

## Correspondence

### *Awareness during anaesthesia - patient view*

To the Editor:

I have recently been operated upon for an ovarian cyst. Although I survived, the experience was traumatic. During the operation I was awake and experienced severe pain and the trauma of being unable to move, scream or communicate.

After relating my experience to the anaesthetist I was told that I was one of 2% of patients who have recall although this figure is contradicted by a UK study that recognizes one in a thousand. Whatever the percentage, it seems to be accepted by the Medical Profession but it is not acceptable to anyone who has endured this complication.

I understand that when I awoke and was aware and in pain, it was most likely when the ultra short acting agent was wearing off and the lightest of anaesthetics had not yet kicked in. Why is this chance at the expense of the patient allowed? Was it for technical prowess and rapid awakening that I was not given enough anaesthesia? Is it possible to return to the practice of slightly deeper anaesthesia without compromising safety? The patients' welfare should take precedence over a "rapid turn around" and other measures to minimize hospital stay. I was also told that there is no way of determining if the patient is awake. Surely, knowing this and accepting that some patients will be paralyzed and aware, pre-medication, longer acting drugs and a deeper anaesthetic should be given?

I am writing to ask you, the anaesthetists, *please* reassess the philosophy and practice of modern anaesthesia so that no more patients suffer.

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### *Desflurane and nausea following ondansetron premedication*

To the Editor:

We wish to report our experience in 120 patients at the University of Wisconsin following laparoscopic tubal banding at risk for postoperative nausea, with either

desflurane or propofol anaesthesia with ondansetron premedication, 4 mg *iv*.

All patients were premedicated with 0.03 mg.kg<sup>-1</sup> midazolam and 15 µg.kg<sup>-1</sup> alfentanil *iv*. 60 had desflurane anaesthesia following anaesthesia induction with 10 mg.kg<sup>-1</sup> methohexitone *iv* and the remaining propofol. Atracurium was used and reversed if indicated. Postoperatively the incidence of nausea and vomiting was assessed at 60, 90 and 120 min and compared using a student's t test,  $P < 0.05$ . Nausea was more frequent in the desflurane group (Table).

It appears that, following *iv* ondansetron premedication, in patients at risk for nausea, desflurane may be an inferior choice as compared to propofol.

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TABLE

Time	<i>Propofol</i>			<i>Desflurane</i>		
	60	90	120	60	90	120
Nausea incidence	12%	13%	17%	32%	37%	37%
	$P < .05$	$P < .05$	$P < .05$			

### *Sevoflurane for oesophagoscopy in a patient with myotonic dystrophy*

To the Editor:

We report the management of a 62-yr-old man with myotonic dystrophy scheduled for flexible oesophagoscopy. Dystrophia myotonica is the most common of the myotonias and presents the anaesthetist with a variety of problems related to the use of muscle relaxants and unpredictable sensitivity to opioids, barbiturates and benzodiazepines.<sup>1-3</sup> The use of propofol in such patients has been reported as exacerbating the muscular weakness.<sup>4</sup>

Sevoflurane was used as the prime anaesthetic agent. Sevoflurane is non-irritant to the airways with a low blood-gas solubility coefficient, characteristics which lend themselves well to employing a single-breath

inhalational induction technique.<sup>5</sup> As the history was of dysphagia, without regurgitation we considered the risk of aspiration to be small.

No premedication was prescribed. A co-axial Bain breathing system with two 2-litre reservoir bags in series at the machine end was primed with 8% sevoflurane 8% in 50% O<sub>2</sub>:N<sub>2</sub>O. He exhaled to residual volume, breathed in deeply and held his breath. Cricoid pressure was applied with the loss of consciousness and anaesthesia deepened with sevoflurane 8%. Following a brief period of apnoea, manual ventilation was performed until tracheal intubation at four minutes. Ventilation was continued with sevoflurane 2%. The procedure was uneventful.

He was extubated in the left lateral position, after the resumption of spontaneous respiration, waking approximately 18 min after induction. Four hours later he was clinically fully recovered. Sevoflurane may have a place in the induction and maintenance of anaesthesia in patients with myotonic dystrophy.

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#### REFERENCES

- 1 Aldridge LM. Anaesthetic problems in myotonic dystrophy. A case report and review of the Aberdeen experience comprising 48 general anaesthetics in a further 16 patients. *Br J Anaesth* 1985; 57: 1119–30.
- 2 Russell SH, Hirsch NP. Anaesthesia and myotonia. *Br J Anaesth* 1994; 72: 210–6.
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- 4 Kinney MAO, Harrison BA. Propofol-induced myotonia in myotonic dystrophy (Letter). *Anesth Analg* 1996; 83: 665–6.
- 5 Smith I, Nathanson M, White PF. Sevoflurane- a long-awaited volatile anaesthetic. *Br J Anaesth* 1996; 76: 435–45.

### *Falsely reassuring readings with conventional pulse oximeters*

To the Editor:

McCrorry *et al.* report a case of falsely elevated pulse oximetry readings in a neonate without apparent explanation.<sup>1</sup> This case emphasizes the limitations of traditional pulse oximetry. The authors provide references to

several potential causes of such an anomaly.<sup>2</sup> The list of potential causes can be expanded to include other dyshaemoglobinaemias (e.g. methaemoglobinaemia) and congenital haemoglobinopathies (e.g. Haemoglobin Köln and Haemoglobin Hammersmith).<sup>3</sup> Were these possibilities considered in the differential diagnosis? I commend the authors on reporting this case as it is important to investigate cases such as the one described to ensure that we understand the limitations of the tools we rely on to indicate normal physiology.

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#### REFERENCES

- 1 McCrorry C, Ryan M, Doherty P. Falsely reassuring pulse oximetry in the presence of severe hypoxia (Letter). *Can J Anaesth* 1997; 44: 1323–4.
- 2 Poets CF, Southall DP. Non-invasive monitoring of oxygenation in infants and children: practical considerations and areas of concern. *Pediatrics* 1994; 93: 737–46.
- 3 Lang SA, Chang PC, Laxdal VA, Huisman THJ. Haemoglobin Hammersmith precludes monitoring with conventional pulse oximetry. *Can J Anaesth* 1994; 41: 965–8.

#### REPLY:

*Thank you for your interesting comments. We were not able to measure the congenital haemoglobinopathies Hammersmith or Köln at the time. Although in this case the neonate was beyond recovery and, therefore, oximetry did not effect outcome, in a less severely ill patient reliance on oximetry could have lead to suboptimal therapy and avoidable patient deterioration. This case report highlights the inherent dangers of placing too much reliance on pulse oximetry or any other piece of monitoring equipment. It is crucial that doctors understand the limitations of the monitoring equipment they use.*

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### *Complications of PA catheter*

To the Editor:

A recent publication demonstrating increased mortality in patients receiving pulmonary artery catheters (PA)<sup>1</sup> questions whether the use of pulmonary artery catheters (PAC) does more harm than good. Based on this report, we performed a retrospective analysis of 102 PAC insertions to examine our complication rate.<sup>2</sup> Complications were observed in 31 patients