CORRESPONDENCE

10. Bracken MB, Holmof TR: Effects of timing of methylprednisolone or naloxone administration on recovery of segmental and long-


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Reference

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Nonanesthetic Haloalkanes and Nicotinic Acetylcholine Receptor Desensitization Kinetics

To the Editor.—Raines reports the effect of volatile anesthetics (enflurane and isoflurane) and 'nonanesthetics' (2,3-dichloroocta-
fluorobutane and 1,2-dichlorohexafluorocyclobutane) on the desen-
sitization kinetics of the Torpedo nicotinic acetylcholine receptor
(nAChR).1 The less pronounced effect of nonanesthetics on the desen-
sitization kinetics of this membrane protein is interpreted as evi-
dence that the system is a valid model of the volatile anesthetic site
of action, based on a somewhat contentious criterion suggested by
Eger and colleagues.2 Indeed, these nonanesthetics were described
recently as inducing at least one component of the anesthetic state.3

The nonanesthetics studied were chosen, in part, because they
lack a bromine atom that may quench fluorescence. However, halo-
genated molecules that contain chlorine atoms also may quench
fluorescence,4 in some cases with an efficiency (the probability that
an encounter between quencher and fluorophore will result in energy
transfer) that exceeds that of brominated compounds. Therefore,
using quenching of indole fluorescence in methanol (as a model for
tryptophan residues in proteins), 1,2-dichlorohexafluorocyclobutane
is found to be a superior quencher compared with halothane, whereas
2,3 dichlorooctafluorobutane is 84% as efficient as halothane (fig. 1).
The effective fluorescence quenching exhibited by these bichlor-
inated compounds is presumably due to the electron withdrawing
influence of the neighboring fluorines, which results in favorable
conditions for electron transfer to the chlorine atoms. The lack of a
bromine atom, therefore, is not a valid reason to exclude direct flu-
oscence quenching by the test compound.

An alternative interpretation of the results reported by Raines1 is
that quenching of a portion of the native nAChR tryptophan fluore-

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