RESUSCITATION 142 (2019) 184–185

Available online at www.sciencedirect.com

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

Editorial

Quantitative EEG after cardiac arrest: New insights from an old technology

Since the 1950's electroencephalography (EEG) has been used in the settings of hypothermia and anoxia. It is a rich source of clinical and prognostic information. Structural damage to hypoxia-sensitive corticothalamic white matter tracts or other brain regions, sedative or other medications, body temperature, adequacy of cerebral blood flow, and other determinants of cortical neuronal energetics may all alter EEG features.[1–4] Qualitative expert interpretation of complex EEG signals is standard of care, and the prognostic value of post-arrest EEG when viewed through the filter of expert eyes is well-established.[5–7] However, qualitative EEG interpretation is laborious, requires special training, and is difficult to broadly implement without a delay of at least minutes to hours (sometimes more) from signal acquisition to usable clinical information. Subtle changes may also be inapparent to the unassisted eye. Quantitative EEG (qEEG) analysis can be used to summarize an infinite variety of signal features, although measures of frequency and amplitude are among the most commonly explored. As computational power, machine learning and complex statistical techniques, and enthusiasm for data-driven knowledge discovery grow, qEEG research is expected to be an increasingly important area of exploration. Whether or not qEEG-based analyses add prognostic information when added to standard tools (e.g. qualitative EEG or other modalities); be used to interrogate underlying pathophysiological mechanisms or determine dominant drivers of abnormal EEG signals (e.g. differentiate medication-induced changes from those that result from structural damage); or be used to titrate patient care in the intensive care unit (ICU) setting remain active areas of research.

In this issue of *Resuscitation*, Kustermann et al., report the results of their analysis leveraging qEEG measures of band-pass filtered spectral power to predict outcome after resuscitation from cardiac arrest.[8] Using statistical methods common in laboratory-based neurophysiology research but infrequently applied to ICU-derived data, they identified and validated that lower power in the theta and alpha frequency ranges (specifically, 5.2–13.2 Hz with spectral power dichotomized at the optimal threshold value) was a highly specific predictor of persistently unfavorable Cerebral Performance Category (CPC) during the first three months after cardiac arrest. Using spectral power in this frequency range as a binary predictor performed better than qualitative assessment of the continuity or reactivity of EEG background. The authors are to be commended for the rigor of their analytical approach, and these findings add substantively to a growing body of literature exploring EEG-based analyses as early (<72 h) prognostic tools after anoxic brain injury.[9,10] Since most patients who die after resuscitation from cardiac arrest do so after withdrawal of life-sustaining therapy for perceived poor neurological prognosis[11] and current prognostic tools leave much to be desired,[12] this work helps meet an important need faced by clinicians daily.

Frequency decomposition is an established method of qEEG analysis that has been computationally feasible for some time using fast Fourier transformations.[4] The authors rightly conclude that preservation of alpha oscillations suggests integrity of both cortical and subcortical networks. As expected, preserved alpha power strongly predicted favorable outcome. Multiple other reversible factors including oligemic cerebral blood flow, sedative medications and temperature may also alter frequency spectrograms,[1–4] perhaps explaining the fact that many patient who ultimately enjoyed favorable outcomes had power spectra below threshold, although few measurable factors appeared to differ between outcome groups among patients below threshold in this cohort. We look forward to future work by this group and others that may quantify the incremental information added by adding qEEG-based measures to a multimodal prognostic algorithm. As a resuscitation science community, we may also wish to explore the myriad of other qEEG features available using standard or novel signal processing algorithms, or multivariable time series analyses that account for other contemporaneous physiological or medication data available, may reveal information that can further differentiate the mechanistic cause of loss of spectral power in this range.

Understanding the mechanistic underpinning of observed changes in qEEG is nontrivial. Beyond improving prognostic performance, such research could allow qEEG to grow from prognostic to a tool to identify early signs of reversible or ongoing secondary brain injury, thereby allowing individualization of patient care. Such qEEG-derived data may reveal phenotypic signatures of patients amenable to specific clinical interventions, for example deeper or longer targeted temperature management or a higher mean arterial pressure. Future clinical trials might also use such a tool to identify those likely to respond to the intervention being tested, while avoiding enrolling patients likely to recover or die irrespective of treatment. As a community of physicians and scientists, we sit poised to leverage previously unavailable methods to analyze complex physiological data, improving both clinical care and patient outcomes. Continued exploration of quantitative approaches to patient phenotyping and prognostication are certain to build upon Kustermann's work and advance the field of resuscitation science.

Conflict of interest

The authors declare they have no conflict of interest.

Acknowledgements

Dr. Elmer's research time is supported by the NIH through grants 5K12HL109068 and 1K23NS097629.

REFERENCES

1. Hofmeijer J, Tjepkema-Cloostermans MC, van Putten MJ. Burst-suppression with identical bursts: a distinct EEG pattern with poor outcome in postanoxic coma. Clin Neurophysiol 2014;125:947–54.
2. Niedermeyer E, Sherman DL, Geocadin RJ, Hansen HC, Hanley DF. The burst-suppression electroencephalogram. Clin Electroencephalogr 1999;30:99–105.
3. Thomke F, Brand A, Weilemann SL. The temporal dynamics of postanoxic burst-suppression EEG. J Clin Neurophysiol 2002;19:24–31.
4. Foreman B, Claassen J. Quantitative EEG for the detection of brain ischemia. Crit care 2012;16:216.
5. Hofmeijer J, Beernink TM, Bosch FH, Beishuizen A, Tjepkema-Cloostermans MC, van Putten MJ. Early EEG contributes to multimodal outcome prediction of postanoxic coma. Neurology 2015;85:137–43.
6. Rossetti AO, Carrera E, Oddo M. Early EEG correlates of neuronal injury after brain anoxia. Neurology 2012;78:796–802.
7. Backman S, Cronberg T, Friberg H, et al. Highly malignant routine EEG predicts poor prognosis after cardiac arrest in the Target Temperature Management trial. Resuscitation 2018;131:24–8.
8. Kustermann T, Nguepnjo Nguissi NA, Pfeiffer C, et al. Electroencephalography-based power spectra allow coma outcome prediction within 24 h of cardiac arrest. Resuscitation 2019;142:162–7.
9. Elmer J, Jones BL, Zadorozhny VI, et al. A novel methodological framework for multimodality, trajectory model-based prognostication. Resuscitation 2019;137:197–204.
10. Nagaraj SB, Tjepkema-Cloostermans MC, Ruijter BJ, Hofmeijer J, van Putten M. The revised cerebral recovery index improves predictions of neurological outcome after cardiac arrest. Clin Neurophysiol 2018;129:2557–66.
11. Elmer J, Torres C, Aufderheide TP, et al. Association of early withdrawal of life-sustaining therapy for perceived neurological prognosis with mortality after cardiac arrest. Resuscitation 2016;102:127–35.
12. Sandroni C, Cariou A, Cavallaro F, et al. Prognostication in comatose survivors of cardiac arrest: an advisory statement from the European Resuscitation Council and the European Society of Intensive Care Medicine. Resuscitation 2014;85:1779–89.

Jonathan Elmer
Jon C. Rittenberger*
University of Pittsburgh, Department of Emergency Medicine, 3600 Forbes Avenue, Suite 400A, Pittsburgh, PA 15213, United StatesUniversity of Pittsburgh, Department of Emergency Medicine, 3600 Forbes Avenue, Suite 400A, Pittsburgh, PA 15213, 412-647-3078 (office)

* Corresponding author.
E-mail address: jcrst19@pitt.edu (J. Rittenberger).

http://dx.doi.org/10.1016/j.resuscitation.2019.06.287