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Effective Topical Anesthesia for Awake Tracheal Intubation

To the Editor—We administered 2% lidocaine jelly (fig. 1) with a 10-ml syringe attached to a 4–5-inch long plastic suction catheter to the base of the tongue and pharynx of the patient. A total of up to 10 ml 2% lidocaine jelly is squirted on the tonsillar pillar area on each side. Administration of the topical anesthetic on the laryngeal side of the epiglottis and the larynx is avoided to preserve protective reflexes of the superior laryngeal nerve. The jelly, as opposed to solutions, adheres to the mucosal surfaces more effectively with quicker penetration. We see an almost immediate effect with the patients after application, allowing us to perform direct laryngoscopy and tracheal intubation smoothly.

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Reconciling Differences between In Vitro and In Vivo Effects of Propofol

To the Editor—Bansinath et al. reported the effects of propofol on seizures induced in mice by a variety of chemical agents. They demonstrated that propofol and GABAa, glycine, or glutamate receptor subtypes were examined. The authors correctly emphasize the importance of using both in vivo and in vitro paradigms to understand the mechanism of anesthesia. However, they suggest that there is a disparity between their results and the information available from in vitro studies regarding the effects of propofol on excitatory amino acid receptors.

It is evident from Bansinath et al.’s table 3 that propofol reduced the incidence of NMDA-induced convulsions, and this anticonvulsant effect was observed at all concentrations of NMDA tested. Moreover, the slopes of the lines in the log–probit plot (their fig. 2C) were similar, suggesting that the anticonvulsant property of propofol was mediated in part through modulation of the NMDA receptor. The calculated effective dose of NMDA that induced seizures in 50% of mice was apparently similar in the presence or absence of propofol. These data are consistent with in vitro studies that indicate propofol...
causes a noncompetitive inhibition of the NMDA receptor. Propofol is thought to modulate the NMDA receptor at a domain other than the agonist recognition site and, hence, would not influence the affinity of receptor for agonist. Accordingly, in vitro studies predict that propofol would reduce the incidence of NMDA-induced seizure activity without affecting the calculated EC50.

Bansinath et al also observed that propofol increased the incidence of kainate- and quisqualate-induced seizures. Propofol failed to produce a consistent effect on kainate-evoked responses recorded from cultured mouse hippocampal neurons, possibly because of the variety of kainate receptor subunits present in native neurons. However, examination of specific subtypes of non-NMDA receptors expressed in *Xenopus* oocytes demonstrated that propofol enhanced the currents recorded from the α1 (GluR1) subfamily of AMPA/quisqualate-sensitive channels. The effects of propofol on non-NMDA receptors are highly dependent on the subunit examined. Furthermore, kainate activates the AMPA/quisqualate receptors, whereas quisqualate activates the metabotropic receptor, a G-protein coupled glutamate receptor. These chemoconversants are not sufficiently selective to make inferences regarding their behavioral properties and specific receptor populations.

Because of the enormous complexity of neuronal circuitry, it is generally difficult to link the clinical effects of anesthetics to specific receptors. However, the data presented by Bansinath et al are consistent with propofol’s demonstrated ability to inhibit the NMDA receptor and enhance certain subtypes of non-NMDA glutamate receptors.

References

1. Bansinath M, Shukla VK, Turnendorf H: Propofol modulates the effects of chemoconversants acting at the GABAergic, glycergic, and glutamate receptor subtypes. Anesthesiology 1995; 83:809–15

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In Reply — We thank Orser and MacDonald for their opinion, which highlights the reliability of our results on modulation of glutamate receptor subtypes by propofol. A similar interpretation of our data on modulation of NMDA-induced convulsions by propofol was made by one of the reviewers of the manuscript. The data suggest some inhibitory action of propofol against NMDA-induced convulsions. We deemed it inappropriate to be conservative in our inference on data that were not statistically significant, especially because these results were from an in vitro paradigm and thus vulnerable to modulation by multiple factors. On the contrary, the in vitro results have the advantage of being immune to the impact of multiple factors working in concert. Hence, it is reassuring to note that some in vitro data, published after our manuscript was processed, bolster our in vivo findings.

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References

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