Regionaalhaigla

ECT for Refractory *Status Epilepticus*

Margus Lõokene

NACT Conference 2021

Snekkersten, October 15th


IMPULSSRAVI

ECT in NEMC

- 1944 May
- 1946: 2117 ECT sessions
- 1951: „Soviet Psychiatric Theory“: reduce ECT!
- Next decades 4 times less, but never restricted
- 1980: 5 ECT devices
- 1994: modified ECT
- 2010: RUL ub ECT
- Nowadays: ~1500 ECT sessions/y

Definitions

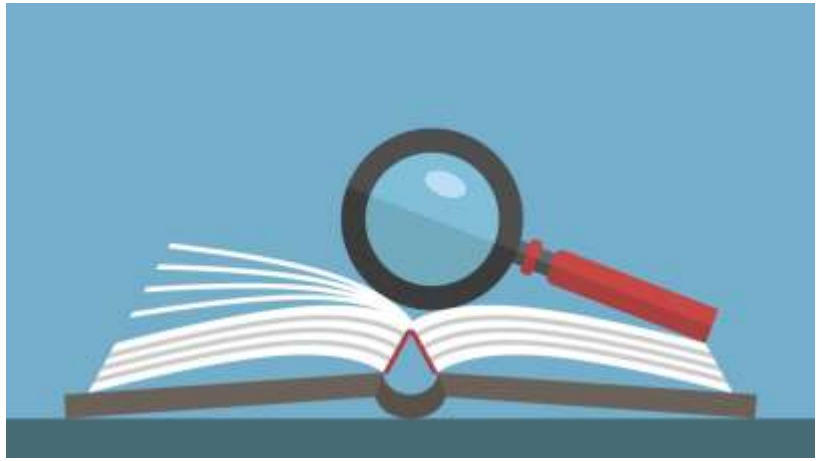
- *Status epilepticus* (SE) is a condition resulting either from the failure of the mechanisms responsible for seizure termination or from the initiation of mechanisms which lead to abnormally prolonged seizures
  - ✓ SE can lead to long-term consequences, incl neuronal death, neuronal injury, and alteration of neuronal networks depending on the type and duration of seizures
- If first- and second-line treatment is unsuccessful, SE is considered refractory (RSE)
  - ✓ Drugs must be administered promptly and in adequate doses
  - ✓ 10-30 % SE become RSE
- If third-line treatment is unsuccessful and/or SE recurse the condition is interpreted as super-refractory status epilepticus (SRSE)
  - ✓ 20 % RSE become SRSE

Neurotherapeutics (2018) 15:697–712
https://doi.org/10.1007/s13311-018-0640-5

CURRENT PERSPECTIVES

Treatment of Refractory and Super-refractory Status Epilepticus

Samhitha Rai[1] · Frank W. Drislane[1]



Egor, 6 years old

- Several colds reported in 2015
- January 10th, 2016
  - high fever (39 °C), CRP 32 -> tonsillitis
- January 13th
  - Seizures -> i/v diazepam -> referred to Children's hospital
  - Intubated, intensive care unit
- January 14th
  - CT: hypodensity in temporal lobe
  - EEG: focal start of the seizure from right posterior temporal lobe + secondary generalization
  - Propofol -> no effect
- January 15th
  - EEG: multifocal irritation, 2nd epileptic foci left fronto-temporal

  …
  - S. Tiopenthal 2,5% 4-5 mg/kg/h
  - Topiramate, carbamazepine, pregabaline and levetiracetam in maximal dosages
  - MRI: bilateral swelling in hippocampal regions
  - Topiramate, carbamazepine, pregabaline and levetiracetam in maximal dosages
  - Plasmapheresis, IVIG, i/v steroids, cytostatics
  - Tiopenthal 2,5% 4-5 mg/kg/h
- March 04th
  - \+ vigabatrin
  - ➢ „Super-refractory!“

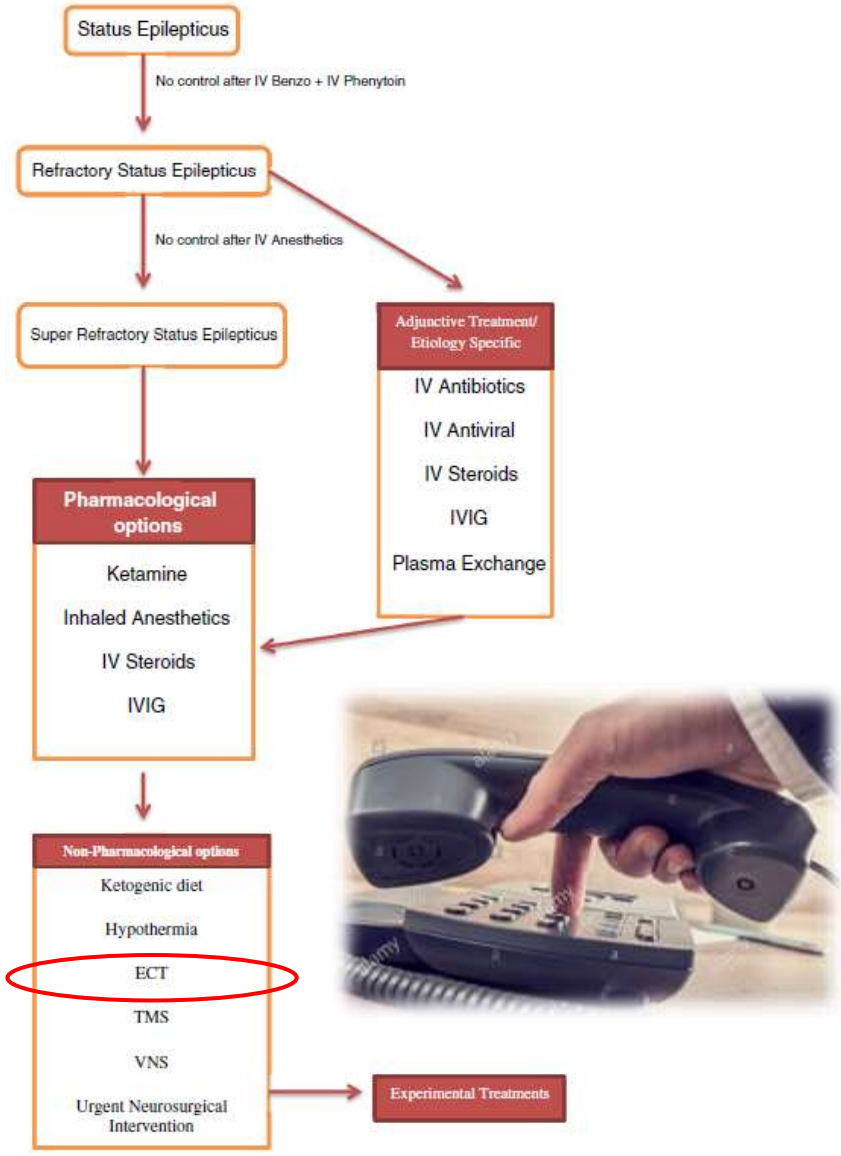
Curr Neurol Neurosci Rep (2015) 15: 66
DOI 10.1007/s11910-015-0589-2

EPILEPSY (CW BAZIL, SECTION EDITOR)

Treatment of Super-Refractory Status Epilepticus

Ahmad Bayrlee[1] · Nimalya Ganeshalingam[2] · Lisa Kurczewski[3] · Gretchen M. Brophy[4]

Intervention	Studied doses	Adverse effects	Clinical pearls and considerations
Ketogenic diet	4:1 (the ratio of fat to carbohydrates and protein)	Hyperlipidemia, weight loss. Contraindicated in pyruvate carboxylase and beta-oxidation deficiency	Unlikely compliance with long-term use of the diet due to social and dietary restrictions, cost, and the complexity involved. Lack of well-designed trials
Hypothermia	Goal temperature of 32–35 °C×24 h with rewarming of no more than 0.5 °C per hour	Coagulation disorders, venous thrombosis, cardiac arrhythmia, electrolyte abnormalities, infections, pharmacokinetic and pharmacodynamics changes, and acute intestinal ischemia/necrosis	Hypothermia can potentially be used as an alternative to two or more unsuccessful EEG burst suppression trials. Goal temperature aimed at appropriate burst suppression pattern on EEG
Electroconvulsive therapy	Protocols vary	Can induce convulsive and non-convulsive status epilepticus after treatment, cognitive impairment, amnesia, and headache	EEG monitoring required Routine use not well established, further studies are needed
Transcranial magnetic stimulation	Can be performed in the ICU setting	Rare seizures, headache, dizziness, and other neurological side effects	Considered a very safe intervention and does not require surgery or device implantation. Still investigational therapy
Vagal nerve stimulator	Surgical implantation	Voice hoarseness, infection risk at the implantation site, and rare bradycardia	No strong evidence to support its use in the acute settings



„Seizures against seisures?“

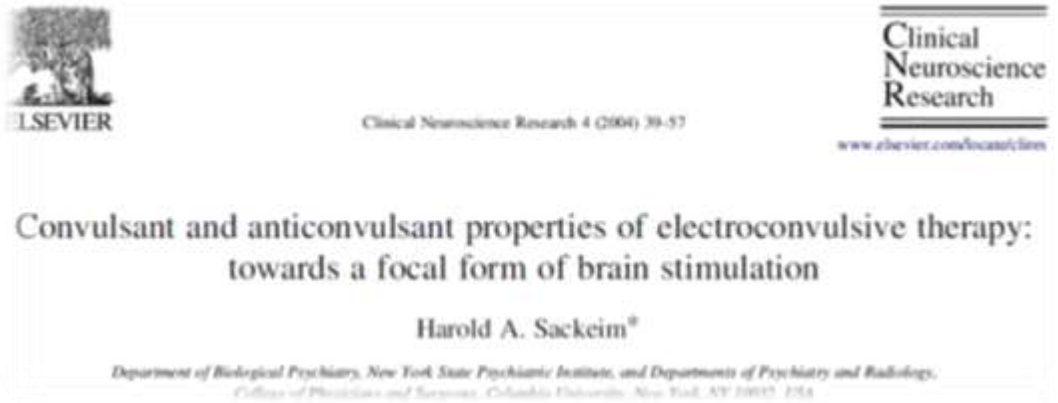
A compensatory increase in the function of inhibitory neurotransmission (GABA) in the brain responsible for ECT´s antidepressant and anticonvulsive properties

- Evidence pro
  - Increase in ST
  - Change in the nature of the seizure
  - Topographic changes in blood supply and metabolism
  - Animal models (*kindling hypothesis*)
  - Increase in inhibitory neurotransmission (GABA, opioid)
  - Neurogenesis

Anticonvulsant and antidepressant properties of electroconvulsive therapy: a proposed mechanism of action

H A Sackeim, P Decina, I Prohovnik, S Malitz, S R Resor

Biol Psychiatry. 1983 Nov;18(11):1301-10.


ELSEVIER

Clinical Neuroscience Research 4 (2004) 39–57

Clinical Neuroscience Research

www.elsevier.com/locate/clinn

Convulsant and anticonvulsant properties of electroconvulsive therapy: towards a focal form of brain stimulation

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ECT vs SE in the literature?

- Limited scientific data published
- Retrospective case-reports
- Place in ECT handbooks
- Place in RSE guidelines

Seizure 20 (2011) 433–436

Contents lists available at ScienceDirect

Seizure

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Case report

Successful ECT treatment for medically refractory nonconvulsive status epilepticus in pediatric patient

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ABSTRACT

Status epilepticus is a life threatening condition with a high mortality rate in spite of aggressive treatment. There is little consensus on third and fourth line approaches in refractory cases. While electroconvulsive therapy (ECT) has been employed successfully as a treatment for refractory epilepsy and status epilepticus (SE) after exhausting conventional therapy, its use for pediatric patients is limited. We describe a 7-year-old pediatric case in which ECT was used successfully to treat medically refractory nonconvulsive status epilepticus (NCSE) without complete withdrawal of antiepileptic drugs (AED).

Review

Refractory status epilepticus: Electroconvulsive therapy as a possible therapeutic strategy

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Review

Electroconvulsive therapy for refractory status epilepticus: A systematic review

F.A. Zeiler [a,*], M. Matuszczak [b,c], J. Teitelbaum [c,d], L.M. Gillman [d,e,f], C.J. Kazina [a,g]

Review

Electroconvulsive therapy for refractory status epilepticus: A systematic review

F.A. Zeiler [a,*], M. Matuszczak [b,1], J. Teitelbaum [c,2], L.M. Gillman [d,e,3], C.J. Kazina [a,4]

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ABSTRACT

Background: Our goal was to perform an extensive systematic review of the literature on the use of electroconvulsive therapy (ECT) for refractory status epilepticus (RSE).
Methods: Articles from MEDLINE, BIOSIS, EMBASE, Healthstar, Global Health, Scopus, Cochrane Library, the International Clinical Trials Registry Platform, clinicaltrials.gov (inception to August 2015), reference lists of relevant articles, and gray literature were searched. The strength of evidence was adjudicated using both the Oxford and GRADE methodology by two independent reviewers.
Results: We identified 14 original articles with a total of 19 patients receiving ECT for RSE. Of the 19 patients, 15 were adult, and 4 were pediatric. All studies were retrospective in nature. Seizure reduction/control with the application of ECT occurred in 11 of the 19 patients (57.9%), with 4 (21.0%) and 7 (36.8%) displaying partial and complete responses respectively. Seizures control lasted for variable duration, with the most commonly quoted duration ranging from 2 weeks to 3 months. Data on patient functional outcome was available in 13 patients, with 10 patients falling into the categories of dead or severely disabled. All studies were an Oxford level 4, GRADE D level of evidence.
Conclusions: Oxford level 4, GRADE D evidence exists to suggest an improvement in seizure control with ECT application for RSE. Routine use of ECT cannot be recommended at this time. Further prospective study of this therapy is required in order to determine its efficacy in this setting.

Table 1
Study Characteristics and Patient Demographics.

Reference	Number of patients treated with ECT	Study type/ design	Article location	Mean age (years)	Etiology of seizures and type of SE/RSE	Mean # meds prior to ECT	Mean tim until ETC treatmen (days)
Carrasco et al.[9]	1	Retrospective Case report	Journal Manuscript	25	Etiology: post traumatic changes to frontal/temporal lobes Type: GRSE	5	40
Cline et al.[10]	1	Retrospective Case Report	Journal Manuscript	39	Etiology: HSV encephalitis Type: GRSE	8	103
Fernandez-Torre et al.[11]	1	Retrospective case Report	Journal Manuscript	59	Etiology: post traumatic epilepsy Type: FRSE	14	>25
Griesemer et al.[12]	2	Retrospective Case Series	Journal Manuscript	11.5 (13 yrs and 10 yrs)	1—Etiology: microgyria Type: GRSE 2— Etiology: microcephaly Type: FRSE	5 (8 and 2)	NA
Kamel et al.[13]	3	Retrospective Case Series	Journal Manuscript	33 (32, 41, and 26 yrs)	1— Etiology: viral encephalitis Type: GRSE 2— Etiology: viral encephalitis Type: GRSE 3— Etiology: infectious cerebritis Type: GRSE	7 (6, 5, and 7)	44
Koong et al.[14]	1	Retrospective Case Report	Journal Manuscript	54	Etiology: epilepsy with psychosis Type: NCRSE	4	NA
Lisanby et al.[15]	1	Retrospective Case Report	Journal Manuscript	36	Etiology: bifrontal cortical dysplasia Type: FRSE	7	26
Moddel et al.[16]	3	Retrospective Case Series	Meeting Abstract	68 (59, 65, and 80 yrs)	1— Etiology: bitemporal encephalitis Type: NCRSE 2— Etiology: resection of meningiomas Type: GRSE 3— Etiology: subdural hematoma Type: GRSE	8 (8, 9, and 6)	44
Morales et al.[17]	1	Retrospective Case Report	Journal Manuscript	8	Etiology: lipofuscinosis Type: FRSE	7	NA
Regenold et al.[18]	1	Retrospective Case Report	Journal Manuscript	71	Etiology: primary epilepsy Type: FRSE	5	9
Savard et al.[19]	1	Retrospective Case Report	Meeting Abstract	27	Etiology: POLG1 Epilepsy Type: GRSE	11	NA
Shin et al.[20]	1	Retrospective Case Report	Journal Manuscript	7	Etiology: refractory epilepsy Type: NCRSE	7	14
Vipanelli et al.[21]	1	Retrospective Case Report	Journal Manuscript	19	Etiology: primary epilepsy Type: FRSE	3	NA
Wusthoff et al.[22]	1	Retrospective Case Report	Journal Manuscript	29	Etiology: Rasmussen's Encephalitis Type: FRSE	10	NA
Shin et al.[23]	1	Retrospective Case Report	Meeting Abstract	7	Etiology: refractory epilepsy	NA	NA

Review

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Table 2
ECT Treatment Characteristics and Outcomes.

Reference	# of patients Treated with ECT	Electrode Positioning	ECT Treatment Regimen	Other AED On Board	Electrographic Seizure Response	Duration of Response	Adverse Effects to ECT	Patient Outcome
Carrasco et al.[5]	1	NA	6 sessions (3/week)	Phenytoin Diazepam Carbamazepine Pentobarbital Phenobarbital	Complete resolution	NA	NA	Completely recovered within 1 month
Cline et al.[6]	1	Bifronto-temporal	9 sessions (3/d for 3 consecutive days) Current=800 mA Charge=576 mC Pulse frequency = 90 Hz Pulse Width = 1 s	Valproate Pentobarbital Levetiracetam Phenytoin Oxcarbazepine Topiramate Lorazepam Felbamate	Improvements with diffusely slow activity with a delayed response over time	16 months	NA	Patient continues to have focal seizures. Significant neurological deficits from which there is slow recovery. Died
Fernandez-Torre et al.[11]	1	NA	NA	Phenobarbital Oxcarbamazepine Valproate Clonazepam Levetiracetam Carbamazepine Topiramate Phenytoin Lamotrigine Vigabatrin Propofol Midazolam Thiopental Lidocaine	Failed	NA	NA	
Grieserner et al.[12]	2	NA	1→ 4 total series. #1 (week 1) 4 sessions over 9d #2: (week 5,6) 3 sessions over 3d #3: (week 82) 3 daily sessions over 2d #4: (week 84) 3 sessions in 1d. 2→ 3 total series #1 (week 1) 3 sessions over 5d #2 (week 3) 3 sessions over 2d #3 (week 5) 3 sessions over 2d	1→ Phenobarbital Phenytoin Acetazolamide Clonazepam Valproate Gabapentin Lamotrigine Felbamate 2→ Gabapentin Valproate	1→ mild improvement in seizure frequency 2→ transient reduction in the number of spontaneous seizures	1→ 1 year for 1st series, 1 month for 2nd series. 3rd and 4th series were effective for 2 weeks 2→2 weeks	1→ lethargic between seizures 2→ NA	ECT response was transient in both patients
Kamel et al.[13]	3	Bifronto-temporal	All 3 patients received the following protocol: 4 sessions over 3d Current = 0.9 A Charge = 507 mC Pulse frequency = 70 Hz Pulse Width = 0.5ms Patient 3 received 2 courses of this protocol.	1→ Valproate Levetiracetam Topiramate Ketamine Phenobarbital Phenytoin 2→ Phenytoin Levetiracetam Valproate Pentobarbital Ketamine 3→ Phenytoin Valproate Topiramate Phenobarbital Levetiracetam Isoflurane Ketamine	1→ complete resolution 2→ no response 3→seizures decreased in frequency	1→ indefinite 2→ NA 3→ indefinite	1→ retrograde amnesia 2→ NA 3→ mild retrograde amnesia	1→ seizures resolved 2→ died. Multi-organ complication leading to acute kidney failure 3→continues to have seizures (no SE) but is able to live independently

Table 2 (Continued)

Reference	# of patients Treated with ECT	Electrode Positioning	ECT Treatment Regimen	Other AED On Board	Electrographic Seizure Response	Duration of Response	Adverse Effects to ECT	Patient Outcome
Koenig et al.[14]	1	NA	Received ECT sessions 2/week for 6 weeks	NA	Transient seizure cessation	~1 week.	NA	Seizures gradually reappeared and patient was maintained with weekly ECT sessions SE resolved, but patient remains comatose
Usseby et al.[15]	1	Right frontotemporal & left parietal	5 consecutive daily sessions Current = 0.8A Charge = 576-1370mC Pulse frequency = 86-120 Hz Pulse Width = 1-1.4ms	Vigabatrin Phenobarbital Nitrazepam Phenytoin Midazolam Phenobarbital Pentobarbital	Resolution of continuous epileptiform activity	2 months	NA	
Shaldel et al.[16]	3	NA	Patient 1 & 3: 3 sessions Patient 2: 1 session	1→ Midazolam Propofol Phenytoin Valproate Levetiracetam Topiramate Oxycarbazepine Lidocaine 2→Lorazepam Phenytoin Propofol Valproate Levetiracetam Lamotrigine Topiramate Lidocaine Thiopental 3→Lorazepam Phenytoin Propofol Thiopental Levetiracetam Valproate	no response	NA	NA	ECT did not terminate SE and functional outcome was poor in all patients
Morales et al.[17]	1	Bilateral	5 consecutive daily sessions Current = NA Charge = 64-1698mC Pulse frequency = 40-120 Hz Pulse Width = 1-1.5 ms	Lorazepam Fosphenytoin Oxycarbezapine Lamotrigine Zonisamide Phenobarbital Levetiracetam	no response	NA	None	No clinical improvement. Patient died 3 months later
Rogenski et al.[18]	1	Bilateral	8 sessions over 16 days	Phenytoin Phenobarbital Diazepam Lorazepam Carbamazepine	Resolution of SE after 8th session	6 months	None	Seizure disorder was under control, but patient continues to partial-complex seizures
Sanord et al.[19]	1	NA	NA	Oxycarbazine Levetiracetam Lacosamide Clobazam Topiramate Valproate Midazolam $MgSO_4$ Ketamine Isoflurane Barbiturates	no response	NA	NA	Died 75d post admission

Table 2 (Continued)

Reference	# of patients Treated with ECT	Electrode Positioning	ECT Treatment Regimen	Other AED On Board	Electrographic Seizure Response	Duration of Response	Adverse Effects to ECT	Patient Outcome
Shea et al.[20]	1	Bilateral	3 daily sessions on 2 consecutive days. This regimen was repeated twice/5 days apart Current = 0.8A Charge = NA Pulse frequency = 60-120Hz Pulse Width = 0.27-2ms	Valproic acid Topiramate Levetiracetam Clobazam Midazolam Ketamine Pentobarbital	NCSE stopped with generalized slowing and bilateral independent periodic lateralized epileptiform discharges.	1st round: 2 days 2nd round: 11 weeks	NA	Patient improved to awful status and alertness over 11 weeks before suddenly passing away
Vigneli et al.[21]	1	NA	2 sessions 48h apart Current = sinusoidal Voltage = 140V Duration = 0.6s	Clonazepam Diazepam Phenytoin	Resolution of spike activity after 2nd session.	8 years	NA	Patient remained seizure free on carbamazepine
Westhoff et al.[22]	1	NA	10 sessions	Pentobarbital Phenytoin Levetiracetam Phenobarbital Midazolam Valproate Topiramate Pregabalin Felbamate Perampanel	No response	NA	NA	Patient responded to ketogenic diet
Shin et al.[23]	1	Bilateral	3 daily sessions on 2 consecutive days. This regimen was repeated twice/5 days apart	Valproic acid Topiramate Levetiracetam Clobazam Midazolam Ketamine Pentobarbital	Seizure cessation	1st round: 2 days 2nd round: 11 weeks	NA	Patient is more alert but continues to have absence seizures.

Review

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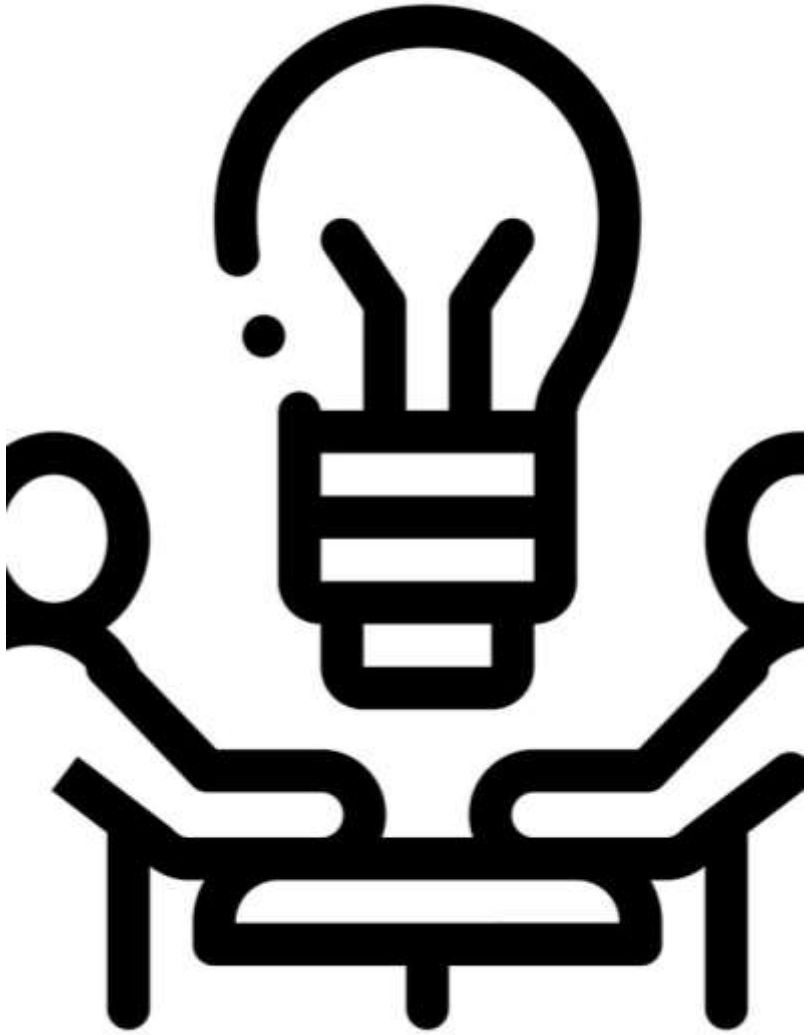
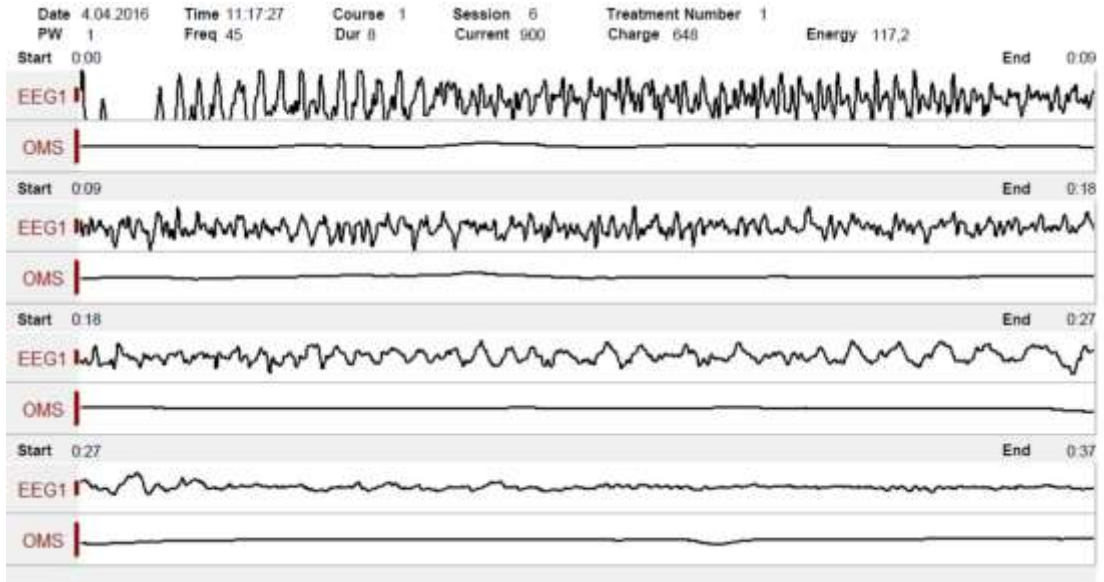
F.A. Zeiler [a,*], M. Matuszczak [b,1], J. Teitelbaum [c,2], L.M. Gillman [d,e,3], C.J. Kazina [a,4]



Egor, 6 years old

- March 24th: 1st ECT
  - BL, titration: 288 mC -> 648 mC
- March 24th, 26th: thiopental stopped
- + 7 ECT sessions 3/week
- + 2 ECT sessions 2/week
- Last ECT: April 20th



Egor, 6 years old

- April 20th –> neurology department
- Started to talk (though incoherently) , got better on motor activity.
- Main focus on physiotherapy
- Occasional seizures remained
- Remained in combined antiepileptic treatment.
- EEG (several): no intericatal epileptiformic activity
- May 26th: discharged from hospital

- **The cause remained unsolved**: diagnosed autoimmune encephalities without known cause

Egor, 12 years old

- Goes to special school for students with learning difficulties
- Emotional blunting, cognitive disabilities
- Needs assistance in complex activities
- Short seizures occur occasionally
- IVIG once a month
- Topiramate, pregabaline, levetiracetam, phenobarbital, carbamazepine

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journal homepage: http://www.journals.elsevier.com/brain-stimulation

Neuromodulation techniques for status epilepticus: A review

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ABSTRACT

Background: Electroconvulsive therapy (ECT), Vagal Nerve Stimulation (VNS), Transcranial Magnetic Stimulation (TMS) and Deep Brain Stimulation (DBS) are neuromodulation therapies that have been used to treat Status Epilepticus (SE).
Objective: Review the literature about the efficacy and safety of neuromodulation therapies in SE in humans.
Methods: We searched studies in PubMed, Scopus, Google Scholar and Science Direct (inception to June 2018). Four review authors independently selected the studies, extracted data and assessed the methodological quality of the studies using the recommendations of the Cochrane Handbook for Systematic Reviews of Interventions, PRISMA guidelines, Oxford and GRADE scales, and Murad et al., 2018 methodological quality and synthesis of case series and case reports.
Results: We analyzed 27 articles (45 patients) with 4 different neuromodulation therapies. In ECT we found 80% rate of disruption of SE and 5% of adverse events was reported. Using iVNS 15/16 (93.7%) patients resolved the SE. All patients who underwent TMS and DBS aborted SE, however, 50% of patients with DBS had severe adverse events.
Conclusions: Case series and case reports suggest that neuromodulation therapies can abort SE in 80–100% of patients (Oxford scale and GRADE were level 4 and D) with a wide range of adverse effects, which claims for prospective studies on the relationship be-tween efficacy and safety.

Epilepsia, 52(Suppl. 8):61–63, 2011
doi: 10.1111/j.1528-1167.2011.03240.x

FUTURE PERSPECTIVES, NOVEL THERAPY, AND INNOVATION

The potential of brain stimulation in status epilepticus

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Summary

There is a long history of the use of brain stimulation in the treatment of epilepsy but relatively little experience for its use in status epilepticus. Electroconvulsive therapy, transcranial magnetic stimulation, subcortical and cortical stimulation have all been tried with varying degrees of success in single cases or small case series. It remains unclear, however, which brain areas should be stimulated and the parameters that should be used. Moreover, the aim (stopping status epilepticus) is different from preventing seizures and so the brain areas and parameters that are useful in epilepsy may not directly translate to the treatment of status epilepticus.

KEY WORDS: Status epilepticus, Thalamic stimulation, Electroconvulsive therapy, Transcranial magnetic stimulation.

Conclusions

- ECT has a role in the treatment of RSE
  - ✓Add-on treatment
  - ✓In case of an urgent need
  - ✓C-ECT should be considered
- ECT is very safe!