Sir.—We compared two groups [1]; the only factor we varied was whether alfentanil or suxamethonium was given. The quoted abstract [2] used thiopentone or propofol for induction, both of which have different properties.

An inadequate dose of suxamethonium was not responsible for the observed effects. We used the currently recommended dose of suxamethonium to produce profound neuromuscular block. The study by Meakin and colleagues [3] stated that ‘‘calculated equipotent dose for children is 1.2–1.8 mg kg−1’’ (table I), albeit then suggesting using the upper end of the range. The other study [4] quoted ‘‘optimum conditions for tracheal intubation exist in children given suxamethonium 1 mg kg−1 during a period averaged less than 3 min, but with a minimum of 1.7 min’’. Thus they suggested that many undergo tracheal intubation at a time when effective neuromuscular block has terminated and increasing the dose would increase the duration of action. However, we intubated the trachea at 45 s.

The dose for infants is not relevant, as the youngest child we studied was 2.2 yr of age. It is important that new methods to facilitate tracheal intubation in children are validated correctly by less experienced anaesthetists.

Measurement of the cardiovascular effects are important to examine the potential of a technique to cause bradycardia and hypotension or tachycardia and hypertension. Finally, in our study we used strict criteria for scoring the conditions for tracheal intubation and although the trachea of all children was intubated at the first attempt, any disparity from perfect conditions was noted.

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Ketamine and muscle spasm

Sir.—A young boy was anaesthetized before surgical exploration of his right radial nerve for wrist drop, when during induction of anaesthesia he developed full extension of the right wrist for 10–20 s. This is described below and we would be interested in any comments on the mechanism involved.

An 11-yr-old boy sustained a simple supracondylar fracture of his right humerus. After failed closed reduction he had an open reduction and internal fixation (ORIF) using a lateral incision, and internal fixation by a trans-articular Steinmann pin. After operation he was noted to have complete radial nerve palsy and when this failed to recover spontaneously, surgical exploration was planned. Two months from the time of the ORIF he was again anaesthetized. Anaesthesia was induced with ketamine 50 mg, atracurium 0.4 mg i.v. and halothane by mask. He then received pancuronium 2 mg before tracheal intubation and anaesthesia was maintained with halothane in air using manual ventilation of the lungs. During the induction period the patient was noted to have full active right wrist extension for 10–20 s in the absence of a more generalized "tonic" reaction. The nerve was explored through the previous lateral incision. There was a 3-cm segment of the radial nerve entrapped in scar tissue and fixed to the peristeum at the site of the previous supracondylar fracture. External neurolysis was performed and the nerve was found to be in continuity. The boy subsequently made a full recovery from his wrist drop and all signs of compressive neuropathy resolved completely.

How does a patient suffering from radial nerve palsy suddenly develop full sustained wrist extension? Ketamine certainly increases general muscle tone and has been reported to cause myoclonic movements (muscle spasms) [1]. Could this have been a myoclonic movement? It is not known how ketamine mediates muscle spasm. Does this originate centrally and ketamine has its effect via the normal physiological pathways of CNS to muscle? Ketamine has been shown to increase indirectly evoked twitch tension [2, 3] but decreases the twitch response on directly stimulated skeletal muscle [4]. In children, ketamine in large doses caused generalized extensor spasms in infants, 30 min after the last dose of ketamine [5]. Or does ketamine have a direct peripheral effect causing muscle spasm or increased tone? Recent studies suggest that ketamine acts on neuromuscular transmission, mainly by interfering with acetylcholine-activated ionic channels on the postsynaptic membrane [6, 7]. Low concentrations it facilitates and in higher concentrations it blocks neuromuscular transmission [3, 6]. So what happened in this patient? Why did he have a myoclonic spasm only in the right radial nerve innervated muscles? For any patient with a nerve palsy after closed trauma or an anasthesiologist, the question remains is whether or not the nerve is crushed, crushed or divided? Usually after 4–6 weeks if there is no evidence of recovery, surgical exploration is indicated in order to differentiate the nerve that needs neurolysis or repair from one that will recover spontaneously. Because the patient had complete recovery after neurolysis, this implies that the lesion was a conduction block caused by compressive neuropathy. Ketamine could have overcome this block pharmacologically, and the compressed radial nerve transmitted a centrally initiated myoclonic spasm? Or did ketamine have a direct stimulatory effect on the right radically innervated muscles because of denervation hypersensitivity?

In this observation could there be the basis for a diagnostic test of an intact peripheral nerve?  

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