Electroencephalogram and Anesthetics

To the Editor:

I was intrigued by the article by Warnaby et al., who reported that electroencephalographic slow-wave activity saturation is observed for both intravenous and volatile anesthetics.1 Furthermore, they found that opiates reduced the concentration of anesthetic at which slow-wave saturation was observed. In contrast, they reported that muscle relaxants did not alter the anesthetic concentration at which electroencephalographic slow-wave saturation occurred. Their results may lead to the erroneous conclusion that muscle relaxants do not alter the electroencephalographic effects of anesthetics. By comparison, our own study in dogs demonstrated that pancuronium neuromuscular blockade potentiated electroencephalographic burst suppression elicited by isoflurane.2 This effect on electroencephalography was reversed by the administration of neostigmine. Furthermore, the description of electroencephalographic burst suppression by Warnaby et al. as an “artefactual disturbance” greatly obscures this issue. Most certainly, electroencephalographic burst suppression is not an artefactual disturbance, as multiple reports confirm that dose-dependent electroencephalographic burst suppression is elicited by a wide variety of anesthetic agents of diverse chemical structure.3 For all of these anesthetics, electroencephalographic burst suppression is associated with a dose-related decrease in cerebral metabolic rate. Indeed, altered electroencephalographic burst suppression in elderly patients confirms the accepted principal of age-related shifts in the pharmacodynamics of volatile anesthesia.4,5 It should also be noted that in their clinical study, Warnaby et al. failed to control for dose, timing, or even the identity of muscle relaxants. They also fail to explain why values of N in their own work, cited by this article, do not always agree with values of N reported in the original publications.

In Reply:

We thank Colin et al. and Schwartz for their interest in our recently published work in Anesthesiology.1 In the referenced manuscript, we fitted slow-wave activity drug dose-response curves to electroencephalographic data acquired during anesthesia in a propofol healthy volunteer study and three patient studies. By applying these techniques to induction and emergence, we presented two distinct, but related, findings. First, as described by Schwartz, we confirmed that our experimental finding of slow-wave activity saturation also occurs during surgical anesthesia. While we entirely agree that exploration of the transition to burst suppression is important, this was not the focus of our article. Slow-wave activity saturation occurs at considerably lower levels of anesthesia than burst suppression (fig. 1G of Warnaby et al.)—hence why the presence of burst suppression was considered to be artefactual in the fitting of slow-wave activity–concentration curves. Furthermore, we did not imply that muscle relaxants have no influence on the electroencephalogram, only that they had no influence on the slow-wave activity saturation parameters (i.e., the power and concentration of slow-wave activity saturation, defined by the electroencephalographic dose-response curve fit). Neuromuscular blocking

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