CORRESPONDENCE

Anesthesiology
68:296, 1988

Midazolam in Obstetric Anesthesia—A Reply

To the Editor:—In the initial letter to the editor on the subject of midazolam use as an adjunct drug with regional anesthesia in obstetrics, Camann *et al.*[1] noted that its "superior amnestic qualities . . . may be counterproductive in the obstetric unit." They employed doses of from 2–7 mg iv immediately after the umbilical cord was clamped, but failed to correlate dose employed with the mentioned undesirable side effect of maternal amnesia.

In a follow-up letter, Heyman *et al.*[2] confirmed the problem of unwanted amnesia for the birth of the baby. They suggested delaying administration of midazolam "until after pediatric ministrations to the neonate in the operating room are completed, and the baby is brought to the mother and shown to her . . ." Using this technique, they report no further complaints of amnesia for the birth experience. They failed to mention the elapsed time from delivery until the administration of midazolam.

Most recently, Seidman *et al.*[3] made the rather strong statement that "the use of midazolam in obstetrics should, therefore, be limited to special indications." This statement is not supported by any references but, rather, appears to be based upon the particular philosophy of the authors regarding maternal participation during the entire peripartum period. In addition, they neglect to mention what any of these special indications might be.

We have been successfully using midazolam at our institution in the delivery suite for the past 9 months. The drug is used as an adjunct to epidural anesthesia/analgesia for delivery. The dose of midazolam ranges from 2–4 mg iv. We do not administer midazolam until after the baby has been removed from the mother following the initial bonding period. Typically, this period lasts 15–20 min from delivery. We routinely employ two sources of patient follow-up for the birthing experience: 1) postoperative rounds by the anesthesiologist, and 2) a patient questionnaire sent to the patient's home following discharge. From both sources, we attempt to elicit any unpleasant experiences by directed, leading questions related to the perioperative period. All patients are seen within the first 24 h post-delivery, and the response from the questionnaires is above 80%. While we certainly have our share of problems, nobody to date has mentioned or complained of an unpleasant birthing experience that we can relate to midazolam's amnestic properties. We strongly feel that midazolam is a very useful adjunct for increasing maternal comfort during the birthing experience with regional anesthesia/analgesia. Like all drugs, it must be used at the appropriate time and in the appropriate dose. In their hands, the other modalities noted by Seidman *et al.* may be equally effective in providing maternal comfort.

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(Accepted for publication August 6, 1987.)

Anesthesiology
68:296–297, 1988

Epidural Ketamine Does Not Produce Analgesia

To the Editor:—I read with great interest the article by Ravat *et al.*[1] They compared the epidural ketamine with that of morphine, and could not confirm the analgesic action. We reported previously that the neural mechanism, in the spinal cord, of the analgesic action of anesthetics must be considered by two mechanisms: the direct action on the neural transmission at the spinal cord dorsal horn, and the indirect action exerted through the action on the supraspinal pain inhibition system.[2] It is well known that the opioid analgesia is exerted

through these two mechanisms. Using decerebrate cats, we studied the neural mechanism of analgesic action of anesthetics, such as ketamine, barbiturates, nitrous oxide, halothane, and enflurane. The analgesic action was assessed by the suppression of the neural response, in the spinal cord, to the intra-arterially administered bradykinin. The drug action was compared before and after the spinal cord transection above the recording site. A small dose of ketamine, 2 mg/kg iv, produced a significant suppression of the bradykinin-induced response when the spinal cord was intact. However, after the spinal cord transection, the suppression disappeared almost completely, even with a huge dose of 40 mg/kg iv. Such a lack of direct suppression by the anesthetic was also observed with enflurane. All other anesthetics, on the other hand, showed the direct suppressive action of various degrees. These indicated that the neural mechanism of ketamine-analgesia was rather exceptional as an anesthetic, and was produced solely by the activation of the supraspinal pain inhibition system. The possibility of ketamine-analgesia produced by the direct action on the spinal cord was, thus, ruled out.[3,4] We referred the activation of this system by ketamine to its CNS excitant action we observed in the brain stem reticular core.[5] The controversy to this postulate is the report by Kitahata *et al.*,[6] who observed a significant suppression in the cell activity of the spinal cord dorsal horn in the spinal cord-transected cats. However, our view supported Conseiller *et al.*,[7] who, using spinal cord-transected cats, observed little suppression, by ketamine, of the dorsal horn cell response to the noxious stimuli, and could not explain the clinically observed potent analgesic action of ketamine. The study by Ravat *et al.*, if it is alone, also fails to explain the clinically observed potent analgesia produced by its systemic administration. However, their findings in man are in accordance with those in cats reported by us and Conseiller *et al.*, and can be explained better by the lack of direct suppression of pain transmission at the spinal cord dorsal horn.

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(Accepted for publication August 27, 1987.)

Anesthesiology
68:297–298, 1988

In Reply:—"Selective spinal analgesia" which was observed with ketamine[1] was explained by binding of ketamine to opioid receptors.[2] However, this point is controversial[3] and, in our study,[4] epidural ketamine was unable to relieve postoperative pain (whereas epidural morphine provided maximal analgesia in all cases) contrary to previous findings.[1,5–7] The purpose of our study is not to explain the potent analgesia produced by systemic action of ketamine, but to compare epidural ketamine to epidural morphine for postoperative pain relief. The animal study conducted by Mori *et al.* demonstrated that ketamine analgesia was not mediated by a direct action on dorsal horn; this could explain the negative results we observed with epidural ketamine. Therefore, epidural ketamine is not a good choice for postoperative analgesia.

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