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Continuous EEG in patients with extracorporeal membrane oxygenation support: Clinical need in multidisciplinary collaboration and standardized monitoring

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To the Editor,

We read the manuscript recently published by Amorim et al with great interest (Amorim, 2022). In this article, Amorim et al retrospectively reviewed the records of 92 adult patients on extracorporeal membrane oxygenation (ECMO) who underwent continuous electroencephalography (cEEG) during ECMO. Of these, they reported a high proportion of patients (63%) having epileptiform activity or ictal-interictal continuum patterns on electroencephalography (EEG), including 3 (3%) with non-convulsive status epilepticus, 33 (36%) generalized periodic discharges, and 4 (5%) lateralized periodic discharges. Patients with or without these epileptiform findings reportedly did not have significant differences in in-hospital mortality or neurological outcome. This study highlights the importance of cEEG in patients with ECMO support with common discovery of epileptiform activity or ictal-interictal continuum patterns.

However, this study has some limitations that need to be addressed in future studies.

First, this study was a retrospective study without a standardized neuromonitoring protocol. In this setting, not only were the timing and duration of EEGs not standardized across patients, but the indications for obtaining EEGs were reportedly driven by high clinical suspicion for seizures, thereby introducing significant bias. In contrast, our own study of 40 adult ECMO patients with sedation-cessation protocol and standardized cEEG monitoring protocol revealed no epileptiform discharges or seizures (Hwang, 2022). A standardized neuromonitoring approach in the ECMO patient population is recommended due to unreliable neurological exam, frequent use of sedatives, and limited ability to perform

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timely neuroimaging. Such approach can improve early detection of acute brain injury and neurological outcome at discharge.

Second, sedating and analgesic medications, including continuous propofol and midazolam infusions, were used as needed by the treating clinician, and a sedation-cessation protocol was not included in the study. These medications can significantly confound the EEG signals. For example, it is well-known that propofol can directly cause burst suppression, while benzodiazepines can increase EEG frequency and amplitude (Billard, 1997). Thus, their reports of 39% of patients having burst suppression is likely significantly affected by the fact that 86% of those patients were also sedated with propofol. The very low proportion of patients (4.4%) having well-defined sleep architecture may also be confounded by sedating medications. In contrast, our own aforementioned study with sedation-cessation protocol revealed 4 (10%) patients with burst suppression and another 4 (10%) patients with suppressed backgrounds without periodic discharges (Hwang, 2022). If Amorim et al account for the patients sedated with propofol, the percentage of patients with burst suppression may be more similar to our findings. Additionally, propofol, especially as it is being weaned, is known to cause periodic discharges as a marker of an anesthesia-induced encephalopathy (Husari, 2022). It is therefore not completely clear whether the observed, mostly bilateral/generalized IIC patterns were indeed related to ECMO or were instead a marker of an anesthesia-related encephalopathy. This differentiation is crucial, as it has significant treatment implications. Thus, the authors may have significantly overestimated the frequency of ictal-interictal patterns on EEG associated with ECMO.

Third, the ECMO information could have been presented more carefully. The authors reported “ECPR at any point” in Table 1, however this is not consistent with the standard definition of extracorporeal cardiopulmonary resuscitation (ECPR) and introduces many questions (Richardson, 2021). For instance, post-cardiotomy shock is a common indication for venoarterial-ECMO (VA-ECMO) and these patients often have cardiac arrest prior to or after ECMO cannulation. However, their resuscitation is, by definition, not ECPR even if cardiac arrest is the reason for ECMO cannulation. Also, it is important to present VA- and venovenous-ECMO (VV-ECMO) patients separately for the purposes of ECMO research as they are completely distinct populations with different brain injury mechanisms. In addition, the logistic regression model could have been further optimized with established ECMO risk factors for mortality and neurological outcome, such as important variables in the SAVE score (VA-ECMO) and RESP score (VV-ECMO). As EEG variables can be collinear, 5 different regression models with only one of these 5 EEG variables in each model may have been more informative.

Despite these limitations, the study by Amorim et al. presents valuable information and we welcome the authors’ effort in understanding neurophysiological patterns in critically ill ECMO patients. We also appreciate the range of variability among centers and practice patterns for the treatment of these patients. With the increasing use of cEEG in this patient population, we hope that further multi-center, prospective studies will be performed to better understand the impact of ECMO on brain health and brain injury.

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