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Letter to the Editor

It was not true under therapeutic hypothermia

To the Editor,

A recently published article in Switzerland by Kustermann et al.[1] has reported a high level of the predictive performance of electroencephalography-based power spectra within 24 h from coma onset in patients following cardiac arrest (CA). The authors evaluated the ability of electroencephalography (EEG) to predict long-term outcomes and claimed that on the first day of coma following CA, low power spectra values around 10 Hz, typically linked to impaired cortico-thalamic structural connections, are highly specific of unfavourable outcome. However, despite this exemplary work, we have some concerns in regards to their study conclusion.

Mild therapeutic hypothermia (TH) is an effective treatment approach for organ protection following CA.[2] Studies have confirmed that cooling can cause peripheral vasoconstriction and shivering, reduce vascular volume and lead to skin breakdown, infection and increased oxygen consumption, and the patient seems relatively hypovolemic reductions.[2] To prevent the above complications (pain, vasoconstriction and shivering), anesthetics, neuromuscular blocker and sedatives were usually administered during TH in patients following CA.[2] Under such the setting, a key question is raised: the true and accurate neurological function status was not reflected by the EEG during the phase of cooling while patients were administered sedative, neuromuscular blocker agents and anesthetics after CA.

First, new indexes using part of the cortical frontal electroencephalographic (EEG) signal (e.g., Bispectral Index, Entropy, Patient State Index) have failed to demonstrate their reliability and superiority.[3] Indeed, current brain monitors available for intraoperative analysis of the cortical frontal EEG may not adequately reflect an attempt of movement from the patient under anesthesia, especially if paralytics (neuromuscular blocker agents) are used.[3]

Second, the value and amplitude of EEG could be affected by sedatives and anesthetics, and change with the changes in the depth of anesthesia. In addition, how to accurately control the depth of sedation is still a difficult problem during cooling in patients following CA. Furthermore, hypothermia may alter the pharmacokinetics of anesthetics and result in prolonged systemic clearances of anesthetic agents.[4] During reduced core temperature, a doubling of vecuronium-induced neuromuscular block duration has been demonstrated in humans.[5] The anesthetic/analgesic regimen after traumatic brain injury (TBI) may dramatically influence the response to hypothermia.[5] Mild TH plus the accumulative effect of sedatives could have a significant impact on the activity of neurological function.

Therefore,EEG would not be the native representation of neurological function during the interventional phase of cooling in patients following CA and its predictive value becomes questionable.

Conflict of interest

No conflict of interest exits in the submission of this manuscript, and manuscript is approved by all authors for publication.

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