Correspondence

Further improvements in the technique of fiberoptic intubation

To the Editor:

Recent letters[1] have stressed fine points in the sophisticated art of awake fiberoptic endotracheal intubation. Our own experience leads us to report several modifications of established technique, which we believe offer useful alternatives in certain clinical settings.

A 30-year-old female with severe rheumatoid arthritis presented for total shoulder arthroplasty under general anaesthesia. A previous attempt under regional anaesthesia had been abandoned because of inadequate block. A difficult intubation was anticipated, because of temperomandibular joint involvement, and decreased neck mobility. Awake fiberoptic intubation was planned. Surgery was once again cancelled, however, because although the bronchoscope could be passed orally into the trachea, a 7.0 mm PVC endotracheal tube could not be advanced past the lower pharynx.

Two factors were deemed responsible for failure. First, the patient had a hyperactive gag reflex. Second, indirect laryngoscopy the next day suggested that the tip of the endotracheal tube may have been impinging upon a protruding cervical vertebral body displaced anteriorly.

A third procedure was planned. Premedication included intramuscular morphine and hyoscine, and intravenous diazepam. Topical anaesthesia was achieved with a four per cent lidocaine gargle, and intranasal five per cent cocaine. Bilateral superior laryngeal and glossopharyngeal nerve blocks were performed using standard techniques.[2,3] The cricothyroid membrane was punctured with a 20 g IV catheter, and 3 ml of four per cent lidocaine injected. Fiberoptic nasal intubation was successfully undertaken with a 7.5 mm armoured endotracheal tube. The connector had been removed to allow passage of the bronchoscope, and was replaced after the bronchoscope was withdrawn at the end of the procedure.

Three points are emphasized:

1 The glossopharyngeal nerve block diminished the gag reflex.
2 The flexibility and softness of the armoured tube allowed unimpeded passage through the pharynx into the trachea.
3 Use of a 20 g IV catheter to puncture the cricothyroid membrane allowed rapid injection of local anaesthetic without the fear of lacerating the larynx or trachea during vigorous coughing.

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REPLY

Oxorn and Whatley's comments about a 30-year-old patient with severe rheumatoid arthritis mentioned further options when fiberoptic intubation does not proceed smoothly because of difficulty advancing the endotracheal tube. In such cases, it may be helpful to withdraw the tube a few centimeters, and readvance while rotating the tube in order to orient the tip away from the obstruction. Airway anatomy should be carefully examined as the bronchoscope is passed initially. Abnormal obstructions, such as a protruding cervical vetebral body will be noted. This allows better planning of the actual endotracheal tube passage and a better immediate understanding of problems which may arise as the tube is passed.

Adequate airway anaesthesia is always a prerequisite for an adequate endoscopic examination or elective intubation. We have had excellent success using two per cent lidocaine. The lidocaine solution is placed into the naris and the patient is encouraged to swallow any excess. This provides partial pharyngeal anaesthesia. The bronchoscope is then advanced into the pharynx and additional two per cent lidocaine is injected through the

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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