patients with heterogenous injury in whom cerebral perfusion pressure (CPP) decreases into the 70–90-mmHg range. They make a very persuasive argument that increased ICP in this subset of patients is due to the exponential increase in cerebral blood volume that occurs when CPP decreases into the 70–90-mmHg range. When an exponential increase in cerebral blood volume is produced in a patient whose intracranial pressure-volume relationship is on the exponentially increasing portion of that curve, it is not surprising that a small decrease in MAP, through reflex vasodilatation of brain areas with intact autoregulation, might result in an increase in ICP.

The authors appropriately indicate the possibility that seizure activity could have contributed to their observation, but they report that electroencephalographic evidence of this in humans is lacking. In a recently published study, however, Tempelhoff *et al.* gave fentanyl doses of 7.7–35.7 µg/kg to epileptic patients and observed hippocampal seizure activity with intracranial electrode recordings. This study circumvented the pitfall of missing deep seizure activity with the commonly used surface scalp electrodes or intranasal electrodes. Although the seizure activity was observed in patients with epilepsy, it occurred in the temporal lobe, which was not the epileptic focus. In addition, abstracts by Kearse *et al.*<sup>5.6</sup> have reported dose-related spike activity detected by 20-channel scalp-electrode recordings in humans undergoing induction of opioid anesthesia for cardiac or carotid surgery.

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In Reply:—Kofke and Tempelhoff refer to the possibility that a modestly decreased cerebral perfusion pressure may explain the observed increase in intracranial pressure (ICP) is an important observation. It is true that autoregulatory responses to decreased cerebral perfusion pressure may be associated with an increased ICP. While this may be a contributing factor, in this instance, it is unlikely to be the only causative factor. The standard deviation for the ICP response in our patients was 10 mmHg. This heterogeneity did not correlate with the decrease in cerebral perfusion pressure. Thus, I find it unlikely that decreased cerebral perfusion pressure was anything more than contributory.

Kofke and Templehoff add very recent information to our discussion

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of potential mechanisms for our observations. Their insights on subcortical seizure activity and neuroexcitation associated with opioid administration are a significant addition to our discussion.

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