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In Reply.—We recognize Tommasino’s expertise in the field of brain imaging related to altered states of consciousness and thank her for her comments. We agree that it is always preferable to be semantically as precise as possible when communicating at the scientific level. Although her comments do not affect the results of our study, she is correct to point out the distinction between the coma state and the vegetative state. Therefore, our statement, which attempted to place the magnitude of the metabolic reduction seen during propofol anesthesia into an easily understood frame of reference, could be clarified as follows:

In our study, the anesthetic state produced by propofol was associated with a reduction of whole-brain cerebral metabolic rate of glucose utilization (CMRglu) of about 55%. Non-REM sleep in humans is associated with a CMRglu reduction of about 12.6-23%. One study suggests coma is associated with a 45% CMRglu reduction. Additionally, the vegetative state appears to be associated with an approximately 35-65% CMRglu reduction. Thus, the CMRglu reduction associated with the anesthetic state produced by propofol is somewhat larger than that seen in non-REM sleep, is in the range (or slightly more than) that seen with coma, and may be somewhat less than that seen with the vegetative state.

References


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Carbon Dioxide Reactivity and Cerebral Ischemia: Is the Cortical Somatosensory Evoked Potential a Sensitive Detector of Cerebral Ischemia?

To the Editor.—Thiel et al. attempted to establish a relationship between preoperative changes in cerebrovascular reactivity to carbon dioxide as determined by transcranial Doppler sonography and intraoperative changes in the cortical somatosensory evoked potential (SEP) waveforms. The authors concluded that in patients with impaired carbon dioxide reactivity, the risk of cerebral ischemia, as assessed by intraoperative SEP recordings during carotid endarterectomy, was not increased. We are not sure, however, that the monitor used to detect cerebral ischemia is sensitive enough to warrant that conclusion. The investigators used cortical SEPs to monitor for cerebral ischemia based on the rationale that there is consensus that SEP monitoring is both highly sensitive and specific in detecting ischemia during carotid surgery. In our opinion, that statement contains a bias according to the results of two other studies, a laboratory study examining the threshold at which SEP changes during ischemia and a clinical study comparing SEP monitoring with electroencephalography (EEG) during carotid vascular surgery. The data from these studies suggest that SEP is less sensitive than EEG in detecting cortical cerebral ischemia. Nevertheless, data from humans do not show that one monitor is superior to the other in predicting clinically detectable postoperative neurologic deficit. Answering that question would require a very large study. In the absence of such a study, EEG monitoring is considered by many clinicians involved in neurologic monitoring, including us, to be more sensitive in detecting cerebral ischemia during carotid surgery. SEP monitoring likely misses mild ischemia, which might be reflected by EEG changes. Using continuous EEG as a monitor of ischemia would better test the primary hypothesis that preoperative loss of cerebral reactivity predicts...
an increased risk of intraoperative cerebral ischemia. In our opinion, the authors can conclude only that, in patients with impaired carbon dioxide reactivity, the risk of severe cerebral ischemia is not increased during carotid cross-clamping.

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In Reply.—Although Mahla and Sulek suggest that the “ideal” monitor to detect cerebral ischemia during carotid endarterectomy has not been found, there are convincing data from human studies that somatosensory evoked potentials (SEPs) after median nerve stimulation are highly specific and sensitive to detect cerebral ischemia during carotid surgery.1,2 which, in our opinion, may make SEP monitoring superior to electroencephalography (EEG) for three reasons. First, EEG monitoring appears to be “oversensitive” as ischemic changes will occur in up to 10% intraoperatively, whereas new postoperative neurologic deficits even with a “no-shunt” regimen will not exceed 8–10% in experienced centers. The latter percentage is remarkably close to the findings of our group,1,3 which indicates that SEP changes indicating ischemia (amplitude reduction >50%, central conduction time (CCT) prolongation >20%) will occur in 10–15%. Thus, it is not surprising that Kears et al.4 found SEP much less sensitive when compared with the high incidence of ischemic EEG changes (45%) in their study. Second, EEG monitoring methods may not be sensitive enough to detect ischemia (false negative) in patients suffering from preoperative neurologic symptoms.2,5 whereas false-negative SEP findings in carotid surgery have been reported in only one study.11 Third, EEG monitoring relies on the detection of multiple patterns that, in the absence of computer-aided analysis, are difficult to quantify and require an experienced neurophysiologist. SEP recordings, in contrast, allows the identification of ischemia using only a few quantitative parameters (latency and/or CCT, amplitude).

However, in the absence of carefully controlled clinical studies demonstrating the superiority of one monitoring method, EEG remains undoubtedly an appropriate means to detect cerebral ischemia during carotid surgery. We agree with Mahla and Sulek.

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