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Hypothermia and the Electroencephalogram

To the Editor:—In their article on hypothermia and thiopental, Quasha *et al.*[1] observed burst-suppression in their control group, and attributed this EEG finding to the hypothermia common to all groups. This is remarkable, because neither our clinical experience nor the published reports of others[2,3] supports the observation that moderate hypothermia (25–30° C) *by itself* produces prominent burst-suppression. Their study was performed at the start of cardiopulmonary bypass, a period when many physiologic changes are being imposed upon the previously stable cerebral conditions. Included are thermal gradients (not just hypothermia) and acute hemodilution, with associated changes in serum proteins, electrolytes, glucose, osmolarity, and blood viscosity and oxygen-carrying capacity. Halothane was administered during the study period to maintain anesthetic level; however, the anesthetic potency of this agent doubles when the temperature is reduced to 27° C.[4] Thus, an anesthetic steady state may not have been present. The presence of an unusual, and possibly abnormal, control state raises questions about the general applicability of their conclusions. Accordingly, studies under stable hypothermic conditions seem to be indicated before these conclusions (no matter how reasonable) can be accepted.

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In reply:—Dr. Warren Levy's letter raises a number of important questions and indirectly suggests likely answers. First, as Dr. Levy indicated, it is known that hypothermia significantly increases the potency of halothane. As noted, this, plus the various transient changes that occur at the start of cardiopulmonary bypass, may well explain why we observed some transient burst suppression in our control group at moderate hypothermia (25–30° C) that by itself may not produce prominent burst suppression. Further, since hypothermia has been demonstrated to potentiate the effect of halothane, it should not be too surprising that it likely will also potentiate the effects of thiopental, which is the central point of our paper. Nonetheless, we appreciate the possible confounding effect that can be introduced by the various uncontrolled transient changes in numerous variables at the start of cardiopulmonary bypass. Therefore, although we consider our current conclusions as reasonable, we fully recognize them as tentative and in need of further verification based on more controlled studies, including basic animal studies, as well as clinical trials.

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Nalbuphine and Droperidol Combination for Local Standby Sedation

To the Editor:—During the daily practice of anesthesiology, it is not uncommon to be called upon to deliver sedative medications to the patient undergoing therapeutic and/or diagnostic procedures under local anesthesia. These "local standby" anesthetics, as practiced at this institution, consisted largely of intravenous diazepam and a narcotic. An alternative regimen that avoids some of the therapeutic hazards associated with these agents is presented below.

A safe and consistent regimen well-suited for procedures requiring sedation is the combination of 30 mg/70 kg nalbuphine given as a slow intravenous push followed in five minutes by 2.5 mg droperidol, iv. After one hour, an additional 10 mg nalbuphine and 2.5 mg droperidol may be given. This regimen appears to provide superlative sedation with easy arousability associated with minimal respiratory depression. As a side benefit, patients can be expected to remain comfortable for a period of time up to six hours. Minor side effects, including perinasal and perioral pruritis, may be seen, and patients may complain of burning at the iv site if the nalbuphine is given too rapidly.

The nalbuphine/droperidol combination is a logical choice of agents based upon the predictability of side reactions expected from each of these agents. The respiratory depression commonly seen with narcotics is less of a concern with nalbuphine. There is a ceiling respiratory depression seen with nalbuphine at the dosage of 30 mg/70 kg, and corresponds with a mean displacement of the CO_2 response curve of 9.2 mmHg Pa_{CO_2}.[1] The degree of sedation expected with a 30 mg/70 kg dosage of nalbuphine is seen under the circumstances as a desirable therapeutic effect.*

The use of droperidol in place of diazepam is based upon the antiemetic properties of droperidol[2,3] and the small but significant incidence of respiratory arrest associated with intravenous diazepam. If droperidol is administered five minutes after the first administration of nalbuphine, dysphoric reactions that occasionally accompany the use of droperidol appear to be prevented. Further, droperidol enhances the degree of sedation provided by nalbuphine and diminished patient recall can be expected. This dosage regimen has been used with equally adequate results on both alcoholic and nonalcoholic patient populations. One additional benefit is the convenience of avoiding additional paperwork through the use of these non-controlled pharmaceuticals.

In conclusion, the combination of nalbuphine and droperidol appears to provide an adequate, safe alternative to diazepam/narcotic combinations for sedation in procedures performed under local anesthesia.

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* Magruder MR, Christofforetti R, Difazio CA: Balanced anesthesia with nalbuphine hydrochloride. Anesthesiology Review 7:25–29, 1980.