Airway management in adult epiglottitis

To the Editor:

The management plan of acute epiglottitis in children is generally agreed upon.[1] It calls for airway control by tracheal intubation following mask induction of general anaesthesia using halothane and oxygen. Guidelines for the management of acute epiglottitis in adults, on the other hand, are less well defined. A protocol involving tracheal intubation following inhalational induction of general anaesthesia has been recommended.[2] I would like to describe a case of adult epiglottitis that was managed according to that protocol, but not without difficulty.

A 41 yr, well-developed man presented with acute respiratory distress. He was sitting upright, drooling, and had an audible inspiratory stridor. His upper airway seemed adequate otherwise. A lateral radiograph of the neck was diagnostic of acute epiglottitis.

The patient was taken to the OR to provide an artificial airway by oral tracheal intubation. Following *iv* glycopyrrolate, inhalational induction was started using a low concentration of halothane in 100% oxygen. The halothane concentration was gradually increased. When the patient started to lose consciousness, his respiratory efforts were gently assisted. Airway obstruction worsened as the depth of anaesthesia increased and finally became complete, despite chin lifting and forceful jaw thrusting. Direct laryngoscopy was attempted and tracheal intubation was achieved with difficulty.

When considering a management plan for this case, it was felt that direct laryngoscopy or fibreoptic intubation would have been poorly tolerated by the already distressed patient. Manipulation of the irritable upper airway also runs the risk of precipitating laryngeal spasm. Sedating patients with compromised airways could increase the obstruction or precipitate apnoea. In the meantime, anaesthetizing the larynx might increase the risk of pulmonary aspiration in the presence of severe distress, sympathetic stimulation and delayed gastric emptying. Intravenous induction of anaesthesia and the use of muscle relaxants were ruled out for fear of precipitating complete airway obstruction without the ability to intubate the trachea or ventilate the lungs.

Based on these considerations, inhalational induction was chosen. This case exemplifies its major risk under these circumstances – acute airway obstruction when consciousness is lost. Inhalational induction can also be more difficult in adults than in children due to the longer induction time and the stronger mandibular muscles in the adult. The favourable record of inhalational induction in children with epiglottitis may not, therefore, imply that it is as suitable for adults.

All the alternative approaches that could be employed for the airway management of adult epiglottitis might also be associated with difficulties. Thus, facilities to perform transtracheal jet ventilation and emergency tracheostomy should be available immediately. To a considerable extent, the choice of technique will depend on the clinician's skill and degree of comfort with the options available.

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Negative pressure pulmonary oedema

To the Editor:

We read with interest the report of negative pressure pulmonary oedema in two awake infants given muscle relaxants.[3] We agree with the authors that airway management during induction of general anaesthesia in the infant less than two months of age "requires a high level of experience and skill." We do not agree with their recommended practice of achieving airway control by giving non-depolarising muscle relaxants to awake infants.

It is a fundamental principle of any anaesthetic that before a muscle relaxant is given the anaesthetist should be able to maintain the patient's airway. In infants of this age, with an indwelling intravenous cannula, anaesthesia can be safely induced either by the intravenous or the inhalational route.

In either case, the airway should be clear and maintained by the anaesthetist before muscle relaxants are given.

In neither case was the infant anaesthetized before the administration of the muscle relaxant. In the first case, the infant received 0.25% halothane in 50% N_2O/O_2. The second case received 100% O_2. To say that the slower onset of non-depolarising drugs permits their earlier administration "so that maximal paralysis is present when