in anaesthetising children and young adults with the Treacher Collins Syndrome who presented for evaluation of their external ears and hearing and for corrective surgery on their eyes and ears. Because awake intubation requires a balance between sedation and cooperation of the patient, it is extremely difficult to use this technique in children over the age of six months and under the age of eight. Even in the older age groups those who have had bad experiences refuse to cooperate in this technique.

I have found that these children may be safely anaesthetized with halothane, using a mask induction technique. Once the patients are in a deep plane of anaesthesia, I spray the posterior part of their tongue and their pharyngeal structures and, if visualized, the epiglottis, with four per cent lidocaine. Once these structures have been anaesthetized, the children will not have airway difficulties or laryngospasm in the lighter planes of anaesthesia. With the child breathing spontaneously a laryngoscope is placed, exposing the glottic structures. The endotracheal tube is then advanced by using the breath sounds through the tube as a guide. As long as the breath sounds are continuous and loud through the endotracheal tube, the tube is advanced until it is passed through the glottis. I have used this technique many times, without difficulty. I write this as an agreement with the suggestions brought forth in the article by the authors and add another word from experience with children who have the Treacher Collins Syndrome. The anaesthetic manoeuvre and procedure has proven to be satisfactory.

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REPLY

I would agree that inhalational induction may be an option for anaesthetizing these patients under certain conditions. Some criteria for use of this technique might be: (1) patients previously anaesthetized by yourself or an associate who were not difficult to ventilate by bag and mask, and in whom no operative procedure had been done in the interim that might change airway anatomy (e.g., cleft palate repair, pharyngeal flap, etc.), (2) patients without evidence of cor pulmonale or other obstructive symptoms in the unanaesthetized state, and (3) patients in whom an "awake look" has been done, revealing adequate exposure of the glottic structure.

In addition, our discussion was directed toward anaesthetists who have little or no experience in dealing with this group of patients, so our recommendations were somewhat conserva-

tive. Nonetheless, even healthy children with normal airway anatomy can suddenly become upset during the induction of anaesthesia and with crying, develop copious secretions and laryngospasm. If ventilation by bag and mask or intubation of the trachea are unsuccessful, the child may be committed to transtracheal puncture for jet ventilation, or tracheostomy.

In regard to topical anaesthesia of the airway, unless the lidocaine spray is placed on the cords or below (as in transtracheal instillation) motor innervation to the vocal cords are not blocked, and, therefore, one cannot guarantee that laryngospasm will not occur during airway manipulation or light anaesthesia.¹ Topical anaesthesia at the level of epiglottis and above will block sensory innervation only. Another option in the awake child is to nebulize $4 \text{ mg} \cdot \text{kg}^{-1}$ of lidocaine in saline. This will often effectively anaesthetize the entire airway down to the level of the vocal cords. With judicious sedation, which I admit is time-consuming and sometimes difficult to judge, one can often get valuable information about the airway with an awake look. There will always be the case, as implied by Dr. Mayhew, where visualization of the vocal cords and glottic structures will be impossible and breath sounds via the endotracheal tube will be necessary to guide proper placement of an artificial airway.

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Combined subarachnoid and epidural block for Caesarean Section

To the Editor:

With reference to the letter by Rawal describing single segment combined subarachnoid and epidural block (CSE) for Caesarean section¹ I would like to report my own experience using a similar technique. I use the CSE technique described by Nickalls and Dennison² where the epidural space is identified using a Tuohy needle through which the spinal is performed with a spinal needle (26 gauge, 150 mm long) held in place by an artery forceps. Puncture of the dura is very distinct, but flow of cerebrospinal fluid is not always seen. The dose requirement of 0.5 per cent bupivacaine is 1.5 to 1.6 ml, depending on the height of the patient.³ The extent of the block at the end of 15 minutes is usually T₄ and good surgical anaesthesia has been produced invariably. Rarely an additional injection of the epidural drug has been required to extend the level of block.

Once the baby is delivered an injection of 8 ml of 0.25

per cent bupivacaine plain with 0.3 mg buprenorphine is given epidurally to provide postoperative pain relief. The epidural injection is given early in the operating room while the subarachnoid block exists in order to minimize cardiovascular changes and to allow the patients to be monitored for additional time in the operating room where full facilities are available to deal with any problems. The epidural catheter is removed at the end of operation. The postoperative pain relief obtained is usually of 8 to 12 hours duration. Further postoperative analgesia is obtained by oral or intramuscular analgesics.

Two cases of post-spinal headache of a mild nature have been seen in 300 cases. Headache only occurred when repeated punctures of the dura were performed, due to technical difficulties in defining the distinct feel of dural punctures. The subsequent epidural injections were not given due to fear of extension of block.

My use of this technique has led to the following observations. First, the dose requirement of local anaesthetic to achieve a satisfactory level of anaesthesia for Caesarean section was low, compared to another study⁴ where a "single shot" spinal was administered. Secondly, the incidence of post-spinal headache was very low and the headaches were of a minor nature.

One possible explanation for the reduced requirement of local anaesthetic for the CSE technique is that the epidural pressure becomes atmospheric and this change in pressure somehow interferes with the circulation and volume of the cerebrospinal fluid and hence better spread. The injection of local anaesthetic through the narrow and long spinal needle is very slow and hence better "fixing" of the local anaesthetic likely occurs.

The incidence of post-spinal headache associated with use of a 25-gauge needle has been reported to be between 1 per cent⁵ and 20 per cent.⁶ Although the number of cases in my study is not sufficient to come to a scientific conclusion the low incidence (1:150 cases) must be recognized. This low incidence of spinal headache could be due to the technique of meticulous puncture of the dura with a fine needle, avoiding possible repeated dural puncture with the "single shot" technique in an attempt to elicit free flow of cerebrospinal fluid. The injection of fluid into the epidural space is a reported method for prophylaxis after dural puncture.⁷ Perhaps the injection of local anaesthetic into the epidural space, for postoperative pain relief, might exert a protective effect against further loss of cerebrospinal fluid.

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Efficacy of priming with atracurium

To the Editor:

In their paper on priming with atracurium, Naguib *et al.*¹ conclude that a priming dose of $0.05 \text{ mg} \cdot \text{kg}^{-1}$ is optimal. The authors used a three-minute interval between the priming dose and the intubating dose, on the basis of a previous study on priming intervals.² That study, however, evaluated a priming dose of $0.06 \text{ mg} \cdot \text{kg}^{-1}$. The optimal priming interval for the $0.05 \text{ mg} \cdot \text{kg}^{-1}$ dose may not be the same as that for the $0.06 \text{ mg} \cdot \text{kg}^{-1}$ dose.

Another discrepancy is that in their earlier study Naguib *et al.*² list the time to onset of maximum neuromuscular blockade using single twitch stimuli at 1 Hz as 90.9 seconds for atracurium $0.5 \text{ mg} \cdot \text{kg}^{-1}$, whereas in their more recent study,¹ employing train-of-four stimuli every ten seconds, it was 141.4 seconds for the same dose. Such a large difference may not be explainable on the basis of the type of stimulus employed.³

The authors' conclusion that a $0.05 \text{ mg} \cdot \text{kg}^{-1}$ priming dose of atracurium given three minutes prior to the main dose of $0.45 \text{ mg} \cdot \text{kg}^{-1}$ gives a reduction of 50 per cent in onset time compared to that of a $0.50 \text{ mg} \cdot \text{kg}^{-1}$ bolus is rather surprising in light of two other studies. Ramsey *et al.*⁴ evaluated a $0.05 \text{ mg} \cdot \text{kg}^{-1}$ priming dose of atracurium given 4.4 minutes before an intubating dose of $0.35 \text{ mg} \cdot \text{kg}^{-1}$. As compared to a group receiving a $0.40 \text{ mg} \cdot \text{kg}^{-1}$ bolus, onset time was not reduced. Weinberg *et al.*⁵ found that a $0.05 \text{ mg} \cdot \text{kg}^{-1}$ priming dose of atracurium given five minutes before a $0.10 \text{ mg} \cdot \text{kg}^{-1}$ dose again provided no reduction in the time to maximal