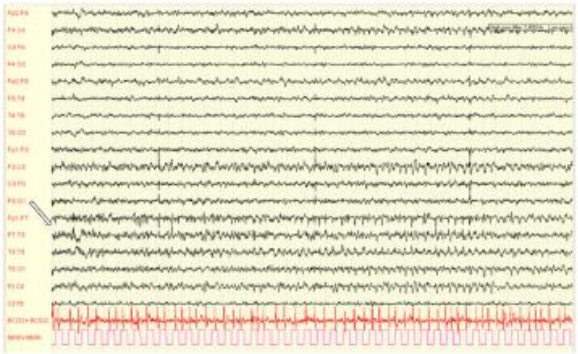
In practice, the labelling of episodic loss of consciousness errs more towards diagnosing epilepsy when it is not, rather than failing to diagnose epilepsy when it is. Thus, epilepsy mimics are more problematic in practice than are epilepsy chameleons. Nevertheless, our patients need us to recognise and treat their epilepsy promptly when it occurs, even if it comes in disguise.

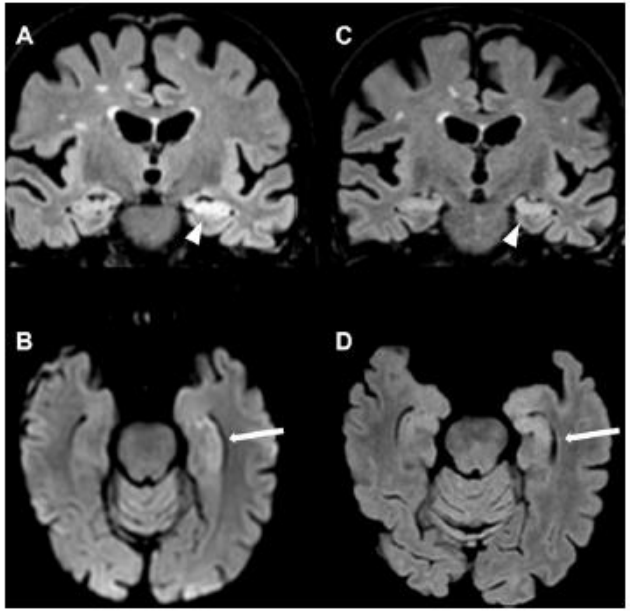
This review briefly explores mimics of adult epilepsy, the epilepsy borderland, and focuses particularly on epilepsy chameleons. The main focus here is in on adults: paediatric presentations are more complex, with greater propensity for misdiagnosis of paroxysmal events at either of the epilepsy spectrum.

- Syncope
- Reflex
  - Vasovagal, micturition, swallow, carotid sinus, orgasmic and laughing
- Cardiac
  - Arrhythmogenic
  - Elderly: scar-related ventricular tachycardia
  - Young: long QT syndrome, short QT syndrome, arrhythmogenic right ventricular cardiomyopathy
  - Structural, aortic stenosis, hypertrophic cardiomyopathy
- Orthostatic
  - Autonomic failure
- Psychogenic non-epileptic attack disorder
  - Panic disorder (especially in people with epilepsy)
  - Dissociative
  - Factitious and malingering
- Sleep disorders
  - Narcolepsy syndrome and cataplexy
  - Parasomnias (see Borderland of epilepsy section)
- Paroxysmal symptoms of structural brain disease
  - Multiple sclerosis
  - Tumour, eg, brainstem glioma
- Vascular
  - Migraine (hemiparetic, occipital, 'basilar artery')
  - Shaking transient ischaemic attack (critical bilateral stenosis)
  - Subclavian steal syndrome
  - Moyamoya (combination of TIA and seizures)
  - *Not* vertebrobasilar insufficiency
- Hypoglycaemia
  - Behaviour disturbance
  - Hemiparesis
- Movement disorder
  - Paroxysmal kinesigenic dystonia/dyskinesia
  - Myoclonus following hypoxia
- Hydrocephalus
  - Colloid cyst
  - Chiari malformation
- Drop attacks
  - Postural instability
  - Psychogenic

Hyperosmolar hyperglycaemic state causing atypical status epilepticus with hippocampal involvement

Emanuele Bartolini ,[1] Raffaella Valenti,[1] Josemir W Sander [2,3]





Key Points

- Diabetes mellitus may abruptly precipitate a hyperosmolar hyperglycaemic state.
- An insidious status epilepticus can very rarely occur, impairing vigilance despite metabolic recovery.
- Clinicians should monitor the electroencephalogram (EEG) in people with a hyperosmolar hyperglycaemic state whose alertness is not fully restored after correcting metabolic abnormalities.

Bartolini E, *et al*. *Pract Neurol* 2022;**22**:117–119. doi:10.1136/practneurol-2021-003222

Epilepsia, 54(Suppl. 6):28–29, 2013
doi: 10.1111/epi.12270

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

Modified from Kaplan (2007).
EDs, epileptiform discharges (spikes, poly spikes, sharp-waves, sharp-and-slow-wave complexes); IV AEDs: intravenous antiepileptic drugs.
[a]If EEG improvement occurs without clinical improvement, or if fluctuation without definite evolution, this should be considered possible NCSE.
[b]Incrementing onset (increase in voltage and change in frequency), or evolution in pattern (change in frequency >1 Hz or change in location), or decrementing termination (voltage or frequency).

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

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

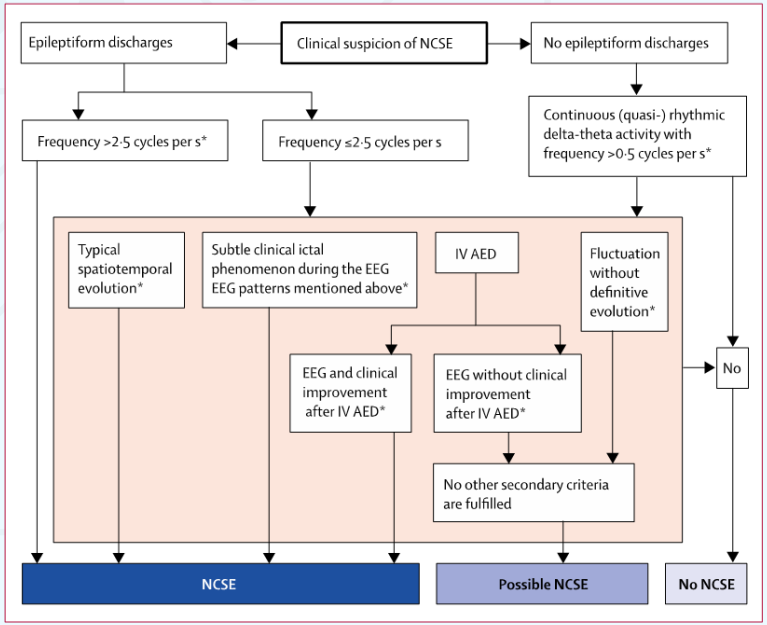


***Figure* 1: Salzburg EEG criteria for the diagnosis of NCSE**
To qualify for a diagnosis of NCSE, the whole EEG recording should be abnormal, and EEG criteria have to be continuously present for at least 10 s. If criteria are not fulfilled at any stage, EEG recording will not qualify for a diagnosis 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).

■ Multimedia teaching material

Epileptic Disord 2021; 23 (2): 425-426

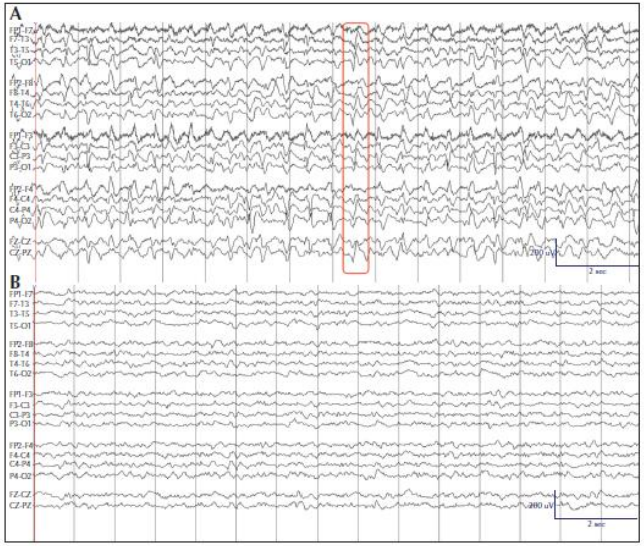
Epileptic Disorders

Generalized periodic discharges with triphasic morphology: to treat or not to treat?

Fábio A. Nascimento, Patrick M. Chen, Joseph Cohen, Brandon M. Westover

We report an 82-year-old woman with recurring episodes of confusion, a left posterior fossa meningioma, and chronic hyponatremia due to syndrome of inappropriate antidiuretic hormone secretion (SIADH). She presented with a habitual episode of confusion. Basic laboratory workup was unremarkable except for hyponatremia (132 mEq/L sodium); head imaging was unrevealing. Continuous EEG showed 2-Hz generalized periodic discharges (GPDs) with triphasic morphology, or "triphasic waves" (*figure 1A*). Her EEG and mental status improved following 1 mg of lorazepam suggesting non-convulsive status epilepticus (NCSE).

Although traditionally associated with toxic metabolic infectious encephalopathies, GPDs with triphasic morphology may be associated with NCSE. Select patients should therefore undergo a trial with antiseizure medication to assess for electroclinical improvement [1, 2]. Importantly, electrographic improvement alone does not imply a diagnosis of NCSE [3]. ■



■ **Figure 1.** EEG: pre-lorazepam trial (A), and 10 minutes following the lorazepam trial (B). Sensitivity: 10 mV/mm; LF: 1 Hz; HF: 70 Hz; notch: 60 Hz; timebase: 15 mm/second. (A) Bipolar montage showing 100 mV 2 Hz generalized periodic discharges with triphasic morphology. (B) Bipolar montage showing diffuse irregular 50 mV delta slowing of the background, excessive beta frequency activity, and resolution of generalized periodic discharges.

NCSE in unconscious patients

neurocritical care society Neurocrit Care (2015) 23:S136–S142
DOI 10.1007/s12028-015-0172-3

CrossMark

REVIEW ARTICLE

Emergency Neurological Life Support: Status Epilepticus

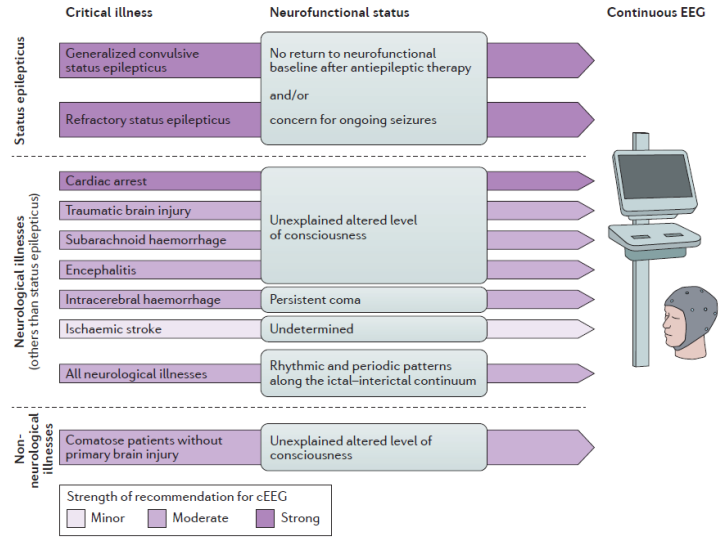
Jan Claassen[1] · James J. Riviello Jr.[2] · Robert Silbergleit[3]

Table 1 Status epilepticus checklist for the first hour

Checklist
☐ Fingerstick glucose
☐ Obtain IV access
☐ Pulse oximetry, BP monitor, supplemental O_2 and fluid as needed, cardiac monitor
☐ Labs: Complete blood count, Basic metabolic panel, Calcium, Magnesium, AED levels
☐ Head CT
☐ Continuous EEG (if available); notify EEG tech if available (as soon as available unless patient returns to pre-status epilepticus baseline)

Nonconvulsive status epilepticus in adults — insights into the invisible

Raoul Sutter[1,2], Saskia Semmlack[1] and Peter W. Kaplan[3]



In 2017, based on a prospective multicenter database, the 2HELPS2B score was created to estimate the seizure risk in acutely hospitalized patients receiving cEEG. According to the 2HELPS2B score calculated from the first hour of cEEG results, patients can be stratified into 3 categories: low-(2HELPS2B = 0), medium-(2HELPS2B = 1), and high-risk (2HELPS2B ≥2) risk. Each category has an associated minimum recommended duration of cEEG monitoring to avoid missed seizures with a certainty of 95% and 98%

Struck AF, Ustun B, Ruiz AR, et al. Association of an electroencephalography-based risk score with seizure probability in hospitalized patients. *JAMA Neurol*. 2017;74:1419-1424.
Struck AF, Tabaeizadeh M, Schmitt SE, et al. Assessment of the validity of the 2HELPS2B score for inpatient seizure risk prediction. *JAMA Neurol*. 2020;77:500-507.

TABLE 2 2HELPS2B score

Risk factors	Score
Frequency >2 Hz[a]	1
Independent sporadic epileptiform discharges	1
LPD/BIPD/LRDA	1
Plus features (superimposed rhythmic, fast, sharp)[b]	1
Prior seizure[c]	1
BIRD	2

Note: Adapted from Aaron F. Struck. JAMA Neurol. 2020;77(4):500–507.[45] & JAMA Neurol. 2017;74(12):1419–1424.[44]

Abbreviations: BIPD, bilateral independent periodic discharges; BIRD, brief potentially ictal rhythmic discharge; LPD, lateralized periodic discharge; LRDA, lateralized rhythmic delta activity.

[a]Frequency of any periodic or rhythmic pattern of more than 2 Hz except GRDA.

[b]Plus features include superimposed rhythmic, fast, or sharp activity only on LRDA, LPDs, or BIPDs.

[c]Prior seizure includes a remote history of epilepsy or recent events suspicious for clinical seizures.

TABLE 3 Seizure risk based on 1-h screening EEG

Seizre risk group	Recommend duration of EEG monitoring	
	For seizure risk <5% (h)	For seizure risk <2% (h)
Low: 2HELPS2B = 0	1	3.3
Medium: 2HELPS2B = 1	12	29
High: 2HELPS2B ≥ 2	≥24	≥30

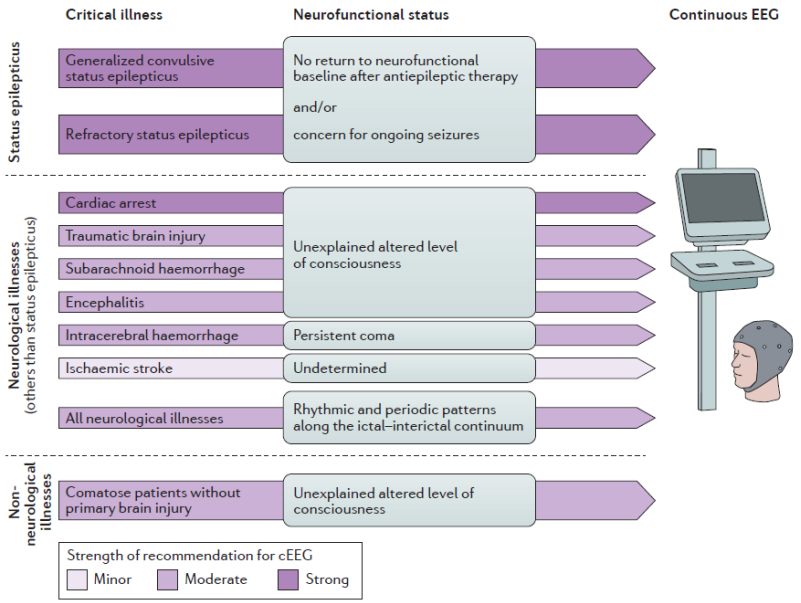
Note: Excludes cardiac arrest patients.

Information obtained from Aaron F. Struck. JAMA Neurol. 2020;77(4):500–507.[45] & Pablo Bravo. Drugs (2021) 81:749–770.[1]

Abbreviation: EEG, Electroencephalogram.

Nonconvulsive status epilepticus in adults — insights into the invisible

Raoul Sutter[1,2], Saskia Semmlack[1] and Peter W. Kaplan[3]



First 2 to 5 minutes

Emergent initial treatment

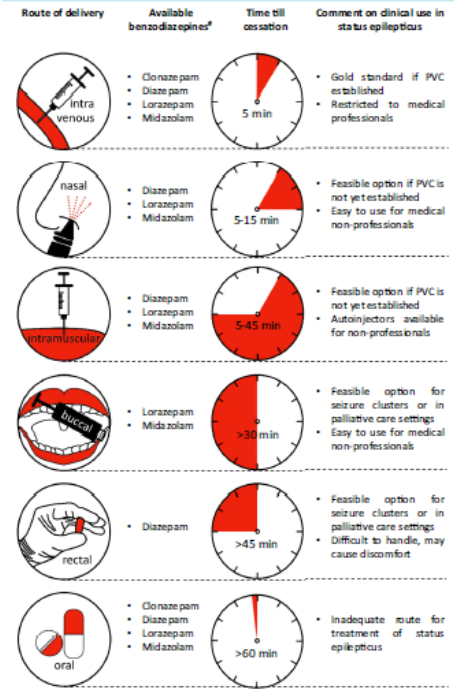
- First-line AEDs (benzodiazepines orally, intramuscular, or intravenous -bolus)

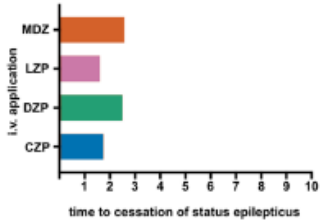
CNS Drugs (2022) 36:951–975
https://doi.org/10.1007/s40263-022-00940-2

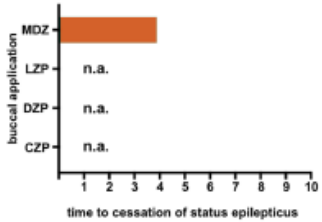
REVIEW ARTICLE

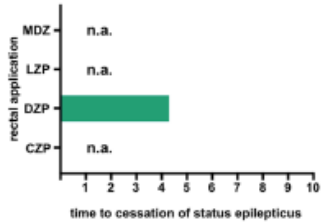
Benzodiazepines in the Management of Seizures and Status Epilepticus: A Review of Routes of Delivery, Pharmacokinetics, Efficacy, and Tolerability

Ricardo Klein[1,2] · Lara Kay[1,2] · Isabelle Beuchat[1,2,3] · Sarah Gelhard[1] · Sophie von Brauchitsch[1] · Catrin Mann[1,2] · Alexandra Lucaciu[1] · Jan-Hendrik Schäfer[1] · Kai Siebenbrodt[1,2] · Johann-Philipp Zöllner[1,2] · Susanne Schubert-Bast[1,2,4] · Felix Rosenow[1,2] · Adam Strzelczyk[1,2] · Laurent M. Willems[1,2]



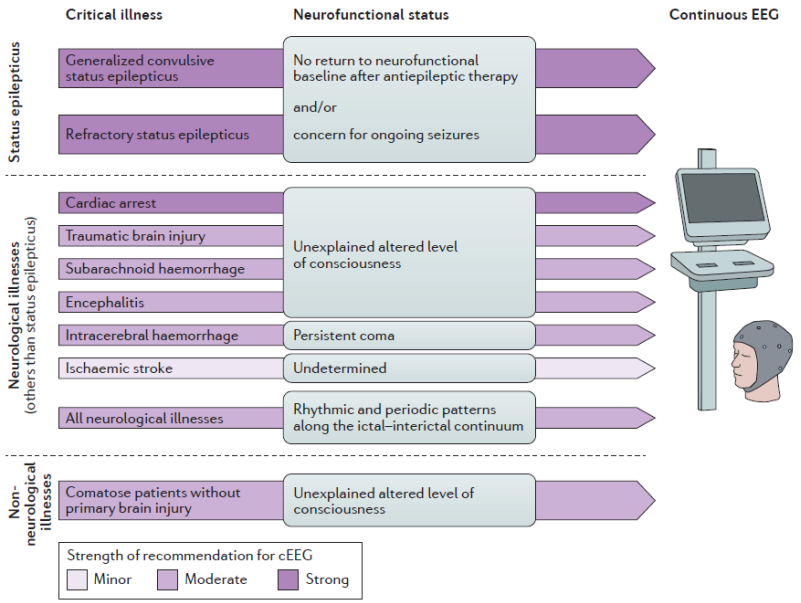


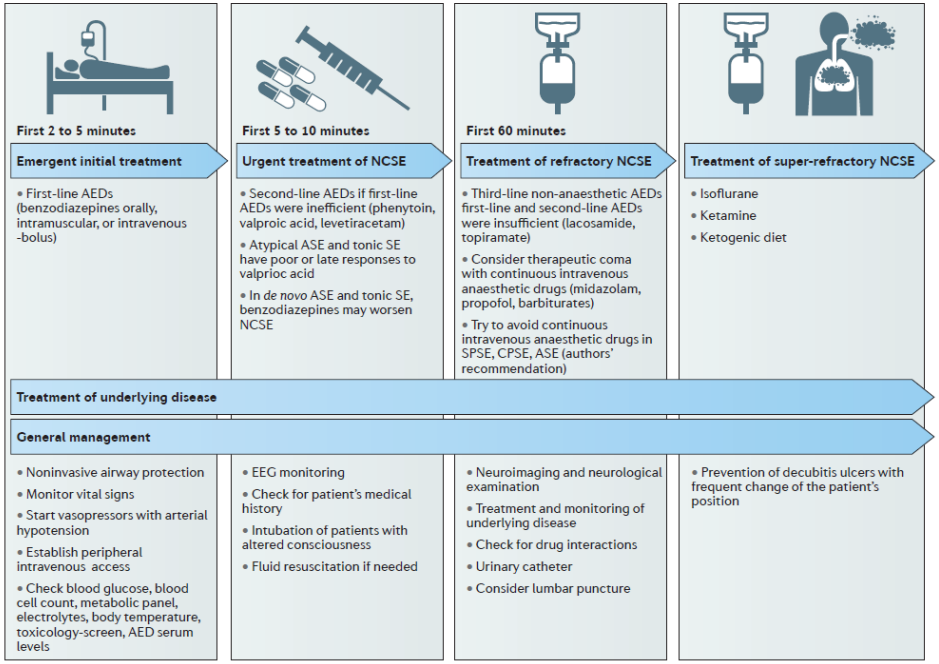




Nonconvulsive status epilepticus in adults — insights into the invisible

Raoul Sutter[1,2], Saskia Semmlack[1] and Peter W. Kaplan[3]





New-onset refractory status epilepticus (NORSE)

Laura Mantoan Ritter,[1,2] Lina Nashef [1]

Box 1 Definitions

Consensus definition of new-onset refractory status epilepticus (NORSE)

'New-onset refractory status epilepticus is a clinical presentation, not a specific diagnosis, in a patient without active epilepsy or other pre-existing relevant neurological disorder, with new-onset of refractory status epilepticus without a clear acute or active structural, toxic or metabolic cause. This includes patients with viral or autoimmune causes. If no cause is found after extensive evaluation, this is considered "cryptogenic NORSE" or "NORSE of unknown cause"'.

Consensus definition of febrile infection-related epilepsy syndrome (FIRES)

'FIRES is a subcategory of NORSE that requires a prior febrile infection, with fever starting between 2 weeks and 24 hours prior to onset of refractory status epilepticus, with or without fever at onset of status epilepticus'.

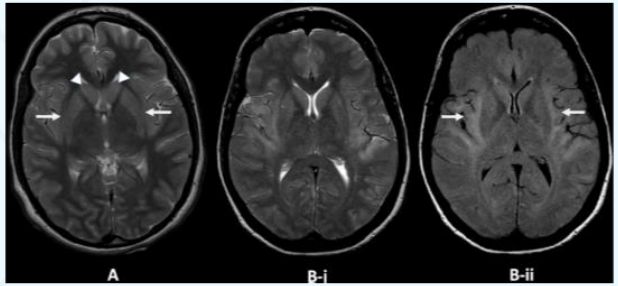


Figure 2 Claustrum changes on MR in two adult patients from the King's College Hospital new-onset refractory status epilepticus database. (A) Axial T2-weighted image, patient 1: there is faint T2 high signal in the claustrum on both sides (arrows) with faint T2 high signal and slight swelling in both caudate nuclei (arrowheads). (B) Axial T2-weighted (i) and fluid-attenuated inversion recovery (ii) images, patient 2. There is T2 high signal involving the claustrum, external capsule and insula and temporal opercula on both sides (arrows). Image courtesy Dr Jo Jarosz, King's College Hospital.

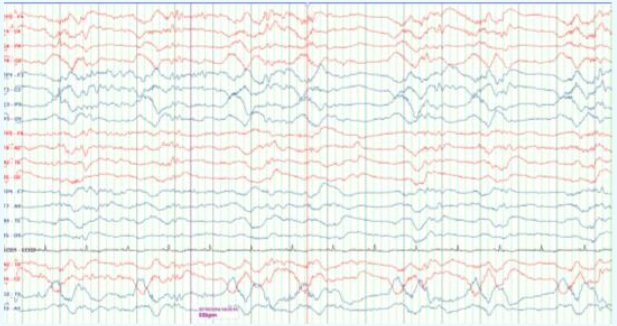


Figure 3 Electroencephalography (EEG): a periodic pattern can be seen consisting of delta waves preceded by fast activity and followed by electrodecrement in this routine EEG (bipolar montage) performed on an adult female patient with new-onset refractory status epilepticus. Abeta-delta complex resembling extreme delta brushes is highlighted (red line). Image courtesy Marisa Pinada Pinto, King's College Hospital.

Seizure: European Journal of Epilepsy 68 (2019) 72–78

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Seizure: European Journal of Epilepsy

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

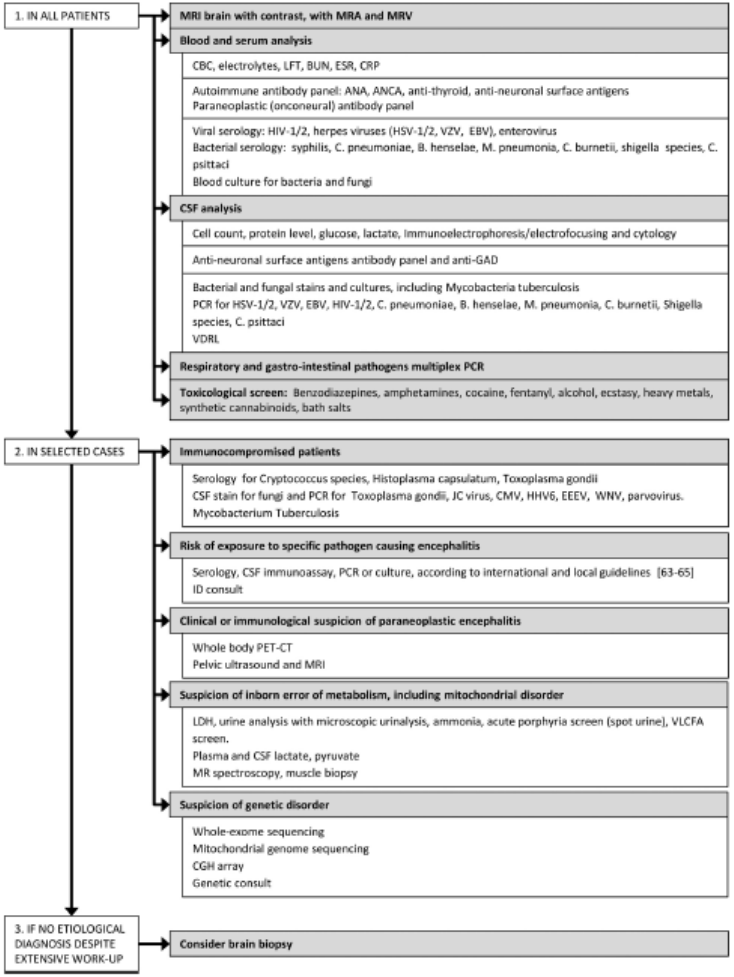
Review

New onset refractory status epilepticus (NORSE)

Claudine Sculier[a,b,*], Nicolas Gaspard[a,c]

NORSE: Prominent presentation features of the most frequent etiologies.

Categories	*	Most frequent findings	Clinical clues
Unknown	50%		No specific findings Prodromal mild febrile illness in 65% of cases Typically severe and prolonged SE
Inflammatory and auto-immune encephalitis	40%	Paraneoplastic limbic encephalitis (Anti-Hu, -Ma2/Ta, -CV2/CRMP-5, -amphiphysin, -VGCC, -mGluR5)	Cognitive, especially memory impairment, behavioral changes, temporal lobe seizures, sleep disturbance Hu: often more diffuse encephalomyelitis Ma2/Ta: hypothalamic dysfunction CV2/CRMP5: diffuse encephalomyelitis, chorea
		Surface-binding autoantibodies	
		Anti-NMDAr	Mostly young females Prodromal fever, short-term memory loss, psychiatric symptoms, hallucinations, oro-lingual dyskinesia, autonomic and respiratory failure *Children*: behavioral changes, movement disorders *EEG*: extreme delta brushes (50%)
		Anti-VGKC complex	Mostly elderly males LGI-1: limbic encephalitis, facio-brachial dystonic seizures, SIADH Caspr2: episodic ataxia
		Anti-GABA(B)r	Limbic encephalitis
		Anti-GABA(A)r	Multifocal neocortical encephalitis
		Anti-AMPAr	Prominent psychiatric symptoms, cerebellar ataxia
		Anti-Glycine-r	No specific features
		Anti-GAD	No specific features
		Steroid responsive encephalopathy with autoimmune thyroiditis	Rapid-onset dementia, myoclonus, stroke-like episodes Anti-TPO, anti-TG
Infectious encephalitis	10%	HSV1	Temporal involvement
		Enterovirus	Rash, acute lower motor neuron syndrome
		CMV	*Immunodeficiency*: Gastro-intestinal symptoms, retinitis, pneumonitis
		EBV	Adenopathies, ataxia
		VZV	*Immunodeficiency*: CNS lymphoma
		Mycoplasma pneumoniae	Rash
		Bartonella henselae	Respiratory symptoms, EEG: extreme spindles
		Arboviruses (West Nile virus, tick-borne virus etc..)	Children. Cat-scratch disease with skin lesion and regional adenopathy Flu-like episode; WNV: parkinsonism, acute lower motor neuron syndrome, EEG: triphasic waves
Genetic disorders	Rare	SCN1A	Dravet syndrome
		PCDH19	Epilepsy and mental retardation limited to female
		CADASIL	Migraine, strokes, visual problems, cognitive deterioration
		Mitochondrial disorders	Elevated CSF lactate and stroke-like episodes.
		MELAS	Occipital seizures, *epilepsia partialis continua*, liver failure, nystagmus, ataxia.
		POLG1	



NORSE treatment algorithm: Commonly used drugs in NORSE and FIRES with most frequently reported doses (expert opinion)

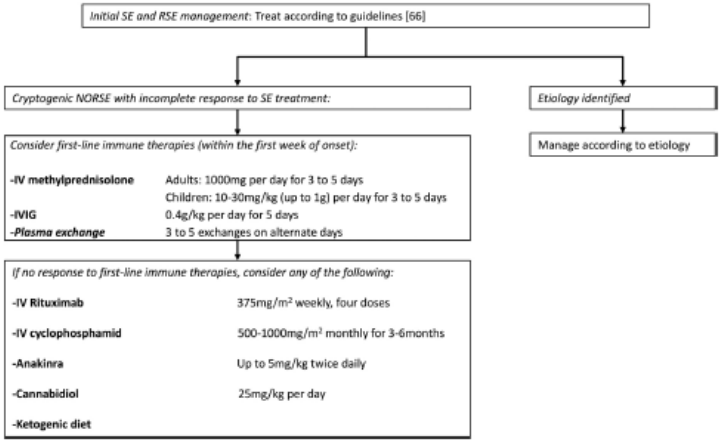


Fig. 2. NORSE treatment algorithm: Commonly used drugs in NORSE and FIRES with most frequently reported doses (expert opinion) [66].
Adapted from Gaspard et al, 2018 [39] and van Baalen et al, 2017 [10].
IV = intravenous; IVIG = intravenous immunoglobulin; RSE: refractory status epilepticus, SE: status epilepticus.

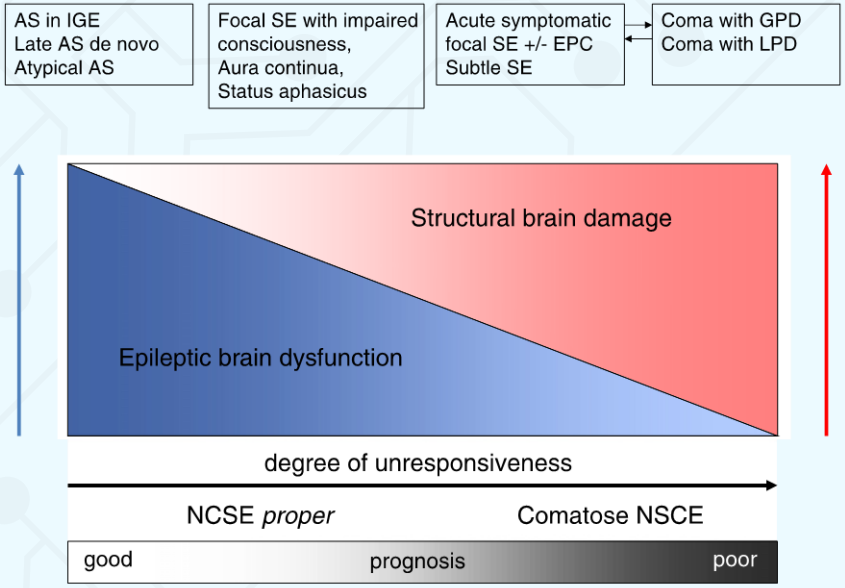
NCSE in Intensive Care Units

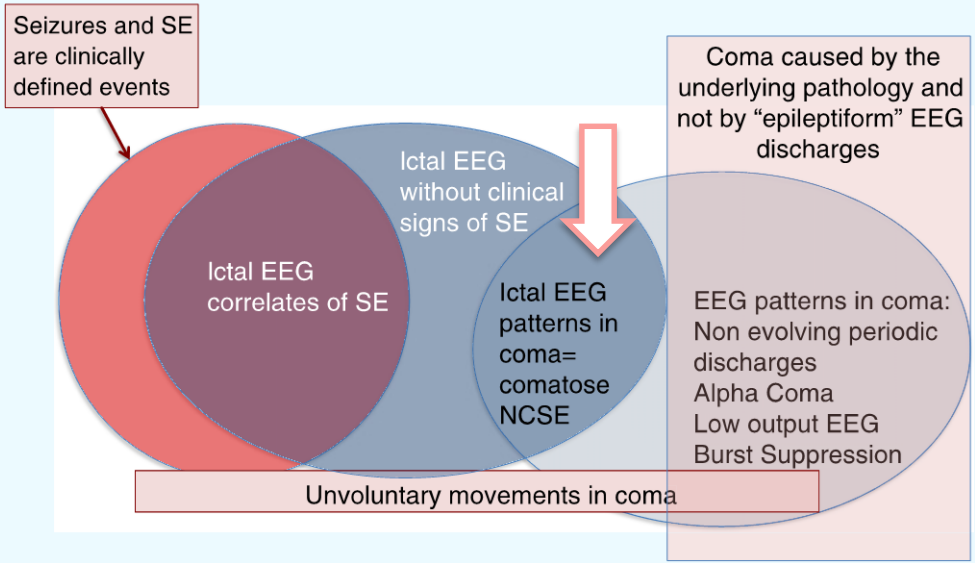
Review

Epilepsy & Behavior 49 (2015) 203–222

Which EEG patterns in coma are nonconvulsive status epilepticus?

Eugen Trinka [a,b,*], Markus Leitinger [a]





Clinical Neurophysiology Practice 4 (2019) 170–177

Contents lists available at ScienceDirect

Clinical Neurophysiology Practice

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

Systematic review article

Nonconvulsive seizures and nonconvulsive status epilepticus in the neuro ICU should or should not be treated aggressively: A debate

Andrea O. Rossetti [a,1], Lawrence J. Hirsch [b], Frank W. Drislane [c,*]

Patients appear most likely to die from complications encountered in prolonged ICU courses (partly occasioned by the use of highly sedating drugs, but also by underlying illnesses) than they are from the SE itself or the direct effects of those drugs, e.g. hypotension (Sutter et al., 2014; Lai et al., 2015).

1. **The proper diagnosis of both the SE type and its etiology should be determined quickly, and treatment should start as soon as possible, at appropriate, adequate doses.**
2. The management and treatment of refractory GCSE and SRSE is now clearly established (Brophy et al., 2012). If early ASDs fail, it is appropriate to use BDZs, propofol, and pentobarbital, as necessary (Brophy et al., 2012; Shorvon and Ferlisi, 2011).
3. Patients with the continuation of GCSE in its later, 'subtle SE' form (where the EEG shows definite or probable NCSE by current EEG definitions) are seriously ill and warrant the same treatment (Treiman et al., 1990, 1998).
4. When NCSE follows a generalized convulsion or GCSE and early ASDs fail, lean toward aggressive treatment, with eventual taper.
5. SE with prior epilepsy syndromes (absence, simple partial, CPSE, and 'benign' MSE) often does well and should rarely be treated aggressively. Treat with higher doses of the patient's earlier ASDs. Other options include i.v. DPH, PB, VPA, LEV, LCM, or other non-sedating i.v. ASDs or other BDZs.
6. If there was NO earlier convulsion or definite seizure, or if seizure activity has been entirely nonconvulsive, or it is unclear how worrisome the NCSE is, try to rely on non-sedating ASDs agents or non-pharmacologic therapy.
7. For the Non-Classic NCSE [or "NCSE in coma (or in sick patients)"], if there was an earlier convulsion or definite clinical seizure, lean toward aggressive treatment if early ASDs fail.
8. Beyond the type of SE, consider also the etiology, EEG pattern (discharge frequency, "seizure burden" etc.), likely side effects of medications and especially of the prolonged ICU course; plus age, social (family) setting, medical comorbidities, and overall prognosis.
9. In all cases, treat vigorously, following clinically, and on EEG.

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https://doi.org/10.1007/s00415-022-11368-5

REVIEW

Nonconvulsive status epilepticus following cardiac arrest: overlooked, untreated and misjudged

Pia De Stefano[1,2] · Peter W. Kaplan[3] · Hervé Quintard[1] · Margitta Seeck[2] · Raoul Sutter[4,5,6,7]

Table 1 Studies describing or indirectly reporting the emergence and frequency of NCSE within the first hours after ROSC

Number of patients	Total SE		Outcome of total SE	NCSE			Sedation and TTM	Outcome of NCSE	EEG type	Paper	Study type
	N	%		*N*	% of post-CA patients	% of SE patients					
Studies directly reporting on NCSE											
74 (prosp. cohort)	28	38%	1 CPC 2 2 CPC 2–3 25 CPC 5	4 (described)	5%	14%	Sedation TTM (33 °C)	1 CPC 2 1 MCS 2 CPC 5	s-EEG	Rossetti et al. [9]	Prospective and Retrospective
101	33	33%	1 CPC 4 32 CPC 5	12 (described)	12%	36%	Sedation TTM (33 °C)	1 CPC 4 11 CPC 5	c-EEG	Rittenberger et al. [32]	Retrospective
60	17	28%	7 CPC < 4 10 CPC ≥ 4	12 (described)	20%	70%	Sedation TTM	4 CPC ≤ 3 8 CPC ≥ 4	s-EEG	Lettieri et al. [33]	Retrospective
38	7	18%	1 CPC 3 1 CPC 4 5 CPC 5	2 (described)	5%	29%	Sedation TTM (33 °C)	2 CPC 5	c-EEG	Mani et al. [34]	Retrospective
25	NA	NA	NA	3 (described)	12%	NA	NA	NA	c-EEG	Claassen et al. [35]	Retrospective
Studies with information that allow indirect conclusions to be drawn regarding NCSE											
106	33	31%	2 CPC 1–2 31 CPC 3–5	4 (inferred*)	4%	12%	Sedation TTM (32–34 °C)	1 CPC 1–2 3 CPC 3–5	s-EEG and c-EEG	Legriel et al. [37]	Prospective observational
95	26	27%	1 CPC 2 1 CPC 3 24 CPC 5	1 (inferred*)	1%	4%	Sedation TTM (33 °C)	1 CPC 2	c-EEG	Rundgren et al. [11]	Prospective observational
51	5	10%	5 CPC 5	1 (inferred*)	2%	20%	Sedation TTM (32–34 °C)	1 CPC 5	s-EEG	Legriel et al. [38]	Prospective observational
127	41	32%	1 CPC 1 2 CPC 2 1 CPC 3 37 CPC 5	5 (inferred*)	3%	12%	Sedation TTM (33 or 36 °C)	2 CPC ≤ 3 3 CPC 5	c-EEG	Backman et al. [36]	Retrospective
288	47	16%	10 CPC 1–2 37 CPC 3–5	14 (inferred*)	5%	30%	Sedation TTM (33 °C)	9 CPC 1–2 5 CPC 3–5	c-EEG	Ruijter et al. [19]	Retrospective
127	41	32%	1 CPC 1 2 CPC 2 1 CPC 3 37 CPC 5	6 (inferred*)	5%	15%	Sedation TTM (33 or 36 °C)	2 CPC ≤ 3 4 CPC 5	c-EEG	Dragancea et al. [14]	Retrospective

CA cardiac arrest, *SE* status epilepticus, *NCSE* non-convulsive status epilepticus, *N* number, *NA* not available, *CPC* cerebral performance category, *MCS* minimally conscious state, *TTM* target temperature management, *c-EEG* continuous EEG, *s-EEG* standard EEG

*Number of patients with presumed NCSE as no motor symptoms were described

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Neurological outcome of postanoxic refractory status epilepticus after aggressive treatment

Simone Beretta [a,*], Anna Coppo [b], Elisa Bianchi [c], Clara Zanchi [a], Davide Carone [a], Andrea Stabile [a], Giada Padovano [a], Endrit Sulmina [b], Alice Grassi [b], Graziella Bogliun [a], Giuseppe Foti [b], Carlo Ferrarese [a], Antonio Pesenti [d], Ettore Beghi [c], Leonello Avalli [b]

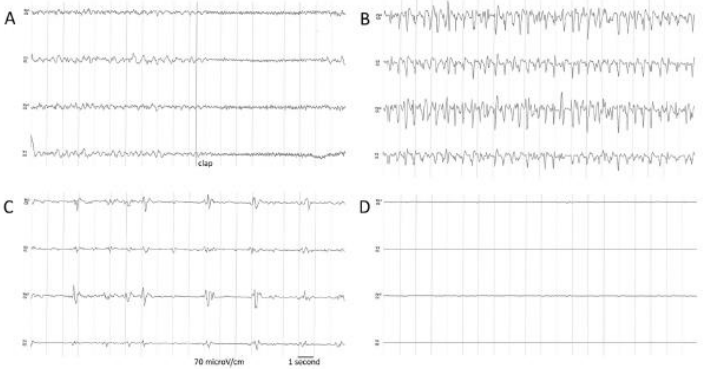
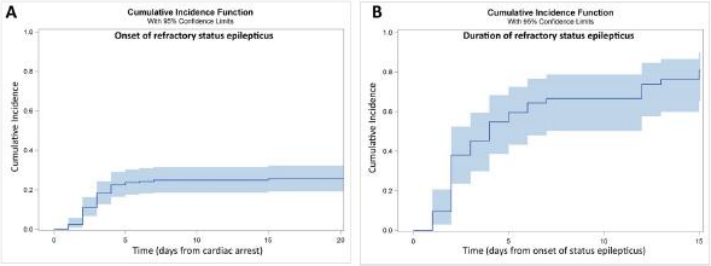
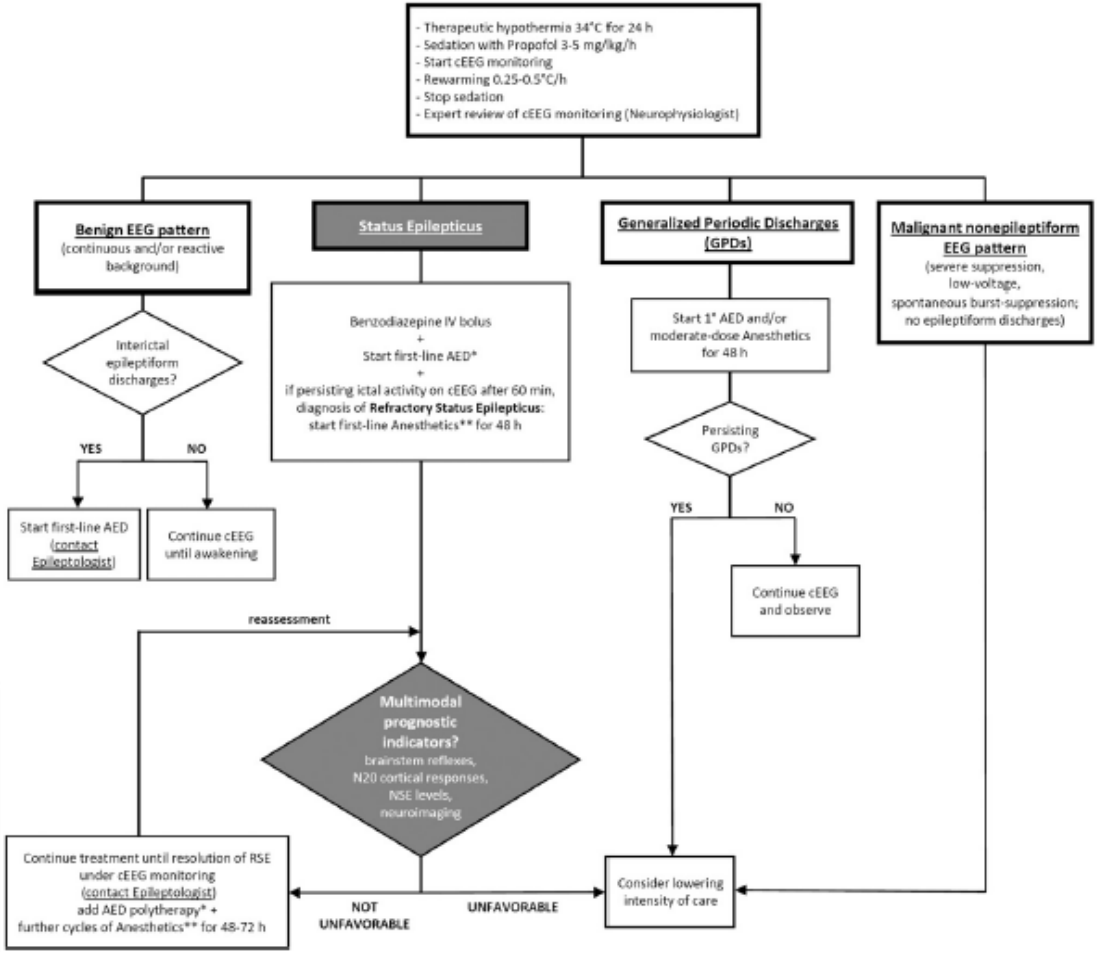


Fig. 2. Prognostic EEG patterns in postcardiac arrest patients. Representative epochs from 4-channel continuous EEG monitoring of patients with benign EEG pattern (A), RSE pattern (B), GPDs pattern (C), and malignant nonepileptiform EEG pattern (D).





ABSENCE STATUS EPILEPTICUS

Contents lists available at ScienceDirect

Seizure: European Journal of Epilepsy

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

Short communication

De novo Absence Status Epilepticus in a pediatric cohort: Electroclinical pattern in a multicenter Italian patients cohort

C. Pepi[a,b,*], E. Cesaroni[c], P. Striano[d], D. Maiorani[e], D. Pruna[f], S. Cossu[f], M. Di Capua[g], F. Vigevano[a], N. Specchio[a], R. Cusmai[g]

This table summarizes the electroclinical and pharmacologic features of our patients.

Patient No. (sex)	Age at last FU (ys)	Follow up (ys)	Age at AS (ys)	Family History	Previous seizures	EEG	AS	Imaging	Comorbility	Genetic Exam	Acute AED	Chronic AED
1 (F)	8	1	7	cousin with epilepsy	1 FS	Ictal AS	3 AS	CT, MRI	no	karyotype aCGH	MDZ 3 mg IV	LEV > VPA
2 (F)	9	2	7		no	Ictal AS	1 AS	CT, MRI	no	karyotype aCGH	DZP 10 mg ER	VPA (withdrawn)
3 (M)	16	7	9	aunt with epilepsy	no	Ictal AS	4 AS	CT	no	karyotype aCGH NGS	MDZ 3 mg IV	VPA (withdrawn)
4 (M)	15	4	11		no		2 AS	MRI	no	karyotype aCGH	MDZ 2 mg IV	VPA (withdrawn)
5 (F)	9	2	9		no	Ictal AS	1 AS	CT	no	WHE	MDZ 10 mg buccal	ETS
6 (F)	9	2	9		no	Ictal AS	1 AS	CT	no	WHE	MDZ 2 mg IV	ETS
7 (M)	13	4	9	grandfather and cousin with epilepsy	no		1 AS	CT, MRI	no	karyotype aCGH	MDZ 2 mg IV	VPA (withdrawn)
8 (F)	11	1	11	aunt with epilepsy and ID	no		1 AS	CT, MRI	no	NGS		ETS
9 (M)	21	13	8		1 FS	Ictal AS	1 AS	MRI	no	WHE	MDZ 2 mg IV	VPA (withdrawn)
10 (M)	14	5	9	cousin with FS	no		3 AS	no	no			VPA
11 (F)	22	14	9	cousin with FS	no	Ictal AS	1 AS	no	no		LZP 4 mg IV	VPA (withdrawn)
12 (F)	22	7	16	no	no	Ictal AS	2 AS	MRI	hyperandrogenism	karyotype aCGH	DZP 10 mg ER	LEV > VPA
13 (M)	16	7	9	no	no		1 AS	MRI	no			VPA

FS: Febrile Seizure; AS: Absence Status; WHE: Whole Exome Sequencing; MDZ: Midazolam; DZP: Diazepam; LZP: Lorazepam; IV: intravenous; ER: endorectal.

Seizure: European Journal of Epilepsy 73 (2019) 79–82

Neuropsychiatric Disease and Treatment

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ORIGINAL RESEARCH

Status epilepticus in patients with genetic (idiopathic) generalized epilepsy

This article was published in the following Dove Press journal:
Neuropsychiatric Disease and Treatment

Magdalena Bosak[1]
Dominika Pawełczak[2]
Agnieszka Słowik[1]

[1]Department of Neurology, Jagiellonian University Medical College, Krakow, Poland; [2]Department of Neurology, Jagiellonian University Medical College, Krakow, Poland

Aim of the study: Genetic (idiopathic) generalized epilepsies (GGEs) account for nearly one-third of all epilepsies. The frequency of status epilepticus (SE) in patients with GGEs has been poorly studied. Therefore, this study aimed to evaluate the frequency of different forms of SE in a cohort of patients with GGEs.

Materials and methods: Among 153 patients with GGEs treated at the university epilepsy clinic in the period between 1998 and 2018, those with SE were retrospectively identified.

Results: Absence SE was diagnosed in 8 patients (13 episodes), while myoclonic SE was found in 2 patients (2 episodes). No cases of tonic–clonic SE were detected in the study cohort. Most SE episodes were found to be provoked by ill-advised antiepileptic drugs or changes in drug regimen. In all the subjects, SE was stopped by intravenous administration of diazepam and/or valproate. Long-term outcome of epilepsy was good, with most patients (70%) being seizure-free.

Conclusion: Status epilepticus is not a rare phenomenon in patients with genetic generalized epilepsies, with absence SE being the most common type. Most cases of SE are provoked by ill-advised AEDs or changes in drug regimen. Status epilepticus in GGEs can be easily treated with benozdiazepines and/or valproate. Status epilepticus in GGEs can be easily treated with benozdiazepines and/or valproate.

Keywords: absence status epilepticus, myoclonic status epilepticus, genetic generalized epilepsy

Case (sex, age)	GGE syndrome (age of onset in years, type or first seizure)	Type of SE (approximate duration)	Cause of SE	AEDs treatment at SE	Treatment of SE	Outcome and follow-up (years)
1.M, 39	JAE (11, AS)	Absence status epilepticus (24 hrs)	Relapse after seziure-free period off medication	None	Diazepam iv	Seizure-free on VPA 1000 mg (4)
2.M, 66	JAE (11, AS)	Absence status epilepticus (6 hrs)	VPA tapering due to side effects	VPA 300 mg/ LTG 300 mg	Diazepam iv, VPA iv	Seizure-free on VPA 1000 mg, LTG 200 mg (2)
3.F, 30	JAE (15, AS)	Absence status epilepticus (5 hrs)	Ill-advised AEDs	TGB 30 mg/ CBZ 800 mg	Diazepam iv	Seizure-free on VPA 600 mg (10)
4.F, 34	GTCSA (17, TCS)	Absence status epilepticus (8 hrs)	Ill-advised AEDs	CBZ 1600 mg/ VGB 2000 mg	VPA iv	Seizure-free on VPA 2000 mg (3)
5.F, 70	JAE (16, AS)	Absence status epilepticus (12 hrs)	AEDs missed for 3 days	LTG 300 mg	Diazepam iv, VPA iv	LTG 300 mg/VPA 600 mg at discharge, lost to follow-up
6.F, 42	JAE (17, AS)	Absence status epilepticus (48 hrs)	Ill-advised AEDs	CBZ 1600 mg/ GBP 3600 mg/ TPM 800 mg	Diazepam, iv, VPA iv	Seizure-free on VPA600 mg/LTG 400 mg (4)
7. F, 31	JAE (11, AS)	Absence status epilepticus – 5 episodes (30–180 mins)	1 episode related to infection with fever	LTG 200 mg	Diazepam iv	Infrequent AS on LTG 500 mg (1)
8. F, 28 at first episode. 35 at second episode	JAE (15, AS)	Absence status epilepticus First episode (7 hrs) Second episode (60 mins)	Before diagnosis, sleep deprivation Adding hormonal contraception to LTG	None LTG 100 mg	Diazepam iv Diazepam iv	Infrequent AS on LTG 200 mg (4)
9.F, 26	JME (TCS, 21)	Myoclonic status epilepticus (3 hrs)	Ill-advised AEDs	GBP 3600 mg/ VGB 1000 mg	Diazepam iv	Seizure-free on VPA 600 mg/LEV 2000 mg (16)
10. F, 20	JME (MS, 14)	Myoclonic status epilepticus (2 hrs)	Sleep deprivation, alcohol	None	Diazepam iv	Seizure-free on LEV 1000 mg (6)

Abbreviations: SE, status epilepticus; JAE, juvenile absence epilepsy; JME, juvenile myoclonic epilepsy; GTCSA, generalized tonic–clonic seizures alone; AEDs, antiepeilptic drugs; AS, absence seizures; MS, myoclonic seizures; TCS, tonic-clonic seizures; CBZ, carbamazepine; GBP, gabapentin; LTG, lamotrigine; TGB, tiagabine; TPM, topiramate; VGB, vigabatrin; VPA, valproate; iv, intravenous.

RESIDENT & FELLOW SECTION

Section Editor
John J. Millichap, MD

Teaching NeuroImages: De novo absence status epilepticus in an adult

Proleta Datta, MD, PhD
Omotola Hope, MD
Giridhar P. Kalamangalam, MD, DPhil

Correspondence to Dr. Datta: proleta.datta@uth.tmc.edu

A 52-year-old man was found wandering at night. He was oriented to name only with no other neurologic signs. Laboratory work, CSF analysis, and brain MRI were unremarkable. EEG (figure) showed profuse generalized spike wave activity. His symptoms and EEG normalized with levetiracetam. On recovery, the patient denied previous seizures but admitted to a benzodiazepine habit.

De novo absence status epilepticus is a rare form of nonconvulsive status epilepticus typically encountered in older adults without prior epilepsy in the setting of benzodiazepine withdrawal, metabolic derangement, or alcoholism.[1] Treatment with anticonvulsants used in the idiopathic generalized epilepsies is effective.[2]

Download teaching slides: Neurology.org

AUTHOR CONTRIBUTIONS

Dr. Datta contributed to concept, analysis and interpretation, and drafting/revising of the manuscript. Dr. Hope contributed to concept, analysis and interpretation, and drafting/revising of the manuscript. Dr. Kalamangalam contributed to concept, analysis and interpretation, and drafting/revising of the manuscript.

STUDY FUNDING

No targeted funding reported.

DISCLOSURE

The authors report no disclosures relevant to the manuscript. Go to Neurology.org for full disclosures.

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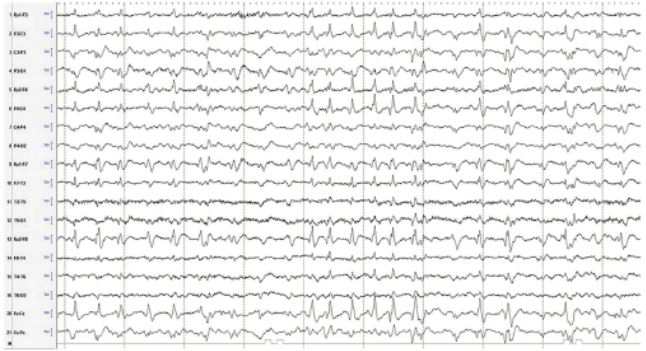
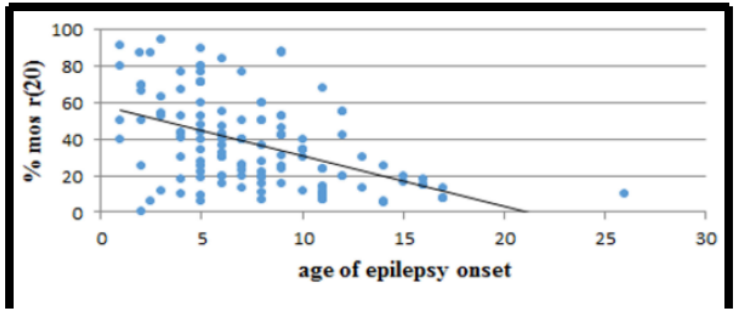
Figure Absence status epilepticus in a 52-year-old man with confusion



Frequent bursts of generalized spike-wave activity superimposed on a slow background (10-second EEG epoch, bipolar longitudinal montage, gain 10 μV/mm).

Ring Chromosome 20 Syndrome: Genetics, Clinical Characteristics, and Overlapping Phenotypes

Angela Peron[1,2,3], Ilaria Catusi[4], Maria Paola Recalcati[4], Luciano Calzari[5], Lidia Larizza[4], Aglaia Vignoli[2] and Maria Paola Canevini[2]*



Analysis extended up to 200 metaphases to detect low-level mosaicism

Core phenotype	• Refractory seizures and frequent non-convulsive status epilepticus (NCSE) are the most common seizure types • Cognitive decline following seizure onset in a previously normally developing child is frequent • Terrific hallucinations are frequent • Growth is usually normal, and dysmorphisms and congenital malformations are uncommon
Inheritance	r(20) occurs sporadically in most patients, but mosaic-to-mosaic transmission has been reported
Diagnosis	Karyotype with high number of metaphase count is the gold standard for diagnosis
Miscellaneous	• Constitutional non-supernumerary r(20) can be mosaic (more frequently) or non-mosaic • In mosaic r(20) the percentage of cells containing the ring chromosome inversely correlates with the age of seizure onset

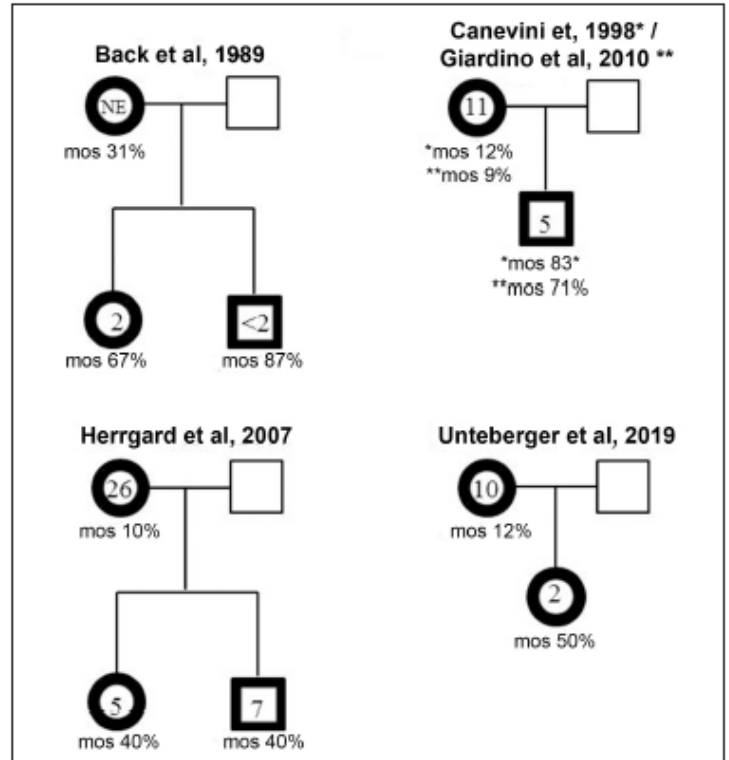


FIGURE 4 | Mosaic-to mosaic maternal transmission of r(20) in four reported family trees. The age of epilepsy onset and the percentage of r(20) mosaicism are indicated within and below the affected individuals (bold framed symbols).

All the patients with a confirmed cytogenetic diagnosis of r(20) syndrome had a triad of signs and symptoms (drug-resistant frontal lobe seizures, recurrent NCSE, and typical EEG), giving this electro-clinical triad a high sensitivity and negative predictive value (100%). The differential diagnosis might be challenging especially with: (1) Frontal Lobe Seizures; (2) Rolandic Epilepsy treated with sodium channel blockers (NCSE during wakefulness); and (3) Lennox-Gastaut syndrome (LGS).

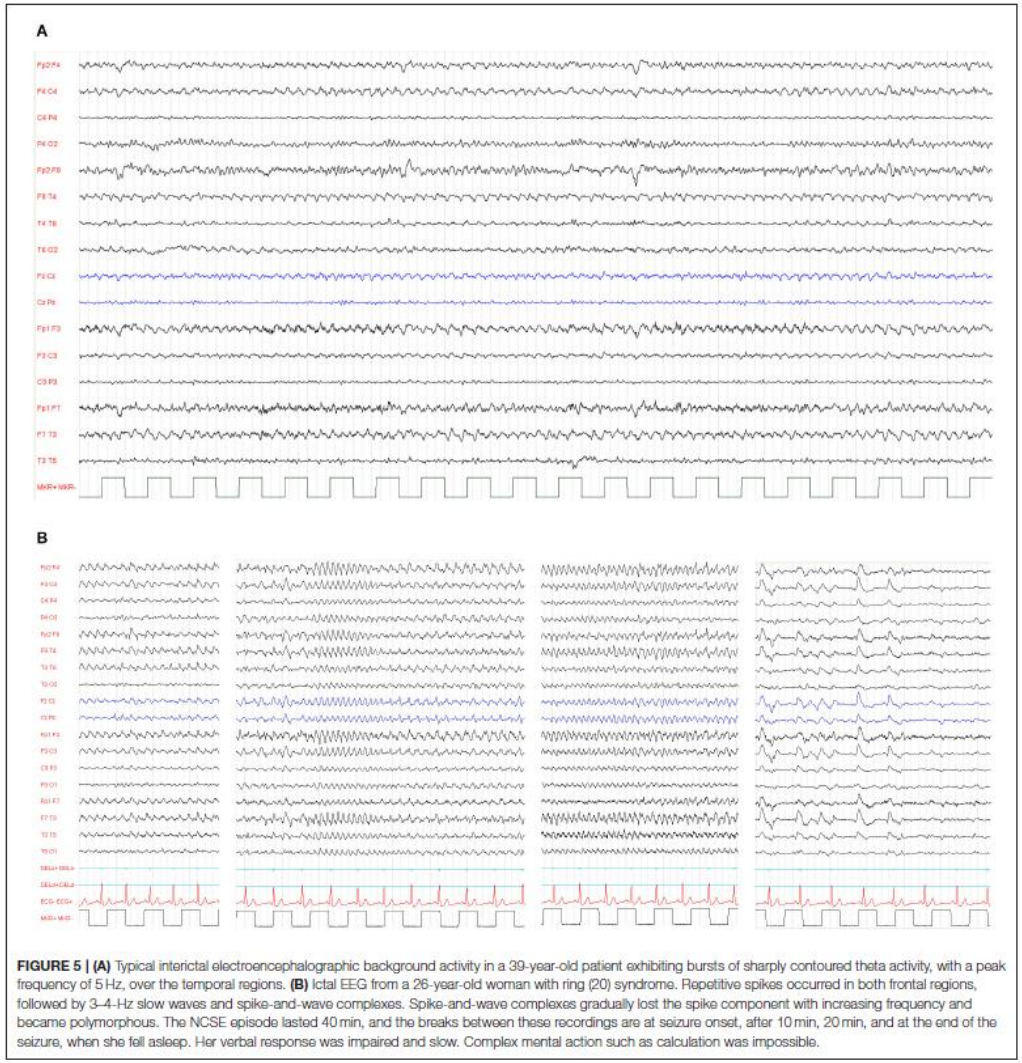


FIGURE 5 | (A) Typical interictal electroencephalographic background activity in a 39-year-old patient exhibiting bursts of sharply contoured theta activity, with a peak frequency of 5 Hz, over the temporal regions. **(B)** Ictal EEG from a 26-year-old woman with ring (20) syndrome. Repetitive spikes occurred in both frontal regions, followed by 3–4-Hz slow waves and spike-and-wave complexes. Spike-and-wave complexes gradually lost the spike component with increasing frequency and became polymorphous. The NCSE episode lasted 40 min, and the breaks between these recordings are at seizure onset, after 10 min, 20 min, and at the end of the seizure, when she fell asleep. Her verbal response was impaired and slow. Complex mental action such as calculation was impossible.

Epilepsy & Behavior 80 (2018) 215–220

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

Specificity of electroclinical features in the diagnosis of ring chromosome 20

A.B. Gago-Veiga [a], R. Toledano [b], I. García-Morales [b], M.A. Pérez-Jiménez [c], J. Bernar [d], A. Gil-Nagel [b,*]

Patients with r(20) experience very frequent NCSE, which can present even daily. The clinical semiology during NCSE consists of altered state of vigilance, staring, loss of emotional facial expression, reduced spontaneous motor activity and speech production, with a slow response to questions. Associated motor symptoms, such as myoclonus, tonic posturing, oral automatisms, and frightened facial expression have been reported

Original article

Epileptic Disord 2000; 10 (4): 254-9

Refractory and lethal status epilepticus in a patient with ring chromosome 20 syndrome

Julia Jacobs[1], Geneviève Bernard[2], Eva Andermann[3], François Dubeau[1], Frederick Andermann[1,2]

The patient experienced up to 30 seizures a day there being four recognizable types: 1) prolonged atypical absences; 2) tonic posturing of the arms, extension of the neck and head turning to the left; 3) nocturnal hypermotor seizures with agitation; and 4) focal seizures with terror, visual hallucinations and impaired consciousness.

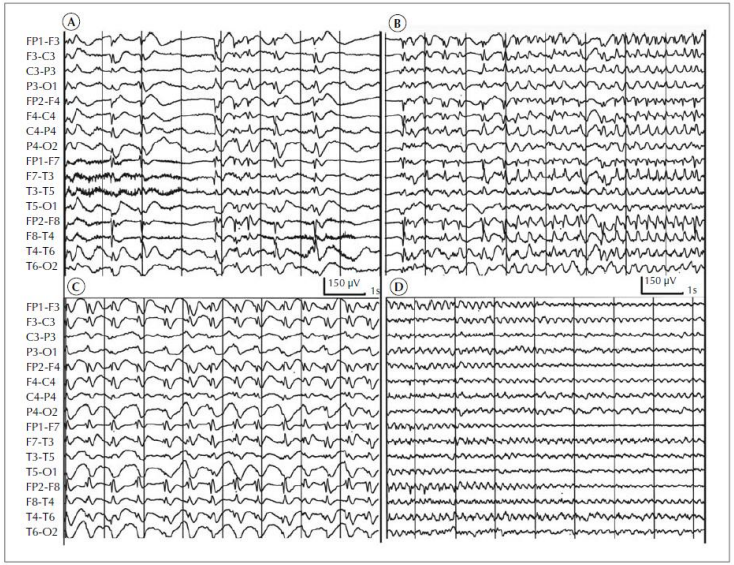
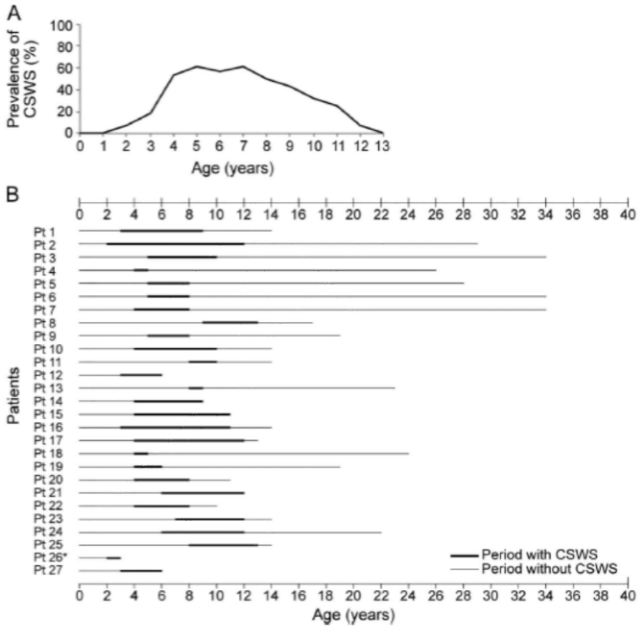


Figure 2. Variable EEG patterns during non-convulsive status epilepticus. Despite intravenous medication with propofol, phenobarbital, midazolam, and induction of coma with thiopental and pentobarbital, the SE could not be controlled.

BOUNDARY CONDITIONS

The syndrome of polymicrogyria, thalamic hypoplasia, and epilepsy with CSWS
Emanuele Bartolini, Melania Falchi, Francesco Zellini, et al.
Neurology published online March 4, 2016
DOI 10.1212/WNL.0000000000002526

Figure 2 Cumulative prevalence of continuous spikes and waves during sleep (CSWS)



CSWS syndrome may develop from heterogeneous etiologies. Children with initially typical ‘rolandic’ seizures may progress towards “mixed form of atypical evolutions” whose EEG hallmark is CSWS.

European Journal of Paediatric Neurology 30 (2021) 121–127

Contents lists available at ScienceDirect

European Journal of Paediatric Neurology

ELSEVIER

Clinical spectrum and treatment outcome of 95 children with continuous spikes and waves during sleep (CSWS)

Benedikt Sonnek [a], Jan Henje Döring [a], Ulrike Mütze [b], Susanne Schubert-Bast [c], Thomas Bast [d], Doreen Balke [a], Gitta Reuner [b, e], Elisabeth Schuler [b], Annick Klabunde-Cherwon [a], Georg F. Hoffmann [a, b], Stefan Kölker [b], Steffen Syrbe [a, *]

Results: Ninety-five children were included with a median age at diagnosis of 5.4 years. A structural/metabolic aetiology was found in 43.2%, genetic alterations in 17.9%, while it remained unknown in 38.9%. The proportion of patients with genetic aetiology increased from 10.3% (1998–2007) to 22.8% (2008–2018). On average, each patient received 5 different treatments. CSWS was refractory in >70% of cases, steroids and neurosurgery were most effective. No difference was observed between children with CSWS or Near-CSWS (Spike-Wave-Index 40–85%).

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ABSTRACT

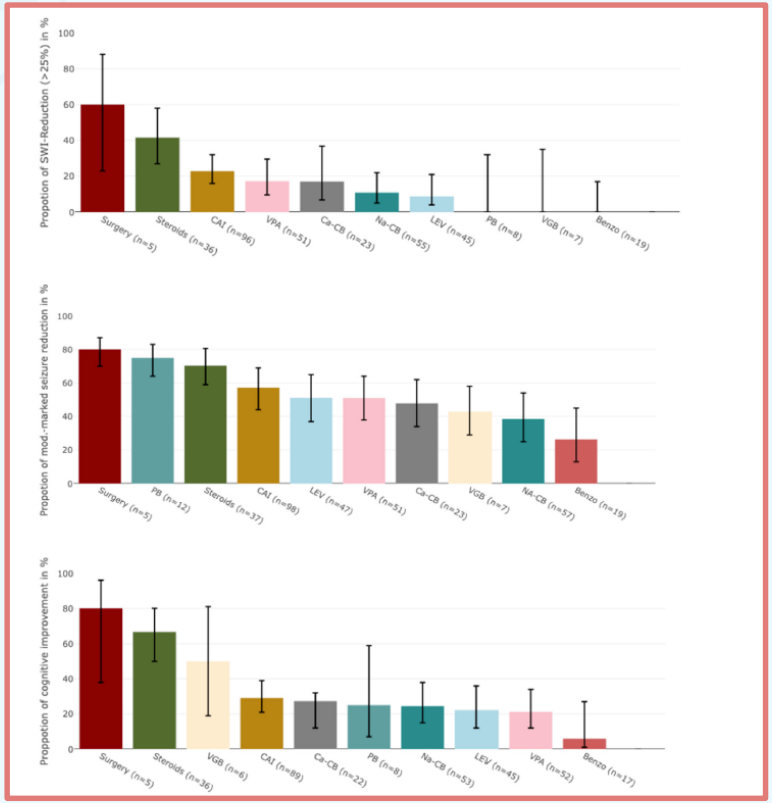
Objective: Continuous spikes and waves during sleep (CSWS) is an epileptic encephalopathy characterized by generalised epileptiform activity and neurocognitive dysfunction. Causes and outcome are diverse and treatment is mainly empirical.

Methods: Retrospective descriptive analysis of clinical and EEG data of children with CSWS diagnosed between 1998 and 2018 at the University Hospital Heidelberg.

Results: Ninety-five children were included with a median age at diagnosis of 5.4 years. A structural/metabolic aetiology was found in 43.2%, genetic alterations in 17.9%, while it remained unknown in 38.9%. The proportion of patients with genetic aetiology increased from 10.3% (1998–2007) to 22.8% (2008–2018). On average, each patient received 5 different treatments. CSWS was refractory in >70% of cases, steroids and neurosurgery were most effective. No difference was observed between children with CSWS or Near-CSWS (Spike-Wave-Index 40–85%).

Conclusions: Our cohort confirms CSWS as an age-dependent epileptic encephalopathy. Structural brain abnormalities were most frequent, but genetic causes are increasingly identified. More specific criteria for the diagnosis and treatment goals should be elaborated and implemented based on evidence.

Significance: This study is the largest monocentric observational study on treatment effects in children with CSWS, providing data for diagnostic and therapeutic decisions.



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