

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 ad-

ministration. 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 ketamine¹ was explained by binding of ketamine to opioid receptors.² However, this point is controversial³ 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.* demon-

strated 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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(Accepted for publication October 6, 1987.)

Anesthesiology
68:298, 1988

Percutaneous Transtracheal High-frequency Jet Ventilation as an Aid to Fiberoptic Intubation

To the Editor:—Boucek *et al.* have described an innovative technique using transtracheal jet ventilation during difficult intubations.¹ While this method may be useful in unusual circumstances, it requires special equipment, is unfamiliar to many anesthesiologists, and is not without risk. Furthermore, simpler alternatives are available.

We disagree that one must necessarily ventilate the anesthetized patient during fiberoptic laryngoscopy. Spontaneous inhalation of anesthetic agents, oxygen, and even nebulized lidocaine can be easily achieved by connecting the breathing circuit to a nasopharyngeal tube or a "dual purpose connector."² Transparent adhesive dressing will provide a seal where needed. This technique, preceded by an inhalation induction, is applicable to patients with difficult airways except in the presence of: 1) decreased intracranial compliance, where hyperventilation is most reliably achieved by conventional positive pressure ventilation, and 2) a "full stomach," where jet ventilation offers no demonstrated advantage, and awake intubation is indicated.

The safety and efficacy of topical anesthesia for awake fiberoptic intubation should not be downplayed. Stating that "potentially toxic doses of local anesthetic may be necessary," the authors have quoted a paper which, in fact, demonstrated low peak plasma concentrations of lidocaine (mean $0.6 \pm 0.3 \mu\text{g/ml}$) despite the high administered doses (mean $5.3 \pm 2.1 \text{ mg/kg}$).³

We have found that the simple, familiar, and, therefore, safer adjuncts to fiberoptic laryngoscopy allow for successful intubation under most circumstances. Therefore, the use of percutaneous transtracheal jet ventilation should be reserved for extraordinary situations.

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(Accepted for publication September 8, 1987.)

Anesthesiology
68:298-299, 1988

In Reply:—We appreciate the comments of Drs. Todesco and Williams. We recognize that multiple strategies are possible in dealing with patients who have difficult airways. Our technique does require special equipment—a fiberoptic laryngoscope and a HFJV—both now commonly found in modern operating suites.

In our report, we clearly mention the possibility of anesthesia induction *via* mask followed by fiberoptic intubation; this technique is frequently inappropriate when ventilation *via* mask is anticipated to be difficult, as in case 2 of our report, or when intracranial pressure may be elevated, as in our case 3. Although there is