bronchoscopic suction channel into the pharynx and the larynx. The airway is visualized during the injection to assure adequate delivery of the lidocaine. If the patient gags when the bronchoscope is passed into the trachea, this is a sign of inadequate airway anaesthesia. Further injection of lidocaine through the bronchoscope, or inhaled aerosolized lidocaine are good options at this point. Superior laryngeal and glossopharyngeal nerve blocks and cricothyroid membrane puncture with lidocaine injection are accepted techniques to use in the older cooperative subject.

These techniques are more invasive and should be rarely be necessary. Drs. Oxorn and Whatley should be praised for their thoughtful approach to their patient. They avoided compounding the initial problems with intubation, and they stopped to analyze the case rather than proceeding aggressively.

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Does nalbuphine reverse opioid obtuned laryngeal reflexes?

To the Editor:

We read the paper by Ramsay *et al.*[1] with great interest since it contains information that may support a clinical impression that we have formed in our use of nalbuphine over the past two years. They describe a sympathetic response when nalbuphine was given to their intubated patients a few hours after fentanyl-supplemented general anaesthesia. We have also sometimes observed an increase in blood pressure and heart rate after 0.05 to 0.1 $mg \cdot kg^{-1}$ of nalbuphine administration at emergence from fentanyl-supplemented anaesthesia. Such a "sympathetic response" has also been reported by others[2–4]

However, contrary to the earlier authors who attribute that response to the reversal of analgesia by nalbuphine, it has been our impression that it has related to the presence of the endotracheal tube in the trachea of our patients. Ramsay *et al.* present data that may support that hypothesis rather than the reversal of analgesia by nalbuphine. All but three of their patients stabilised after extubation and "did not require more analgesia in the following 12 hours than those who did not receive the drug." Furthermore, it seems that the three patients who required morphine and sedation where those who remained intubated. We believe that these three patients might also have "settled down" and stabilised if they had been extubated since they were likely responding to the presence of the endotracheal tube rather than suffering from incisional pain.

Martin *et al.* showed that low dose fentanyl blunts circulatory responses to tracheal intubation. They suggested that fentanyl blunts the response to laryngeal stimulation by its agonistic activity at the opioid receptors, as found by Atweh and Kuhar[6] in the solitary nuclei and the nuclei of the ninth and tenth cranial nerves. They believed that these opioid receptors were associated with visceral afferent fibres of these nerves which originate in the pharynx and larynx.

We propose that nalbuphine may have a low degree of intrinsic activity, or an antagonistic activity, at these receptor sites. Furthermore, even though the subtype of these opioid receptors found by Atweh and Kuhar is still unknown, we propose that they are of the same subtype as those responsible for the respiratory depression and/or pruritus, since nalbuphine has been shown to have an antagonistic activity at these receptors subtypes.[7,8] These have yet to be identified as being mu_1, mu_2, sigma, delta, epsilon or another subtype.

Our hypothesis deserves further study since the possibility of nalbuphine reversing opioid-induced obtundation of laryngeal reflexes while preserving analgesia could have significant clinical advantages.

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REPLY

Drs. Samson, Baxter and Penning present an interesting hypothesis which we did not consider during our study. We did not elaborate in our "Methods" section on further questioning we did of our patients who responded negatively to the question "Are you comfortable?" If patients indicated that they were not comfortable then they were indeed asked if they were having pain. The scale in the "Methods" section and Table IV use the word "pain." Those patients experiencing severe discomfort pointed emphatically to their surgical wound. This does not rule out the possibility of laryngeal discomfort, but we were convinced that patients 13, 18 and 20 had incisional pain.

The authors speculate whether the three patients who required morphine would have "settled down" had they been extubated immediately after their small dose of nalbuphine. Clinical judgment suggested, and our experimental protocol dictated the more conservative approach, however. Seven of the ten patients who received nalbuphine tolerated their endotracheal tubes for some time after the nalbuphine was administered. The tubes were left in place so that a ventilatory response to CO_2 could be performed. The nature of this latter procedure (progressive hyperventilation) is such that if the endotracheal tubes were the major source of discomfort, our patients should have become acutely distressed. Such was not the case.

We agree that the hypothesis of these authors warrants further investigation. From the above it should be clear that we are not as enthusiastic about how our results support the hypothesis, and remain convinced that nalbuphine reversed analgesia in several of our patients.

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Anaphylactic reaction to fentanyl or preservative

To the Editor:

We read with interest the recent paper by Bennett *et al.*[1] which represents, for the first time, a case report of vascular collapse, generalized erythema and urticaria following the administration of fentanyl. In their patient fentanyl allergy was later confirmed by intradermal test. Recently we encountered a case of anaphylactoid reaction following the administration of fentanyl during anaesthesia.

Our patient was a 45-year-old female who underwent three operations for dissecting aneurysm and pituitary tumour during a nine month period. During each of the three anaesthetics she developed anaphylactoid reactions including an urticaria-like skin rash and arterial hypotension (<60 mmHg systolic) following the administration of different anaesthetic agents. Based upon the time course of these events, 0.5 per cent lidocaine used for skin analgesia was suspected as a causative factor in the first event; 4.0 per cent lidocaine spray, which was administrated into the larynx and trachea, was suspected as a causative factor in the second; and in the third event, fentanyl was primarily suspected to have caused the anaphylactoid reaction.

It was later discovered that each of the preparations of the three drugs contain methylparaben as a preservative. Although methylparaben allergy has not been previously proven by several tests, including the intradermal test and the Prausnitz-Küstner test, this preservative was strongly suspected as a causative agent in our patient.

Stoelting[2] and Moudgil[3] suspect that anaphylaxis to local anaesthetics and muscle relaxants is mainly due to reaction to methylparaben. Swanson[4] proposed using small test doses (0.1 ml) of pure