3rd Nordic Status Epilepticus Meeting

Oslo, Norway, 22-23 May 2025

Radisson Blu Scandinavia Hotel

Holbergs gate 30, Oslo

Norway

The meeting is financially supported by Desitin.

Welcome

Dear Colleagues

It is a great pleasure to welcome you all to the third Nordic status epilepticus meeting (3rd NSEM) to be held in Oslo, May 22-23, 2025.
Status epilepticus is the most serious and most feared seizure type with still a high mortality and morbidity rate. Early and correct treatment is of utmost importance for outcome and long-term prognosis. This time, we will therefore focus on the prehospital and early phase. The overarching theme is therefore: "*Time To Control – from basics to the clinic in status epilepticus*" and the goal is to improve and standardize early status epilepticus treatment. We will also discuss factors involved in the development and maintenance of status epilepticus including autoimmunity, biomarkers and molecular mechanisms, and also touch upon the difficult problem of functional/dissociative seizures and status epilepticus.
We are proud to have some of the leading experts in the field both internationally and from the Nordic countries and look forward to two exciting days in Oslo in May 2025.
This is an independent scientific meeting sponsored by an unrestricted grant from Desitin Pharma. You can attend either in person or digitally and the meeting is free of charge.

We look forward to seeing you in Oslo in May 2025!

Erik Taubøll
Norway

Johan Zelano
Sweden

Christoph P Beier
Denmark

Reetta Kälviäinen
Finland

The meeting is endorsed by the European Reference Network for Rare and Complex Epilepsies, ERN EpiCARE, funded by the European Commission.

Program

3rd Nordic Status Epilepticus Meeting (3rd NSEM meeting), Oslo, Norway, 22-23 May 2025

Overarching theme: Time To Control – from basics to the clinic in status epilepticus

Organising committee: Erik Taubøll (Head of committee, Norway), Johan Zelano (Sweden), Christoph P Beier (Denmark), Reetta Kälviäinen (Finland). From Norway: Kjell Heuser, Line Bédos Ulvin, Morten Horn, Bernt Engelsen, Gyri Veiby, Kjersti Nesheim Power, Christian Samsonsen, Ketil Berg Olsen, Ellen Molteberg. From Desitin: Gro Ellen Albrigtsen.

Day 1

09.00-10.00 Registration, coffee and fruit
10.00-10.10 Opening. *Erik Taubøll, Oslo, Norway*

Session 1. Prehospital SE treatment and the role of Time to Control

Chairs: Kjersti Nesheim Power and Gyri Veiby, Bergen, Norway

10.10-10.30 The prehospital experience – a report from the scene. *Rune Trøftmoen, Oslo, Norway*
10.30-10.40 Questions
10.40-11.00 Factors related to delays in pre-hospital management of status. *Leena Kämppi, Helsinki, Finland*
11.00-11.10 Questions
11.10-11.30 Prehospital EEG in the ambulance. *Sampsa Lohi, Joensuu, Finland*
11.30-11.40 Questions
11.40-12.00 Coffee break

Session 1 – cont. Prehospital SE treatment and the role of Time to Control

Chair: Reetta Kälviäinen, Kuopio, Finland

12.00-12.20 Early intubation or a new drug? *Gunhild Holmaas, Bergen, Norway*
12.20-12.30 Questions
12.30-12.50 How can prehospital seizure management be improved? From protocols to implementation. The preCTRL study. *Ingrid Hustad, Oslo, Norway*
12.50-13.00 Questions
13.00-14.00 Lunch

Session 1 – cont. Prehospital SE treatment and the role of Time to Control

Chair: Kjell Heuser, Oslo, Norway

14.00-14.30 The changing definitions of SE. Has the new definitions changed treatment to a more offensive strategy? *Eugen Trinka, Salzburg, Austria*

14.30-14.40 Discussion

14.40-14.55 The need for a standardized measure for acute treatment. The concept of TTC; time to control. *Morten Horn, Oslo, Norway*

14.55-15.30 General discussion: How can prehospital seizure treatment be improved? *Moderator Johan Zelano, Gothenburg, Sweden*

15.30-16.00 Coffee break

Session 2. Status epilepticus – from the past to the future

Chair: Erik Taubøll, Oslo, Norway

16.00-16.30 Status epilepticus – the past. The evolution of concepts of status epilepticus. *Simon Shorvon, London, UK*

16.30-16.40 Discussion

16.40-17.10 Status epilepticus – the future. The role of artificial intelligence for diagnostication and prognostication of SE. *Sándor Beniczky, Aarhus, Denmark*

17.10-17.20 Discussion, the past, present and future

17.20-17.25 Closing remarks, day 1

19.30 Dinner

Day 2

Session 3. Factors involved in the development of SE. Can we stop epileptogenesis?

Chair: Johan Zelano, Gothenburg, Sweden

08.45-09.20 Immune mechanisms in refractory status epilepticus - what is the role of early immunotherapy? *Ronny Wickström, Stockholm, Sweden*

09.20-09.30 Discussion

09.30-09.50 Biomarkers to predict early and late posttraumatic seizures after traumatic brain injuries. *Hild Flatmark Sødal, Oslo, Norway*

09.50-10.00 Discussion

10.00-10.20 Coffee break

Chair: Ronny Wickström, Stockholm, Sweden

10.20-10.40 What makes seizures stop? - a superficial look into the molecular basis of seizure termination. *Kjell Heuser, Oslo, Norway*

10.40-10.50 Questions

10.50-11.10 The interplay between age, inflammation and neuronal damage in SE. Consequences for treatment? *Sofie Bloch Mangaard, Odense, Denmark*

11.10-11.15 Questions

11.15-11.35 Factors responsible for long-term outcome after SE. *Christoph P Beier, Odense, Denmark*

11.35-11.40 Questions

11.40-12.40 Lunch

Session 4. Functional/Dissociative seizures and SE

Chair: Christian Samsonsen, Trondheim, Norway

12.40-13.20 Not everything that shakes is epilepsy: How to identify and treat prolonged functional/dissociative seizures. *Markus Reuber, Sheffield, UK*

13.20-13.35 Discussion

13.35-13.55 Functional/dissociative seizures and SE – epidemiological aspects. *Antonia Villagran, Sandvika, Norway*

13.55-14.05 Discussion

14.05-14.15 Coffee

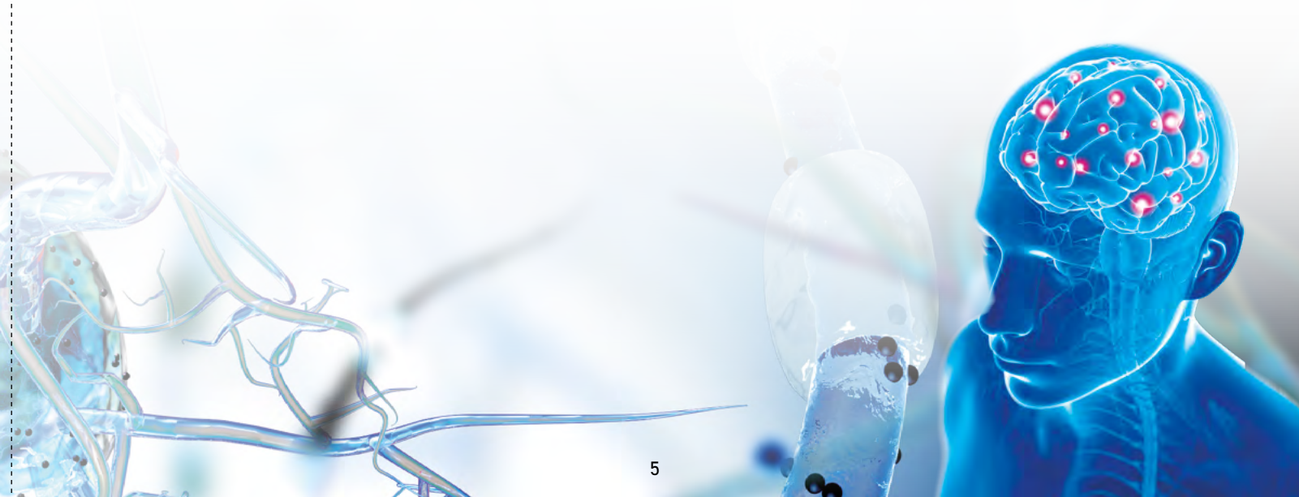
Session 5. Late-breaking news – projects in the pipeline

Chair: Morten Horn, Oslo, Norway

14.15-15.15 Four presentations, one from each of the Nordic countries.
10 min presentation + 5 min discussion.

- Post-ictal MRI changes – first results of a systematic review and single patient meta-analysis. *Andrea Enerstad Bolle, Odense, Denmark*
- Refractory and Super-refractory Status Epilepticus in Children and Adolescents: A Population Based Study from Western Norway. *Omar Hikmat, Bergen, Norway*
- Risk of epilepsy after a first post-traumatic seizure. *Markus Karlander, Gothenburg, Sweden*
- Adherence to current protocols of prolonged epileptic seizures in prehospital setting. *Lauri Eerola, Kuopi, Finland*

15.15-15.20 Closing remarks. *Erik Taubøll, Oslo, Norway*



Faculty and speakers

Simon Shorvon
is Emeritus Professor in Neurology at UCL Queen Square Institute of Neurology and Consultant Neurologist at the National Hospital Queen, London. His major interests are in epilepsy and especially status epilepticus, and in the history of neurology.

Sándor Beniczky
MD PhD is neurologist, clinical neurophysiologist and epileptologist. He is professor at Aarhus University Hospital, the head of the Clinical Neurophysiology Department at the Danish Epilepsy Centre, and editor-in-chief of Epileptic Disorders. Main research interest: EEG and epilepsy, source imaging, seizure detection, automated analysis, artificial intelligence, standardisation and quality assurance. He has supervised 11 Ph.D. students, authored over 260 peer-reviewed papers and 26 book chapters.

Johan Zelano
Professor of Neurology at the Univeristy of Gothenburg and consultant at the Sahlgernska University Hospital. Head of a translational epilepsy research group with a focus on biomarkers and big data, with particular interest in improved diagnosis and treatment.

Christoph Patrick Beier
Professor of neurology and consultant for general neurology with specialist interest in epilepsy. Head of the epilepsy research group and affiliated to the Center for Innovative Medical Technology at the Odense University Hospital, Denmark. His research interest are prognostication and optimization of treatment of status epilepticus, patho-physiology of drug resistant idiopathic generalized epilepsies, and new approaches for optimized ambulatory patient care.

Ronny Wickström
is professor of Child Neurology at Karolinska Institutet in Stockholm, Sweden. His work on NORSE and FIRES led to the international consensus publications on work-up and treatment in the last years. He is also part of the NORSE Institute and the Steering Committee of the NORSE Biorepository at Yale University.

Sampsa Lohi
He is a neurologist and clinical neurophysiologist with special interest in epilepsy, EEG, and sleep medicine. In addition to clinical work, he develop open access tools for clinical neurophysiology education and research.

Eugen Trinka
Univ.-Prof. Dr. Mag. Eugen Trinka, FRCP is Professor and Chairman of the University Department of Neurology, Deputy Medical Director of the Christian Doppler University Hospital and President of the Salzburg Medical Society. He is also currently Member of the Executive Committee of the International League Against Epilepsy.

Markus Reuber
is a Professor of Clinical Neurology at the University of Sheffield and Honorary Consultant at the Royal Hallamshire Hospital in Sheffield, United Kingdom. His clinical work focuses on the treatment of patients with complex seizure disorders. In his research, he has been particularly interested in the phenomenology and treatment of functional / dissociative seizure disorders.

Reetta Kälviäinen
is Full Professor and Chair of Neurology in the University of Eastern Finland and Director of the Kuopio Epilepsy Center in Kuopio University Hospital. Her special research interest is clinical epileptology. She serves in the executive committee of the European Reference Network for rare and complex epilepsies EpiCARE. She is the chair of the board of the Neurocenter Finland and the Finnish Neurological Society.

Erik Taubøll
Professor of Neurology and Senior Consultant, Department of Neurology, Oslo University Hospital - Rikshospitalet, Oslo, Norway. Head of ERGO, Epilepsy Research Group of Oslo, in Department of Neurology. The group is active in both clinical, translational and basic epilepsy research, see: https://www.ous-research.no/ergo/

Leena Kämppi
Associate Professor and adult neurologist working in Epilepsia Helsinki in HUS Neurocenter. Her research group is working on acute epileptic seizures and status epilepticus with the focus on delays in the treatment, pre-hospital care, NORSE and outcome of SE.

Kjell Heuser
MD, PhD, is a Senior Consultant and Clinical epileptologist in Department of Neurology, Oslo University Hospital. He is Head of Translational Epilepsy Research Group which is part of ERGO (Epilepsy Research Group of Oslo), with a special interest in translational and clinical epilepsy research.

Faculty and speakers

Gyri Veiby
MD, PhD, is a senior consultant neurologist at Haukeland University Hospital in Bergen, working at the section for acute neurological diseases. Veiby has a special clinical interest in the treatment of refractory status epilepticus, and specifically in relation to POLG disease. Main research is focused on different female aspects of epilepsy, in particular teratogenic effects of antiseizure medications.

Kjersti Nesheim Power
is a neurologist with a PhD in treatment and consequences of status epilepticus, and has a particular interest in cognitive sequelae. She works at the Department of Neurology at Haukeland University Hospital.

Christian Samsonsen
Head of department/neurologist at Department of Neurology and Clinical Neurophysiology, St Olav's hospital; Associate professor, INB, NTNU.
Special fields of interest: Clinical epileptology, seizurerecipitants.

Morten Horn
MD, PhD, is a consultant in neurology at Oslo University Hospital. He is a lecturer in medical ethics at the University of Oslo, and a member of the Medical Ethics Council for the Norwegian Medical Association.

Ellen Molteberg
MD, Neurologist and Head of the Adult Department at the National Center for Epilepsy in Norway. Special interest in non-medical treatment, i.e modified Atkins diet for adult patients.

Rune Trøftmoen
Paramedic at Oslo University Hospital and MSc student in Prehospital Critical Care, University of Stavanger, Norway.

Sofie Bloch Mangaard
M.D. and PhD-student at Odense University Hospital and University of Southern Denmark. Her research focuses on age-dependent immune changes after status epilepticus.

Bernt Engelsen
Professor of Neurology since 1989. Research background; neurotransmission research on glutamate in the 1980ies-90ies, and since 1994 head of the clinical epilepsy unit at Haukeland University Hospital. Research interests; clinical epileptology, POLG related epilepsy and Status epilepticus.

Ketil Berg Olsen
Senior Consultant in Neurology and Clinical Neurophysiology, Østfold Hospital Trust and Oslo University Hospital, Rikshospitalet. Member of ERGO, Epilepsy Research Group of Oslo. Many years experience in epileptology/ neurology/clinical neurophysiology, many years experience in SEEG and IOM.

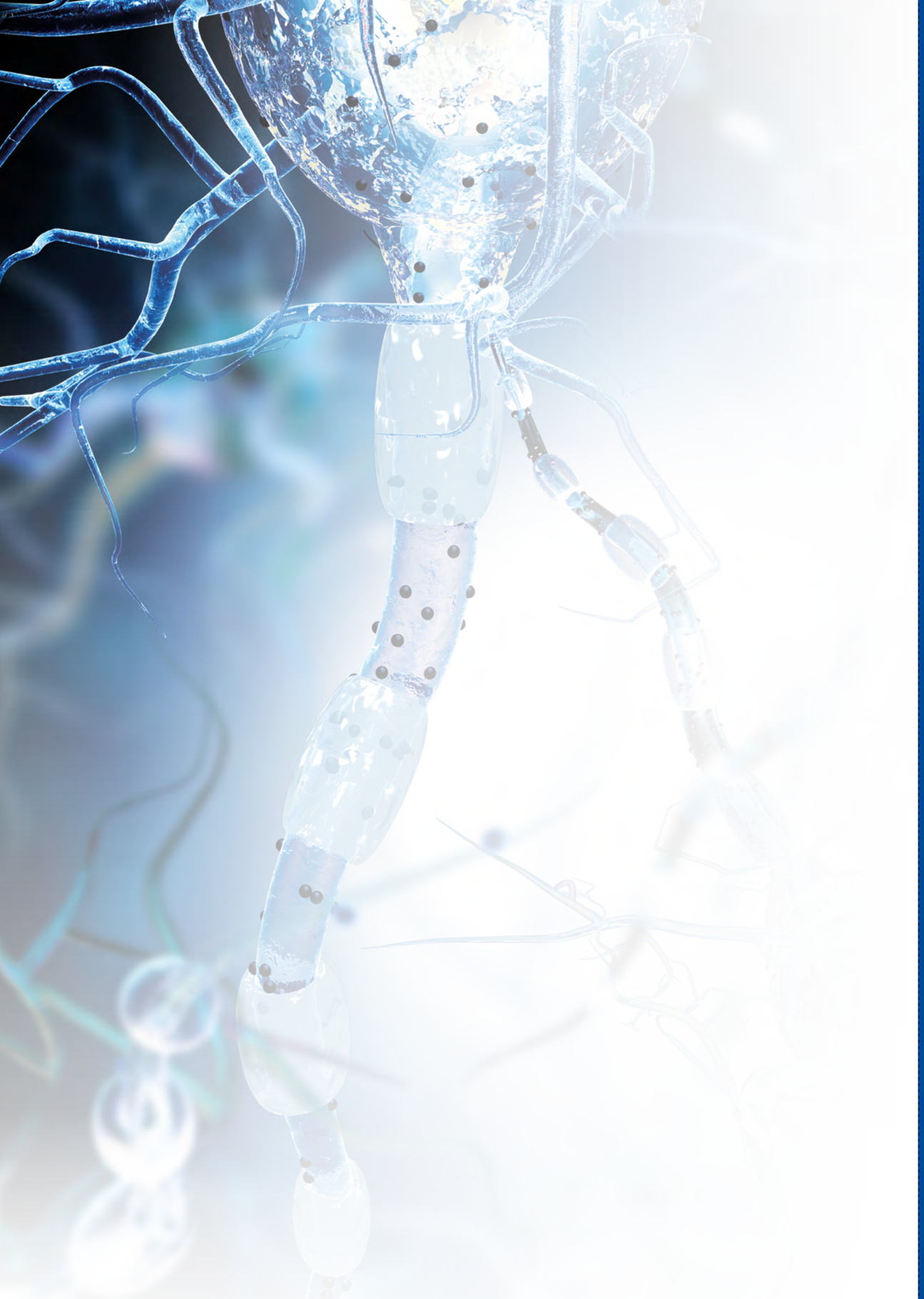
Line Bédos Ulvin
is a neurologist and clinical neurophysiologist working at Oslo University Hospital. Her field of interest is epilepsy and EEG, and she is involved in several research projects on status epilepticus.

Ingrid Hustad
MD, PhD candidate with the Norwegian Air Ambulance Foundation and Oslo University Hospital, focusing on prehospital seizure management through the Prehospital Seizure Control Trial (PreCTRL). The study aims to map prehospital and early hospital care for seizure patients with the goal of contributing to improved treatment pathways for this patient group.

Gunhild Holmaas
is specialist in anaesthesiology and intensive care and works as senior consultant at Department of Anaesthesia and Intensive Care, at Haukeland University Hospital, Bergen, Norway.

Antonia Villagrán
MD, phD, is a consultant in neurology at the National Center for Epilepsy, Oslo University Hospital, Norway.

Hild Flatmark Sødal
MD, PhD student and consultant in neurology at Oslo University Hospital. Member of ERGO, Epilepsy Research Group of Oslo. Special interest in clinical epileptology and post-traumatic epilepsy



Abstracts

for the 3rd Nordic Status Epilepticus meeting 2025

Factors related to delays in pre-hospital management of status epilepticus

Leena Kämppi, Helsinki, Finland

Status epilepticus (SE) is the most extreme form of an epileptic seizure. It is considered to be a life-threatening neurological emergency situation, which requires immediate treatment actions to cease the excessive electric activity in the brain. Increased understanding of the pathophysiology of status epilepticus has led us to recognize that time is brain also in status epilepticus. 30 minutes of continuous seizure results in receptor trafficking in synapses and thereby leading to pharmacoresistance and refractoriness. SE is an exceedingly dynamic process and several factors during the process have been proposed to influence the patients' outcome: 1) patient's pre-existing characteristics, 2) factors related to the current SE episode and 3) treatment and complications. Most of the influencing factors are pre-existing and cannot be affected, therefore, treatment and complications should be in the focus when aiming to improve SE patients' outcome.

Pre-hospital management of SE has been studied mainly with the focus on medication selection, safety and efficacy of the given treatment. Pre-hospital treatment has been considered to be safe and efficient in reducing SE duration and refractoriness, with the benefit of the treatment exceeding the risks. Adherence to treatment protocols, quality of treatment, proper drug sequence and management within the suggested timeframes seem to have a significant impact on the prognosis of SE. It has even been suggested that rapid administration of the medication per se is probably more important than the actual agent. Still, in many countries the medication availability out-of-hospital in emergency medical services is low and the treatment protocol variability, even within one country, is high.

Studies focusing on the actual management of SE out-of-hospital, delays in the treatment and adherence to treatment protocols are scarce. These studies have shown unexpectedly and unacceptably long median delays in the management and highlighted that the significant prehospital delays can adversely affect patient outcomes. These long delays have been related to the remarkable inadequacy detected in recognition of SE both among laity and medical professionals, and suboptimal triaging of SE patients. Even 30.5% of the SE cases, mainly other types of SE than generalized tonic-clonic SE, may not be recognized during the prehospital phase leading to delayed initiation of the medication.

Published studies collectively underscore the critical nature of early recognition and treatment of SE, as prolonged prehospital delays can lead to worse clinical outcomes. Strategies to enhance prehospital symptom recognition and streamline the treatment process are necessary to mitigate these delays and improve patient care. SE should be given a compeer position with stroke and cardiac infarction and acknowledged as a medical emergency with similar resource allocation in the pre-hospital management.

1. Kämppi L, Mustonen H, Soinila S. Analysis of the delay components in the treatment of status epilepticus. Neurocritical Care (2013) 19:10-18.
2. Kämppi L, Mustonen H, Soinila S. 2015. Factors related to delays in pre-hospital management of status epilepticus. Neurocritical Care (2015) 22: 93-104.
3. Sairanen J, Kantanen AM, Hyppölä H, Kälviäinen R. Treatment delay in status epilepticus – more effective prehospital symptom recognition warranted. Scand J Traum Resusc Emerg Med 2019; 7;27(1):28. doi: 10.1186/s13049-019-0605-7.
4. Hustad I, Horn M, Rehn M, Tauboll E, Hov M. Prehospital seizure management protocols need standardized guidelines. A descriptive study from Norway. Seizure (2024) 123:92-96.

Prehospital EEG in the ambulance

Sampsa Lohi, Joensuu, Finland

As an investigational method, EEG could be useful in assessing altered metal state and suspected brain insults in the field. In particular, EEG could help emergency medical personnel identify stroke-like epileptic seizures, in administering seizure medications during the transit, and in choosing which centre the patient should be transferred to.

Due to technical limitations, recording EEG in the ambulance has been impractical until recently. Published research on prehospital EEG is still scarce, but a couple of groups have already published promising results using dry electrodes. While the systems used in these studies proved to be technically viable, both groups reported issues with maintaining good signal quality for extended periods of time. While dry electrodes are user-friendly and reusable, in the hospital environment, wet electrodes have traditionally been used to record EEG due to their better signal-to-noise ratio and adhesion to the patient's scalp. No literature currently exists on using wet electrodes to record EEG in the ambulance or prehospital setting in general.

We have tested the feasibility of using a portable EEG recorder-amplifier and a screen-printed, self-adhesive wet electrode set (Muraja-Murro et al. 2015) to record EEG in a moving ambulance with good results. In this system, the electrodes are attached to predetermined locations on the forehead and temples, below the patient's hairline. The electrode set is very easy and quick to apply and can stay in place for several hours. This setup is capable of measuring EEG in the frontal and fronto-temporal brain regions. Up to four additional electrode leads can be added to measure activity from more posterior parts of the brain, if needed. We are now carrying out a clinical trial where we measure EEG on suspected acute stroke patients in the ambulance during their transport from the field to the hospital.

References

Muraja-Murro A, Mervaala E, Westeren-Punnonen S, Lepola P, Töyräs J, Myllymaa S, Julkunen P, Kantanen AM, Kälviäinen R, Myllymaa K. Forehead EEG electrode set versus full-head scalp EEG in 100 patients with altered mental state. Epilepsy & Behavior. 2015;49: 245–249. doi:10.1016/j.yebeh.2015.04.041.

Sedation, or a new drug?

Gunhild Holmaas, Bergen, Norway

Deep enough general anaesthesia moderate and eventually stops electrical activity in the brain and may thereby abort status epilepticus.

Several sedative agents are used in general anaesthesia. Most of them have antiepileptic effect, and bi-spectral index (BIS), a kind of EEG analysis, is used to monitor anaesthesia depth. At the same time propofol and fluorinated gases are known to alter the seizure threshold, and seizures during recovery after general anaesthesia is a well-known complication. The effect of general anaesthesia sedative drugs is dose dependent, and minor doses of benzodiazepines may stop epileptic seizures without affecting the ventilation and gag reflex. A bolus dose of propofol or thiopentone, however, may stop an epileptic attack but give apnoea and should be administered by anaesthesia personnel only. The status epilepticus may restart when the drugs are ceased, and a sustained effect is time dependent, often 24 to 48 hours. Continuous EEG-monitoring guides the necessary sedative dosage to suppress epileptic activity, and burst suppression is easily recognisable for non-neurophysiology specialists. The necessity of burst suppression in non-mitochondrial diseases is debated, but overtreatment may be better than insufficient treatment.

No RCTs comparing sedation with more or higher doses of non-sedating antiepileptic drugs exist. Increasing delay of anaesthesia was associated with decreased odds for return to premorbid function in a retrospective cohort study (1). Another cohort study, where logistic regression analyses excluded cofounding factors, revealed increased odds for death and unfavourable outcome in survivors receiving general anaesthesia (2). However, excluding all confounding factors in retrospective cohort-studies is a difficult task, and what is the alternative to anaesthesia when other antiepileptic drugs fail?

Intubation may lead to aspiration as well as preventing it. Ventilator associated pneumonia, lung atelectasis and barotrauma are common in intubated patients. Deep sedation stops seizure related muscle contractions and lactic acidosis. At the same time sedation and artificial ventilation may give muscle atrophy, and the risk of venous thrombosis increases substantially. Propofol infusion syndrome (propofol) and malignant hyperthermia (thiopentone and fluorinated gases) are extremely rear events. Opiate- and benzodiazepine withdrawal symptoms, however, is common, especially in children.

The question is not sedation or a new drug, since sedation represents new antiepileptic drugs with side effects demanding intubation, ventilation and detailed monitoring. The question is how much time we are allowed to use waiting for effects from the non-sedating antiepileptic drugs, before we seize the sedating ones.

Literature:

1. De Stefano P et al. Early timing of anesthesia in status epilepticus is associated with complete recovery: A 7-year retrospective two-center study. Epilepsia. 2023 Jun;64(6):1493-1506.
2. Sutter R et al. Anesthetics and Outcome in Status Epilepticus: A Matched Two-Center Cohort Study. CNS Drugs. 2017 Jan;31(1):65-74.

Norwegian Prehospital Seizure Management – The PreCTRL study

Ingrid Hustad, Oslo, Norway

Background: Patients with convulsive seizures constitute a significant group in acute neurology. Most patients are brought to hospital for seizure treatment, with great variation in which prehospital treatment is provided. Status Epilepticus (SE) is often not recognized during the prehospital phase. Only 33% of SE patients receive a benzodiazepine as first anti-seizure medication (ASM), and underdosing of benzodiazepines is common.
In Norway, specialist healthcare services are organized into four Regional Health Authorities comprising 19 Health Trusts, 18 of which have an ambulance service. Historically, they operate with their own treatment protocols designed at a local level.

A recent analysis found disparities in the prehospital seizure management protocols within the Norwegian healthcare system, including a lack of SE definitions, unclear timing for benzodiazepine administration, and variation in drugs, dosages, administration routes, and re-dosing strategies. Only a minority of protocols specify a first-line treatment. These findings are suggestive as to *why* paramedics could fail in recognizing the condition and why first-line treatment remains delayed or suboptimal in the prehospital setting – though further investigation is needed.

This study aims to describe the prehospital and initial in-hospital management of seizure patients, focusing on EMS assessment and factors influencing treatment decisions. Incidence data on prehospital seizure assessment and hospital admission in Norway is limited; this retrospective study will provide a baseline.

Method:
We are conducting a retrospective analysis of patients in the Oslo region for whom an ambulance was dispatched using seizure-related criteria by the Emergency Medical Communication Centre (EMCC). Hospital records are used to identify the prehospital pathway of additional patients discharged from Oslo University Hospital (OUH) with a seizure-related diagnoses. Data is sourced from the EMCC, Acute Medical Information System (AMIS), and emergency department records over a 12-month period. Exclusion criteria include patients under 18, referrals from GPs or other hospitals, admissions outside Oslo, and incomplete EMCC records.

Results: Preliminary data shows 5318 emergency calls due to suspected seizure, resulting in 1323 ambulance dispatches. EMS assessed and left 481 patients (36.4%) on scene. Of those transported by EMS, 345 were taken to an out-of-hours clinic and 497 directly to hospital. During the same period, 31 624 patients presented to the OUH Emergency Department;
508 had suspected seizures or were discharged with seizure-related diagnoses.
Ongoing analysis will evaluate EMS decision-making, on-scene treatment, and admission outcomes from the population left on scene or transported to the out-of-hours clinics.

The changing definitions of Status Epilepticus; Has the new definitions changed treatment to a more offensive strategy?

Eugen Trinka, Salzburg, Austria

Despite that Status Epilepticus is known since antiquity and the clinical term had recently it's 200th birthday (1824 etat de mal). It is still one of the most intriguing conditions in medicine. It has been traditionally defined as seizures, that merge into a continuum, where the patient does not recover between attacks, without given at definite time frame. Since the 1990s a 30-minute definition has been progressively modified to 20 minutes, 10 minutes and now 5 minutes for convulsive Status Epilepticus. The reason why this progressive modification took place is to prevent brain damage, which occurs in various types of Status Epilepticus. However, the precise time, when this occurs, is not known for every case. So the time point T2, which is defined as the time, when you can expect neuronal damage or alteration of neuronal networks, has changed our view in the attitude of Status Epilepticus and guides us to start treatment earlier to prevent later damage of Status Epilepticus. Whether this can be translated in a better outcome, has still to be determined. In this presentation the treats and opportunities with the new definition will be discussed. In clinical terms, every treatment decision has to be balanced between presumed risks and expected benefits. The impact of burden concept can help the clinicians in their treatment considerations.

The need for a standardized measure for acute treatment. The concept of TTC; time to control

Morten Andreas Horn, Oslo, Norway

When patients experience seizures that won't cease spontaneously within five minutes ("T1"), the situation is considered as status epilepticus, a potentially life-threatening emergency. According to both clinical experience and modern definitions, time is urgent when it comes to controlling ongoing seizures. One may envision a "golden half-hour" during which seizure control should and could be achieved. Seizures lasting for more than 30 minutes become increasingly difficult to control, and ongoing seizures are causing progressively more neuronal and systemic damage. This is reflected in the "T2" milestone in modern status epilepticus definitions.

Even though the underlying pathology is of great importance for the prognosis of status epilepticus, swift control of seizures matters too. Most status epilepticus treatment protocols use a stage-based approach, with first-, second- and third-line interventions that are supposed to be administered in a systematic and timely fashion. In spite of the common knowledge that time matters, seizure management in clinical practice may frequently seem to lack a clear consciousness of how much time has passed, what stage the patient is in, and how to adapt the theoretical guidelines to the patient at hand. Often, treatment may seem to be "too little, too late".

A particular – and very frequent – challenge arises when seizures start out of hospital, are first attended to by prehospital medical services, and the patient is then brought to hospital with ongoing, uncontrolled seizures. Often, there is a lack of cohesion between what has been done to control seizures in the prehospital phase, and what is being done in the emergency room and in the intensive care unit.

When training junior physicians and nurses in team-based management of status epilepticus, using simulated scenarios, we saw the need for a standardized measure of success. Obviously, cessation of seizures is the paramount measure of success. However, we needed another measure that took into account the timeliness of interventions in stage-based management. We needed participants – both in simulation scenarios and in clinical practice – to be conscious of time elapsed.

Therefore, we have formulated the concept of "Time To Control" (TTC): This time measure of effectiveness starts with the onset of seizures, and ends with the cessation of clinical seizures, either spontaneously or following treatment. The lecture will present the rationale for this measure in management of status epilepticus.

Reference:

Horn MA, Hov MR, Heuser K, Taubøll E. "Time to Control – A goal in seizure management". Seizure 2023;106:76.

Status epilepticus – the past. The evolution of concepts of status epilepticus

Simon Shorvon, London, United Kingdom

It has been known since the earliest records that prolonged seizures can end fatally. This was famously recorded in the Babylonian cuneiform text recorded on 40 tablets known as Sakikku, or 'All diseases', dating from the middle of the first millennium BC. Then the disease was often considered due to possession. Other examples of fatal episodes of prolonged epilepsy are well recorded throughout the medieval and renaissance periods. In the 17th and 18th centuries, careful clinical descriptions were published, for instance by Thomas Willis who first documented the progression of signs and symptoms in convulsive forms. The term *état de mal* first appeared in the in the medical literature in 1824, in the doctoral thesis of Dr Louis Florentin Calmeil, attributed the name to the patients in the Paris asylums who had coined the term, but he considered the 'state' to be due simply to a succession of normal seizures. Thus for over 2000 years, episodes of status were not conceptualised to be in anyway distinct from other epileptic seizures, but seen simply as prolonged and severe variants which often ended fatally.

It was Trousseau in 1868 who first considered the condition 'special' and distinctive, with signs not seen in ordinary seizures. The greatest descriptions though were by Bourneville who 1876 defined the stages of status epilepticus and conceived of it, not as a seizure, but as a 'grave complication' of epilepsy'. The pathological changes in the brain were then described (notably by Crichton-Brown in 1873) and were of a type not seen in ordinary epilepsy. Clark and Prout then saw the condition 'more a true climax of the disease [epilepsy] and not a chance termination….. an epileptic is foredoomed to die of the status as the maximum development of the disease'.

EEG then had a profound effect on the concept of status epilepticus. Henri Gastaut famously wrote in 1971 that for every seizure type there is a status variant, and the condition was again viewed as a form of epileptic seizure – albeit one not confined to convulsive seizures. It is only recently, with the rise of molecular biology that the concept has again arisen that the condition is quite distinct from ordinary seizures, not only in its form and prognosis, but also in its mechanism. Fundamental questions are now posed. Is status epilepticus due to an intrinsic 'failure' of the mechanisms which terminate seizures, or are the initiating mechanisms different in some way from those initiating self-limiting seizures, or are normal mechanisms simply overwhelmed by the 'severity' of the seizure. It could be argued that the emphasis on EEG has inhibited novel thinking, and it seems to me that shaking off the 'seizure' analogy is fundamental to making further conceptual advance. It seems likely that molecular biology or cellular physiology will furnish better explanations and status epilepticus should be conceptualised as a distinctive condition in its own right, with differing mechanisms, clinical features, consequences and treatment from run-of-the-mill epilepsy.

Suggested reading:

For historical references, see: Shorvon S. Status epilepticus: Its clinical features and treatment in children and adults. Cambridge: Cambridge University Press; 1994.

For modern definition, see: Trinka E, Cock H, Hesdorffer D, Rossetti AO, Scheffer IE, Shinnar S, Shorvon S, Lowenstein DH. A definition and classification of status epilepticus--Report of the ILAE Task Force on Classification of Status Epilepticus. Epilepsia. 2015 Oct;56(10):1515-23.

The role of artificial intelligence for diagnostic and prognostic assessment of patients with status epilepticus

Sandór Beniczky, Aarhus, Denmark

Artificial intelligence (AI) has the potential to revolutionize medicine, including the management of status epilepticus. AI models have been developed and validated to identify high-risk patients for resource-intensive investigations (such as continuous EEG), assist in automated and semi-automated EEG analysis, and predict clinical outcomes. This presentation will provide an overview of the current state of AI-based analysis of clinical and electrographic data in critically ill patients at high risk for status epilepticus.
Using AI, nomograms and models have been developed to predict the risk of seizures. These tools are useful to select the patients who would benefit most from cEEG, especially when resources are scarce.

EEG is needed to diagnose non-convulsive status epilepticus. AI models are available for automated and semiautomated interpretation of EEG, including the special application of cEEG in critically ill patients, at high risk for status epilepticus.

AI models are available for improved prediction of clinical outcome: progression to refractory status epilepticus, mortality and cortical performance category.

Immune mechanisms in refractory status epilepticus - what is the role of early immunotherapy?

Ronny Wickström, Stockholm, Sweden

Refractory status epilepticus (RSE) is a severe and prolonged seizure state that persists despite administration of first-line (benzodiazepines) and second-line (intravenous antiseizure medications like phenytoin, valproate, or levetiracetam) treatments. It is a neurologic emergency associated with high morbidity and mortality, requiring aggressive management, including anesthetic agents and intensive care unit admission. RSE can result from various causes, including structural brain lesions, metabolic disturbances, infections, autoimmune encephalitis, or cryptogenic factors.

It is becoming increasingly clear that neuroinflammation plays a critical role in the pathophysiology for both epilepsy in general and RSE, contributing to both seizure persistence and neuronal injury. This may be particularly true in new-onset cases (NORSE) with its subcategory Febrile Infection Related Epilepsy Syndrome (FIRES). Potential mechanisms include release of cytokines and chemokines (enhancing excitatory neurotransmission and promoting neuronal hyperexcitability), an increased permeability of the blood-brain barrier (allowing immune cells, antibodies, and inflammatory mediators to enter the central nervous system) and autoimmune mechanisms associated with autoantibodies, which impair synaptic function and neuronal communication. The mechanisms also include microglial and astrocyte activation.

Understanding these mechanisms is essential to allow initiation of appropriate management of RSE which in addition to anti-seize medications also may warrant early immunotherapy, including corticosteroids, IVIG, plasmapheresis or second-line treatments. This lecture will review the theoretical background for immunological pathways, appropriate diagnostic work-up and immunological treatment options in RSE.

Suggested reading

Foiadelli T, Santangelo A, Costagliola G, Costa E, Scacciati M, Riva A, Volpedo G, Smaldone M, Bonuccelli A, Clemente AM, Ferretti A, Savasta S, Striano P and Orsini A (2023) Neuroinflammation and status epilepticus: a narrative review unraveling a complex interplay. Front. Pediatr. 11:1251914. doi: 10.3389/fped.2023.1251914

Wickstrom R, Taraschenko O, Dilena R, Payne ET, Specchio N, Nabbout R, et al. for the International NORSE Consensus Group. International consensus recommendations for management of new onset refractory status epilepticus including febrile infection-related epilepsy syndrome: Statements and supporting evidence. Epilepsia. 2022;63:2840– 2864. https://doi.org/10.1111/epi.17397

Biomarkers to predict early and late post-traumatic seizures after traumatic brain injury

Hild Flatmark Sødal, Oslo, Norway

Common for all acquired epilepsies is the development from a normal brain into one that produces recurrent epileptic seizures. This process, called epileptogenesis, starts with an insult followed by complex processes that are initiated in the injured brain. After a latent period, clinical seizures arise and represents the onset of epilepsy. Elucidation of the processes involved in epileptogenesis have led to identification of potential targets for treatment that may prevent the development and progression of epilepsy. Currently, no such treatment exists.
Post-traumatic epilepsy (PTE) accounts for 5% of all epilepsies and are one of the most commons forms of acquired epilepsy. PTE is often considered a model disease to understand human epileptogenesis and to study potential preventive therapies. Previous studies on preventive treatment in TBI patients have so far been unsuccessful. Clinical trials are challenging due to several reasons. First, most patients suffering TBI do not develop PTE, even in the presence of clinical risk factors. Second, the latency period before seizures occur may last for years, requiring long term follow up. Discovery and validation of biomarkers is crucial to aid the identification of patients at high risk of PTE who might benefit from therapeutic interventions.
Clinical risk factors and potential biomarkers for post-traumatic seizures will be discussed, with focus on blood-based biomarkers. Preliminary data from an ongoing multicenter study on genetic and protein biomarkers of PTE will be presented.

Suggested reading:

Klein P, Tyrlikova I. No prevention or cure of epilepsy as yet. Neuropharmacology. 2020 May 15;168:107762.
Bruckhaus AA, Asifriyaz T, Kriukova K, O'Brien TJ, Agoston DV, Staba RJ, Jones NC, Moshé SL, Galanopoulou AS, Duncan D. Exploring multimodal biomarker candidates of post-traumatic epilepsy following moderate to severe traumatic brain injury: A systematic review and meta-analysis. Epilepsia. 2025 Jan;66(1):6-32.

What makes seizures stop? - a superficial look into the molecular basis of seizure termination.

Kjell Heuser, Oslo, Norway

The spontaneous termination of seizures remains a key unresolved question in epileptology (Lado and Moshé, 2008; Löscher and Köhling, 2010). Proposed mechanisms include increased GABAergic signaling, neurotransmitter or ATP depletion, ionic imbalance, and adenosine release (Lado and Moshé, 2008; Kramer et al., 2012).

Recently it has been hypothesized that spreading depolarization (SD) may have an important role in seizure termination and may even serve as part of the brain's natural machinery to curb hyperexcitability and prevent epileptic seizures (Enger and Heuser, 2024).
SD waves are slow-moving waves of complete depolarization in gray matter followed by neuronal silencing (Takano and Nedergaard, 2009; Charles and Baca, 2013).
The phenomenon is believed to be the cellular substrate of the migraine aura.

While the role of SD in migraine has been quite extensively explored, the role for SD in epilepsy and seizures is much less studied, although SD was first discovered during studying of seizure activity (Leao, 1943). Recent evidence from both studies in acute and chronic animal models and humans sheds new light on the role of SD in epilepsy. We will focus on the role of SD in co-occurrence with epilepsy and highlight its putative role in terminating seizures and preventing seizure generalization.

The interplay between age, inflammation and neuronal damage in SE. Consequences for treatment?

Sofie Bloch Mangaard, Odense, Denmark

Status epilepticus (SE) is the second most common neurological emergency with a significant morbidity and mortality. SE characterizes by prolonged or recurrent seizures that does not stop spontaneously leading to neuronal damage with high inter-individual variability. The underlying causes of SE are diverse, but SE-induced necrosis of cortical neurons is in humans associated with proliferation and activation of microglia and astrocytes. In healthy rodent models, induced SE causes a rapid onset (min) and long-lasting (days/weeks) neuro-inflammatory response, highlighting the role of glial cells in epileptic driven neuro-inflammation. In humans, age and SE duration is key predictors of neurological deficits – the older the patients the shorter SE episodes is required to induce severe new neurological deficits. This aligns with the changes seen in microglia and astrocytes during aging in e.g. neurodegenerative diseases, where microglia's response shift from an anti-inflammatory to a more pro-inflammatory phenotype.

Given the possible critical role of neuro-inflammation in SE, this raises the question: Could age-related changes in glial cells explain the vulnerability of older patients to SE-induced neurological deficits? Moreover, can targeting these inflammatory processes offer new therapeutic avenues? This talk will explore the interplay between SE, neuro-inflammation, and aging, with implications for treatment strategies in vulnerable populations.

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Factors responsible for long-term outcome after SE

Christoph P Beier, Odense, Denmark

Status epilepticus (SE) is associated with high long-term mortality, reaching 50% at 30 months, with no significant improvement in recent years. This presentation will focus on how different clinical characteristics influence long-term survival based on data from Odense University Hospital and a retrospective multicentre study from Denmark, Norway and Germany analysing factors influencing outcome. Aetiology, age and established prognostic markers were important, but influenced outcomes at different times. The burden of complications during acute treatment had only a transient effect on outcome. Seizure duration is a modifiable factor, but whether aggressive treatment and rapid seizure termination affect survival remains controversial. This talk will present data on short and long-term outcomes as a function of seizure duration, suggesting that seizure duration has little effect on short-term survival. Conversely, longer seizure duration was increasingly associated with worse survival over time. Complementary retrospective studies suggest that modifiable organisational factors, such as the timing and delay of acute EEG, may be associated with post-discharge survival, suggesting that improved care of SE patients may have significant potential to improve long-term outcomes.

Not everything that shakes is epilepsy: How to identify and treat prolonged functional/ dissociative seizures

Markus Reuber, Sheffield, UK

Functional / dissociative seizures (FDS) are the main differential diagnosis of convulsive status epilepticus (CSE). Previous studies suggest that at least 10% of patients brought to hospital with continuing seizures have FDS. However many of these patients are initially misdiagnosed: over one quarter of patients initially thought to have presented with CSE are subsequently diagnosed with a prolonged FDS. A correct diagnosis of FDS is even more likely among patients who presenting repeatedly with apparent CSE, or among those who fail to respond to first line treatments for CSE. The risk of misdiagnosis is particularly high in settings without immediate access to neurological expertise. The misdiagnosis and mistreatments of FDS can cause serious iatrogenic damage: one in ten patients with prolonged FDS erroneously treated for CSE with benzodiazepines will end up being intubated. Deaths from inappropriate treatment of FDS as CSE have been reported.

While epileptologists are now familiar with the typical characteristics of FDS, the training of first responders and emergency clinicians in the recognition and treatment of prolonged FDS is an ongoing challenge. Although the motor manifestations of these seizures can superficially resemble those of bilateral tonic clonic seizures, closer observation reveals many differences. Readily observable pointers to a diagnosis of FDS include closed eyes and mouth during a convulsive phase, resistance to eye opening, maintained pupillary light response, sinusoidal instead of clonic movements and partial responsiveness.

Inexpert clinicians tend to feel that they need to “err on the side of caution” when they balance the risks of failing to treat CSE with those of treating FDS inappropriately with benzodiazepines. However, FDS represent a dissociative state in which a person is disconnected from their environment and their level of self-control is diminished. Benzodiazepines may increase the level of dissociation and reduce the patient’s ability to regain self-control: Several studies suggest that the administration of these first line treatments for CSE can cause FDS to persist and escalate and thereby to increase the risk of iatrogenic injury.

In the absence of evidence based on research, clinical experience suggests that the most effective treatment for FDS begins with the recognition of the diagnosis and its communication to patient, accompanying persons and other members of staff. FDS often stop quickly when the level of anxiety surrounding the patient can be reduced. Clinicians can help the patient to reconnect with their body and their environment by using their name, talking to them and by acknowledging their distress.

Functional/dissociative seizures and SE – epidemiological aspects

Antonia Villagrán, Oslo, Norway

Diagnosing patients with prolonged seizures remains challenging. Functional/dissociative seizures (FDS) is an important differential diagnosis to SE. Diagnosing prolonged seizures correctly is crucial for accurate treatment and care.
The talk will focus on the following aspects:

(I) Do health care professionals diagnose patients with prolonged seizures correctly?
(II) How often are prolonged FDS misdiagnosed as SE (and vice versa) and does it matter?
(III) How to handle patients with prolonged FDS

Risk of epilepsy after a first post-traumatic seizure

Markus Karlander MD, PhD. Institute of Neuroscience and Physiology, Department of Clinical Neuroscience, Sahlgrenska Academy, Gothenburg University, Gothenburg, Sweden

Epilepsy is typically diagnosed after two unprovoked epileptic seizures occurring more than 24 hours apart. However, under certain circumstances, epilepsy can be diagnosed already after a first unprovoked seizure. This applies if the risk of seizure recurrence is particularly high. According to the current definition, this is defined as a risk of seizure recurrence of ≥60 % within 10 years (1). A previous brain injury, including a traumatic brain injury (TBI) is a factor that can cause an increased risk of seizure recurrence following a first seizure.

However, the risk of seizure recurrence following a first post-traumatic seizure (PTS) varies quite widely in the literature. According to recently conducted register-based study, the overall risk of seizure recurrence was found to be 41.1% within 10 years (2). This, in contrast to a smaller Italian study that found the risk to be 100% (3).

Although, the risk of seizure recurrence seems to be tightly linked to the severity of the previous TBI. Most studies have focused on individuals following more severe TBIs (4). It also seems that the time from the TBI to the first PTS play a role, with a lower risk of seizure recurrence with a longer duration from the TBI to the first seizure (2).

After the most severe TBIs, previous studies have shown a significantly increased risk of seizure recurrence following a first unprovoked seizure. In a recently conducted American study including 98 individuals with a first PTS following a severe TBI, the 2-year risk of seizure recurrence was 86% (5). In addition to another American conducted study on war veterans with penetrating brain injuries, among which the risk of seizure recurrence was 92% within 15 years (6).

After isolated mild or moderate TBI, the risk of seizure recurrence is less researched. However, it seems that the risk following milder TBI, including concussion and fracture of the face and skull, does not seem to be increased compared to a control group without a previous TBI, and following less severe structural brain lesions without focal deficits, the risk seems to be increased. However, the increased risk does not seem to motivate an epilepsy diagnosis (2).

Furthermore, the overall risk of seizure recurrence seems to decrease with time elapsed from the brain insult, similarly to the risk of post-stroke epilepsy (7).

In conclusion, a previous TBI will, in many cases increase the risk of seizure recurrence following an unprovoked seizure. However, the risk following the mildest TBI, does not seem to be increased. The risk of seizure recurrence seems to increase with the severity of the TBI. However, an epilepsy diagnosis following a first unprovoked seizure should probably in most cases be reserved for individuals with a previous severe TBI, particularly if the first post-traumatic seizure occurred shortly after the TBI.

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5. Pease M, et al. Ann Neurol. 2022;92(4):663-9.
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Refractory and Super-refractory Status Epilepticus in Children and Adolescents: A Population Based Study from Western Norway

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Refractory (RSE) and super-refractory status epilepticus (SRSE) are critical medical emergencies where timely management significantly impacts long-term outcomes. However, population-based clinical and epidemiological data on these conditions remain limited. This study aimed to provide a comprehensive overview of the epidemiology and clinical course of RSE and SRSE in children and adolescents while identifying potential prognostic biomarkers.

In this retrospective, population-based study, we included patients aged one month to 18 years who met the diagnostic criteria for RSE/SRSE and were admitted to the intensive care unit at Haukeland University Hospital between 2012 and 2021, Bergen, Norway. We systematically analyzed clinical and laboratory data, treatment approaches, and patient outcomes.

A total of 43 patients with 52 episodes of RSE/SRSE were identified, corresponding to an incidence rate of 3.13 per 100,000 per year. The median time from seizure onset to the administration of the first rescue medication was 13 minutes, while the median times to second- and third-line treatments were 83 minutes and 66 minutes, respectively.

Delays in treatment were observed throughout various stages of RSE/SRSE management. These findings highlight the need for improved strategies to ensure the prompt administration of rescue medications and timely escalation of treatment.

Reference:
Hepsø SW, Lee M, Noszka K, Wollertsen YM, Holmaas G, Kristensen E, Eichele T, Bjork MH, Griffiths ST, Hikmat O. Refractory and super-refractory status epilepticus in children and adolescents: A population-based study. Seizure. 2024;120:116-123. doi: 10.1016/j.seizure.2024.06.023.

Electronic patient care report of out-of-hospital emergency medical services saves lives and helps identify gaps in the emergency care pathway

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Finland is gradually taking into use a national system, in which patient data concerning out-of-hospital emergency medical care is recorded in common structured format as electronic patient care records (ePCR). In the future, emergency medical service (EMS) professionals will record patient data electronically using KEJO, the Common Authority Field Command System, which is used by all Finnish public protection and disaster relief authorities, including all EMS providers. From KEJO, emergency medical reports are stored in the Patient Data Repository Kanta.

KEJO and the ePCR respond to the challenges in communication between emergency care and other health care providers. From Kanta, EMS records can easily be viewed by health care professionals whenever patient care so requires. This allows information to flow smoothly between emergency care and other health care services. Through KEJO, professionals in emergency medical units also have access to patient data stored in Kanta by other health care service providers. This will make critical patient data available more quickly. In the future, patients will also be able to view their EMS records in MyKanta after their emergency care episode.

Emergency service registry formed from the structured ePCRs can be used for development and education of emergency medical care including EMS for prolonged seizures and status epilepticus. Structured ePCRs are also very useful in status epilepticus trials in determining the duration of the seizure and checking other immediate inclusion and exclusion criteria and in other consent-based status epilepticus research. As secondary use of the data for research purposes the regional and national registry data can be also combined with other relevant registries to determine long term outcomes and effectiveness of EMS.

All wellbeing services counties will deploy the ePCRs until the end of 2025, but the Wellbeing services county of North Savo in Kuopio area has piloted the system and has used it already since 2023. We will report preliminary data of the use of the ePCR in EMS for SE research.

Postictal abnormalities in related to status epilepticus in the adult patient: A systematic review and metaanalysis

Andrea Enerstad Bolle, Department of Neurology, Odense University Hospital, Denmark

Status Epilepticus (SE) is a life-threatening neurological emergency requiring promt intervention. Magnetic Resonance Imaging (MRI) has become essential for the work-up in SE, often revealing the underlying etiology, as well as postictal abnormalities (PMAs). These changes can be both reversible and irreversible, which may contribute to the long-term sequela following SE. Most existing literature derive from case studies, but recently larger cohorts have been published from several clinical centers. However, no studies to date have investigated the potential correlation between the localization of PMAs and mortality and morbidity outcomes at discharge.
We performed a systematic review of the current literature on PMAs and conducted a meta-analysis on the available data, with a focus on patient outcome in relation to PMA placement. This presentation will provide an overview of the results from the meta-analysis, demonstration a significant association between the location of PMAs and patient prognosis.

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Abstracts

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