In conclusion, the available data do not support methylphenidate causing cognitive dysfunction and delirium if it is administered to induce emergence from general anesthesia. On the contrary, available data suggest that methylphenidate could be administered safely to anesthetized patients in the immediate postsurgical period. There are specific instances when it could be very useful. For example, methylphenidate may find use as a “rescue” drug for patients induced with propofol, who cannot be ventilated or intubated, as it increases both arousal and respiratory drive.7 Methylphenidate may also be useful at the end of prolonged total intravenous anesthetics with propofol (e.g., for complicated spine surgeries that require neurological monitoring), after which patients often have delayed emergence. Having a drug that restores arousal and respiratory drive in the anesthesiologist’s armamentarium would improve the safety of propofol administration as well as efficiency in the operating room. Nevertheless, while the available data suggest that methylphenidate administration to patients under propofol anesthesia would be safe, further research is needed to define the therapeutic benefits and possible side effects of this drug combination.

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Utility of Near-infrared Spectroscopy for Assessing Cerebral Oxygen Saturation during Beach Chair Position

To the Editor:

We read with interest the article by Jeong et al.1 on cerebral oxygen saturation measured by near-infrared spectroscopy (NIRS) and jugular venous bulb oximetry during shoulder surgery in the beach chair position. Their results showed a significant lower jugular venous bulb oxygen saturation during beach chair position in the propofol–remifentanil group compared with the sevoflurane–nitrous oxide group. This difference between the two groups was not found for NIRS–measured cerebral oxygen saturation, which led the authors to conclude that NIRS cannot be recommended for assessing cerebral oxygenation during beach chair position.

The authors explain the lack of agreement between cerebral oxygen saturation measured by NIRS and measured with jugular bulb oximetry by emphasizing on the inherent limitations of the NIRS technology, such as the assumed constant arterial/venous ratio and the contamination by extracerebral blood flow and by other tissues that are not the tissues of interest. We would like to suggest an alternative explanation for the observations. In a recent study by Klein et al.,2 it was demonstrated that propofol has a differential effect on the cerebral vessels, and that propofol preserves cerebral oxygen saturation in the frontal cortex, which is the measurement site of NIRS. Therefore, the finding that oxygen saturation measured by NIRS does not differ between the sevoflurane–nitrous oxide and the propofol–remifentanil group, and it does with jugular venous bulb oximetry, could be attributable to the fact that NIRS measures oxygen saturation in the frontal cortex, whereas jugular venous bulb oximetry measures global cerebral oxygen saturation.

Another concern relates to the fact that the authors used the original Bland-Altman method to assess whether cerebral oxygen saturations measured by jugular venous bulb oximetry or by NIRS were interchangeable. However, this Bland-Altman technique accounts for independent data, not for repeated measurements over a period of time, such as in this study. For both the linear regression and Bland-Altman analysis performed in this study, a technique that accounts for the within-patient–dependent data is mandatory. Consequently, the agreement estimation might have been obscured by the use of repeated-measures data.3

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