Received: 2 August 2021 | Accepted: 6 August 2021
DOI: 10.1111/epi.17070

Epilepsia

LETTER

SUDEP-7 Inventory: Validation in a retrospective cohort study

It is with great interest that we read the report by Tarighati Rasekhi and colleagues: "Improving prediction of sudden unexpected death in epilepsy: From SUDEP-7 to SUDEP-3"[1] This study provides the first external validation of the sudden unexpected death in epilepsy (SUDEP)-7 inventory in a matched-cohort study. The SUDEP-7 inventory is a seven-item weighted inventory derived from the prospective SUDEP study published by Walczak and colleagues.[2] The SUDEP-7 is correlated with biomarkers of SUDEP, including RMSSD (vagus-mediated heart rate variability) and post-ictal electroencephalography (EEG) suppression, and it has good inter-observer correlation.[3-5]

In the retrospective study by Tarighati Rasekhi and colleagues, mean SUDEP-7 scores were significantly higher in persons who dies of SUDEP (SUDEP-7 score = 3.65 standard deviation [SD] = 2.18) than in matched controls (SUDEP-7 score = 2.09 SD 1.82, $p = .016$).[1] In a sub-analysis, the authors used stepwise regression to develop a SUDEP-3, a sub-score of the SUDEP-7. Although the maximum likelihood estimate for the area under the receiver- operating characteristic (ROC) curve (AUC) of the SUDEP-7 is lower than the SUDEP-3 (66% vs 75%), the 95% confidence intervals (CIs) overlap (95% CI 54%–87% vs 95% CI 64%–86%). Because the authors used their data set to select the components and validate the SUDEP-3, this may have inadvertently increased the predictive performance as a result of the relatively small sample size.[7] Therefore, the appropriate interpretation of this finding is that the SUDEP-3 does not differ significantly from the SUDEP-7.

The authors stated correctly that they had insufficient power to evaluate the additive benefit, or lack thereof, of the other four elements of the SUDEP-7. It is very possible that these components of the SUDEP-7 would capture meaningful variation in a larger cohort of children and adults with those associated factors, who were sparsely sampled in this work. For example, very high seizure frequencies of 50 or more per month are common in children with Lennox-Gastaut syndrome and other pediatric syndromes. Yet, the authors only sampled those 14 years and older. Regarding three or more antiseizure medications (ASMs), the odds ratio of 2.6 (95% CI 0.97–7.2) had a p-value of .058, which did not reach the canonical threshold of $p < .05$. However, the American Statistical Association recommends that these significance thresholds not be viewed as binary.[8] In addition, in the context of prior significant results from the North American SUDEP registry that better sampled patients on polytherapy,[9] a Bayesian perspective or meta-analysis would likely conclude that ASM polytherapy is meaningfully associated with a risk of SUDEP. Therefore, although the SUDEP-3 may be more targeted to the sample of Tarighati Rasekhi and colleagues, we believe the SUDEP-7 may be more applicable to a broader pediatric and adult population, which is under-represented in this study.

Despite these concerns, we remain enthusiastic that Tarighati Rasekhi and colleagues provide the first external validation in a retrospective study of the SUDEP-7 inventory. These data and their relevant discussion of the recent literature highlight that the SUDEP-7 can be refined to better capture quantifiable and modifiable factors associated with SUDEP. We believe it is time for multiple stakeholders to collaboratively re-evaluate these factors and develop an evidence-based consensus revision of the SUDEP-7.

FUNDING INFORMATION

National Institute of Neurological Disorders and Stroke, Grant/Award Number: R25 NS065723; and an unrestricted gioft to the the UCLA Department of Neurology from Beverly and james Peters and family.

CONFLICT OF INTEREST

Dr. DeGiorgio and Ms. Markovic were involved in the development of the SUDEP-7 and have no commercial incentive for its use. The authors have no relevant conflicts of interest. Dr. Kerr's time was partially supported by National Institutes of Health (NIH) R25 NS065723 and an unrestricted gioft to the UCLA Department of Neurology from Beverly and James Peters and family. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

Wesley T. Kerr[1,2]
Daniela Markovic[3]
Christopher M. DeGiorgio[2,3]

[1]*Department of Neurology, University of Michigan, Ann Arbor, Michigan, USA*
[2]*Department of Neurology, University of California, Los Angeles, California, USA*
[3]*David Geffen School of Medicine, University of California, Los Angeles, California, USA*

Correspondence
Christopher M. DeGiorgio, UCLA Department of Neurology, University of California, Olive View-UCLA Medical Center, 14445 Olive View Dr., @c136, Sylmar, CA 91342, USA.
Email: cmd@mednet.ucla.edu

ORCID

Wesley T. Kerr https://orcid.org/0000-0002-5546-5951

REFERENCES

1. Tarighati Rasekhi R, Devlin KN, Mass JA, Donmez M, Asma B, Sperling MR, Nei M. Improving prediction of sudden unexpected death in epilepsy: from SUDEP-7 to SUDEP-3. Epilepsia. 2021;62:1536–45.
2. Walczak TS, Leppik IE, D'Amelio M, Rarick J, So E, Ahman P, et al. Incidence and risk factors in sudden unexpected death in epilepsy: a prospective cohort study. Neurology. 2001;56:519–25.
3. Moseley BD, DeGiorgio CM. The SUDEP Risk inventory: association with postictal generalized EEG suppression. Epilepsy Res. 2015;117:82–4.
4. Novak JL, Miller PR, Markovic D, Meymandi SK, DeGiorgio CM. Risk assessment for sudden death in epilepsy: the SUDEP-7 inventory. Front Neurol. 2015;6:252.
5. McCarter AR, Timm PC, Shepard PW, Sandness DJ, Luu T, McCarter SJ, et al. Obstructive sleep apnea in refractory epilepsy: a pilot study investigating frequency, clinical features, and association with risk of sudden unexpected death in epilepsy. Epilepsia. 2018;59:1973–81.
6. DeGiorgio CM, Miller P, Meymandi S, Chin A, Epps J, Gordon S, Gornbein J, Harper RM. RMSSD, a measure of vagusmediated heart rate variability, is associated with risk factors for SUDEP: the SUDEP-7 Inventory. Epilepsy Behav. 2010;19:78–81.
7. Pulini AA, Kerr WT, Loo SK, Lenartowicz A. Classification accuracy of neuroimaging biomarkers in attention-deficit/hyperactivity disorder: effects of sample size and circular analysis. Biol Psychiatry Cogn Neurosci Neuroimaging. 2019;4:108–20.
8. Wasserstein RL, Lazar NA. The ASA statement on p-values: context, process, and purpose. Am Stat. 2016;70:129–33.
9. Verducci C, Hussain F, Donner E, Moseley BD, Buchhalter J, Hesdorffer D, et al. SUDEP in the North American SUDEP Registry: the full spectrum of epilepsies. Neurology. 2019;93:e227–36.

Received: 3 September 2021 | Accepted: 3 September 2021

DOI: 10.1111/epi.17073

Epilepsia®

LETTER

Response: SUDEP-7 Inventory: Validation in a retrospective cohort study

We sincerely thank Dr. DeGiorgio and his colleagues for their comments on our article[1] and we will address their critiques. Our subjects were drawn from a large prospective surgical and nonsurgical epilepsy database (over 1500 patients) as well as a sudden unexpected death in epilepsy (SUDEP) database. We believe that our sample size was adequate to report and formulate a new inventory, particularly when compared with the original SUDEP-7 inventory (28 SUDEP patients in our study vs 20 SUDEP patients in the original inventory).[2,3]

Regarding seizure frequency: the authors reference the age of the patients at the time of admission (14 years and older). They argue that inclusion of younger patients might lead to a greater likelihood of finding that higher seizure frequency (50 or more per month) is associated with SUDEP risk. The authors stated that not including young patients may exclude patients with epilepsy syndromes, for example, Lennox-Gastaut syndrome, with very high seizure frequencies. However, in the Walczak study, which forms the basis of the SUDEP-7 Inventory, the age range of SUDEP patients was between 20 and 59 years.[2] Our study includes younger patients, but this predictor (having had 50 or more seizures per month) remained nonsignificant. It should also be emphasized that four of our SUDEP cases (14%) have been classified as developmental and epileptic encephalopathy, with no statistically significant difference with our control patients. Moreover, in their multivariate analysis, the Walczak study found that although the occurrence of tonic-clonic seizures remained a strong SUDEP risk factor, high seizure frequency itself did not. These are consistent with our findings.

Regarding the significance and inclusion of polytherapy ($p = .058$) as a SUDEP predictor, our statistical threshold of $p < .05$ was chosen based on its wide use and acceptance across a variety of disciplines. We examined a SUDEP-4 inventory that included polytherapy, but its predictive value was comparable to that of the SUDEP-3 inventory. Thus polytherapy was not retained in our new inventory. That said, we appreciate the comments of DeGiorgio and colleagues regarding the utility of moving beyond binary classifications of significance, and we concur that future studies should utilize alternative (eg, Bayesian) approaches to assessing SUDEP risk factors.

Regarding the relative predictive validity of the SUDEP-3 and SUDEP-7 inventories, our article included an error in the upper bound of the confidence interval for the area under the curve (AUC) of the SUDEP-7. It is .78, rather than .87. Therefore, the AUCs (95% confidence interval [CI]) are .66 (.54–.78) for the SUDEP-7 and .75 (.64–.86) for the SUDEP-3. Thus, although there is overlap in these confidence intervals, there is considerably less overlap than the original version of the article suggests. This difference appears to be clinically meaningful even if not statistically significant because of the small sample size.

We recognize that the use of the same data set to construct and validate the SUDEP-3 inventory might inflate its predictive performance. Given the low incidence of SUDEP in our large prospective database, the use of separate testing and validation samples was not possible, and we look forward to further validation of SUDEP inventories in other cohorts.

Although DeGiorgio et al. note that the SUDEP-7 has been correlated with potential biomarkers of SUDEP, unfortunately, these findings remain of questionable importance because the quoted biomarkers are still not confirmed to correlate with SUDEP. In addition, the only other published study of the SUDEP-7 found that it had poor correlation with SUDEP (Odom and Bateman).[4]

We appreciate the feedback to date regarding the SUDEP-3 inventory, including a recent report highlighting the utility of the SUDEP-3 inventory[5] and are enthusiastic about further evaluations of the new inventory in larger cohorts.

ACKNOWLEDGMENTS

None.

CONFLICT OF INTEREST

Michael R. Sperling is a consultant/advisor for Medtronic (fee to institution); received research support (to institution) from Eisai, Engage Therapeutics, Medtronic, Neurelis, Pfizer, SK Life Science, Inc., Takeda, UCB, Cerevel, and Xenon; and has been a speaker for Eisai, Medscape, NeurologyLive, UCB, and Projects in Knowledge. The remaining authors have no conflicts of interest to disclose. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

Roozbeh Tarighati Rasekhi[1]
Kathryn N. Devlin[2]
Michael R. Sperling[3]
Maromi Nei[3]

[1]*Department of Radiology and Imaging Sciences, School of Medicine, Emory University, Atlanta, Georgia, USA*
[2]*Department of Psychology, Drexel University, Philadelphia, Pennsylvania, USA*
[3]*Department of Neurology, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania, USA*

Correspondence
Maromi Nei, Department of Neurology, Jefferson Comprehensive Epilepsy Center, Sidney Kimmel Medical College at Thomas Jefferson University, 901 Walnut Street, Suite 400, Philadelphia, PA 19107, USA.
Email: maromi.nei@jefferson.edu

ORCID

Roozbeh Tarighati Rasekhi https://orcid.org/0000-0003-3868-7752
Michael R. Sperling https://orcid.org/0000-0003-0708-6006
Maromi Nei https://orcid.org/0000-0001-7197-3094

REFERENCES

1. Tarighati Rasekhi R, Devlin KN, Mass JA, Donmez M, Asma B, Sperling MR, et al. Improving prediction of sudden unexpected death in epilepsy: from SUDEP-7 to SUDEP-3. Epilepsia. 2021;62(7):1536–45.
2. Walczak TS, Leppik IE, D'Amelio M, Rarick J, So E, Ahman P, et al. Incidence and risk factors in sudden unexpected death in epilepsy: a prospective cohort study. Neurology. 2001;56(4):519–25.
3. DeGiorgio CM, Miller P, Meymandi S, Chin A, Epps J, Gordon S, et al. RMSSD, a measure of vagus-mediated heart rate variability, is associated with risk factors for SUDEP: the SUDEP-7 Inventory. Epilepsy Behav. 2010;19(1):78–81.
4. Odom N, Bateman LM. Sudden unexpected death in epilepsy, periictal physiology, and the SUDEP-7 inventory. Epilepsia. 2018;59(10):e157–60.
5. Barguilla A, Panadés-de Oliveira L, Principe A, Rocamora R. SUDEP-3: probable improvement in risk stratification for sudden death in epilepsy. Epilepsia. 2021. Letter. https://doi.org/10.1111/epi.17033

How to cite this article: Tarighati Rasekhi R, Devlin KN, Sperling MR, Nei M. Response: SUDEP-7 Inventory: Validation in a retrospective cohort study. Epilepsia. 2021;62:2873–2874. https://doi.org/10.1111/epi.17073

Received: 17 June 2021 | Accepted: 16 August 2021
DOI: 10.1111/epi.17053

Epilepsia®

LETTER

The true prevalence of psychogenic nonepileptic seizures is much higher than this

To the Editors,

Epilepsia published an article entitled, "Incidence and prevalence of psychogenic nonepileptic seizures in a Norwegian county: A 10-year population-based study" authored by Dr. Antonia Villagrán and colleagues.[1] The authors reported a population-based estimate of the prevalence of psychogenic nonepileptic seizures (PNES) for the first time. They found a PNES prevalence of 23.8/100 000 (95% confidence interval [CI] = 17.9–29.6).[1] Although this study tries to provide an important and missing piece of the data on the issue of interest (i.e., PNES) and it achieves this objective to some extent, hereby, I would like to discuss important limitations of this work. The authors of this study used the Norwegian patient registry and identified patients diagnosed with "conversion disorder with seizures or convulsions" or "convulsions, not elsewhere classified" in the period from January 2010 to January 2020. Although this approach may identify those who had a diagnosis of PNES or those who had uncertain seizure types for further scrutiny, it would miss a significant number of patients for the following reasons.

First, most patients with PNES (about two thirds) are being diagnosed as having epilepsy and are prescribed antiseizure medications for years.[2] Delay in the definite diagnosis and appropriate management of patients with PNES is a common occurrence in both developed and developing countries.[2,3] This delay may last for years or even for decades.[2,3] Therefore, it is highly likely that these patients receive inappropriate diagnostic codes in the registries for years. Second, a significant minority of patients with PNES have comorbid epilepsy. A recent systematic review suggested that the mean frequency of epilepsy in patients with PNES across all studies was 22% (95% CI = 20%–25%).[4] This means that approximately 20% of patients with PNES may receive inappropriate diagnostic codes and may mistakenly not be included in any epidemiological study with this methodology (of studying registries). Finally, a significant minority (more than one quarter) of patients with PNES may have focal abnormalities in their brain imaging studies[5]; this may result in a mistaken diagnosis of "focal epilepsy," and therefore even a diagnostic code of "convulsions, not elsewhere classified" may not detect these patients for an epidemiological study.

In a recent analytical study of the incidence and prevalence of PNES (functional seizures), I considered all these variables and confounding factors and also other variables such as the outcome, and mortality of PNES.[6] The calculated prevalence rate of PNES in 2019 was 108.5 (95% CI = 39.2–177.8) per 100 000 population in the United States.[6] Therefore, I believe that the true prevalence of PNES is much higher than the rate reported by Dr. Antonia Villagrán and colleagues.[1] Any future field study of the epidemiology of PNES should consider all the discussed confounding variables.

KEYWORDS

dissociative, functional, psychogenic, seizure

ACKNOWLEDGMENT

None.

CONFLICT OF INTEREST

A.A.A.-P. reports honoraria from Cobel Daruo, Tekaje, and RaymandRad, and a royalty from Oxford University Press (for a book publication) outside the submitted work. I confirm that I have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

Ali A. Asadi-Pooya[1,2]

[1]*Epilepsy Research Center, Shiraz University of Medical Sciences, Shiraz, Iran*
[2]*Department of Neurology, Jefferson Comprehensive Epilepsy Center, Thomas Jefferson University, Philadelphia, Pennsylvania, USA*

Correspondence
Ali A. Asadi-Pooya, Epilepsy Research Center, Shiraz University of Medical Sciences, Shiraz, Iran.
Email: aliasadipooya@yahoo.com

ORCID

Ali A. Asadi-Pooya https://orcid.org/0000-0002-2598-7601

REFERENCES

1. Villagrán A, Eldøen G, Duncan R, Aaberg KM, Hofoss D, Lossius MI. Incidence and prevalence of psychogenic nonepileptic seizures in a Norwegian county: a 10-year population-based study. Epilepsia. 2021;62(7):1528–35.
2. Asadi-Pooya AA, Bahrami Z. Risk factors for the use of antiepileptic drugs in patients with psychogenic nonepileptic seizures. Epilepsy Behav. 2019;90:119–21.
3. Asadi-Pooya AA, Tinker J. Delay in diagnosis of psychogenic nonepileptic seizures in adults: a post hoc study. Epilepsy Behav. 2017;75:143–5.
4. Kutlubaev MA, Xu Y, Hackett ML, Stone J. Dual diagnosis of epilepsy and psychogenic nonepileptic seizures: systematic review and meta-analysis of frequency, correlates, and outcomes. Epilepsy Behav. 2018;89:70–8.
5. Asadi-Pooya AA, Homayoun M. Structural brain abnormalities in patients with psychogenic nonepileptic seizures. Neurol Sci. 2020;41:555–9.
6. Asadi-Pooya AA. Incidence and prevalence of psychogenic nonepileptic seizures (functional seizures): a systematic review and an analytical study. Int J Neurosci. 2021;4:1–6.

Received: 11 August 2021 | Accepted: 16 August 2021

DOI: 10.1111/epi.17054

Epilepsia

LETTER

Response: The true prevalence of psychogenic nonepileptic seizures is much higher than this

To the Editors

We thank Dr. Asadi-Pooya for his comments on our recently published article by Villagrán et al.[1] We note that he comprehensively agrees with our view, stated clearly in our paper, that our study may have underestimated the prevalence of psychogenic nonepileptic seizures (PNES). In our paper, we discuss potential reasons for this, helpfully reiterated by Dr. Asadi-Pooya.

He does raise an interesting point: that of the inaccessibility of some parts of the PNES population to epidemiological study. If a patient does not have a diagnosis of PNES, then it is difficult to include him or her in an epidemiological study of PNES. The study of misdiagnosis rates can usefully estimate the error stemming from this, but we think that few would contend that this has epidemiological meaning without some basis in population data.

Dr. Asadi-Pooya also refers to his own article,[2] in which he attempts to extrapolate prevalence from published incidence studies, rather than carrying out an epidemiological study. On a basic level, prevalence is linked to incidence by duration of disease, and nowhere in his somewhat complicated calculation do we see any understandable measure or estimate of this. Thus, although we do of course respect Dr. Asadi-Pooya's opinion that the prevalence of PNES may be very high, we are not of the view that the figures he presents provide meaningful support for that opinion.

KEYWORDS

epidemiology, psychogenic nonepileptic seizures

ACKNOWLEDGMENT

None.

CONFLICT OF INTEREST

None of the authors has any conflict of interest to disclose. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

Antonia Villagrán[1,2]
Roderick Duncan[3]
Dag Hofoss[1]
Morten Ingvar Lossius[1,2]

[1]*Division of Clinical Neuroscience, National Center for Epilepsy, Oslo University Hospital, Oslo, Norway*
[2]*Institute of Clinical Medicine, University of Oslo, Oslo, Norway*
[3]*Department of Neurology, Christchurch Hospital, Christchurch, New Zealand*

Correspondence
Antonia Villagrán, Division of Clinical Neuroscience, National Center for Epilepsy, Oslo University Hospital, Postbox 4950 Nydalen, Oslo 0424, Norway.
Email: antvil@ous-hf.no

ORCID

Antonia Villagrán https://orcid.org/0000-0001-7310-3746

REFERENCES

1. Villagrán A, Eldøen G, Duncan R, Aaberg KM, Hofoss D, Lossius MI. Incidence and prevalence of psychogenic nonepileptic seizures in a Norwegian county: a 10-year population-based study. Epilepsia. 2021;62(7):1528–35.
2. Asadi-Pooya AA. Incidence and prevalence of psychogenic nonepileptic seizures (functional seizures): a systematic review and an analytical study. Int J Neurosci. 2021;28:1–6.

DOI: 10.1111/epi.17103

Epilepsia®

ANNOUNCEMENT

Epilepsia – November 2021 – Announcements

ILAE CONGRESSES

20–25 March 2022
3rd International Training Course on Neuropsychology in Epilepsy
Bordeaux, France
https://www.ilae.org/congresses/3rd-international-training-course-on-neuropsychology-in-epilepsy

10–13 April 2022
EEG in the First Year of Life – from newborn to toddler
Cambridge, UK
https://www.ilae.org/congresses/eeg-in-the-first-year-of-life1

9–13 July 2022
14th European Congress on Epileptology (ECE)
Geneva, Switzerland
https://www.epilepsycongress.org/eec/

8–11 September 2022
11th Summer School for Neuropathology and Epilepsy Surgery (INES 2022)
Erlangen, Germany
https://www.ilae.org/congresses/11th-international-summer-school-for-neuropathology-and-epilepsy-surgery-ines-2021

WEBINARS

Canadian Epilepsy Teaching Network of the CLAE
The Canadian League Against Epilepsy is proud to launch of the Canadian Epilepsy Teaching Network (CETN). We are excited to showcase monthly virtual rounds to be given by national and international experts in epilepsy. Sessions were designed based on the survey results conducted among the CLAE members and follow the ILAE competency-based curriculum. Sessions will be held Fridays, usually at 12 noon Eastern Time.
https://www.claegroup.org/CETN-Program

OTHER CONGRESSES

2–4 November 2021
Epilepsy Society of Australia 35th Annual Scientific Meeting
Hobart, Tasmania, Australia OR virtual meeting
https://www.ivvy.com.au/event/ESA21/

19 November 2021
Dravet Syndrome UK Conference
Virtual conference
https://www.dravet.org.uk/events/dsuk-2021-conference-professional-day/

27 November 2021
3rd Educational Symposium of the Psychiatry Commission: Diagnosis and Treatment of Psychiatric Disorders in Persons with Epilepsy throughout Life
Virtual symposium
https://www.ilae.org/congresses/3rd-educational-symposium-of-the-psychiatry-commission-diagnosis-and-treatment-of-psychiatric-disorders-in-persons-with-epilepsy-throughout-life

3–7 December 2021
AES Annual Meeting
Chicago, Illinois, USA
https://www.aesnet.org/2021-annual-meeting

8–11 December 2021
European Congress of NeuroRehabilitation 2021 jointly with 27. Jahrestagung der Deutschen Gesellschaft für Neurorehabilitation
Virtual congress
https://www.efnr-congress.org/

2022

24–28 January 2022
11th EPODES–Epilepsy Surgery – Basic
Brno, Czech Republic
http://www.ta-service.cz/epodes2021

3–8 April 2022
9th Eilat International Educational Course: Pharmacological Treatment of Epilepsy
Jerusalem, Israel
https://www.eilatedu2021.com/

8–10 April 2022
1er Curso Latinoamericano Teórico práctico de Electrocencefalografía Clínica
Santiago, Chile
https://www.clinicaepilepsia.cl/curso_electroencefalografia_clinica

27–30 April 2022
60. Jahrestagung der Deutschen Gesellschaft für Epileptologie
Leipzig, Germany
https://www.epilepsie-tagung.de/

28 April – 2 May 2022
14th European Paediatric Neurology Society (EPNS) Congress: Precision in Child Neurology
Glasgow, UK OR virtual congress
https://epns-congress.com/

22–25 May 2022
16th EILAT Conference on New Antiepileptic Drugs and Devices
Madrid, Spain
https://www.eilatxvi.com/

27–28 May 2022
Neurophysiology, neuropsychology, and epilepsy in 2022: Hills we have climbed and hills ahead
Honoring Professors Jean Gotman and Marilyn Jones-Gotman
Montreal, Canada
https://www.ilae.org/congresses/neurophysiology-neuropsychology-and-epilepsy-in-2022-hills-we-have-climbed-and-hills-ahead

17–20 June 2022
10th Migrating Course on Epilepsy
Lviv, Ukraine
https://www.ilae.org/congresses/10th-migrating-course-on-epilepsy

25–28 June 2022
8th Congress of the European Academy of Neurology (EAN)
Vienna, Austria
https://www.ilae.org/congresses/8th-congress-of-the-european-academy-of-neurology-ean

16–23 July 2022
5th Dianalund Summer School on EEG and Epilepsy
Dianalund, Denmark
https://www.ilae.org/congresses/5th-dianalund-summer-school-on-eeg-and-epilepsy

18–29 July 20222022 Advanced San Servolo Epilepsy Course. Bridging Basic with Clinical Epileptology - 7: Accelerating Translation in Epilepsy Research
San Servolo (Venice), Italy
https://www.ilae.org/congresses/2022-advanced-san-servolo-epilepsy-course

September 2022 (dates not finalized)
ILAE British Branch Virtual 18th Specialist Registrar Epilepsy Teaching Weekend
In-person event
https://www.epilepsyteachingweekend.com/

2023

20–24 June 2023
15th European Paediatric Neurology Society Congress (EPNS): From genome and connectome to cure
Prague, Czech Republic
https://www.epns.info/epns-congress-2023/