be of great interest to measure interleukin-1β concentration at the level of neurons; however, this is a challenging task to achieve with the technologies currently available.

The mechanisms by which inflammation causes anesthetic sensitivity in patients are complex and difficult to resolve in a single study. Therefore, in the Discussion, we stated that "These findings from animal studies cannot be directly extrapolated to patients."[1] Nevertheless, we anticipate that our data will further clinical efforts to identify the biologic underpinning of anesthetic hypersensitivity, in order to optimize anesthetic dosing in critically ill patients.

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Competing Interests

The authors declare no competing interests.

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Unmasking of Focal Neurologic Dysfunction Caused by Sedation: Concerns and Ambiguity

To the Editor:

We read with interest the article by Lin *et al.*[1] in the March 2016 issue of Anesthesiology. We appreciate and congratulate the authors for conducting this interesting study and sharing some clinically useful findings. However, there are a few queries, which have intrigued our minds.

1. We remain unclear on whether recruitment of patients with supratentorial mass lesion (with midline shift) in this study for sedation without further diagnostic or therapeutic intention is appropriate.
2. As stated in study protocol, investigators have excluded the patients who were oversedated after giving additional doses of respective sedative agents in each group to get National Institutes of Health Stroke Scale of two points. We are unclear as to how many patients became oversedated with this strategy followed in each group.
3. It would be useful to know the clinical outcome of patients in whom unmasking/exacerbation of neurologic deficits occurred. It would also be meaningful to know whether these deficits were of transient nature or persisted for some time. In this context, the authors should have mentioned the time of onset and duration of neurologic deficits after initiation of the sedation protocol.

Competing Interests

The authors declare no competing interests.

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1. Lin N, Han R, Zhou J, Gelb AW: Mild sedation exacerbates or unmasks focal neurologic dysfunction in neurosurgical patients with supratentorial brain mass lesions in a drug-specific manner. Anesthesiology 2016; 124: 598–607

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In Reply:

We thank Professor Bithal and Dr. Tomar for their interest in our study.[1]

Their first question suggests that these patients had not been appropriately evaluated before and during the scheduled craniotomy. The study, as we indicated, was performed in the operating room immediately before the craniotomy. The patients had undergone all necessary and appropriate evaluations including imaging before surgery being scheduled. We see nothing to be gained by including every patient's preoperative assessment in the article beyond the tumor diagnosis and imaging information. Further, pathologic diagnosis was obtained at craniotomy, and this was the basis for our reporting the pathologies and subgroups of glioma grades.

The answer to their second question, the number of oversedated patients who were excluded from the study, can be found in the article. Figure 2 shows the number of patients who were over- and undersedated and were therefore excluded.

Neurologic evaluation was performed once patients reached Observer's Assessment of Alertness/Sedation scale 4. The speed of onset depends on the pharmacologic characteristic of each sedative, *e.g.*, propofol faster than dexmedetomidine. We did not specifically record the exact time for each patient. The duration of the effects we noted is indeed important. However, assessing the duration of an effect we had not yet demonstrated was not the aim of the study and its design. As the patients were anesthetized as soon as we completed our assessments, we cannot answer the question beyond stating that ongoing studies would address the question.

Competing Interests

The authors declare no competing interests.

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