because the ventilation mechanism is supraglottic, it suffers from the same limitations I outlined in my article for the supraglottic ventilatory mechanisms of the LMA and Combitube (i.e., the ventilatory mechanism can be proximal to a perioidic pathologic obstruction).

Second, use of the nasopharyngeal route may cause nasopharyngeal bleeding, a most unwelcome happenstance in a “cannot ventilate, cannot intubate” situation. Although Burkh may be dismayed that the ASA Difficult Airway Algorithm does not specifically mention his first choice emergency ventilation method, and many other practitioners have reminded me of not specifically-mentioned tricks they have, the ASA Difficult Airway Algorithm does not exclude these methods. It is appropriate to conclude by reminding the anesthesia community that the ASA Difficult Airway Algorithm states that the recommendations therein “may be adopted, modified, or rejected according to clinical needs and constraints” and that “the techniques chosen by the practitioner in a particular case will depend upon specific needs, preferences, skills and clinical constraints.”

References


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Effects of 0.86 mg/kg Cisatracurium in an Infant

To the Editor—Lack of familiarity with a new drug may increase the likelihood of errors in dosing. An instance of this occurred in our operating theater.

A 7-month-old, 7-kg infant received 6 mg cisatracurium intravenously after induction of anesthesia with nitrous oxide and halothane. There was no change in heart rate, noninvasive blood pressure obtained with the cuff on the upper arm, or skin color, but when the surgery was concluded, neuromuscular block persisted to the extent that no posttetanic count could be elicited. The infant received midazolam and mechanical ventilation in the recovery room. One hundred minutes after the dose of 0.86 mg/kg cisatracurium, a very slight response of the adductor pollicis to stimulation of the ulnar nerve with 2 Hz, a train-of-four (TOF) every 60 s was documented by the Datex neuromuscular transmission monitor electromyogram in the uncalibrated mode. One hundred and ten minutes after the bolus of cisatracurium, four responses to the TOF were recorded. At this time, the patient’s diaphragm, fingers, and toes were moving spontaneously. Propofol was administered by infusion to produce immobility. During the following 15 min, the TOF ratio increased to >0.9. Propofol was discontinued, and 0.1 mg atropine followed by 5.0 mg edrophonium was administered to reverse any residual blockade. The trachea of the infant was extubated without complication several minutes later. This conservative approach was taken to be certain that the infant would not be clinically weak after extubation. Because cisatracurium is one of the ten isoesters that compose atracurium, it may be that the relatively minor age-related differences in potency that have been documented for atracurium would also be documented for cisatracurium. The potency of atracurium in infants during halothane anesthesia is greater by 50% than in children. The ED95 of cisatracurium in children is 0.911 mg/kg during halothane anesthesia. Assuming that age-related differences in the potency of atracurium and cisatracurium are similar, the ED95 of cisatracurium in the infant population could be close to 0.027 mg/kg. Given these assumptions, this patient received more than 30 times an ED95 dose of cisatracurium. With regard to neuromuscular blocking potency, this dose of cisatracurium is equivalent to more than 4.5 mg/kg atracurium, 0.75 mg/kg vecuronium, 2.5 mg/kg mivacurium, or 15 mg/kg succinylcholine in an infant during halothane anesthesia. The effects of such a relatively large dose of neuromuscular blocker have not been documented previously.

No data exist regarding the expected duration or the pharmacokinetics of cisatracurium in infants. The elimination half-life of cisatracurium in adults is similar after doses of two and four times the ED95. If the kinetics of cisatracurium are not dependent on dose, and the infant received approximately 32 or 64 times an ED95 dose, then five elimination half-lives of 20 min should pass from administration of the overdose to the beginning of spontaneous recovery. This exemplification is an extrapolation beyond the region of published data regarding cisatracurium, but it is consistent with the events in this patient. Observation of more patients is necessary to appropriately compare variability in recovery from cisatracurium with variability in recovery from other drugs. However, we find it noteworthy that spontaneous recovery from one response to a train-of-four stimulus at the ulnar nerve to a train-of-four ratio of 1.0 occurred in this infant within an interval of 45 min. Similar recovery rates were observed after doses of one and two times the ED95 in children. The recovery index, the time from 25% to 75% recovery of the amplitude of the response of the adductor pollicis to stimulation of ulnar nerve, was an average of 11 min (SEM = 0.6 min) in these children. This suggests that the recovery index was between 15 and 20 min in our infant anesthetic.

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To the Editor — Cardiopulmonary bypass associated with the use of inotropic agents and mechanical ventilation for the treatment of patients with heart failure or cardiogenic shock is a very common clinical situation. The pharmacological properties of dobutamine in this setting have been described by Popp et al., but the use of inotropic agents for the treatment of patients with heart failure or cardiogenic shock is a very common clinical situation. I studied the pharmacological properties of dobutamine in this setting, and I found that the use of inotropic agents for the treatment of patients with heart failure or cardiogenic shock is a very common clinical situation.

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Method of Humidifying Inspired Gas Influences the Type of Ventilator-Associated Pneumonia

To the Editor.—The recent article on the histopathologic and microbiologic aspects of ventilator-acquired pneumonia by Fabregas and colleagues was very interesting and provoked much discussion at our recent journal club.1 We noted that these workers reported a very high incidence of *Pseudomonas aeruginosa* and *P. putida* infections in patients whose lungs were mecanically ventilated. Humidification of the inspired gas is necessary because the patients’ own mechanisms have been bypassed. There are a variety of devices for this, of which the water bath and heat and moisture exchangers are most commonly used. Although the authors do not comment on the method of humidification they used for the ventilator circuits, we assume they are using a water bath device because of the high rates of infection with the above-mentioned organisms. Gallagher and coworkers reported that the use of heat and moisture exchangers in the ventilator circuit reduced the incidence of these infections from 5% to 21%, and, as a consequence, we have abandoned the routine use of water bath humidifiers.2 In addition, Ahlgren and Redding showed that the use of water bath-type humidifiers creates a reservoir for infection, necessitating filters between inspiratory and expiratory ports and between the patient and humidifier.3,4 The heat and moisture exchanger incorporates a filter that prevents bacterial contamination of the circuit and patient, and is an efficient method of humidification itself. However, if we have supposed wrongly, then the levels of *Pseudomonas* and similar infections are high, in comparison with Gallagher’s article.

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References


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