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Editor—We thank Dr Cattano for his interest in our study.¹ We agree that the Cormack–Lehane classification is one of the longstanding laryngoscopy view classifications. However, this classification has never been thoroughly evaluated and the fact that it has been revised quite often shows that users were not really happy with this classification. Interestingly enough, the authors of the original publication in 1984 did not intend to propose a general classification for anaesthetists to document intubation conditions.

The primary aim of our study was to evaluate a classification that is widely used as a gold standard to describe laryngeal view during direct laryngoscopy. You suggest that one should never stop learning to further improve theoretical knowledge about the Cormack–Lehane classification, and in that context that ‘not knowing’ should not automatically lead towards ‘not using’ it anymore. But even if anaesthetists enhance their knowledge about Cormack–Lehane classification, they would still have to deal with the fact that the inter- and intra-observer reliability is fairly poor with this classification. All of our participants in the simulator study were briefed about the Cormack–Lehane classification (definitions and figures of all four grades). However, a major portion could not translate this theoretical knowledge into practice. That limits reliability. In particular, the poor inter-observer reliability under our standardized conditions resembling the clinical situation restricts its clinical usefulness.

Conflict of interest

None declared.

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Obstruction of a reinforced oral tracheal tube

Editor—A 19-yr-old man was admitted to our intensive care unit after surgical fixation of his multilevel cervical spine injury. He had sustained his injury with associated high

spinal cord lesion in a road traffic accident 3 days previously. Intubation for surgery was performed with a halo frame *in situ* after adequate fasting, using an asleep fiberoptic technique. After operation, he was transferred to the intensive care unit intubated and ventilated through a size 8.0 Mallinckrodt® reinforced oral tracheal tube. Shortly after admission, while lightly sedated, planned removal of the halo frame was undertaken by the neurosurgical team. The intensive care team was asked to attend immediately after this as ventilation had become problematic with low tidal volumes and minute ventilation. Initial assessment revealed no evidence of tracheal tube displacement, with reduced but bilateral air entry and adequate sedation. Ventilation using a Mapleson C circuit remained difficult, excluding the ventilator and its circuit as the causative factor. Despite delivery of $F_{I_{O_2}}$ 1.0, the patient began to desaturate and the tracheal tube was therefore removed. Mask ventilation was easy and the airway was subsequently resecured with a standard tracheal tube without complication.

Inspection of the removed reinforced tracheal tube clearly demonstrated the cause of the airway obstruction (Fig. 1) Compression of the reinforcing wire coil had irreversibly narrowed the lumen of the tube, leading to near-occlusion. We hypothesize that this occurred as a result of the patient biting on the tube which went unrecognized during removal of the halo frame.

The indications for use of reinforced Mallinckrodt® tracheal tube include neurosurgical procedures and long-term cases where maintenance of an airway is critical (<http://www.nellcor.com/prod/Product.aspx?S1=AIR&S2=&id=124>). The product features describe the reduced risk of kinking of the tube due to the spiral-wound reinforcing wire. Although the company makes no claim that the wire reduces the risk of compression, reference to the reinforced or armoured nature of these types of tracheal tubes may promote this inaccurate belief. We wish to draw colleagues' attention both to the possibility of compression of these tracheal tubes and importantly, due to its irreversible nature, to the consequent increased potential for airway obstruction.



Fig 1 Compressed Mallinckrodt® reinforced oral tracheal tube.

Conflict of interest

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Rapid awaking after administration of neostigmine in an elderly neurosurgical patient with prolonged recovery from general anaesthesia

Editor—It is postulated that alteration of central cholinergic transmission may play an important role in the mechanism by which general anaesthetic drugs produce unconsciousness.¹

² It has been shown that increasing central cholinergic tone with the anticholinesterase physostigmine antagonizes the hypnotic effect of propofol or sevoflurane shown by the return of consciousness.^{3–4} In contrast, passage of neostigmine across the blood–brain barrier (BBB) is limited.⁵ Therefore, it is reasonable to suppose that neostigmine does not possess arousal effects like physostigmine. However, we experienced an elderly neurosurgical case in which neostigmine probably reversed prolonged recovery from general anaesthesia.

A 70-yr-old woman was undergoing an elective removal of a left temporal lobe tumour. Before operation, she had no neurological deficit, her medical history included hypertension, and her physical examination and laboratory analyses were essentially normal.

No premedication was given. Bispectral index (BIS) was recorded using the Aspect A-2000 EEG monitor (BIS version 3.4; Aspect Medical Systems, Newton, MA, USA) with electrodes (Zipprep; Aspect Medical System) positioned around the lateral corner of the right eye.⁶ Anaesthesia was induced with propofol 100 mg and fentanyl 100 µg, and the trachea was intubated after rocuronium 30 mg. The ventilatory frequency was adjusted to maintain normocapnia. The rectal temperature was monitored and maintained at normothermia. Anaesthesia was maintained with 1–2% sevoflurane in oxygen/air to keep BIS at 50–60. Remifentanyl was given by continuous infusion to achieve adequate analgesia (0.1–0.2 µg kg⁻¹ min⁻¹). No further rocuronium was given during the operation. During craniotomy, 300 ml of 20% mannitol was infused to prevent cerebral oedema. After uneventful surgery (4 h), the patient was asleep, but spontaneous ventilation was sufficient. Repeated measurements of train-of-four ratio with acceleromyography (TOF-WATCHTM, Schering-Plough, Kenilworth, NJ, USA) were 1.0 or more, indicating no residual neuromuscular block.⁷ She remained deeply sedated without response to verbal or tactile stimulation. BIS score was still around 60, although the expiratory sevoflurane concentration was almost zero. The BIS sensor was

relocated in the commercially recommended position; however, BIS score was unchanged. As consciousness was still not present after 65 min, a presumptive diagnosis of alteration of central cholinergic transmission by general anaesthesia was proposed. We had to use neostigmine as physostigmine is not available in Japan. Immediately after the administration of neostigmine (2 mg), BIS score increased 60–95, spontaneous eye opening occurred, and she became responsive to verbal commands. A transient decrease in heart rate from 80 to 60 beats min⁻¹ was observed, but no treatment was necessary. The patient was transferred to the intensive care unit for further postoperative treatment with no evidence of persisting neurological deficit. Additional neostigmine was not required. A single administration of physostigmine is usually efficient for treatment of central anticholinergic syndrome after general anaesthesia.⁸

Central cholinergic transmission can be inhibited to some degree after general anaesthesia.^{9–11} Therefore, it is recognized that postoperative sustained deep sedation is occasionally caused by reduced central anticholinergic transmission.⁸ Postoperative respiratory depression due to opioids and residual neuromuscular block were not presented in our case, and the expired sevoflurane concentration was almost zero. As the recovery from deep sedation relates to the time of neostigmine administration, it is reasonable to consider that central cholinergic transmission played an important role in developing postoperative sustained deep sedation in this case.

Neostigmine given peripherally is thought to be ineffective at reversing central cholinergic inhibition. However, mannitol was given in this case, and this has been used to deliver drugs into the brain parenchyma through its osmotic effect on the BBB.¹² In addition, BBB may have been damaged during the neurosurgical procedures. Therefore, the neostigmine may have entered the brain through a disrupted BBB and restored cholinergic transmission.

The elapsed time (65 min) could itself be an important factor in the recovery from anaesthesia in this case. However, it is not unreasonable to suggest that neostigmine was the pivotal factor in recovery, taking the recovery profile and timing of neostigmine administration into consideration.

In conclusion, we describe a case showing rapid awaking in a patient with prolonged recovery from general anaesthesia after administration of neostigmine. It is proposed that restoration of central cholinergic transmission by neostigmine was responsible for this.

Conflict of interest

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