



**CONVEGNO REGIONALE SIN / SNO
Liguria - Piemonte e Valle d'Aosta**

Ivrea, 6-7 dicembre 2019
Università infermieristica di Ivrea

**NEURO-NEWS:
INNOVAZIONI DIAGNOSTICHE E TERAPEUTICHE IN NEUROLOGIA
LE NOVITÀ DELL'ULTIMO ANNO DA NON PERDERE**

Moderatori: **FABIO BANDINI, MICHELE DOTTA**

Epilessia – Paolo Benna



La nuova nosografia e definizione

ILAE POSITION PAPER

ILAE classification of the epilepsies: Position paper of the ILAE Commission for Classification and Terminology

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Epilepsia, **(*) :1–10, 2017

ILAE POSITION PAPER

Operational classification of seizure types by the International League Against Epilepsy: Position Paper of the ILAE Commission for Classification and Terminology

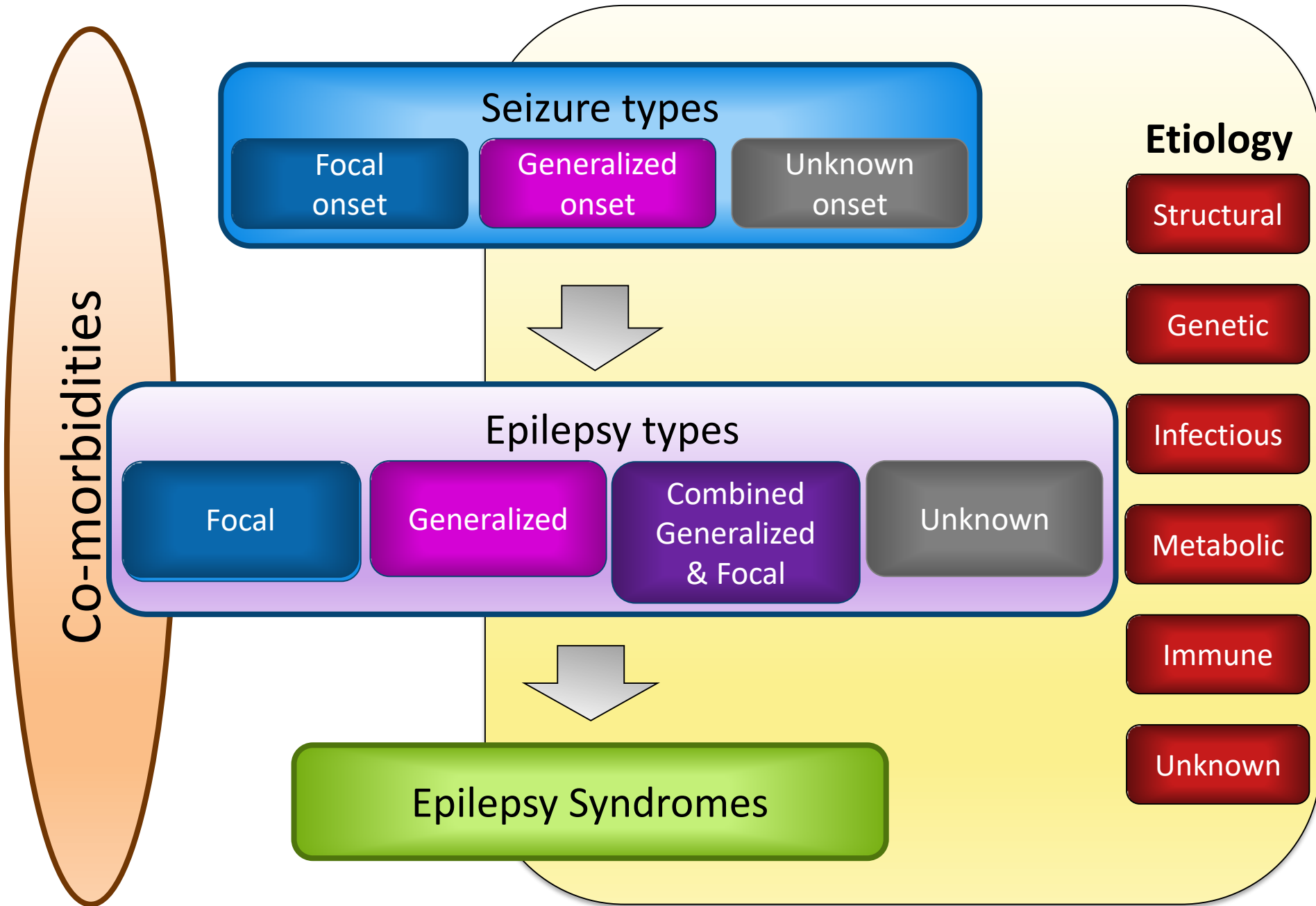
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Epilepsia, **(*) :1–9, 2017
doi: 10.1111/epi.13670

Critique of the 2017 epileptic seizure and epilepsy classifications

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This article critiques the International League Against Epilepsy (ILAE) 2015–2017 classifications of epilepsy, epileptic seizures, and status epilepticus. It points out the following shortcomings of the ILAE classifications: (1) they **mix semiological terms with epileptogenic zone terminology**; (2) **simple and widely accepted terminology has been replaced by complex terminology containing less information**; (3) seizure evolution cannot be described in any detail; (4) in the four-level epilepsy classification, level two (epilepsy category) overlaps almost 100% with diagnostic level one (seizure type); and (5) **the design of different classifications with distinct frameworks for newborns, adults, and patients in status epilepticus is confusing**. The authors stress the importance of validating the new ILAE classifications and feel that the decision of *Epilepsia* to accept only manuscripts that use the ILAE classifications is premature and regrettable.



A practical clinical definition of epilepsy

*Robert S. Fisher, †Carlos Acevedo, ‡Alexis Arzimanoglou, §Alicia Bogacz, ¶Helen Cross, #Christian E. Elger, **Jerome Engel Jr, ††Lars Forsgren, ‡‡Jacqueline A. French, §§Mike Glynn, ¶¶Dale C. Hesdorffer, ###B.I. Lee, ***Gary W. Mathern, †††Solomon L. Moshé, ‡‡‡Emilio Perucca, §§§Ingrid E. Scheffer, ¶¶¶Torbjörn Tomson, ####Masako Watanabe, and *****Samuel Wiebe

Epilepsia, 55(4):475–482, 2014
doi: 10.1111/epi.12550

Table 1. Conceptual definition of seizure and epilepsy – 2005 report

An epileptic seizure is a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain.

Epilepsy is a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures, and by the neurobiologic, cognitive, psychological, and social consequences of this condition. The definition of epilepsy requires the occurrence of at least one epileptic seizure.

Epilepsia, 55(4):475–482, 2014
doi: 10.1111/epi.12550

Table 2. Operational (practical) clinical definition of epilepsy

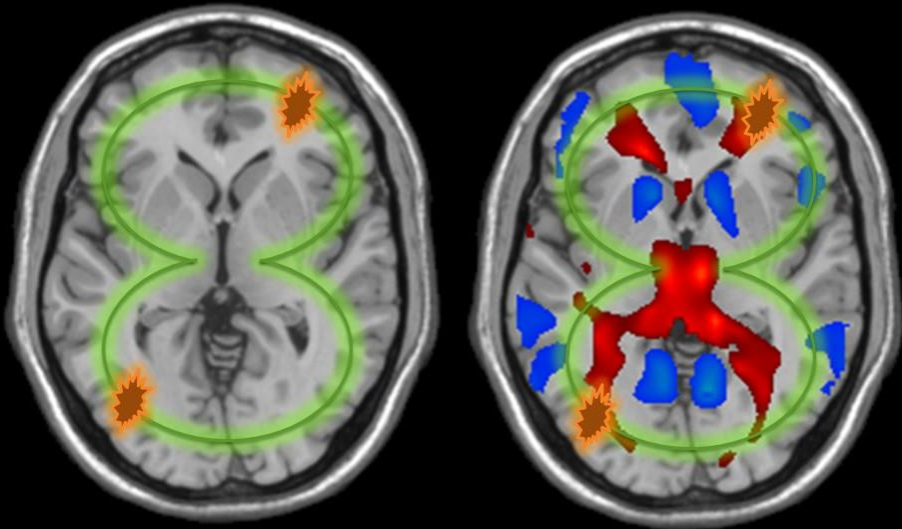
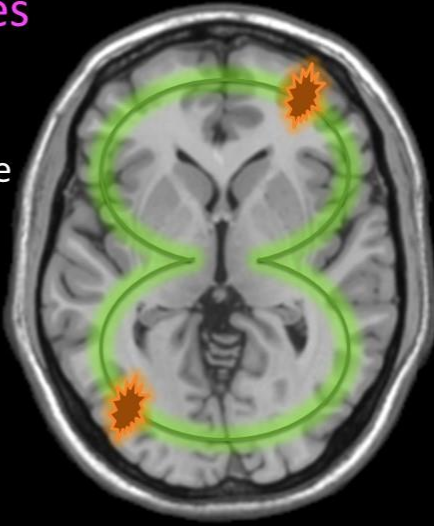
Epilepsy is a disease of the brain defined by any of the following conditions

1. At least two unprovoked (or reflex) seizures occurring >24 h apart
2. One unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years
3. Diagnosis of an epilepsy syndrome

Epilepsy is considered to be *resolved* for individuals who had an age-dependent epilepsy syndrome but are now past the applicable age or those who have remained seizure-free for the last 10 years, with no seizure medicines for the last 5 years.

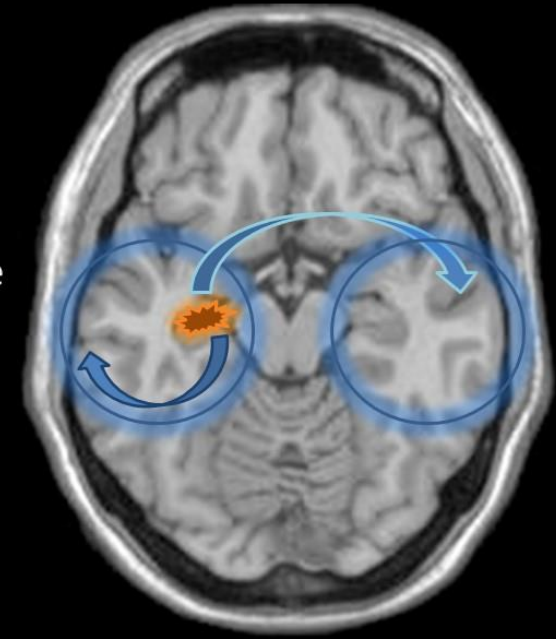
Generalized seizures

- Originate at some point within and rapidly engage bilaterally distributed networks
- Can include cortical and subcortical structures but not necessarily the entire cortex



Focal seizures

- Originate within networks limited to one hemisphere
- May be discretely localized or more widely distributed...



- Le metodiche di esplorazione funzionale con imaging evidenziano la maggiore complessità del processo epilettico, ben più da riferire - anche nelle crisi 'focali' - a circuiti che ad aree corticali limitate.
- *E' possibile che le parole utilizzate per esprimere il concetto non siano quelle più appropriate.*

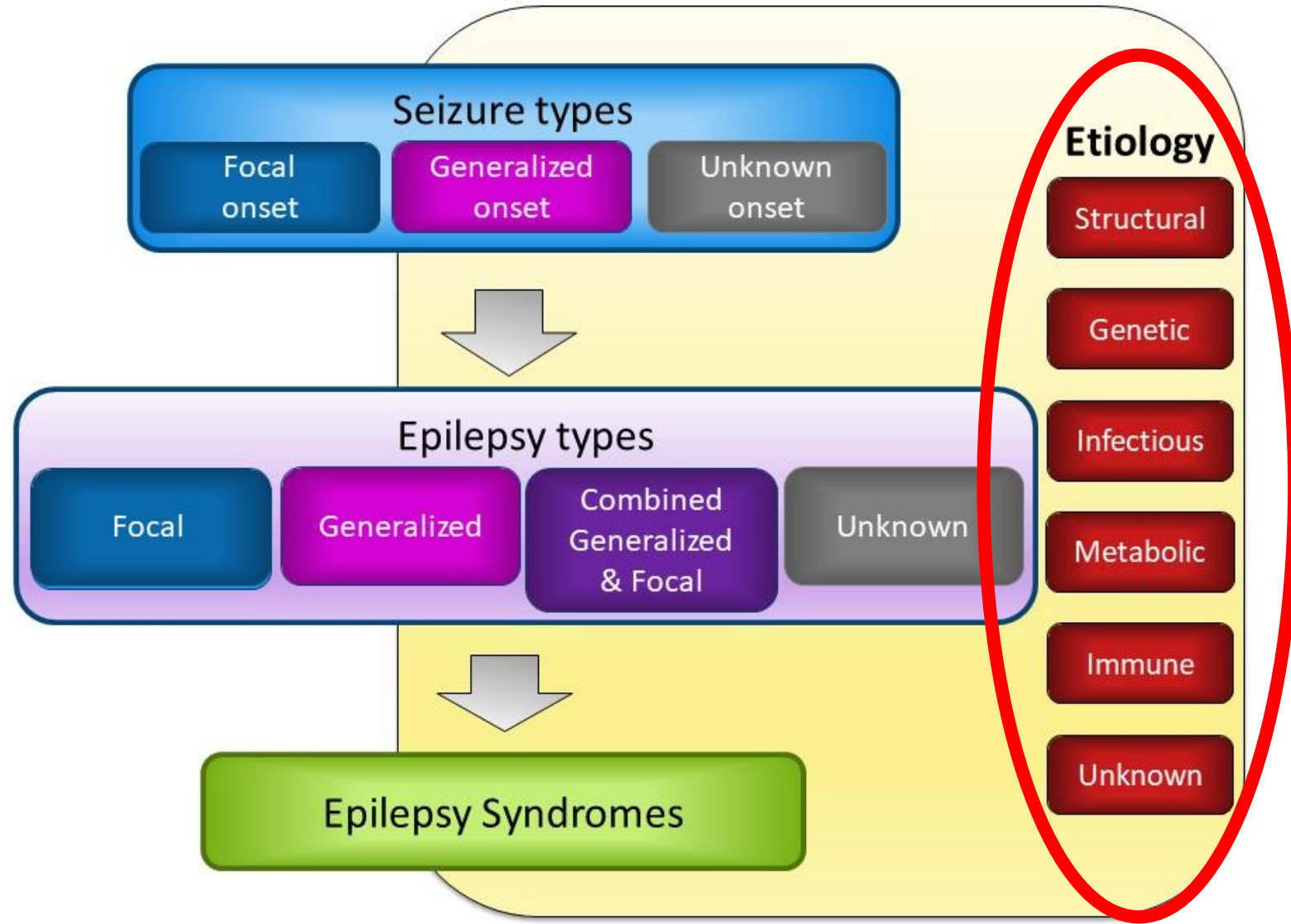
Terms no longer in use

- Complex partial
- Simple partial
- Partial
- Psychic
- Dyscognitive
- Secondarily generalized tonic-clonic



Epilepsy syndromes

- There are **no** approved ILAE epilepsy syndromes



Encefaliti / epilessie disimmuni

REVIEW

The Journal of Clinical Investigation

Autoimmune seizures and epilepsy

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⁴Catalan Institution for Research and Advanced Studies (ICREA), Barcelona, Spain. ⁵Department of Neurology, University of Pennsylvania, Philadelphia, Pennsylvania, USA.

The rapid expansion in the number of encephalitis disorders associated with autoantibodies against neuronal proteins has led to an incremental increase in use of the term “autoimmune epilepsy,” yet has occurred with limited attention to the pathophysiology of each disease and genuine propensity to develop epilepsy. Indeed, most autoimmune encephalitides present with seizures, but the probability of evolving to epilepsy is relatively small. The risk of epilepsy is higher for disorders in which the antigens are intracellular (often T cell-mediated) compared with disorders in which the antigens are on the cell surface (antibody-mediated). Most autoantibodies against neuronal surface antigens show robust effects on the target proteins, resulting in hyperexcitability and impairment of synaptic function and plasticity. Here, we trace the evolution of the concept of autoimmune epilepsy and examine common inflammatory pathways that might lead to epilepsy. Then, we focus on several antibody-mediated encephalitis disorders that associate with seizures and review the synaptic alterations caused by patients' antibodies, with emphasis on those that have been modeled in animals (e.g., antibodies against NMDA, AMPA receptors, LGI1 protein) or in cultured neurons (e.g., antibodies against the GABA_B receptor).

Table 3. Antibody-mediated encephalitis, seizures, and estimated risk of epilepsy^A

Antigen	Seizures	Risk of epilepsy	General outcome
NMDAR	~75% of patients develop seizures, which are often the first symptom in children and young males (53). 11%–30% of adults and 6% of children have a highly characteristic EEG pattern (extreme delta brush) that associates with more severe symptoms (163, 164). Diffuse slowing and focal slowing are the most frequent EEG findings (42). A normal posterior rhythm on the first EEG predicts a favorable clinical outcome, while a severely abnormal EEG associates with poor outcome. In a few patients the EEG can be normal (164).	Low (<5%)	Good. ~80%–85% of patients with substantial or full recovery. Relapses in ~15%–20%.
AMPA	~30%–40% of patients develop seizures in the context of limbic encephalitis.	Low (~5%)	Depends on the control of the tumor. Otherwise, ~70% partial or full recovery. Relapses in ~16%.
GABA _B R	90%–95% of patients have early and prominent seizures in the context of limbic encephalitis. Can present with status epilepticus.	Low (5%)	Depends on the control of the tumor. Otherwise, ~70% partial or full recovery. Relapses can occur (frequency unknown).
LGI1	~40%–50% of patients present with faciobrachial dystonic seizures (FDS). EEG is often normal in patients with isolated FDS (165); some of these patients have MRI T1 and T2 basal ganglia hyperintensity (166). At the stage of encephalitis, multiple types of seizures (temporal lobe, focal, tonic-clonic, or autonomic) can occur (57, 167, 168). Low chance of seizure control unless immunotherapy is used.	~15% (some with hippocampal sclerosis)	~70%–80% partial or complete recovery, but only ~35% able to return to work. Relapses in 27%–34% (57, 155).
CASPR2	24% of patients present with seizures. Overall, 54% develop seizures during the course of the disease (50).	Exact risk of epilepsy unknown; probably low (<10%) (143)	48% full response to treatment, 44% partial response, 7% no response. Relapses in 25% (50).
GABA _A R	Seizures occurred in 88% of patients (48% developed status epilepticus). Compared with adults, children were more likely to have generalized seizures (59).	Exact risk of epilepsy unknown; probably moderate (20%–30%) (58)	23% complete recovery, 64% partial recovery, 13% death (status epilepticus or sepsis) (59).
mGluR5	6 of 11 patients presented with seizures. Compared with adults, children were more prone to develop generalized seizures and status epilepticus (49).	Low (5%)	6 of 11 patients had complete recovery and 5 partial recovery. None developed epilepsy (49).
D2R	2 of 12 patients developed seizures (159).	Low (5%)	5 of 12 patients had full recovery. None developed epilepsy. Relapses in 3 of 12 cases (159).
DPPX	Seizures in 10%–22% of patients (160, 169).	Not available (small number of patients)	60% substantial or moderate improvement, 23% no improvement (most not treated), 17% died (160, 169). Relapses in 23% (160).
GlyR	At disease onset, 13% of patients had seizures. 5 of 45 patients developed only encephalopathy with seizures (161).	Not available	Most patients with substantial or partial improvement; 11% died. Relapses in 14% (161).


^AExcludes neurexin-3α, as fewer than 10 patients reported.

Antigen (ref.)	Age, median years [range]; male:female	Main presenting symptoms	Main syndrome	Frequency (main types of cancer)	Brain MRI FLAIR/T2 sequences ^B
NMDAR (42)	21 [2 months–85 years]; 1:4	Children: seizures, dyskinesias; adults: behavior changes, psychiatric	Anti-NMDAR encephalitis ^C	Varies with age and sex ^D ; 58% of women 18–45 years old have ovarian teratoma	Normal (70%) or nonspecific changes

Antigen	Seizures	Risk of epilepsy	General outcome
NMDAR	~75% of patients develop seizures, which are often the first symptom in children and young males (53). 11%–30% of adults and 6% of children have a highly characteristic EEG pattern (extreme delta brush) that associates with more severe symptoms (163, 164). Diffuse slowing and focal slowing are the most frequent EEG findings (42). A normal posterior rhythm on the first EEG predicts a favorable clinical outcome, while a severely abnormal EEG associates with poor outcome. In a few patients the EEG can be normal (164).	Low (<5%)	Good. ~80%–85% of patients with substantial or full recovery. Relapses in ~15%–20%.

Antigen	Number of patients analyzed	Patients with long-term ^A follow-up	Seizure semiology	Clinical specificities	EEG pattern	Brain MRI	Warnings
NMDAR ^{13,17,19,20,22,25}	927	838	Temporal lobe seizures (impaired awareness and orofacial or manual automatisms) Seizures arising from fronto-central regions (tonic or clonic seizures)	Predominantly women under 45 y, half of them with an ovarian teratoma Stereotyped course Initial symptoms differ according to age and sex Seizures are sometimes difficult to distinguish from abnormal movements	Three EEG stages: (a) Excessive beta activity range 14-20 Hz (EBA); (b) Extreme delta brush (EDB); (c) Generalized delta activity (GDA)	Aspecific abnormalities in about 20% of the cases Demyelinating lesions in a subset of patients	Rhythmic aspect of GDA should not be misinterpreted as status epilepticus Subclinical epileptic discharges are observed in up to 20% of the patients

Seizure specificities in patients with antibody-mediated autoimmune encephalitis

Alberto Vogrig^{1,2,3,4}  | Bastien Joubert^{1,2,3} | Nathalie André-Obadia^{5,6} |
Gian Luigi Gigli^{4,7,8} | Sylvain Rheims^{5,6} | Jérôme Honnorat^{1,2,3}

Key Points

- Epilepsy is part of the clinical spectrum of a wide range of immune-mediated neurologic diseases
- Autoimmune epilepsy is suspected on clinical grounds, prior to autoantibody diagnostics, when a set of criteria are fulfilled
- When high suspicion of autoimmune encephalitis is present, immunotherapy can be discussed before antibody results are obtained
- Sensitivity to treatment and outcome differ greatly according to the associated autoantibodies present
- A personalized therapeutic approach that takes into consideration factors such as age, sex, presence of cancer, and type of antibody is now warranted

Clinical suspicion of autoimmune epilepsy

New-onset, drug-resistant, seizure disorder of unknown etiology

Seizures arising from temporal lobe(s) or multifocal

Faciobrachial dystonic seizures

Coexisting neuropsychiatric features

Autonomic disturbances

Previous history of autoimmune diseases

Associated cancer

Inflammatory pattern on CSF/MRI

Neuroimaging

Received: 11 May 2018 | Revised: 23 April 2019 | Accepted: 24 April 2019
DOI: 10.1111/epi.15612



Epilepsia

SPECIAL REPORT

Recommendations for the use of structural magnetic resonance imaging in the care of patients with epilepsy: A consensus report from the International League Against Epilepsy Neuroimaging Task Force

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Ravnoor S. Gill¹ | Matthias J. Koeppe⁴ | Robert Edward Hogan⁵ | Graeme D. Jackson⁶ |
Paolo Federico⁷ | Angelo Labate⁸ | Anna Elisabetta Vaudano⁹ | Ingmar Blümcke¹⁰ |
Philippe Ryvlin¹¹ | Neda Bernasconi¹

Key Points

- Practices for the use of structural MRI are variable worldwide and may not harness the full potential of technological advances for the benefit of people with epilepsy
- The Neuroimaging Task Force recommends use of the Harmonized Neuroimaging of Epilepsy Structural Sequences (HARNESS-MRI) protocol with isotropic, millimetric 3D T1 and FLAIR images, and high-resolution 2D submillimetric T2 images
- Use of the HARNESS-MRI protocol standardizes best-practice neuroimaging of epilepsy in outpatient clinics and specialized surgery centers alike

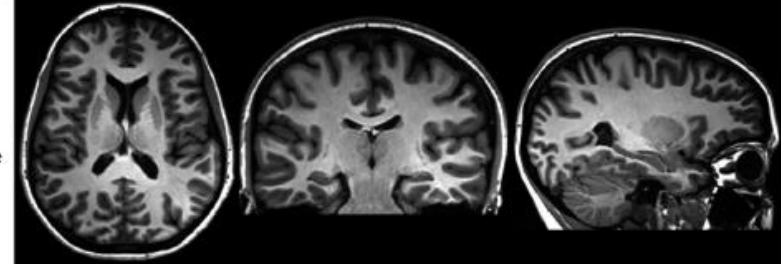
EPILEPSY PROTOCOL – 3D MRI

T1-weighted

Sequence type: gradient echo

Voxel size (mm): 1 x 1 x 1

Best to evaluate: anatomy and morphology (volume, thickness, sulco-gyral shape, grey-white matter interface integrity)



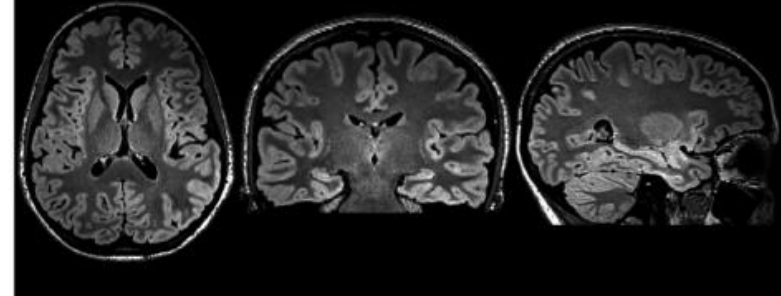
FLAIR

Sequence type: turbo spin echo

Voxel size (mm): 1 x 1 x 1

Best to evaluate: signal intensity

Caveat - Not sensitive in neonates and children <24 months of age due to incomplete myelination



EPILEPSY PROTOCOL – 2D MRI

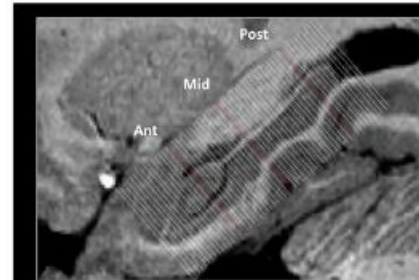
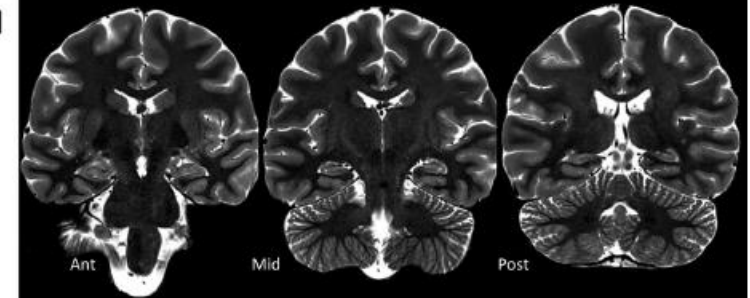
Coronal T2-weighted

Acquired perpendicular to hippocampal long axis

Sequence type: turbo spin echo

Voxel size (mm): 0.4 x 0.4 x 2; no inter-slice gap

Best to evaluate: Hippocampal internal structure (distinction of CA subfields, dentate gyrus), amygdala, and parahippocampal cortices



EEG

Long-term EEG

NeurologyToday®

Home > Epileptic High-Frequency Oscillations Disrupt Cognition in H...

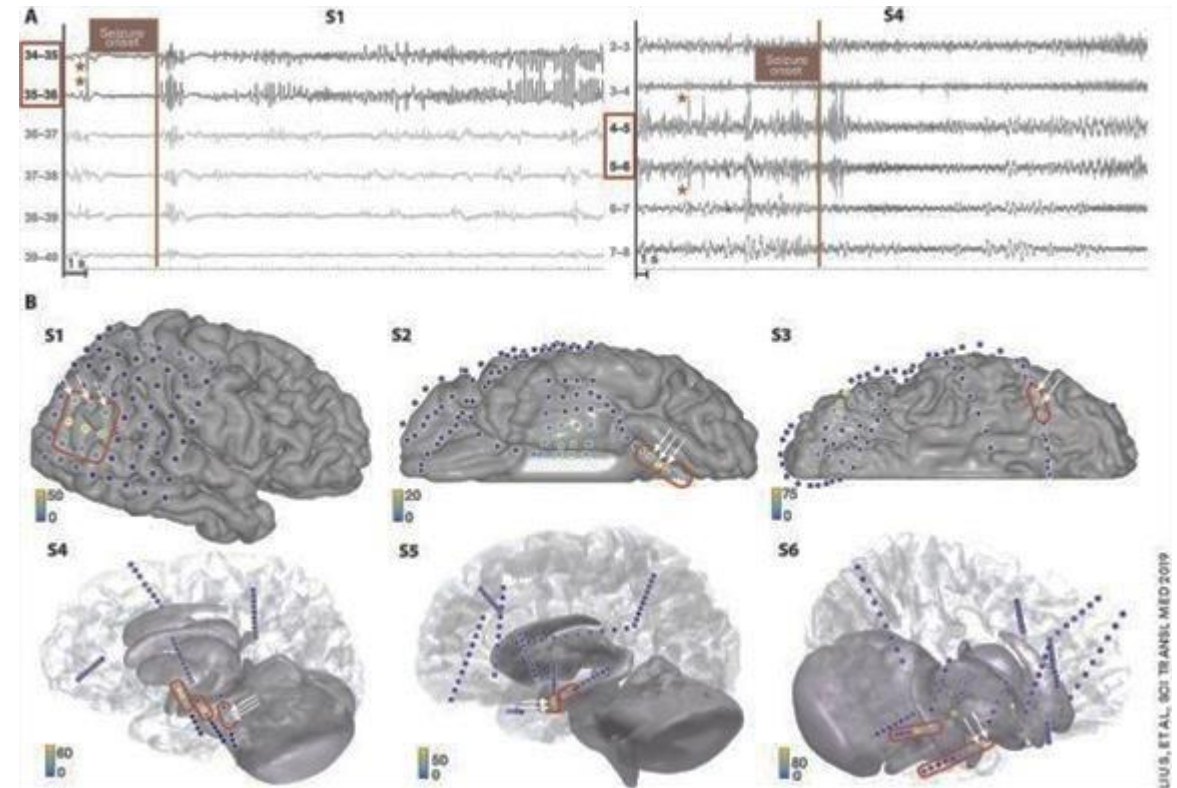


Epileptic High-Frequency Oscillations Disrupt Cognition in Human Brain

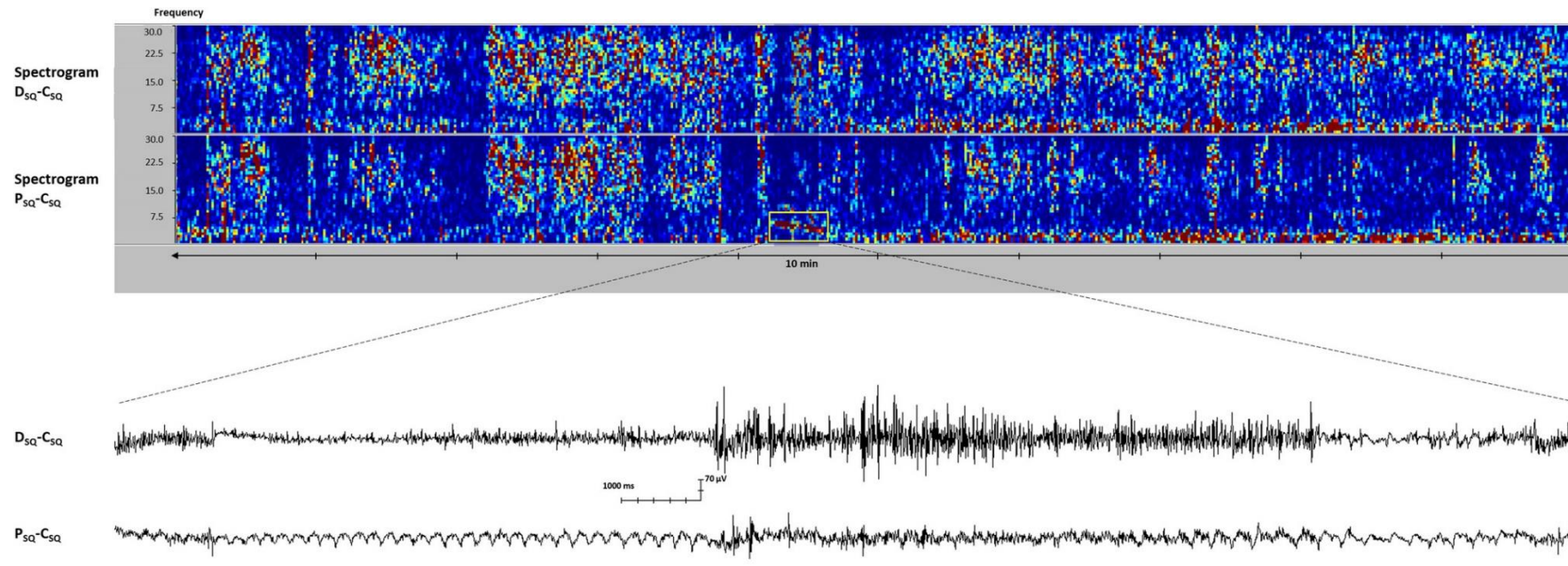
By Jamle Talan
December 5, 2019

Article In Brief

A new study demonstrates that there are normal physiological responses to cognitive stimuli in non-lesional epileptic tissue unless there is ongoing spontaneous high-frequency oscillation (HFOs). These HFOs can disrupt cognitive function, which could explain why controlling seizures does not stop patients from complaining about cognitive problems.



Spatial locations of pathological HFOs and functional HFBs. (A) Data recorded from a subset of channels in two representative subjects, S1 (grid) and S4 (depth), showing early involvement of the HFO site in seizure onset. Epileptiform discharges are marked by red stars. (B) Electrode coverage in all subjects with the load of HFOs in each electrode. Results are derived after co-registration of individual preoperative MRI and postoperative CT images. The selected ROIs in each subject are circled in red; the contacts with HFB activation within the ROI are pointed at by white arrows.



Seizure signature. The top panel shows the spectrograms of the two subcutaneous channels (DSQ-CSQ; PSQ-CSQ) as reviewed in the study. The yellow square highlights the spectrographic seizure signature, which, in this case, can be discerned only on spectrogram PSQ-CSQ. For each type of seizure from each participant, one signature was predefined from previous scalp electroencephalography (EEG) recordings. Shortly after the seizure, there is an increase in delta power (0.5-4 Hz), which could represent a postictal EEG pattern. Any spectrographic pattern resembling the relevant signature would be reviewed in the time domain for confirmation. The bottom panel shows the raw EEG at the time of the seizure with rhythmic theta activity in channel PSQ-CSQ and typical frequency dynamics. Thus, the raw EEG confirms the presence of a seizure

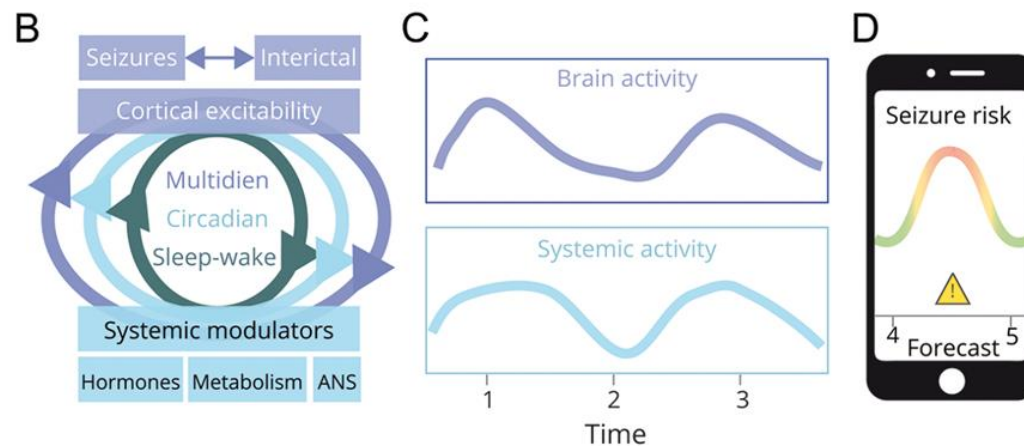
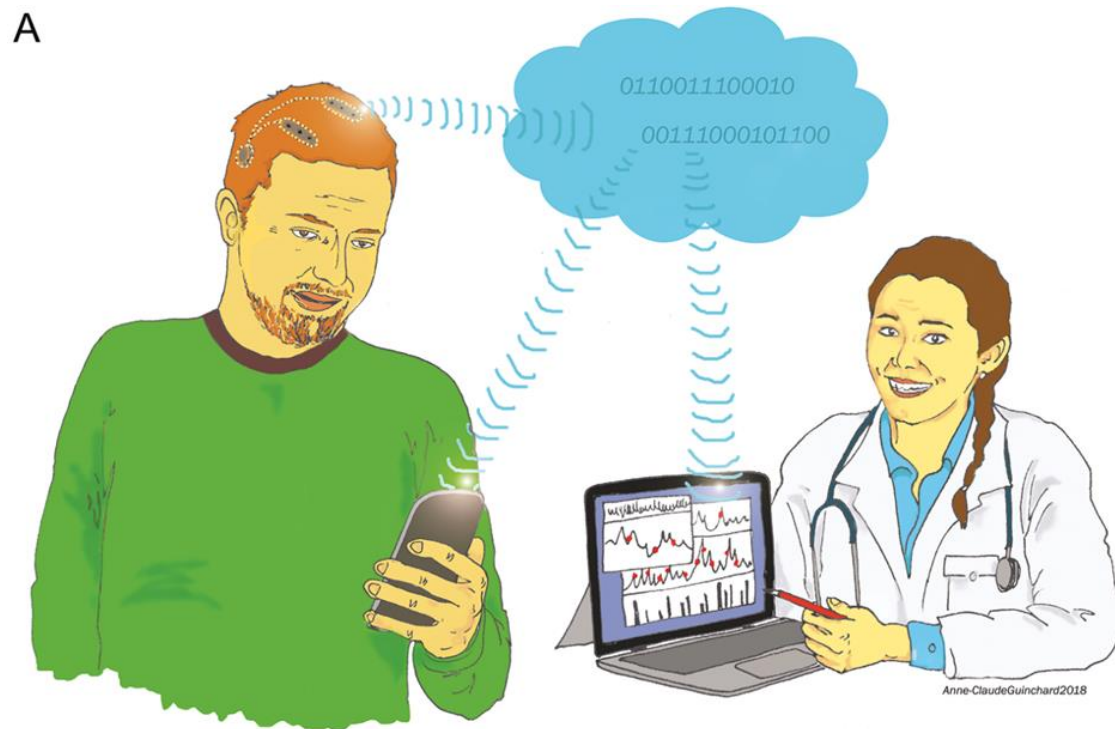
Gauging seizure risk

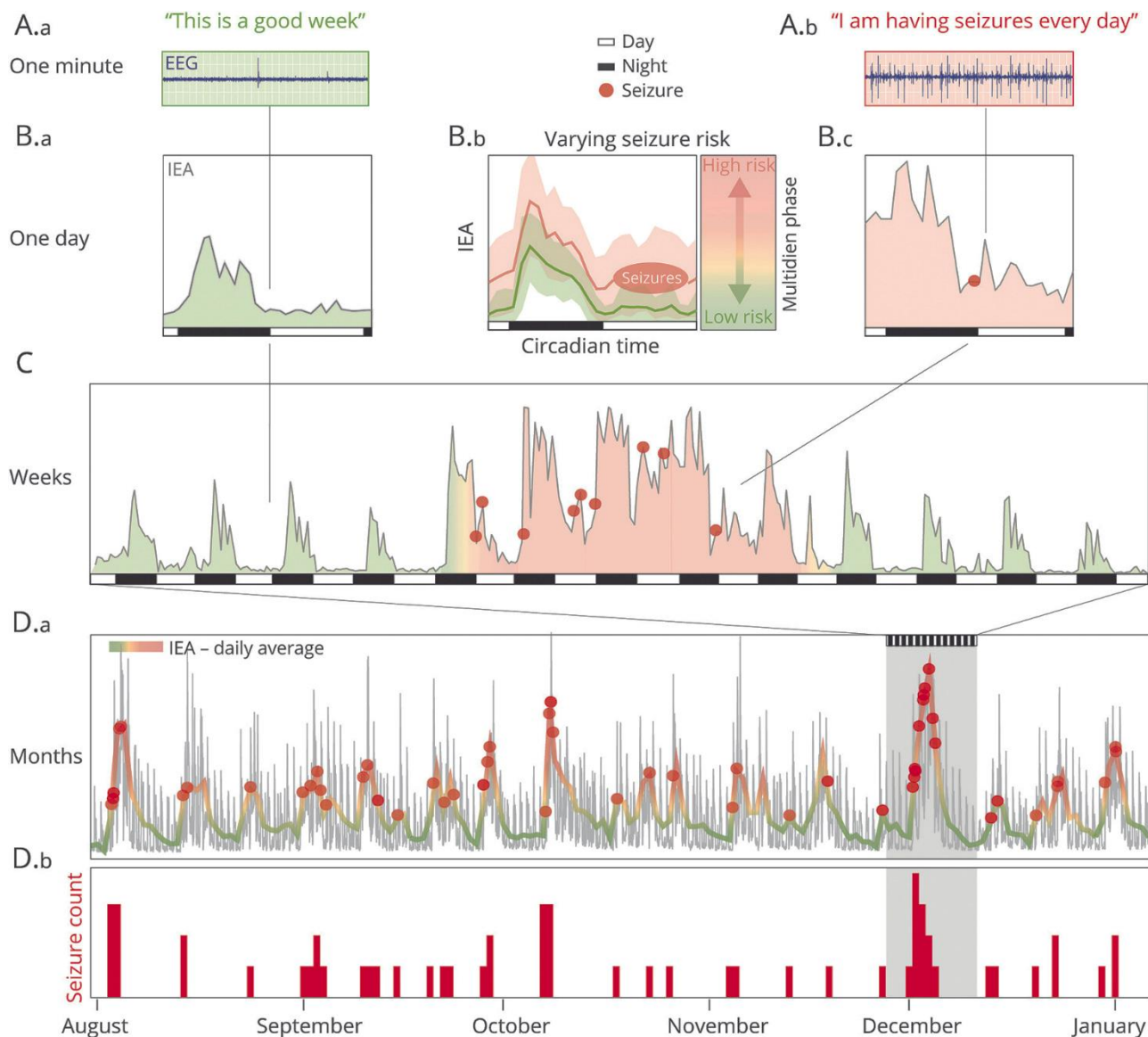
Maxime O. Baud, MD, PhD, and Vikram R. Rao, MD, PhD

Neurology® 2018;91:967-973. doi:10.1212/WNL.0000000000006548

Abstract

The current paradigm for treatment of epilepsy begins with trials of antiepileptic drugs, followed by evaluation for resective brain surgery in drug-resistant patients. If surgery is not possible or fails to control seizures, some patients benefit from implanted neurostimulation devices. In addition to their therapeutic benefit, some of these devices have diagnostic capability enabling recordings of brain activity with unprecedented chronicity. Two recent studies using different devices for chronic EEG (i.e., over months to years) yielded convergent findings of daily and multiday cycles of brain activity that help explain seizure timing. Knowledge of these patient-specific cycles can be leveraged to gauge and forecast seizure risk, empowering patients to adopt risk-stratified treatment strategies and behavioral modifications. We review evidence that epilepsy is a cyclical disorder, and we argue that implanted monitoring devices should be offered earlier in the treatment paradigm. Chronic EEG would allow pharmacologic treatments tailored to days of high seizure risk—here termed chronotherapy—and would help characterize long timescale seizure dynamics to improve subsequent surgical planning. Coupled with neuromodulation, the proposed approach could improve quality of life for patients and decrease the number ultimately requiring resective surgery. We outline challenges for chronic monitoring and seizure forecasting that demand close collaboration among engineers, neurosurgeons, and neurologists.





Rhythms of seizures (red dots) and interictal epileptiform activity (IEA; gray lines) at circadian and multidien timescales in one representative patient.

(A) One minute: raw EEG shows (A.a) low and (A.b) high IEA on different days at the same circadian time.

(B) One day: hourly fluctuations of IEA extracted from raw EEG with (B.a) low and (B.c) high 24-hour average (same scale) as well as day–night fluctuations (white and black boxes). (B.b) Average circadian amplitude of IEA during low (green) and high-risk portions (red) of the underlying multidien rhythm (green–red gradient; colored arrows). In this patient, most seizures (oval shadow centered on mean \pm SD) occur during daytime on high-risk days when IEA is approximately 4 times higher than on low-risk days. Note that although the 24-hour average amplitude is higher on those days, seizures do not align with the circadian peak of IEA.

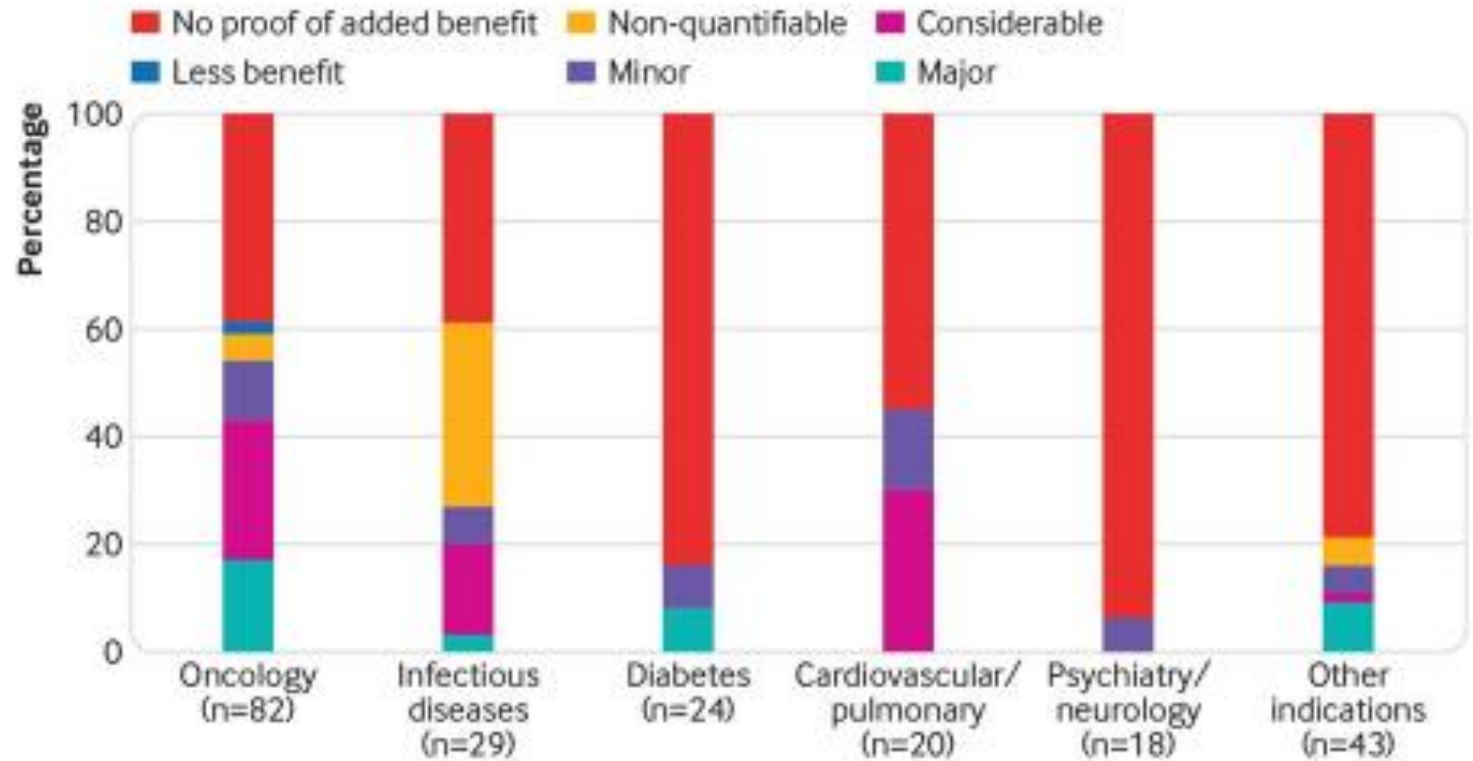
(C) Weeks: hourly fluctuations of IEA during low (green) and high-risk (red) phases of the underlying multidien rhythm.

(D) (a) Months: hourly (light gray) and daily average (green–red gradient) IEA over 5 months shows robust multidien periodicity. Note how seizures tend to cluster during high-risk phases.

(b) Seizure counts show clustering of seizures with 8-day periodicity, as well as larger clusters every 2 months.

I nuovi AED

Results of the assessment of added benefit versus standard care by indication for drugs entering the German market, 2011-17.



Beate Wieseler et al. BMJ 2019;366:bmj.l4340



Brivaracetam

- **Sviluppato da LEV**
- **Ligando selettivo con elevata affinità per proteina 2A delle vescicole sinaptiche (SV2A)**
- **Meccanismo posseduto, in minor misura, anche da LEV (che ha azione anche su canali Ca^{++} e recettori AMPA)**
- **Controversa una qualche azione anche su canali Na^+ (e conseguente efficacia su modello sperimentale MES, su cui LEV non è attivo)**
- **Maggiore lipofilicità rispetto a LEV**
 - penetra più facilmente la barriera
 - azione più rapida (dopo i.v. 3' vs 23')
- **Affinità per SV2A maggiore da 15 a 30 volte**
- **Legame proteico 20%; emivita 9 h**
- **Catabolismo: idrolisi, idrossilazione**

Brivaracetam

- **Dosi efficaci: da 50 a 200 mg/dì**
- **Efficacia ridotta da rifampicina (45%), CBZ PHT PB (26%-19%)**
- **Non modifica altri AED, ma incrementa CBZ-eossido**
- **Approvato da EMA e FDA per trattamento add-on di crisi focali, in pazienti di almeno 16 a.**
- **Studi clinici su 2388 p. (5558 pazienti-anno)**
- **Retention rate: 79,8% a 1 anno**
- **Effetti collaterali: cefalea, vertigini, sonnolenza, astenia**

RESEARCH PAPER

Cannabis use is both independently associated with and mediates worse psychosocial health in patients with epilepsy

Sandra Wahby,¹ Vikram Karnik,¹ Anita Brobbey,² Samuel Wiebe,¹ Tolulope Sajobi,^{1,2} Colin Bruce Josephson^{1,2}

To cite: Wahby S, Karnik V, Brobbey A, et al. *J Neurol Neurosurg Psychiatry* 2019;**90**:945–951.

Conclusions There is a strong and independent association between cannabis use and poor psychosocial health, and it partially mediates the deleterious effect of a psychiatric history on these same outcomes. Inclusion of PROMs in future cannabis trials is warranted.

Source: GW Pharmaceuticals plc

July 26, 2019 06:46 ET

GW Pharmaceuticals receives positive CHMP opinion for EPIDYOLEX™ (cannabidiol oral solution) for the treatment of seizures in patients with two rare, severe forms of childhood-onset epilepsy

If approved cannabidiol oral solution will be the first plant-derived cannabis-based medicine to be approved in Europe for the treatment of any form of epilepsy

GW's cannabidiol oral solution contains highly purified, plant-derived cannabidiol (CBD), a cannabinoid lacking the "high" associated with cannabis

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Evidence on CBD Use for Pediatric Epilepsy Emerges Slowly Less Is Known About 'Artisanal' Products



By Thomas R. Collins

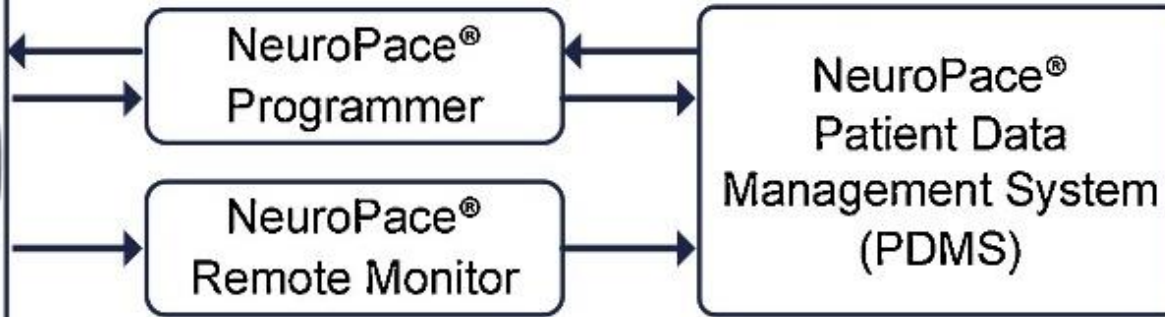
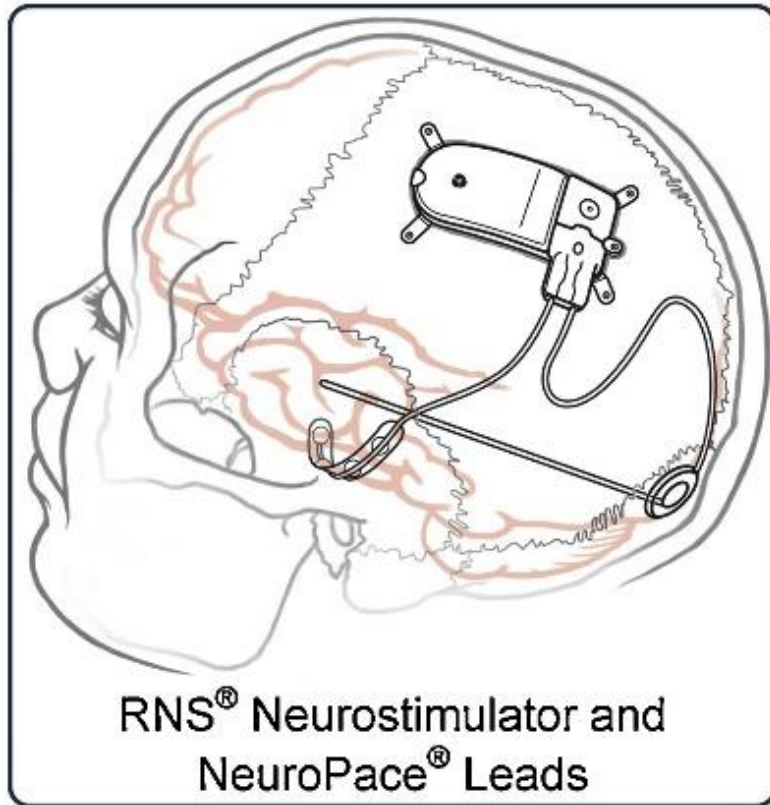
December 5, 2019

Article In Brief

The FDA-approved cannabidiol-based drug was a welcome development for severe pediatric epilepsy syndromes, experts said. But there are still many unanswered questions about the "artisanal" cannabidiol projects that patients and families request.

Terapie non farmacologiche

Responsive neurostimulation for the treatment of epilepsy



The RNS System is a programmable and responsive device that consists of depth or subdural strip leads, a pulse generator and an external programmer.

The RNS System has an algorithm capable of detecting specific patterns of epileptogenic activity and triggering focal stimulation to interrupt the seizure.

Responsive neurostimulation (RNS) is indicated for individuals who have partial-onset seizures with no more than 2 epileptogenic foci, and who have 3 or more disabling seizures per month.

In addition to seizure frequency reduction, RNS may have other applications, such as drug response evaluation and long-term electrocorticography recording.

Responsive Neurostimulation for the Treatment of Epilepsy

Caio M. Matias, MD, PhD^{1,2,*}, Ashwini Sharan, MD³,
Chengyuan Wu, MD, MSBME⁴

Neurosurg Clin N Am 30 (2019) 231–242
<https://doi.org/10.1016/j.nec.2018.12.006>

Therapie innovative, stereo-EEG

Received: 24 January 2019 | Revised: 23 July 2019 | Accepted: 23 July 2019
DOI: 10.1111/epi.16315



FULL-LENGTH ORIGINAL RESEARCH

Epilepsia

Method of invasive monitoring in epilepsy surgery and seizure freedom and morbidity: A systematic review

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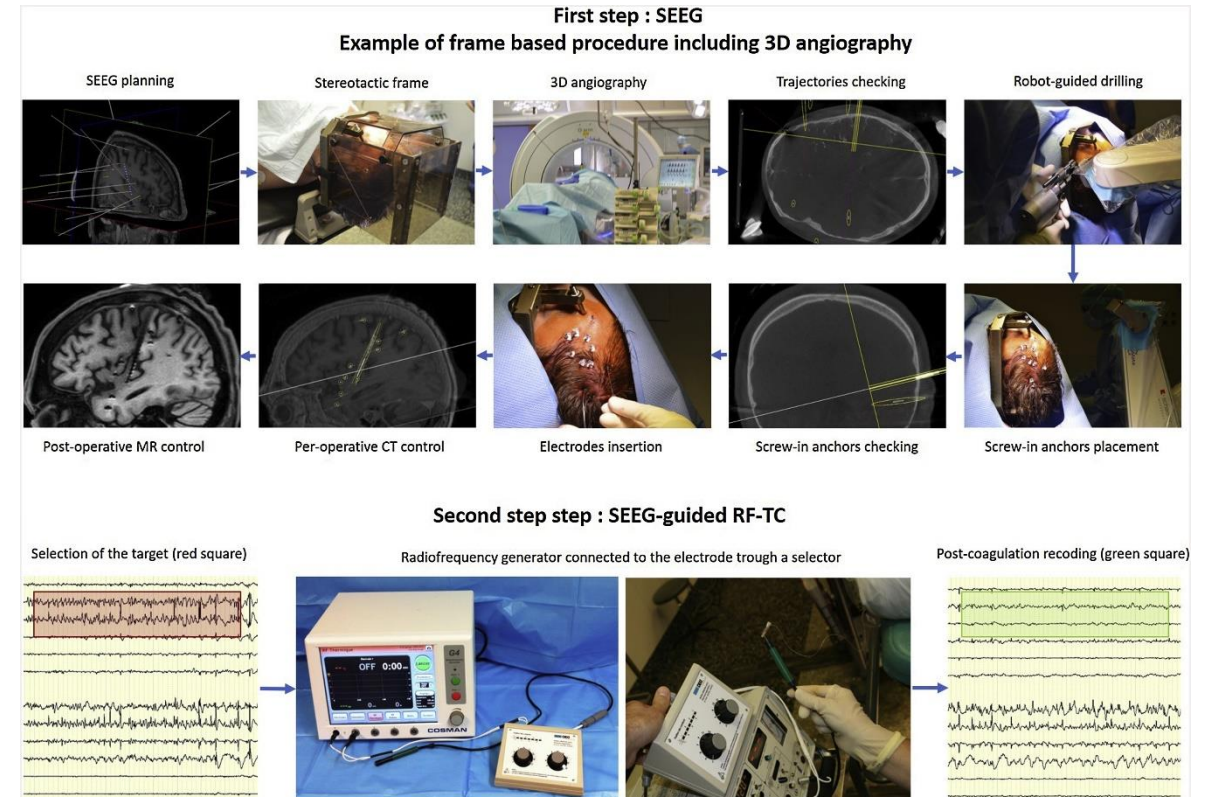
In this systematic review of SEEG and SDE invasive monitoring techniques, SEEG was associated with fewer surgical resections yet better seizure freedom outcomes in those undergoing resections. SEEG was also associated with lower mortality and morbidity than SDE.



Review

Surgical techniques: Stereoelectroencephalography-guided radiofrequency-thermocoagulation (SEEG-guided RF-TC)

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