To the Editor:—Cellular heterogeneity and access to the brain under basal conditions are major problems for investigators concerned with the role of cyclic nucleotides in the central nervous system. The studies cited by Dr. Kraynack do not resolve these problems.

Cohn *et al.*[1] reported reversal of narcosis with intracerebroventricular injection of large amounts of db-cAMP. Amobarbital narcosis could not be prevented by prior administration of db cAMP, nor could righting reflexes be maintained in the early stages of narcosis. Cohn has suggested a role for cyclic nucleotides in arousal from narcosis but not, quite rightly, in a biochemical mechanism of narcosis.

My review outlined principles of cyclic nucleotide physiology established along the lines suggested by Sutherland. Eventually, cyclic nucleotides may be shown to play a role in narcosis, but speculation at the present time is not supported.

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REFERENCE

1. Cohn ML, Yamaoka H, Taylor FH, et al: Action of intracerebroventricular dibutyryl cyclic AMP on amobarbital anaesthesia in rats. Neuropharmacology 12:401–405, 1973

(*Accepted for publication December 4, 1974.*)

Intratracheal Lidocaine—Local Anesthesia or Direct Cardiac Effect?

To the Editor:—Denlinger, Ellison, and Ominsky (ANESTHESIOLOGY 41:409–412, 1974) observed that the "hypertensive response (following intubation in patients anesthetized with morphine and nitrous oxide can be significantly decreased by a simple intratracheal spray with lidocaine, 4 per cent."

The dose used in their study (120 mg/70 kg) is of the same order as that often given intravenously in the treatment of many tachycardias and arrythmias. It has long been known that drugs put into the tracheobronchial tree are absorbed almost as quickly as if they had been injected intravenously.[1] Therefore, we are left wondering whether the reported beneficial effect was caused by local anesthetic blockade of noxious reflex stimuli, as the authors imply, or by direct cardiac sedation from rapid absorption into the circulation. If the latter were true, to inject some lidocaine into a vein would be simpler and quicker than performing the extra laryngoscopy and spray.

I hope that Denlinger and colleagues will carry their work a little further and give us a more clear answer.

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REFERENCE

1. Campbell D, Adriani J: Absorption of local anesthetics. JAMA 168, 873–877, 1958

(*Accepted for publication December 10, 1974.*)

To the Editor:—Intravenous injection of lidocaine during nitrous oxide anesthesia has been reported to increase arterial blood pressure[1] by means of an effect on the autonomic nervous system.[2,3] It is unlikely, therefore, that the beneficial effect of intratracheal lidocaine that we observed is due to the hemodynamic effect of lidocaine *per se*, as Thomas suggests.

To the best of our knowledge, there is no clinical or experimental evidence to suggest that lidocaine, 120 mg/70 kg, iv, would be effective in preventing hypertension or tachycardia in response to a noxious stimulus. We would, of course, be most interested in