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that this represents a reasonable time frame. Also, we have now compared mortality distributions across patients recorded within 36 hours following admission (cEEG group: 38 of 67 patients; rEEG group: 26 of 60 patients; P = .13) and thereafter (cEEG group: 51 of 115 patients; rEEG group: 62 of 122 patients; P = .32), which remain very similar.

The EEG surveillance “during working hours” means that interpretations occurred at least 3 times during workdays (in practice, almost continuously between 8 AM and 6 PM) and 2 times during weekends, with prompt communication to the treating team.1,2 While we recognize that an ideal monitoring may really benefit from this intervention.

While we recognize that an ideal monitoring should imply 24-7 coverage by EEG specialists and our colleagues might have the luck to work in selected environments in which cEEG is continuously interpreted overnight, we believe that our design reflects current practice in most centers.

We acknowledge that antiseizure medication changes were not prespecified, reflecting the pragmatic nature of our trial.1 Their prevalence in the cEEG arm (21%, excluding sedation modifications) is lower than that of the cited, retrospective analysis (52%); these analyses appear even lower than another prospective study with a control group (cEEG, 84%; no cEEG, 27%).3 These discrepancies probably reflect the variability of definitions and ascertainment of treatment modifications; of note, we assessed changes that were specifically triggered by EEG findings within 60 hours of EEG start (a more conservative approach than in other observational studies),1 and according to our practice, antiseizure medications were introduced in virtually all patients with epileptiform EEG results.

Stating that cognitive decline and functional disability were not addressed does not seem entirely correct: Table 2 and the Supplement4 describe Cerebral Performance Category and modified Rankin Score evolution from premadmission estimation until 6 months after intervention as prespecified secondary outcomes.2 We fully agree that ascertainment of epilepsy prevalence should be included in a future trial.

While it seems reasonable to target rapid identification and treatment of nonconvulsive seizures and status epilepticus in patients with critical illness, to our knowledge, there is still no clear evidence that successful treatment of ictal events improves outcome in this setting. The results of our trial3 and some previous observations5,4 suggest that a consistent proportion of patients may not additionally benefit from cEEG: the exploratory analyses of mortality (Figure 2 in our study) did not show any major difference, both for point estimates and confidence intervals, stratifying for different causative mechanisms, comorbidities, or ages. The most promising, albeit nonsignificant, point estimate favoring cEEG appeared in patients with relatively light consciousness impairment (relative risk, 0.36), possibly suggesting that in several patients with deep comatocoonstant structural damage might represent a difficult-to-treat burden influencing the outcome, on top of seizure activity.5

Our trial3 should certainly not dissuade clinicians to perform cEEG with timely interpretation. Besides offering some rationale to accept repeated rEEG in resource-limited settings as a reasonable alternative and reminding us that cEEG by itself cannot improve outcomes,6 it should rather motivate researchers to identify in future studies those patients who may really benefit from this intervention.

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Published Online: February 1, 2021. doi:10.1001/jamaneurol.2020.5343

Correction: This article was corrected in March 11, 2021, to change the phrase “imitated generalizability” to “limited generalizability.”

Conflict of Interest Disclosures: Dr Rossetti reported grants from Swiss National Scientific Foundation paid to the institution during the conduct of the study. Dr Schindler reported grants from Swiss National Science Foundation during the conduct of the study. No other disclosures were reported.

Additional Contributions: We thank Stephan Ruegg, MD, Basel University Hospital, for his thoughtful input. He was not compensated for this contribution.


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CORRECTION

Omission of Group Information: The Original Investigation titled “Characterization of Alzheimer Disease Biomarker Discrepancies Using Cerebrospinal Fluid Phosphorylated Tau and AV1451 Positron Emission Tomography,” published online January 21, 2020,1 was missing the ADNI Investigators in the byline. The group author notation “for the ADNI Investigators” now appears in the byline, and a complete list of the collaborators appears in the eAppendix of the Supplement. This article was corrected online.


Error in Figure and Author Affiliation: In the Images in Neurology by Liu et al, titled “Bilateral Temporal Pole Involvement in Adult-Onset X-Linked Adrenoleukodystrophy,” published online December 21, 2020,2 there were errors in a figure and the author affiliations list. In Figure 1B, the yellow arrowheads on top should be blue, and the blue arrowheads below should be yellow. In the author affiliations list, the first author, Dr Liu, should be affiliated with the Department of Neurology at Tongji Hospital in Wuhan, China, instead of the Department of Radiology. This article was corrected online.