outcome. It simply means that a competent anaesthetist should be present.

Unfortunately, this study does not answer the basic question of whether the LMA is a useful or good alternative to endotracheal intubation in children who undergo adenotonsillectomy.

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REPLY

We appreciate Dr. Hackmann's analysis of our paper, and the opportunity to reply to his observations. Dr. Hackmann is quite correct in pointing out that the greater incidence of difficulty in inserting the LMA was due to our initial unfamiliarity with the technique. The dose of propofol recommended for induction of paediatric anaesthesia ($2.5 \text{ mg} \cdot \text{kg}^{-1}$) did not consistently provide adequate conditions for insertion of the LMA. Once we increased our induction dose, we experienced no further difficulty in positioning the airway. Similarly, the cause of airway obstruction seen after opening the B-D gag and surgical stimulation was not immediately apparent. When we realized that the probable cause was reflex laryngeal closure with inadequate depth of anaesthesia, the incidence of problems diminished. These difficulties did lead early to bias against the LMA but, rather than redesign the study, we reported our problems in order that others may benefit from our experience. No child needed hospitalization or suffered any damage from these or other adverse events.

It has been shown that insertion of the LMA in the adult produces less cardiovascular response than laryngoscopy and intubation.¹ It is reasonable to postulate that similar effects are seen in children. We acknowledge the possible contribution of atropine and succinylcholine to haemodynamic differences in our two study groups. One of the advantages of the LMA is that it is not necessary to give either drug.

The difference in blood loss in the two groups may be attributable not only to differences in systemic arterial pressure, but also in venous pressure. Positive pressure ventilation, during which central venous pressure is increased, was required more often in the tracheal intubation group.

The shorter interval from iv induction to establishment of an airway with the LMA is primarily due to the extra time required to wait for the action of succinylcholine. In the ET

group there was wide variance in the interval before onset of spontaneous breathing, reflected in large SD's. Some patients required assisted ventilation throughout surgery. The Kruskal-Wallis test used in the analysis is a non-parametric test, and does not assume that the data is normally distributed.

An $\text{SpO}_2 \leq 94\%$ defines a desaturation threshold, intended to provide early warning of hypoxic events. Transitory desaturation below this point is unlikely to have clinical significance. We agree with Dr. Hackmann that the difference in oxyhaemoglobin saturation between groups on arrival in the PACU, reported as mean values and SD, is probably not clinically relevant.

The significance of stridor is that it may herald the more serious complication of laryngospasm. Although it can readily be handled by the alert anaesthetist, most of us would prefer to avoid it. Controversy continues concerning the optimum condition for tracheal extubation in children. We extubated the trachea and the LMA with the child sufficiently deeply anaesthetized to suppress airway reflexes. Incidents of upper airway obstruction before return of consciousness led to insertion of oropharyngeal airways, and further potential upper airway irritation.

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Postoperative recurrent paralysis in an infant after mivacurium infusion

To the Editor:

We wish to report a healthy two-month-old, 6-kg girl who underwent repair of a congenital cleft lip. During general anaesthesia, a mivacurium continuous infusion was used for neuromuscular blockade and was monitored with a nerve stimulator. Fifty minutes after starting the infusion, 60 mg mivacurium had been administered ($0.2 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$). Because of concerns for volume overload, the infusion was discontinued and neuromuscular blockade was maintained with bolus dosing. Additional doses of 0.08, 0.04, and 0.04 $\text{mg} \cdot \text{kg}^{-1}$ were given at approximately 30 min intervals with two to three twitches remaining after each dose. Approximately five minutes after the last dose was given, the surgical procedure ended and neostigmine, 0.07 $\text{mg} \cdot \text{kg}^{-1}$, and atropine, 0.025 $\text{mg} \cdot \text{kg}^{-1}$ were administered. Reappearance of four twitches was confirmed and the trachea was extubated

when the infant demonstrated adequate tone and wakefulness. Upon arrival in the PACU, the infant's temperature was 36.4°C, respiratory rate 30–35 per minute, heart rate 180–190 bpm, and the SpO₂ was 100% breathing 0.4 FiO₂. The infant's nurse, noting that the *iv* solution was not infusing, injected saline through an injection port. Approximately ten minutes later, the patient's cry began to weaken and take on a whispering quality, at which time a house officer was summoned. Within one minute the infant became apnoeic and cyanosed, with a heart rate of 70 bpm. Resuscitation commenced immediately with 100% oxygen administered by bag and mask, followed by tracheal intubation. The infant made no effort to breath spontaneously and a nerve stimulator revealed no response to supramaximal tetanic stimulation. Twelve minutes later one twitch on TOF testing returned and *iv* neostigmine 0.4 mg was given. Within five minutes the patient regained spontaneous ventilation, and good physical strength. Nerve stimulation revealed four strong twitches and sustained tetanus at 50 Hz. The trachea was extubated and the remainder of her postoperative course was uneventful.

Residual neuromuscular blockade is a concern in the PACU,¹ but recurarization after seemingly adequate reversal is rare. A retrospective review of 16,700 children reported no PACU complications related to residual paralysis,² nor did a prospective study involving 10,000 children.³ The weak cry which progressed to apnoea and cyanosis, and absence of twitches on train-of-four monitoring, both of which responded to an additional dose of reversal agent, were clear manifestations of recurrent neuromuscular blockade. Upon careful review of the foregoing events we were unable to find evidence of insufficient reversal dose, undetected subcutaneous infiltration, or administration of other drugs with potential neuromuscular blocking properties (e.g., antibiotics). However, we were unable to exclude unintentional administration of residual mivacurium in the *iv* tubing when saline was flushed through the injection port. To our knowledge, this is the first published report of this postoperative complication in a paediatric patient. Although a technical source of this complication is most likely, an unknown cause related to the use of mivacurium in infants cannot be excluded and thus it is prudent that anaesthetists carefully monitor infants when using and reversing mivacurium, as sufficient data has not yet accumulated in this population.

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Vecuronium neuromuscular block in patients with malignant obstructive jaundice

To the Editor:

Vecuronium has a shorter duration of action than equipotent doses of pancuronium.¹ The plasma clearance of vecuronium is decreased markedly in patients with hepatic cirrhosis,² and in patients with cholestasis.³ The present report demonstrates a prolonged neuromuscular block when vecuronium was used in two patients having malignant obstructive jaundice.

The first patient was a 53-yr-old man, with an adenocarcinoma of the hepatic duct. The second was a 66-yr-old man with cancer of the head of the pancreas. Both were scheduled for cholecystectomy and hepaticojunctionostomy. Anaesthesia was induced with thiopentone 5 mg · kg⁻¹, followed by vecuronium 0.1 mg · kg⁻¹. When complete neuromuscular block was achieved, the trachea was intubated and anaesthesia was maintained with nitrous oxide:oxygen (3:1), supplemented with fentanyl 2 µg · kg⁻¹ and intermittent doses of vecuronium 0.015 mg · kg⁻¹.

Neuromuscular transmission was monitored by train-of-four electromyography (Datex) of the adductor pollicis. In the two patients, vecuronium 0.1 mg · kg⁻¹ resulted in complete neuromuscular block followed by slow recovery. A T₄/T₁ twitch ratio of 0.1 was observed only after 45 min. Subsequent doses of vecuronium 0.015 mg · kg⁻¹ were required every 25–30 min to maintain T₄/T₁ ratio of 0.1.

Vecuronium, being monoquaternary, may be relatively lipophilic and this may favour its rapid entry into hepatocytes.⁴ More than 40% of vecuronium injected *iv* is eliminated, mostly unchanged, in the bile.^{5,6} Hepatic uptake could be the limiting factor for elimination of ve-