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REPLY

Thank you for the comments from Backman et al. on our article Anaesthesia for non-cardiac surgery in heart-transplanted patients[1] concerning the reported lack of effect of anticholinesterase on heart rate. We reported in our series that 11 of the 12 patients who underwent general anaesthesia received intraoperative neuromuscular blocking agents (vecuronium n = 9, pancuronium n = 2). The fact is that no significant haemodynamic effect on heart rate was observed when the block in these patients was reversed with neostigmine with (n = 8) or without (n = 3) atropine. We later stated in our discussion that our result is consistent with the literature: it is generally accepted that heart rate shows no response to drugs like muscle relaxants, anticholinergics, anticholinesterases, etc. However, in the same paragraph, we did mention that slow development of cardiac reinnervation may be possible.[2] I agree with the case report by Backman et al.[3] that one of their heart transplanted patients had a decrease (21%) in heart rate from 95 to 75 bpm after neostigmine administration. In the same report, they stated that two other previously heart-transplanted patients had a reduction of 7% and 14% in heart rate after neostigmine administration. However, we do not know if the decrease in heart rate is a consequence of cardiac reinnervation, prolonged denervation, or direct activation on cardiac ganglionic cells by anticholinesterases.[4] I don't know if this can be justified as a clinically significant bradycardia as no decrease in blood pressure was reported simultaneously. As well, I consider a clinically significant bradycardia as a heart rate <50 bpm. I do not object that muscarinic antagonists be administered with anticholinesterases to block possible muscarinic side-effects of anticholinesterases in heart-transplanted patients. However, I will continue to utilize the heart transplant models for teaching residents regarding denervated heart physiology and pharmacology of anticholinesterase with or with anticholinergic agents.

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The oesophageal tracheal combitube for difficult intubation

To the Editor:

We read with interest the report by Baraka and Salem describing the successful use of an oesophageal tracheal combitube (OTC) following a failed intubation in a patient with a potentially full stomach.[1] As direct laryngoscopy revealed only a Cormack and Lehane grade 4 view, an OTC was inserted and the operation completed using controlled ventilation and a succinylcholine infusion. There are a number of points we would like to make about the use of the combitube in this situation.

Firstly, the authors did not state if facemask (FM) ventilation was attempted. In the "cannot intubate, cannot ventilate" situation, use of the OTC may be appropriate.[2] If, however, adequate ventilation can be achieved with an FM and maintained cricoid pressure, use of the OTC could not be recommended since it is a blind technique and ideal placement is not guaranteed. In this circumstance, the most appropriate course of action is probably to wake up the patient and secure the airway using an awake technique before proceeding with surgery. If difficult tracheal intubation is anticipated, as in this case, we consider than an elective awake intubation technique would be wiser than a rapid sequence induction of anaesthesia where applied cricoid pressure may worsen the view of the larynx.[3]

Secondly, the authors comment that the OTC may be preferred to the laryngeal mask airway (LMA) in the difficult intubation situation whenever the patient is con-