6 *Devos P, Preiser J, Melot C.* Impact of tight glucose control by intensive insulin therapy on ICU mortality and the rate of hypoglycaemia: final results of the GLUCONTROL study. Intensive Care Med 2007; 33(Suppl 2): S189.
7 *Brunkhorst FM, Engel C, Bloos F, et al.* Intensive insulin therapy and pentastarch resuscitation in severe sepsis. N Engl J Med 2008; 358: 125–39.
8 *Arabi Y, Tamim H, Alshimemeri A et al.* Intensive versus standard insulin therapy: a randomized controlled trial in medical surgical critically ill patients. Crit Care Med 2006; 34: A65.
9 *Merz TM, Finfer S.* Pro/con debate: Is intensive insulin therapy targeting tight blood glucose control of benefit in critically ill patients? Crit Care 2008; 12: 212.

Reply:

I appreciate the interest in my editorial regarding a potential link between perioperative hyperglycemia and postoperative cognitive dysfunction (POCD).[1] As previously alluded to, despite the associative link that was demonstrated in the study by Puskas et al.,[2] we are far from understanding whether there is any causative link between hyperglycemia and POCD. Although it is convenient to think of POCD and stroke on different ends of the same cerebral ischemic continuum, this may be an erroneous assumption, particularly as the pathophysiology of POCD is not well understood. As a result, putting the Puskas et al. POCD findings in the same context with the Gandhi et al.,[3] where patients receiving intensive insulin therapy had a higher stroke rate, may be misleading.

I wholeheartedly agree with you that anesthesiologists are at risk of "too quickly adopting a treatment that we do not yet fully understand". Although the adoption of this therapy is clearly well-intentioned, changing practice patterns based on a single positive trial (van den Berghe et al.)[4] or a negative trial (Gandhi et al.)[3] should be cautioned against. Rather, it should be the overall weight of the evidence that determines what therapy is best adopted. To that end, we should await future trials of glycemic control in cardiac surgery[B] to determine how best to deal with these patients; until then, it is prudent to be cautious in how aggressively we manage perioperative hyperglycemia.

B *ClinicalTrial.gov*. A service of the US National Institutes of Health. Available from URL; www.clinicaltrials.gov/ct2/show/NCT00524472?term=hyperinsulinemic&rank=1 (accessed May 2008).

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References

1 *Grocott HP.* Hyperglycemia and postoperative cognitive dysfunction: another call for better glycemic control? Can J Anesth 2008; 55: 140–5.
2 *Puskas F, Grocott HP, White WD, Mathew JP, Newman MF, Bar-Yosef S.* Intraoperative hyperglycemia and cognitive decline after CABG. Ann Thorac Surg 2007; 84: 1467–73.
3 *Gandhi GY, Nuttall GA, Abel MD, et al.* Intensive intraoperative insulin therapy versus conventional glucose management during cardiac surgery: a randomized trial. Ann Intern Med 2007; 146: 233–43.
4 *Van den Berghe G, Wouters P, Weekers F, et al.* Intensive insulin therapy in the critically ill patients. N Engl J Med 2001; 345: 1359–67.

Safeguarding against errors with free flow protection devices of patient controlled analgesia systems

To the Editor:

Patient controlled analgesia (PCA) devices can provide improved treatment of postoperative pain. Despite their apparent safety, adverse drug events (ADE) continue to be associated with their use. Etiologies of ADE include errors in programming, errors in preparation of the narcotic solution, and unintentional free flow of narcotics into the patient's intravenous tubing. Free flow protection devices (FFPD) have been incorporated in these pumps in order to prevent the latter problem, in an effort to improve safety.

I read with interest the recently published letter by Dr. Doyle *et al.*,[1] entitled: "Another failure mechanism leading to patient-controlled analgesia overdoses". In this case, failure of a Baxter PCA pump was attributed to failure of the FFPD, as a result of unrecognized damage to the device. There was a similar case in our hospital, which involved a different manufacturer of PCA. The patient was initially stable in the postanesthesia care unit, following laparoscopic abdominal surgery. Immediately after starting postoperative analgesia with a Curlin Medical PCA (Huntington Beach, CA, USA), the patient became obtunded, and was nearly apneic. The entire content of the PCA medication bag, containing 100 mg morphine, had been administered to the patient. As in the case reported by Dr. Doyle *et al.*, the patient responded to treat-

ment with a bolus, followed by continuous infusion of naloxone, and tracheal intubation was not needed. Subsequent testing of the PCA determined that, when the door of the PCA was closed, due to a defect in the pump, there was unintended pressure on the free flow protection device which allowed for free flow. This defect was not visible to the naked eye. Following this event, all the PCA machines in the hospital were evaluated by the manufacturer, and none was found to have this defect. The same pumps are still used in our hospital, and there have been no reported recurrences. Following this case, our protocol for attaching the PCA was changed, such that, after PCA set up, there is a specific examination to test for the absence of free flow, prior to attaching the tubing to the intravenous line of the patient. In the event described in this letter, the PCA tubing was clamped until after it had been attached to the patient's intravenous tubing. This experience further emphasizes that a free flow protection device is not infallible, and regardless of manufacturer, the absence of free flow should be confirmed prior to attaching the PCA to the patient.

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Reference

1 *Doyle DJ, Keebler A.* Another failure mechanism leading to patient-controlled analgesia overdoses (Letter). Can J Anesth 2008; 55: 319–320.

Use of the Airtraq® with a fibreoptic bronchoscope in a difficult intubation outside the operating room

To the Editor:

No single airway device or technique will be successful in every clinical situation. A comatose 60-yr-old male, with extensive radiation therapy to the neck, required urgent tracheal intubation for respiratory failure (pneumonia). Airway examination revealed hardened neck structures, a limited mouth opening, a fixed mandible, the neck in flexion, and a reduced thyromental distance. The patient was breathing spontaneously with the oxygen saturation maintained at 95% with a nonrebreathing face mask. Considering the limited airway examination and the patient's clinical condition, a decision was made to avoid direct laryngoscopy and the use of a muscle relaxant.

The upper airway was atomized with lidocaine, in preparation for a fibreoptic bronchoscopic (FOB) intubation. Two successful FOB attempts, with an Ovassapian airway (Teleflex, Hudson RCI, Durham, NC, USA), were followed by the inability to slide either an 8.0-mm or a 7.0-mm endotracheal tube (ETT) (Sheridan, Temecula, CA, USA) past the oropharynx. Using the rotational insertion technique, a small Airtraq® (King Systems, Noblesville, IN, USA), loaded with a 7.5-mm ETT, was passed through the limited mouth opening. The glottis was fully visualized ("Cormack and Lehane grade 1 view") in the left upper corner of the viewfinder. However, the hardened pharyngeal tissue did not allow any Airtraq® maneuvring of the glottis to the centre of the viewfinder for an optimal intubation attempt. Similarly, exterior laryngeal manipulation was ineffective. With the Airtraq® *in situ*, the pediatric FOB was advanced through the ETT (Figure 1). The vocal cords were easily identified,

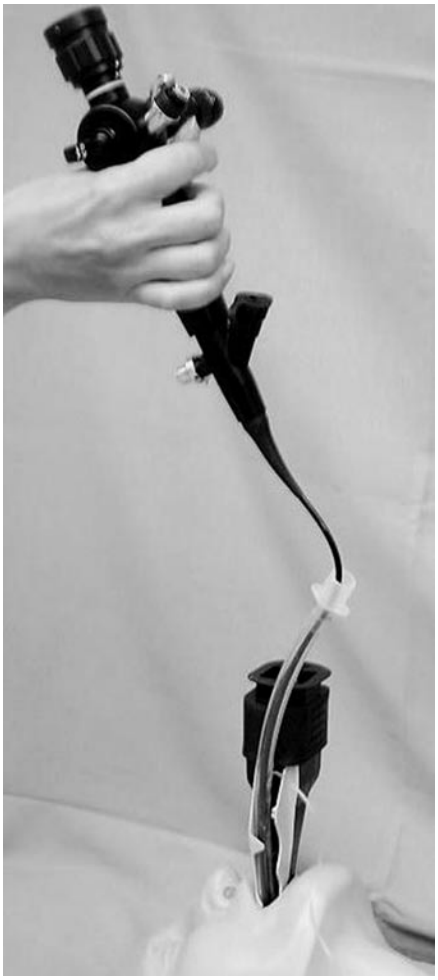

FIGURE 1 The pediatric bronchoscope advanced through the endotracheal tube housed in the Airtraq® channel.