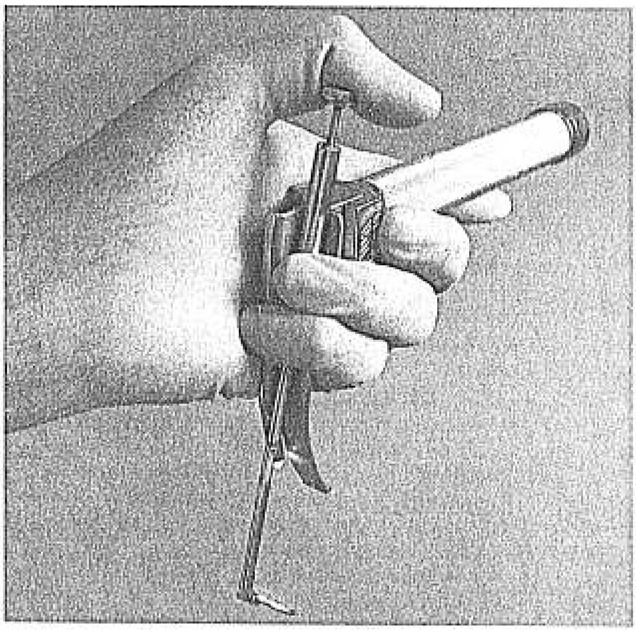

Fig. 2. The Stortz dental mirror is modified to change the angle of the mirror as required to expose the larynx. The mirror is held with the left hand along with the Macintosh laryngoscope.

The tip of the endotracheal tube was advanced into the larynx, looking into the mirror (fig. 1B). The trachea was intubated successfully without trauma.

Although various techniques have been advocated as an alternative to direct laryngoscopy to intubate the trachea in children, to our knowledge the use of a dental mirror as an aid to tracheal intubation in an infant has not been reported.

This technique is very simple and does not need expensive instruments such as a pediatric flexible fiberscope. Insufflation with oxygen and halothane can be instituted easily to prevent the patient from responding during the procedure. A similar technique can be adapted to tracheal intubation in adult patients with anterior larynx or in conditions where the patient's neck cannot be manipulated.

A Stortz #3 dental mirror was used in this case (fig. 2). The Stortz dental mirror can be custom-made to different angles to suit different airway anatomy. A Labordette laryngoscopy speculum,[1] Siker laryngoscope blade,[2] Robert Miller indirect laryngoscope,[3] Huffman prism,* and Bellhouse angulated laryngoscope[4] work on the same principle to visualize the larynx indirectly in adult patients.

In summary, we are reporting an inexpensive, readily available, easy-to-learn technique as an alternative to direct laryngoscopy in difficult airway situations in children to intubate the trachea.

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Increased Intracranial Pressure in Head Trauma Patients Given Fentanyl or Sufentanil

To the Editor:—We read with interest the recent article by Sperry *et al.*[1] describing a modest increase in intracranial pressure (ICP) in head trauma patients given fentanyl or sufentanil. They administered 0.6 μg/kg sufentanil or 3 μg/kg fentanyl in a randomized, double-blind fashion and observed increases in ICP of 7.7 and 6.1 mmHg after fentanyl and sufentanil, respectively. In their discussion, they appropriately point out the importance of the 9–10 mmHg decrement in mean arterial blood pressure (MAP) or neuroexcitation to their observations. We would like to elaborate on these speculations.

The decrease in MAP, besides contributing to ischemia, may in fact have been the primary *cause* of a reflex increase in ICP. Rosner *et al.*[2,3] have demonstrated how ICP can increase in the subset of patients with heterogenous injury in whom cerebral perfusion pressure (CPP) decreases into the 70–90-mmHg range. They make a very persuasive argument that increased ICP in this subset of patients is due to the exponential increase in cerebral blood volume that occurs when CPP decreases into the 70–90-mmHg range. When an exponential increase in cerebral blood volume is produced in a patient whose intracranial pressure-volume relationship is on the exponentially increasing portion of that curve, it is not surprising that a small decrease in MAP, through reflex vasodilatation of brain areas with intact autoregulation, might result in an increase in ICP.

The authors appropriately indicate the possibility that seizure activity could have contributed to their observation, but they report that electroencephalographic evidence of this in humans is lacking. In a recently published study,[4] however, Tempelhoff *et al.* gave fentanyl doses of 7.7–35.7 μg/kg to epileptic patients and observed hippocampal seizure activity with intracranial electrode recordings. This study circumvented the pitfall of missing deep seizure activity with the commonly used surface scalp electrodes or intranasal electrodes. Although the seizure activity was observed in patients with epilepsy, it occurred in the temporal lobe, which was not the epileptic focus. In addition, abstracts by Kearse *et al.*[5,6] have reported dose-related spike activity detected by 20-channel scalp-electrode recordings in humans undergoing induction of opioid anesthesia for cardiac or carotid surgery.

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In Reply:—Kofke and Tempelhoff refer to the possibility that a modestly decreased cerebral perfusion pressure may explain the observed increase in intracranial pressure (ICP) is an important observation. It is true that autoregulatory responses to decreased cerebral perfusion pressure may be associated with an increased ICP. While this may be a contributing factor, in this instance, it is unlikely to be the only causative factor. The standard deviation for the ICP response in our patients was 10 mmHg. This heterogeneity did not correlate with the decrease in cerebral perfusion pressure. Thus, I find it unlikely that decreased cerebral perfusion pressure was anything more than contributory.

Kofke and Templehoff add very recent information to our discussion of potential mechanisms for our observations. Their insights on subcortical seizure activity and neuroexcitation associated with opioid administration are a significant addition to our discussion.

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