Classification of Nonconvulsive Status Epilepticus (NCSE)

Stephan Rüegg,
Division of Clinical Neurophysiology,
Department of Neurology,
University Hospital Basel

Abbreviations:

AC:	aura continua
AS:	absence status
BZD:	benzodiazepines
BSMEI:	borderline severe myoclonic epilepsy of infancy
CISE:	critical illness status epilepticus
CPSE:	complex-partial status epilepticus
CT:	computer tomography
DSE:	dyscognitive status epilepticus
DWI:	diffusion-weighted imaging
EEG:	electroencephalography
ESES:	electric status epilepticus in slow-wave sleep
GCSE:	generalized convulsive status epilepticus
GPED:	generalized periodic epileptiform discharges
ICU:	intensive care unit
IGE:	idiopathic generalized epilepsy
ILAE:	International League Against Epilepsy
LEV:	levetiracetam
LTG:	lamotrigine
LZP:	lorazepam
MDL:	midazolam
MSE:	myoclonic status epilepticus
MRI:	magnetic resonance imaging
NCSE:	non-convulsive status epilepticus
PLED:	periodic lateralized epileptiform discharges
PSE:	postanoxic (myoclonic) status epilepticus
SE:	status epilepticus
SMEI:	severe myoclonic epilepsy in infancy
SSE:	subtle status epilepticus
TPW:	triphasic waves
VPA:	valproic acid

Summary

There is no widely accepted classification of nonconvulsive status epilepticus (NCSE) and any classification has its own strengths and flaws. The following article incorporates the two main proposals of the International League against Epilepsy (ILAE) and Shorvon for the classification of NCSE. In general, NCSE may be subdivided into the focal and (primary) generalized forms, and in those types occurring in critically ill patients with focal or generalized electroencephalographic patterns. In addition, there are age-specific forms of NCSE in the neonatal, early infancy and childhood periods. The generalized forms include typical and atypical absence status, de novo absence status of late onset and the myoclonic SE in idiopathic generalized epilepsies. The focal forms are split into those forms without impairment of consciousness, called "aura continua" (corresponding to the former "simple partial NCSE") and those with impaired consciousness, called "dyscognitive SE" (corresponding to the former "complex-partial" or "psychomotor" SE). The latter is further subdivided into the "mesial temporal" ("limbic") and the "neocortical" forms.

The neonatal, early infancy and childhood forms of NCSE are only briefly discussed and listed in a table. A concise electroclinical case vignette may illustrate the principal features of each type of NCSE in adults.

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Key words: non-convulsive status epilepticus, classification, absence status, aura continua dyscognitive status epilepticus, subtle status epilepticus, postanoxic myoclonic status epilepticus, electroencephalography

Classification du statut épileptique non convulsif

Il n'existe toujours pas de classification généralement acceptée du status epilepticus non convulsif (NCSE). Toute solution proposée jusqu'à ce jour présente ses avantages et ses inconvénients spécifiques. L'article ci-après se réfère à deux schémas actuels de classification du NCSE : celui de la Ligue Internationale contre l'Epilepsie (ILAE) et celui de Shorvon. De manière générale, on peut scinder le NCSE en formes focales et en formes (primaires) généralisées, plus les pathologies très lourdes avec un bilan encéphalographique focal ou généra-

lisé. A cela s'ajoutent encore les types de NCSE âge-dépendants tels que la période néonatale, la première enfance et l'enfance préadolescente. Les formes primaires généralisées englobent l'état d'absence typique et atypique, l'état d'absence « de novo » à début tardif, ainsi que le SE myoclonique dans le cadre des syndromes d'une épilepsie généralisée idiopathique. Les NCSE focaux sont subdivisés en formes sans perte de connaissance, celles à facultés cognitives restreintes, aujourd'hui « aura continua » (anciennement « NCSE partielle simple ») et en « mal épileptique dyscognitif » (autrefois appelé SE « partiel complexe « ou « psychomoteur »). Pour ce dernier état, la distinction se fait toujours entre les formes « mésio-temporales » (jadis aussi « limbiques ») et les formes « néo-corticales ». Les différents types de NCSE de la période néonatale, de la petite enfance et de la préadolescence ne sont mentionnés qu'en marge et inclus dans la synopsis. Une courte vignette électroclinique de cas représentant chaque forme importante de NCSE doit illustrer la discussion.

Mots clés : Statut épileptique non convulsif, état d'absence, aura continua, statut épileptique dyscognitif, statut épileptique « subtil », statut épileptique myoclonique post-anoxique, électroencéphalographie

Klassifikation des nicht-konvulsiven Status epilepticus

Zurzeit besteht nach wie vor keine allgemein akzeptierte Klassifikation des nicht-konvulsiven Status epilepticus (NCSE). Jeder bisherige Vorschlag weist seine spezifischen Vor- und Nachteile auf. Im nachfolgenden Artikel wird auf zwei aktuelle Klassifikations-Schemata für den NCSE, eines der Internationalen Liga gegen Epilepsie (ILAE) und dasjenige von Shorvon, zurückgegriffen. Generell kann der NCSE in die fokalen und (primär) generalisierten Formen unterteilt werden sowie zusätzlich in diejenigen, welche bei schwerst erkrankten Patienten mit fokalem oder generalisiertem elektroenzephalographischem Bild auftreten. Darüber hinaus bestehen altersgebundene Typen des NCSE in der Neugeborenenperiode, im Kleinkindalter sowie in der späteren Kindheit. Die primär generalisierten Formen umfassen den typischen sowie atypischen Absencenstatus, den „de novo“ spät beginnenden Absencenstatus sowie den myoklonischen SE im Rahmen der idiopathischen generalisierten Epilepsiesyndrome. Die fokalen NCSE werden unterteilt in diejenigen Formen mit erhaltenem und in diejenigen mit eingeschränktem Bewusstsein und heutzutage „aura continua“ („früher einfach-partieller NCSE“) beziehungsweise „dyskognitiver SE“ (früher „partiell-komplexer“ oder „psychomotorischer“ SE) genannt. Bei letzterem wird weiter zwischen den „mesial temporalen“ (früher auch „limbischen“) und den „neokortikalen“ Formen unterschieden. Die NCSE-Typen der Neonatalperiode, der Kleinkindphase und der späteren Kindheit werden nur gestreift und tabellarisch aufgeführt. Eine kurze elektroklinische Fall-Vignette zu jeder wichtigen NCSE-Form soll mithelfen, das Besprochene zu veranschaulichen.

Schlüsselwörter: Nicht-konvulsiver Status epilepticus, Klassifikation, Absence-Status, aura continua, dyskognitiver Status epilepticus, „subtle“ Status epilepticus, postanoxischer myoklonischer Status epilepticus, Elektroenzephalographie

Introduction

Any classification of a disorder relies on its definition. Nonconvulsive status epilepticus (NCSE) shares one of the most debated classifications because of the difficulty to define it. The most simple definition of NCSE may be the mathematical formula NCSE = all status epileptici (SE) minus the convulsive SE. Beyond the lack of convulsive signs and the therefore often intriguingly subtle or protean clinical manifestations of NCSE, NCSE essentially needs to be confirmed by electroencephalography (EEG). Thus, a definition of NCSE could read as proposed by Shorvon [1]: “nonconvulsive SE is a term used to denote a range of conditions in which electroencephalographic seizure activity is prolonged and results in nonconvulsive clinical symptoms“. This “range of conditions” was already evoked by Gastaut in 1962 when he stated “there may be as many forms of SE as seizure types exist” during the Xth Marseille Colloquium on Epilepsy [2]. Minimal duration of NCSE is another debated issue among epileptologists. While the former 30 minutes duration for SE was reasonably shortened to 5 minutes for generalized convulsive SE (GCSE) after the results of the large VA study on GCSE [3] and the proposal of Lowenstein [4], the minimal duration of NCSE was kept at a duration of 30 to 60 minutes, although Jordan proposed 15 to 30 minutes [5, 6]. The current Swiss guidelines for the treatment of SE insist on a duration of 5 minutes [7], since more than 90% of seizures stop after 3 minutes [8, 9] and those during longer than 10 minutes have only a minor tendency to stop spontaneously [10]. Also from a brain’s perspective, it does not make a difference for a neuron whether it is involved in convulsive or nonconvulsive activity: prolonged hyperexcitation of both of them carries the risk of neuronal damage or death.

The various manifestations of NCSE and its electroclinically blurred margins leave many doors open to borderline conditions (like the postanoxic (myclonic) SE, the critcal illness SE or the epileptic encephalopathies (discussed in this article) or even imitators of NCSE (discussed in the article of P. Thomas in this issue). The classification of NCSE can be based on age of manifestation [11], on localization and semiology, on focality, and on etiology; the latter may also be subdivided into the groups of symptomatic or idiopathic SE and into

Table 1: Types of nonconvulsive status epilepticus (NCSE) in adults:

focal:

with maintained consciousness:

aura continua:
- dysaesthetic
- painful
- epigastric
- fearful
- déjà-/ jamais-vu
- visual
- olfactory
- gustatory
- auditory
- pilomotor
- aphasic

with impaired consciousness:

dyscognitive SE:
mesial temporal:
- emotional (fear, anger)
- amnestic
- déjà-/ jamais-vu
- confusional
- olfactory
- gustatory
- epigastric
- psychotic

neocortical:
- dysaesthetic
- auditory
- visual
- aphasic
- pilomotor

primarily generalized:

- typical absence status
- atypical absence status
- de novo absence status of late onset
- myoclonic SE (in IGE)

in comatose patients (generalized & focal):

- postanoxic (myoclonic) SE
- subtle SE (after overt convulsive SE)
- critical illness SE

whether an epileptic disorder is already known or absent. Most classifications cannot stringently rely on solely one of these criteria and therefore they were almost always an intermingled conglomerate of all of these factors.

Classification

The most early classification used the terms "absence status" (AS) for generalized NCSE and "complex partial SE" (CPSE) for the focal forms of NCSE. The advent of clinical EEG [12] allowed for better differentiation of the clinically almost indistinguishable states of purely postictal alterations and non-convulsive status epilepticus (NCSE). The typical EEG picture of absence status (AS) was discovered by Lennox and co-workers [13]; Penfield and Jaspers reported the existence of continuous somato-sensory SE which they labelled "aura continua" [14]. Landolt emphasized the usefulness of EEG for the discrimination between AS and complex partial SE (CPSE) which often are clinically hardly to distinguish [15]. The Xth Marseille Colloquium on Epilepsy held in 1962 was the first conference devoted to classify the various forms of SE. This classification, however, submerged in a babylonic use of terms for both conditions given the countless manifestations of NCSE on the one hand and the clinically often very similar nature of AS and "classical, psychomotor" CPSE on the other hand; accordingly, there were more than 20 different expressions for this disorder [1]. This eventually resulted in complete confusion and substantial difficulties to perform (epidemiological) studies. The most recent proposal of an expert panel of the ILAE from 2006 slightly modified and enlarged the subtypes and incorporated them into their classification. They kept the term AS for primarily generalized NCSE, but correctly refined focal NCSE by the terms "aura continua" (AC) [14] for simple partial NCSE and "dyscognitive SE" (DSE) for CPSE properly [16] . However, more recent results of basic science and the progress of modern medicine led to the surge of new forms of NCSE, like the "subtle" SE (SSE) [17] or postanoxic myoclonic SE (PSE) and "critical illness" SE (CISE) [18, 19] which should be incorporated in contemporary classifications. Such a classification is presented in **table 1**. In addition, this article will only briefly touch on those types of NCSE which exclusively occur in the neonatal and early childhood period. The latter form a unique continuum of paroxysmal episodes of NCSE often developing into a more chronic encephalopathic state [20].

Table 2: Types of NCSE and epileptic encephalopathies in children (modified from [1])

Types of NCSE shared between children and adults:

primarily generalized:

typical absence status
myoclonic SE (in IGE)
NCSE in Lennox Gastaut syndrome:
- atypical absence status
- tonic SE

NCSE in other symptomatic encephalopathies or in patients with learning disabilities

focal:

with maintained consciousness:
- aura continua (s. also **table 1**)

with impaired consciousness:
dyscognitive SE (s. also **table 1**):
- mesial temporal
- neocortical

in comatose patients (generalized & focal):

postanoxic (myoclonic) SE
subtle SE (after overt convulsive SE)
critical illness SE

Types of NCSE of exclusively childhood:

NCSE of early-onset benign occipital epilepsy of childhood (Panayiotopoulos syndrome)
NCSE of specific childhood epileptic syndromes (often with genetic background and progressive):
- ring chromosome 20 syndrome
- Angelman syndrome
- Rett syndrome
- myoclonic-astatic epilepsy (Doose syndrome)
- Lafora's disease

Electric status epilepticus in slow-wave sleep (ESES)
Landau-Kleffner syndrome

Types of NCSE confined to the neonatal and early infancy period:

- neonatal SE
- Ohtahara syndrome
- West syndrome
- severe myclonic epilepsy of infancy (SMEI; Dravet syndrome); boundary/"benign" variant (BSMEI)
- NCSE in other forms of epilepsy during the neo natal and early infancy period

Absence status [AS]

Absence status may mainly occur in three different populations of patients. First and important, "typical" AS may develop in patients with an idiopathic generalized epilepsy (IGE) syndrome most frequently due to a medication error (dose-reduction, inadequate drug choice, etc.) [21]. "Atypical" AS occurs in patients with a cryptogenic or symptomatic (generalized) epilepsy syndrome, like in chromosomal disorders, non progressive encephalopathy, Lennox-Gastaut syndrome, etc. "De novo" AS is probably the rarest form of AS, most likely resulting from (un-)intentional benzodiazepine (BZD) withdrawal [22]. An impairment of consciousness, of concentration and of the ability to store and recall memory content is common to all these types of AS. The clinical signs may be susceptible only to the individual, but not to bystanders, to both of them or only to the bystanders, but not the patient itself (**s. case-1; figure 1**) and can impede the diagnosis if an EEG is not available. The EEG shows generalized spike-wave activity of 2.5 Hz or faster, usually no attenuation after the end of the generalized spike-wave activity, and occasionally focal single spike-wave activity. Interictal EEG tracings look out normally, but sometimes bi-temporal slowing may develop, especially in patients with seizures difficult to control. Atypical AS, as well as most forms of cryptogenic and symptomatic myclonic SE (MSE) often manifest as a continuum of clinical absences, intermingled with positive or negative myoclonic manifestations and subtle other (even focal) motor signs. Pure MSE in patients with IGE, especially with juvenile myoclonic epilepsy, is rare and has to be considered as convulsive SE [23, 24].

De novo AS starts in later life and more than two thirds result from BZD withdrawal (s. case-2; **figure 2**).

Aura continua (AC)

Aura continua confers to the focal forms of NCSE without changes of consciousness, and may be also considererd as the nonconvulsive forms of simple partial SE. The clinical manifestations of AC are dependent on the cerebral focal localization: those originating from the parietal lobe will display sensory symptoms (paresthesias, pain), those from occipital lobe visual phenomena, those of neocortical temporal lobe with auditory features, those from mesial temporal lobe with fear, epigastric, gustatory or olfactory sensations, and those from frontal lobe may imitate temporal lobe signs and symptoms, or pilomotor and other autonomic symptoms. The EEG shows continuous focal epileptiform discharges or – depending on the depth of origin of the epileptic activity – rhythmic delta activity (s. case-3; **figure 3**).

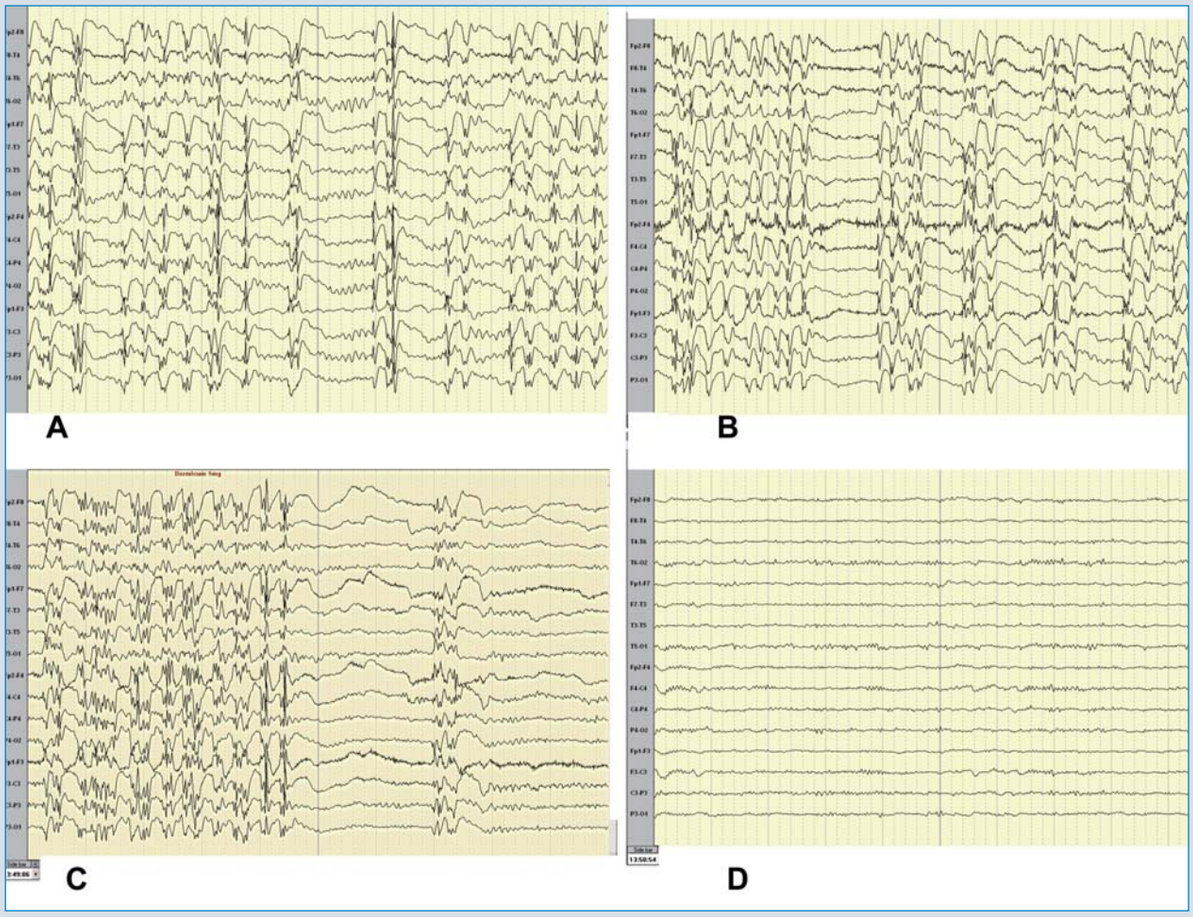


Figure 1: 47 year-old man with generalized tonic-clonic seizures and absences since age 17. Valproic acid (VPA) and primidone never completely controlled his seizures. He developed severe hyperammonemic encephalopathy and had to be switched to levetiracetam (LEV), lamotrigine (LTG), and topiramate. He then experienced several episodes of AS where he was walking around, but was confused. He could speak and responded to questions, but mimicked Ganser's syndromes in that his most answers were "near- correct" (October 17 instead of November 17, for example). The EEG showed almost permanent primary generalized (poly-)spike-wave discharges with short bouts of normal background activity (A). Absence status did not stop after i/v-administration of 8 mg of lorazepam (LZP), but the background activity became flattened and beta activity was increased (B). The subsequent i/v-administration of 1 mg of midazolam (MDL)(C) completely abolished the epileptic activity within 90 sec.(D).

Dyscognitive status (DSE)

Dyscognitive (formerly "psychomotor", "complex-partial") SE is further subdivided into the mesial temporal and the neocortical forms and includes all those forms of NCSE of focal origin with an alteration of consciousness. While limbic signs and symptoms (confusion, amnesia, fear, etc., "limbic" SE) dominate the clinical appearance of mesial DSE [25, 26], the neocortical forms of DSE may also display impairments of vision, language, hearing etc. [27, 28]. The EEG of mesial DSE shows either focal epileptiform discharges (s. case-4; **figure 4**) or prolonged rhythmic delta activity over the temporal regions, sometimes bilaterally.

Clinically, it may be often difficult if not impossible to differentiate AS from DSE, especially the confusional forms; therefore, the role of the EEG and sometimes imaging is crucial for yielding the correct diagnosis.

Subtle status epilepticus (SSE)

Subtle status epilepticus is neither a syndromal nor an etiological type of NCSE, but denotes the clinical situation where focal motor or GCSE apparently stops, but epileptiform discharges continue in the EEG and the patient does not regain consciousness or return to his pre-ictal state (case-5; **figure 5**). The concept of SSE was established by the pivotal animal studies of Treiman et al. where they identified a relatively strictly evolving sequence of electroclinical events during SE [17], later clinically corroborated by DeLorenzo [29].

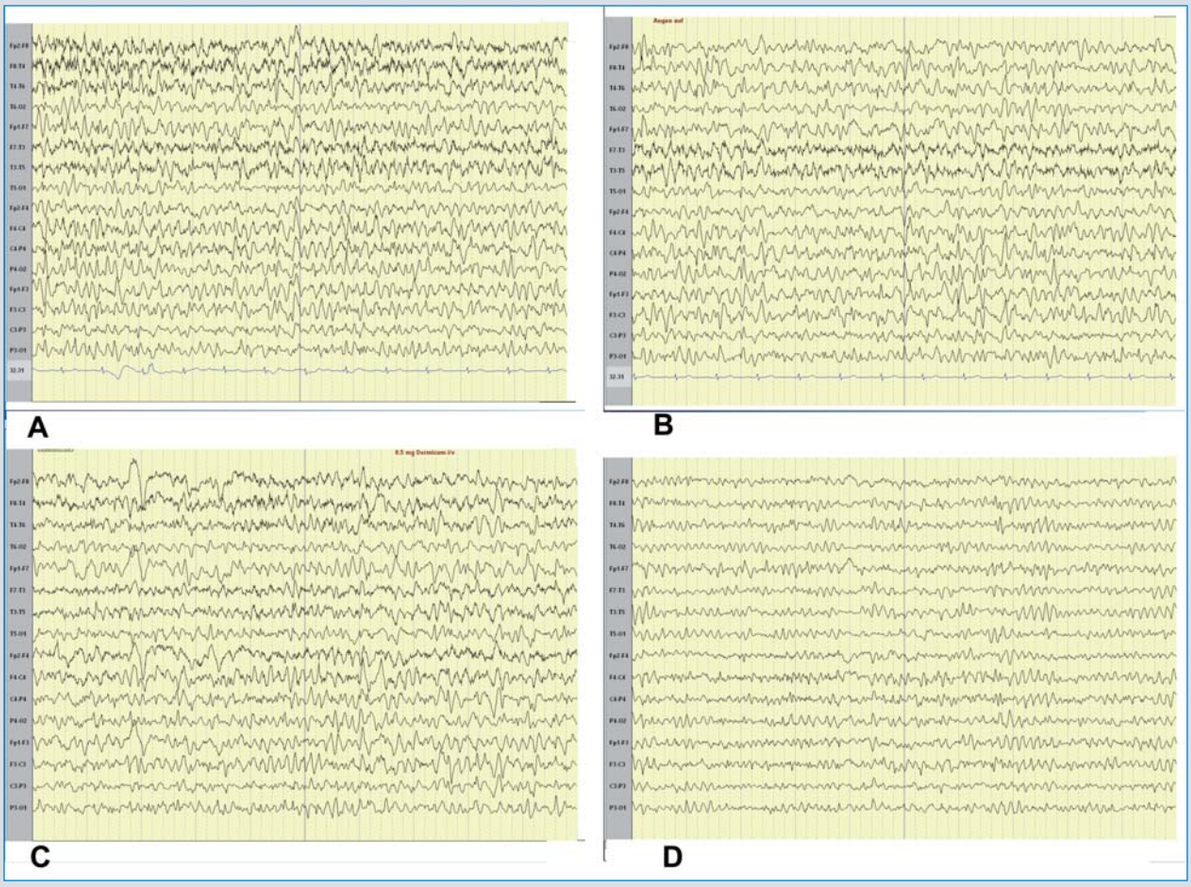


Figure 2: 84 year-old otherwise healthy woman who was found slightly confused in her apartment. A CT scan and the CSF were completely normal. Within 24 hours, she became comatose. The EEG showed diffuse, irregular, sharp-contured, high-amplitude theta- and delta activity, intermingled with multifocal sharp waves (A). This activity did not change upon eye opening (B). The i/v-administration of 0.5 mg MDL (C) markedly reduced the epileptic activity and led to an accelerated, more regular background activity within 90 seconds (D). The patient opened her eyes and briefly talked. Extensive work-up did not reveal another cause than BZD intake for insomnia and an involuntary stop of this medication a few days before admission because of medication run-out.

Postanoxic (myoclonic) status epilepticus (PSE)

Hypoxia after cardiac arrest or prolonged cardiopulmonary resuscitation, severe asthmatic crisis, carbon monoxide poisoning, and near-missed drowning damage the brain, especially the cortex, basal ganglia, and the mesolimbic system resulting in postanoxic encephalopathy. Coma and often bursts or prolonged phases of spontaneous or stimulus-sensitive myoclonus, either subtle (periorbicular, facial or truncal), mutlifocal or generalized may be present [30-32]. The EEG is characterized by almost always lacking background activity (suppression) and either periodic lateralized or generalized epileptiform discharges (case-6; **figure 6**) or bursts of focal or generalized (poly-)spike-wave discharges (cases-7&8; **figure 7 and 8**). The myocloni may be epileptic from cortical islets of malfunctioning cerebral cortex (especially when mutlifocal), they may be of reticular origin (especially when generalized or bilateral), whereby they can be epileptic too by retrograde volleys to the cortex, as well as they may represent a disinhibitive phenomenon of the reticular formation which seems to exert an important "gating" function with regard to seizure propagation [33]. Treatment of PSE is often disappointing and may influence only the EEG and probably reduce the frequency and intensity of myocloni, but not improve the patients comatose state. In addition, PSE has been shown to be an independent outcome predictor after cerebral anoxia in a retrospective study [34]. Some authors argue that PSE is not a form of SE, but the expression of a toxic-anoxic severest dysfunction of the brain.

Critical illness status epilepticus (CISE)

Life-threatening illness with multi-organ failure also influences brain functions and may provoke epileptic activity or unmask an otherwise not manifest propensity of the patient's brain to seize up to build up SE.

According to recent reports, this results mainly from proconvulsive inflammatory cytokines, like IL-1β and fever [35-38], but also from well known factors like hypotension, hypoxemia, and medications, like cefepime, carbopenems, etc.[19]. The EEG shows substantial alteration of background activity intermingled with epileptiform discharges and often also triphasic waves reflecting, for example, uremia and/or hepatic failure (case-9; **figure 9**); both, triphasic waves and epileptiform discharges may disappear upon administration of BZD [39].

Chronic static or progressive epileptic encephalopathies

During the neonatal period, SE exclusively occurs as NCSE, either in its neonatal specific form with a pleomorphic EEG signature (repetitive epileptic discharges of 10 seconds or more over at least one hour) [40] or some days to weeks later in the form of the Ohtahara syndrome, again with a typical EEG tracing (bursts with high-voltage slow-wave activity and multifocal spikes of 1-3 sec duration alternating with suppression of 3-5 sec) [41].

In childhood, NCSE may occur in the form of AS (typical and atypical), AC, DSE; and several progressive and non-progressive encephalopathies, (like ring chromosome 20-, West-, Rett-, Angelman-, Lennox-Gastaut-, and Landau-Kleffner syndrome, etc.), resulting from structural alterations and/or metabolic disturbancies of an often genetic background may be associated with NCSE, frequently lasting for days, if not weeks and months. Thus, classifications were built upon these various, often age-dependent, syndromic encephalopathies (**s. table 2**) [1, 11, 20, 42].

Epileptic encephalopathies developping in adults only are rather uncommon and can reflect a non- or slowly progressive underlying disorder. Due to the often insidious, slow onset and the non-convulsive clinical appearance, the highly epileptic EEG activity – formally SE or often close to it – is detected only when clinical suspicion raises the need for an EEG (case-10; **figure 10**). The electroclinical response to treatment in these patients is very slow and may need several months.

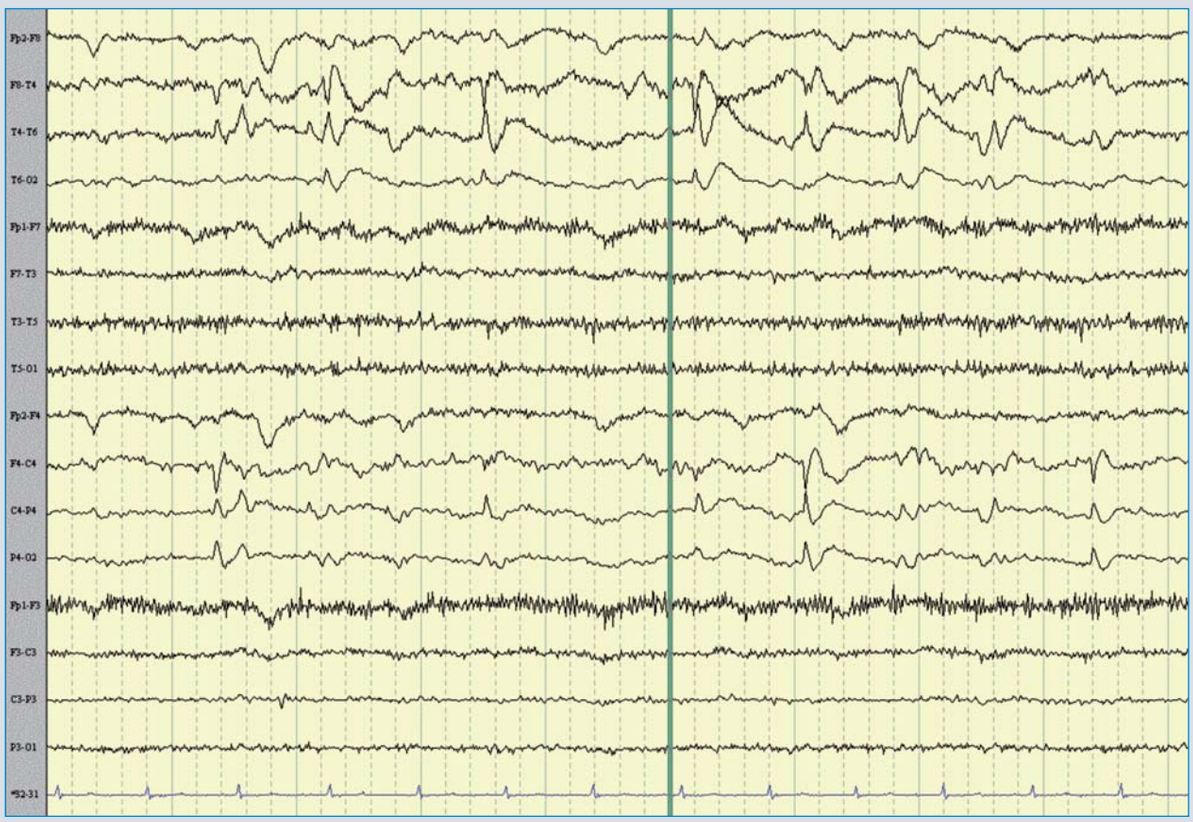


Figure 3: 52 year-old woman with a history of right-sided temporal, parietal and occipital bleeding due to an arteriovenous malformation three years ago. The patient then underwent embolisation of the malformation and experienced no seizures thereafter. Without preceding tonic-clonic movements, she experienced weakness of her left arm and face, but responded well to questions. MRI did not indicate recurrent bleeding, tumor or ischemia (diffusion-weighted imaging inclusively(DWI)). The EEG showed continuous pseudoperiodic right-sided epileptic discharges over the central and temporal regions. Symptoms completely resolved after control of the epileptic activity upon administration of i/v-MDL (inhibitory SE).

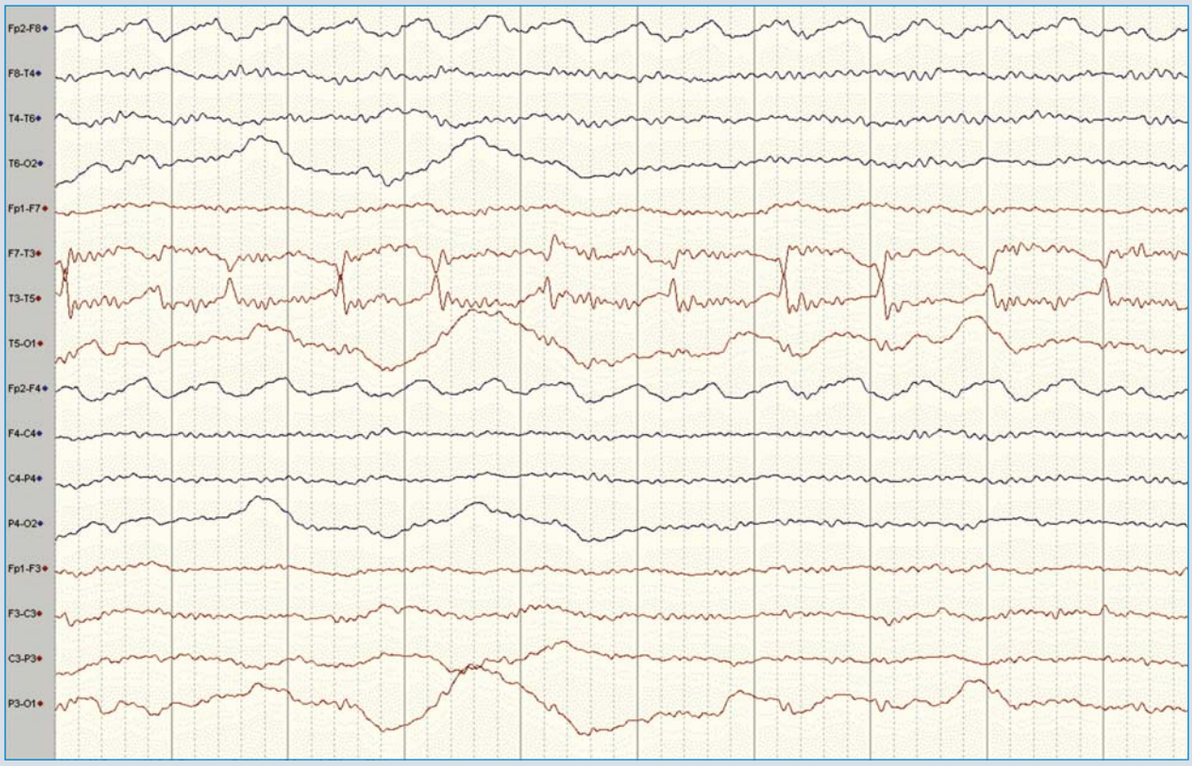


Figure 4: 84 year-old man with a long history of hypertension and depression. He suddenly stopped talking while an appointment with his family doctor. He had no right-sided hemiparesis, but seemed confused and apractic. Cerebral MRI revealed no acute lesion (DWI inclusively), but signs of cerebral microangiopathy; intracranial vessels were without stenosis or occlusion on MR-angiography. The EEG showed periodic lateralized epileptic discharges in the left frontal and temporal region which soon resolved upon the i/v-administration of 3 mg of MDL. The patient had amnesia for the period from entering the family doctor's office until the recovery on the intensive care unit (ICU).

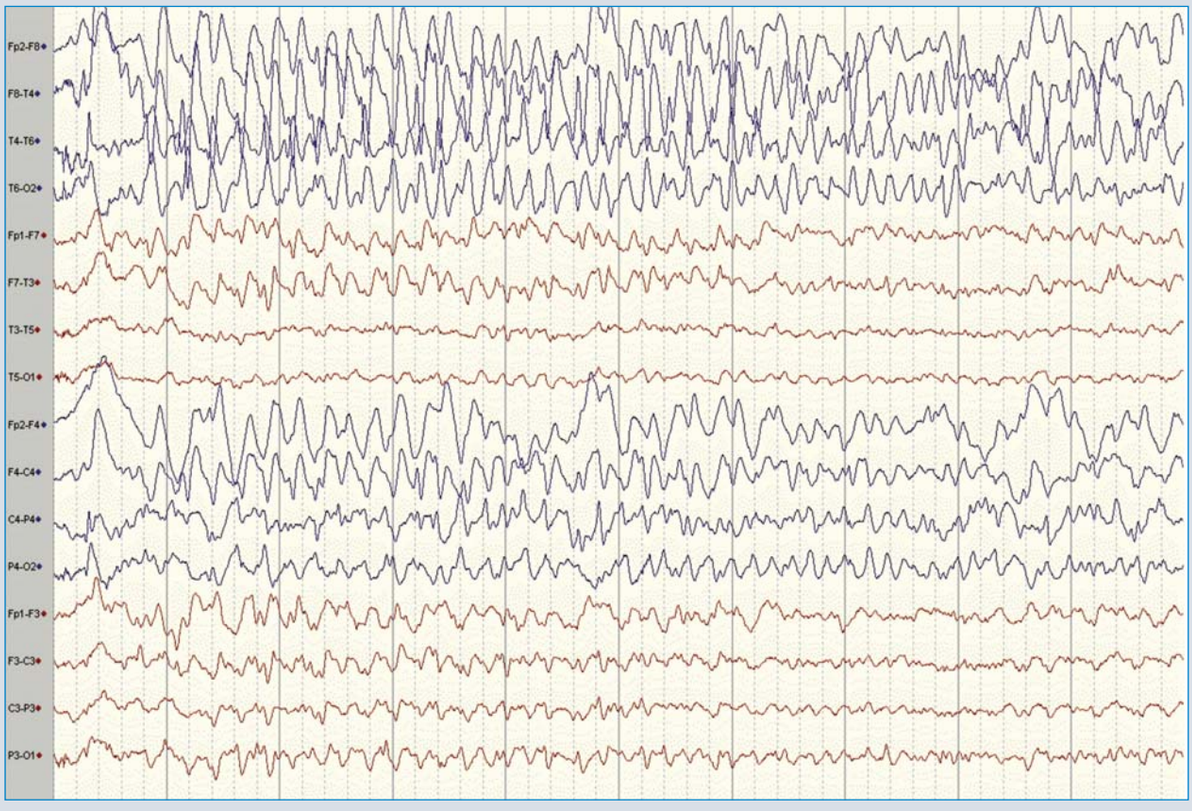


Figure 5: 52 year-old man with large meningeoma of the sphenoidal plane, operated four years ago. No postoperative seizures. On admission he had a series of focal motor seizures with swift secondary generalization. He remained comatose, had minimal perioral twitching, and was transferred to the ICU. The EEG showed continuous epileptic activity over almost the whole right hemisphere with spike-waves in the right frontal, central and temporal regions and propagation of rhythmic theta/delta activity to the left hemisphere. He subsequently experienced highly refractory subtle SE.

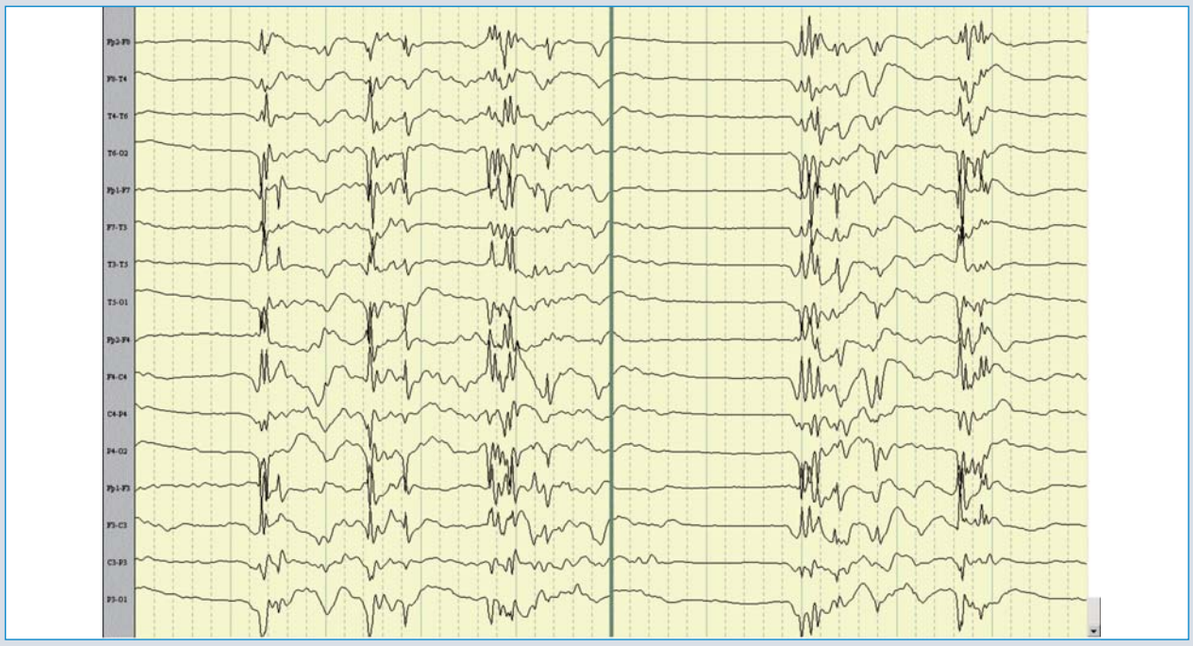


Figure 6:82 year-old man after prolonged cardiopulmonary resuscitation (CPR) following pulseless electrical activity. He experienced severe postanoxic encephalopathy and had recurrent bouts of generalized myocloni. The EEG showed almost absent/flat background activity, interrupted by bursts of generalized poly-spike wave activity, clinically manifesting as myocloni.

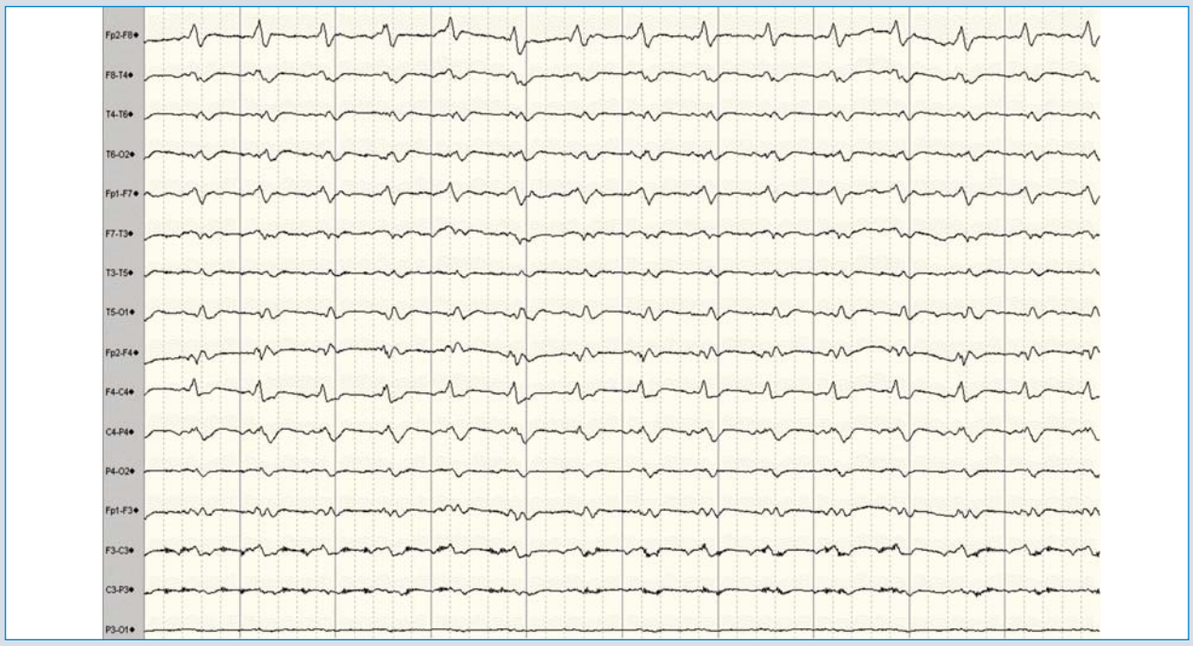


Figure 7:70 year-old man after successful outdoor CPR in the context of known severe coronary heart disease and ventricular fibrillation. Stenting and revascularization failed. He was treated by hypothermia for 24 hours, but remained comatose. The EEG 72 hours after stopping sedation showed generalized periodic epileptiform discharges (GPED) which were not responsive to external stimuli. The patient developed fulminant pneumonia and died from septic shock with multi-organ failure.

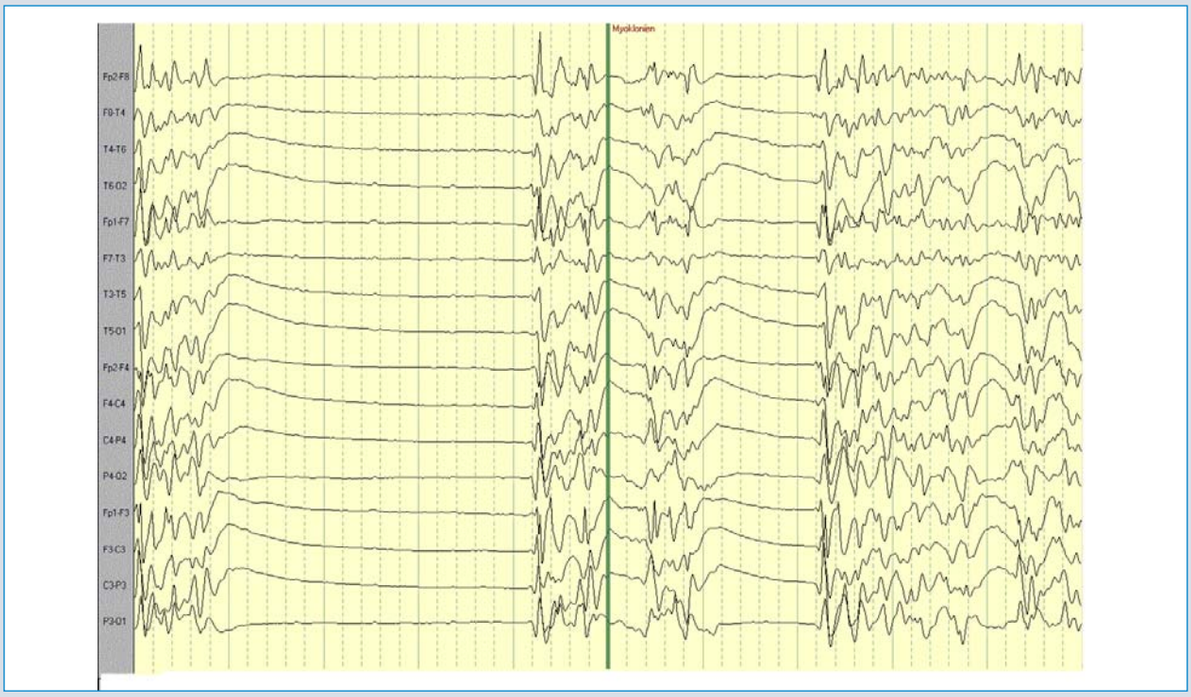


Figure 8:53 year-old woman with acute respiratory exhaustion after left ventricular decompensation and subsequent pulseless electric activity. Successful outdoor reanimation after an estimated time of hypoxia of 35 minutes. She was treated by hypothermia for 24 h. EEG after rewarming without sedative drugs showed a spontaneous burst-suppression pattern with spike-slow- and sharp-slow-waves with clinical myocloni. She remained deeply comatose and somatosensory evoked potentials 48h later showed absence of cortical responses.

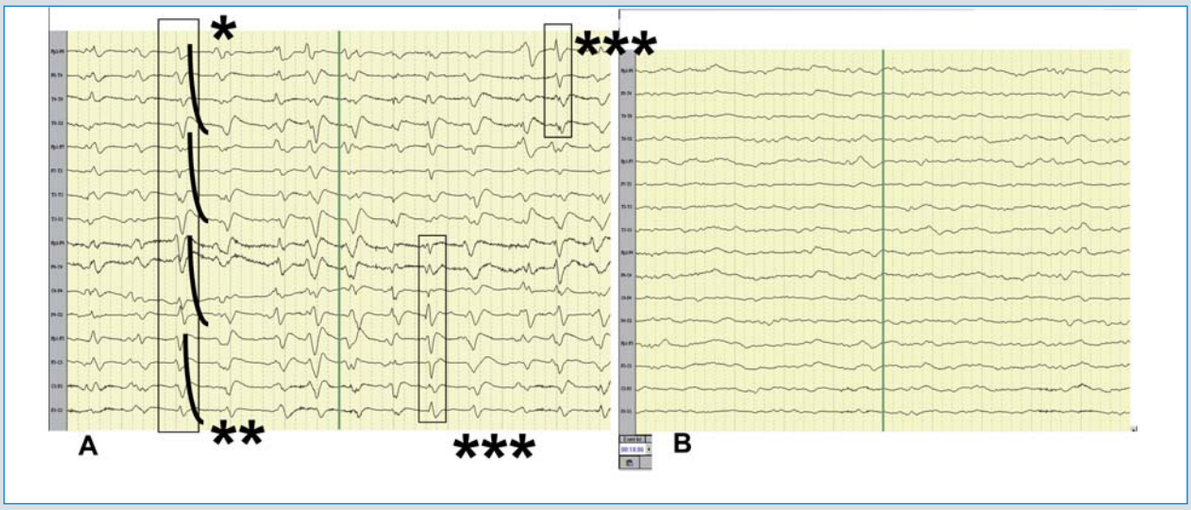
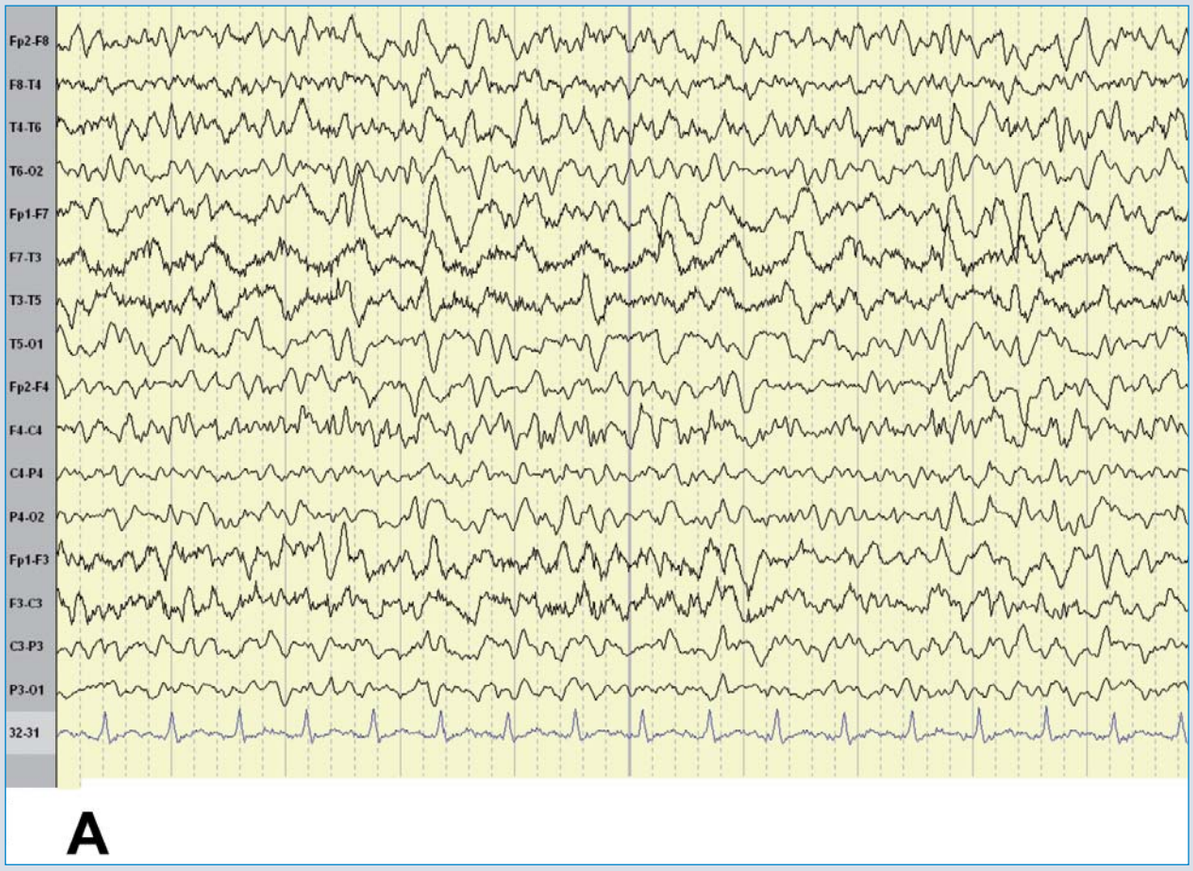
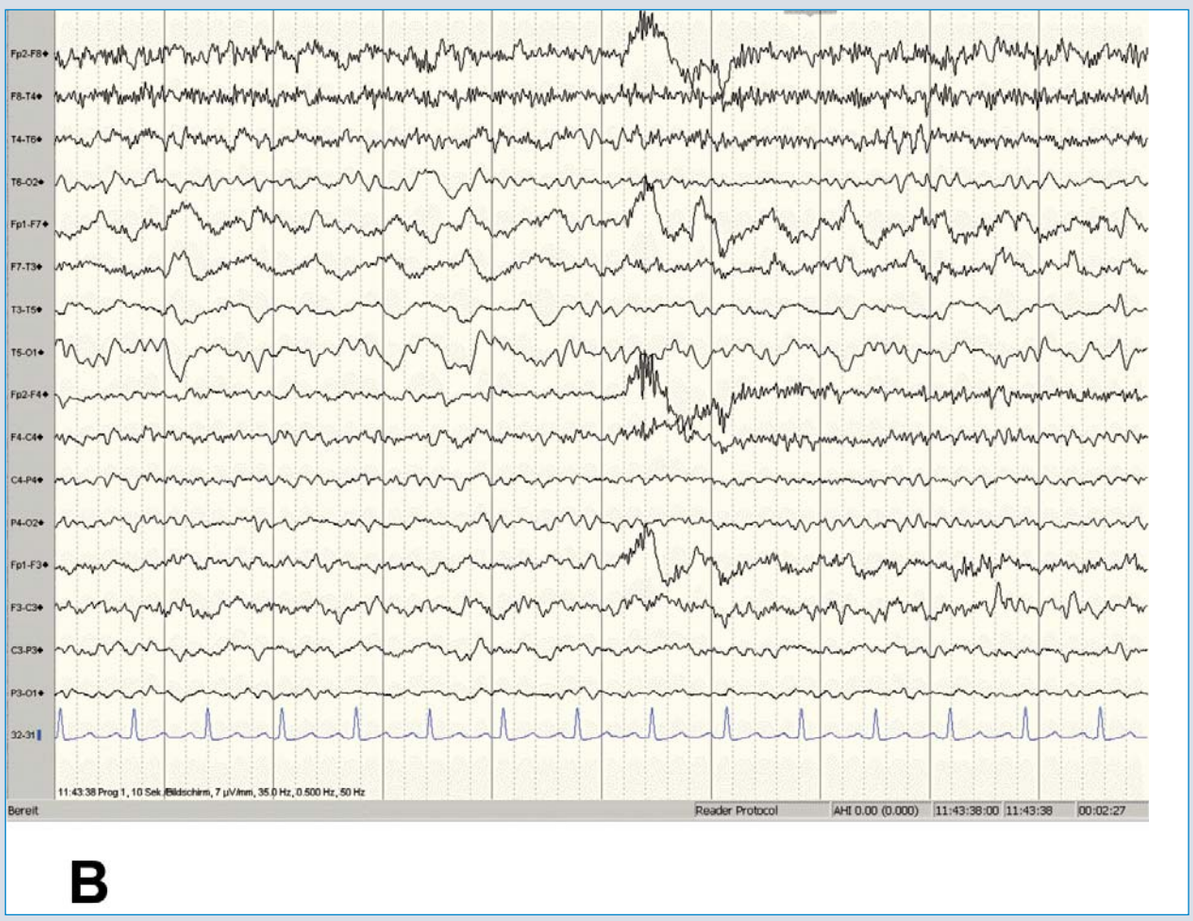


Figure 9: 81 year-old patient with sepsis caused by E. coli, prosthetic hip infection and multiple retroperitoneal abscesses was treated with rifampicine and cefepime; two days later, acute renal failure occurred and the patient was comatose despite immediate dialysis. The EEG (A) showed periodic triphasic waves (TPW) (*left box) with fronto-occipital shift (**); additionally, multifocal epileptic discharges (*, boxes in the middle and at the right) were observed in both paracentral regions and over the right temporal region. Intravenous administration of 1 mg of LZP (B) led to complete abolition of both the TPW and the epileptic discharges.**


Fp2-F8
F8-T4
T4-T6
T6-O2
Fp1-F7
F7-T3
T3-T5
T5-O1
Fp2-F4
F4-C4
C4-P4
P4-O2
Fp1-F3
F3-C3
C3-P3
P3-O1
32-31

A


Fp2-F8
F8-T4
T4-T6
T6-O2
Fp1-F7
F7-T3
T3-T5
T5-O1
Fp2-F4
F4-C4
C4-P4
P4-O2
Fp1-F3
F3-C3
C3-P3
P3-O1
32-31
11:43:38 Prog 1, 10 Sek /Bildschirm, 7 µV/mm, 35.0 Hz, 0.500 Hz, 50 Hz
Bereit
Reader Protocol
AHI 0.00 (0.000)
11:43:38:00
11:43:38
00:02:27

B

Figure 10:56 year-old woman with a history of multi-drug addiction (BZD, tramadol, alcohol). One year before admission, she insidiously became demented with bursts of frantic behaviour, where she tried twice to inflame her house. A giant aneurysm of the left middle cerebral artery was detected and the EEG showed substantial slowing of background activity and severe focal slowing over the left frontal and temporal regions often becoming rhythmic and with propagation also to the right. Epileptiform discharges frequently appeared over the left temporal region (A). Because of a lack of observation of episodic changes indicating overt seizures, epileptic and symptomatic encephalopathy resulting was suspected. She was started on an intensive antiepileptic regimen (VPA, LTG, and LEV) without BZD. Six months later, her cognitive and behavioural state had improved despite a further increase in size of the aneurysm. The EEG revealed an acceleration of background activity and a decrease of epileptic activity (B).

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Address for correspondence:
Stephan Rüegg MD
Division of Clinical Neurophysiology
Department of Neurology
University Hospital Basel
Petersgraben 4
CH 4031 Basel
phone 0041 61 2654757
fax 0041 61 2655638
srueegg@uhbs.ch