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Correspondence

The role of the anaesthetist: replacement brain

The present-day anaesthetist has many roles outside the operating theatre, such as in intensive care, on obstetric units, acute and chronic pain management, teaching, education and resuscitation. We shall always be primarily associated, however, with our role of providing anaesthesia for patients undergoing surgical procedures; yet we are very poor at explaining our function in this role. As Professor Strunin points out, only 55% of the general public think that we are medically qualified (Strunin. *Anaesthesia* 2000; **55**: 941–2). I have been asked if I started as a porter and then gained promotion! Some patients even ask if I am going to be present during the operation. This may explain why some anaesthetists would prefer to be called intensivists or chronic pain doctors for example.

The surgeons simply want a patient to lie still, have an optimal view of their operating field plus a zero turnaround time. The patient simply wants to be unaware of the operation and to be alive at the end. Neither truly appreciates what we do and we are bad at explaining it to our own trainees and other medical practitioners, let alone the general public. I have heard mixed views on the success of the recent National Anaesthesia Day.

So what is the role of the anaesthetist in theatre? It is surely very easy to explain. **We are the patients' replacement brain.** We induce a reversible chemical coma and then take over the functions that the brain would normally perform. This task involves both the cerebral functions and the practical functions that an anaesthetist normally performs. The ease with which these tasks are performed depends on the anaesthetist's skills, knowledge, training and mental state. The following examples may help to elaborate this surrogate brain role.

Airway and breathing

We analyse the competence and adequacy of the airway and breathing.

'*What type of airway is best for me?*' (face-mask, laryngeal mask, tracheal, bronchial tube, venturi system, etc.). '*What type of breathing am I to perform?*' (spontaneous, assisted, one-lung ventilation, insufflation, etc.). '*Am I breathing at the right rate? Is my breathing deep enough? Have I enough oxygen in my circulation?*'

Depending on the operation and the patient, we establish and act upon acceptable parameters. Simple pulse oximetry and capnography may suffice for decision-making, but blood gas analysis may be necessary in some cases (e.g. complex general surgery or thoracic and cardiac surgery).

Circulation

We apply parameters for heart rate and blood pressure. In addition, in some cases we need to decide on the adequacy of blood volume, haematocrit, acid base balance, clotting function and ionic composition. '*My Blood pressure is too high or too low. My heart rate is too fast or too slow. I am getting angina. I am dehydrated. I am bleeding too much.*'

Disability and environment

'*My neck is badly positioned. I am in pain. I am cold. My pressure areas need attention. I am developing clots in my calf veins.*' The list is endless.

The pre-operative visit is a crucial part of the process. This is when we perform the vital medical evaluation of our patient. To which areas of the body do I need to give special attention? Will cerebral perfusion (of the original not the replacement brain) be a problem? Will the cardiovascular system be a problem? Will the airway or breathing be a problem? Will the hepatic or renal function be a problem? Will there be excessive loss of blood or clotting factors? Will the endocrine system be a problem? Are all these in optimal condition? In some cases, we have to refer the patient for further tests in order to perform a complete evaluation.

The need for a complete knowledge of the effects of the drugs that we plan to administer to a patient is obvious. We also need to have a good working knowledge of the surgery and a good understanding of the surgeon performing the operation. This enables us to predict the trauma and hazards the patient, and therefore we, will face during the course of an operation; for example, blood loss during hepatic surgery, cardiovascular changes during cardiac surgery, the need for hypotensive anaesthesia or long and short operations due to the speed of the

All correspondence should be addressed to Professor M. Harmer, Editor of Anaesthesia, Department of Anaesthetics, University of Wales College of Medicine, Heath Park, Cardiff CF14 4XN, UK.

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surgeon. We are constantly monitoring the patient's vital signs during an operation. However, we also need to monitor the progress of the surgery so that we can anticipate critical events and perform pre-emptive measures. This may explain why it is sometimes frustrating when we cannot see what the surgeon is doing under the microscope or deep in the abdominal or thoracic cavity.

Webster clearly illustrated the problems of calling ourselves peri-operative physicians [1]. I agree with his misgivings. We think highly of physicians such as the neurologists and cardiologists because of their understanding of the brain and heart. Yet we do not emphasise our role as the surrogate brain during general or, in some instances, regional anaesthesia. The physician can ponder over various medical aberrations, institute treatment and wait for a response that can take hours or days to occur. We do similar processing, but with response times of seconds and minutes rather than hours. Presenting our role as the replacement or surrogate brain will make it easier for the public, and perhaps other medical professionals to understand what we do (its not just one big syringe and one small syringe followed by a cup of coffee and *The Times* crossword). It also makes it easier to teach trainee anaesthetists to appreciate why they need to see patients pre-operatively and to understand the operation so that their brain can be ready to cope with the anaesthetic period and beyond.

This concept would explain why two different anaesthetists can have two quite different outcomes even when using, apparently, the same anaesthetic drugs and technique. It is another reason for re-emphasizing the importance of training, teaching and experience. Whose brain do you want to look after you when you undergo general anaesthesia? A consultant anaesthetist, a trainee anaesthetist or a nurse anaesthetist? It explains why one anaesthetist will be happy to anaesthetise a patient that another would not. It clarifies why critical incidences can occur due to fatigue, distractions or when anaesthetic staff have disastrously changed in the middle of an operation.

I think that our College and Association are performing a good job of improving our image. It is obviously important that we enlighten the public and others in the medical profession of our role outside the theatre complex. However, when 43% of our time is spent performing routine operating theatre work, it is important that we explain this role more clearly. I feel that the best way of explaining my role to patients is by telling them that 'A general anaesthetic puts you into a reversible coma and I will be your replacement brain for the duration of your operation.'

A. Vohra
Manchester Royal Infirmary,
Manchester M13 9WL, UK
E-mail: avohra@compuserve.com

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Anaesthesia, what's in a name?

We all recognise the difficulty that the rest of the world has in identifying just what an anaesthetist is. In his editorial (Strunin. *Anaesthesia* 2000; **55**: 941–2), Professor Strunin wrestles with the name itself and considers new ways of publicity. However, I believe that the major cause lies within our specialty and is more fundamental than we care to think. It is we ourselves who have problems with our identity.

Thirty years ago the role of the anaesthetist was clear. His job was to render the patients insensible for the surgeon to operate on and almost all of his time was within the confines of the operating theatre. The public had some idea of his identity, though not of his credentials or his importance. He was the one who put you to sleep and woke you up. He was an anaesthetist in the true sense of the word. Whilst the drugs and tools used in anaesthesia fundamentally have changed little, induction of and arousal from anaesthesia are now a minor part of our work. Surgery has become more complex and patients have become older and sicker. Nowadays, caring for the patient before, during and after the pharmacological exposure, surgical assault and associated physiological disturbance is our major role. We have evolved into peri-operative physicians and the term anaesthetist has become an anachronism.

According to the 'Snapshot' survey last year, in-theatre work only accounts for about 50% of our time. A substantial proportion of the specially has moved into the field of intensive care medicine where they undertake a similar type of work on the very sickest patients. Again, rendering the patient insensible is a minor part of their activity. Trauma management in the A & E Department is a variant of this.

The other substantial area of the non-theatre clinical activity is pain management. It departs substantially from the above two roles. The pharmacological expertise and the ability to perform anaesthetic techniques (blocks etc.) are valuable skills. Perhaps the most important are the communication skills honed in theatre during the everyday experience of communicating with anxious, distressed, difficult and sometimes strange patients (and surgeons).

Some have gone even further and immersed themselves in palliative care. This is a long way from the operating room, yet the process by which it occurs has a logic to it. On occasions, I have found myself, on the same day, working in the operating room, ICU and Accident department whilst also caring for a patient, in pain, dying of cancer.

Can we then still call ourselves anaesthetists or are we merely practicing anaesthesia (rendering insensible) whilst we spend most of our time undertaking a range of other activities? Are anaesthetists already peri-operative physicians in contrast to the 'peri-operative physicians' proposed by

others as a peripatetic trouble-shooter [1]?

The dawn of a new century is an appropriate time to look at our specially and to see how its role is changing within the broader pattern of health care that is evolving in our hospitals. If we can spot where it will be in 10 years time, might it not be even further from the original concept of the anaesthetist so beloved by the surgeons. That was the 'gasser' who kept a steady stream of patients flowing through the operating theatre and did nothing else.

Anaesthesia has changed over the last 30 years and is going in new directions. It is no surprise that the outside world cannot grasp what we are all about.

What's in a name? Well, the term anaesthesia doesn't cover my work – which is sad as I am rather fond of the title anaesthetist.

W. Notcutt
James Paget Hospital,
Great Yarmouth NR31 6LA, UK

Reference

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Complete power failure 1

I read with interest the letter (Tye & Chamley. *Anaesthesia* 2000; **55:** 1133–4) describing their experience of a complete power failure as a result of construction work. Such events are fortunately rare but may occur with no obvious precipitating factor. At about midday on a weekday in the middle of January this year, there was a total power failure at this hospital, which affected the 18-bed adult intensive care unit in particular. The cause was found to be fatigue of an old component in the main switching relay, which stopped both the mains electrical supply and the backup generator supply to a large part of the hospital. Looking back at the incident, I would like to highlight several important issues.

1 The number of extra staff required to deal with the situation in a large intensive care unit/theatre complex was significant, notably anaesthetists, anaesthetic support staff, perfusionists, and non-clinical backup from the Estates Department. We were fortunate that the incident occurred in the middle of a weekday with all staff immediately available. It is important to establish quickly who is in charge from both a clinical and a non-clinical perspective so that sensible decisions can be made quickly regarding treatment priorities and the potential need to evacuate the area.

2 The hospital telephone exchange had stopped working and there was no internal or external telephone communication. Extensive use was made of personal mobile telephones (which of course had little opportunity to interfere with medical equipment).

3 In common with the majority of intensive care units in the UK, we depend on electrically driven ventilators. We had brought a number of gas-driven ventilators of the minute volume divider type (Blease Brompton Manley) back into service to cover the potential problems that had been predicted for the New Year, and these were deployed during this incident. It was notable that some of the more junior intensive care nurses had not seen such ventilators in use before. There were no problems with the pipeline supply of oxygen or air during this incident.

4 We were fortunate that this incident occurred in daylight hours. Some natural light was available in the ITU and elsewhere. It was clear that it would be very difficult to manage the situation in the dark with only a handful of torches.

5 The ambient temperature in some areas rapidly fell to approach that outside (which was about average for January).

What was clear from this incident was that had there really been a problem with the much-hyped 'millennium' bug we would have been struggling with the probability of significant ITU mortality. As it was, all members of the 'team' did a good job without loss of life.

J. Mitchell
Harefield Hospital,
Middlesex UB9 6JH, UK

Complete power failure 2

We were most interested to read the letter concerning complete power failure (Tye & Chamley. *Anaesthesia* 2000; **55**: 1133–4), but were disappointed that no reference was made to uninterruptible power supplies (UPS). These devices provide mains-level voltage for a limited period of time after complete power failure. Although they are not new, only relatively recently have Office Grade UPSs been improved to Medical Grade standards by reducing the earth leakage currents to allowable limits. Most medical-grade UPSs are of the order of 300 VA rated, which is calculated by multiplying the mains voltage (V) by the current consumption in amps (A). In the UK, many monitors have a VA rating of $240\ \text{V} \times 1\ \text{A} = 240$. This would give a maximum run time after power failure of $300/240 = 1.25$ h.

For approximately £700, a 300 VA rated device (and a little more for a 600 VA device) can be purchased allowing uninterrupted monitoring of the patient. A further device may be required to power a ventilator.

Many hospitals test their generators regularly, and this has to be done at times of peak usage (i.e. during operating lists). The impact of momentary or medium-term power failures can be eliminated by UPSs and we believe they should be available in all operating theatre suites.

W. J. Fawcett
H. Blowers
G. Wilson
Royal Surrey County Hospital,
Guildford GU2 7XX, UK

The Combitube® should be redesigned for anaesthetic use

We read with great interest the article (Hartmann *et al*. *Anaesthesia* 2000; **55**: 670–5), in which the authors described the use of the Combitube (ETC) for gynaecological laparoscopy. We are also finishing a similar study in elective patients and we would like to comment on ETC use in anaesthesia.

First, we congratulate the authors because they have provided an alternative to tracheal intubation for this type of surgery. In our country, airway management for gynaecological laparoscopy remains a challenge, mainly because we consider that no other device other than a tracheal tube can be used. Although published experience using laryngeal mask airway for gynaecological laparoscopy is available [1], we prefer not to use it for this type of surgery. We think that the laryngeal mask airway does not provide reliable protection of the airway against gastric content regurgitation in patients undergoing gynaecological laparoscopy. Wide variation in gastric content volume among patients is an important issue, especially when gastroesophageal reflux may not be clinically apparent. Other considerations are: high airway pressures may develop during surgery and the laryngeal mask airway may fail to provide adequate ventilation under this condition, and laryngeal mask airway fixation is not reliable and displacements might occur.

Second, similar to Hartmann *et al*. and contrary to previous reports [2], we have observed a very low incidence of mild and transient postoperative pharyngeal symptoms with the ETC 37 F SA, either using blind or laryngoscopical insertion techniques [3, 4].

Third, ETC is a reliable device in managing failed intubations [5] in the anaesthesia setting and is a feasible device for elective patients. Thus, we are including ETC in many more elective surgical procedures, including gynaecological laparoscopy. However, we feel that ETC needs some improvements to enhance its current use in anaesthesia:

1 The pharyngeal balloon is rather rigid and could produce pharyngeal trauma, especially during prolonged surgery. Shifting to a high-volume/low-pressure balloon could solve this problem.

2 The tube should be bent without kinking its upper end.

3 A softer material for the tube would be an advantage

According to a recent publication which suggests that re-sterilisation is possible [6], we re-use the ETC without problems.

No other device provides good ventilation, airway protection, allows blind or laryngoscopic insertion, automatic and reliable fixation, and is so useful in the difficult airway. Thus, we strongly encourage the manufacturers to develop a new special model for anaesthesia use. Minor changes to such a remarkable device would make it more feasible for anaesthetic situations.

R. M. Urtubia
Mutual de Seguridad Hospital,
C.CH.C.,
Alameda 4848,
Santiago, Chile

J. Medina
Padre Hurtado Hospital,
Esperanza 2150,
Santiago, Chile

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Supplementary oxygenation with the laryngeal mask airway 1

We read with interest the paper on the administration of oxygen via the laryngeal mask airway in the recovery room (Peyton *et al*. *Anaesthesia* 2000; **55:** 992–9). Whilst congratulating them on their excellent study in which they directly compared the efficacy of a number of oxygen delivery devices, we would like to comment on a number of issues that this study has raised.

We agree that end-tidal oximetry, as first described by Myles and colleagues, is the optimum method of measuring oxygen delivery in this setting, since they have shown the end-tidal oxygen concentration to approximate closely to the alveolar oxygen concentration [1]. However, Peyton and colleagues were incorrect to state that other studies did not use this method since our description of the breathing system filter or heat and moisture exchanger as an oxygen delivery device also used end-tidal oximetry [2]. In our clinical study of 40 patients, we sampled gas from a catheter positioned just above the laryngeal opening of the laryngeal mask airway, and not, as they implied, at the filter's orifice. At an oxygen flow rate of 4 $l.min^{-1}$, we found an end-tidal oxygen concentration of 36.2%, which is slightly greater than was found in this paper (27.5%).

They also stated that the achievement of a high end-tidal oxygen concentration is important to patient safety. Whilst this and other studies [3, 4] have confirmed the superior performance of the T-bag compared to other devices, how much this improves patient safety is debatable, as the amount of time gained before desaturation in the event of apnoea or airway obstruction is likely to be minimal. However, the T-bag does have the advantage that movement of the rebreathing bag confirms spontaneous respiratory effort. We would also argue that it is only necessary to provide sufficient oxygen to maintain adequate oxygen saturation in order to overcome the effects of reduced functional residual capacity, ventilation-perfusion mismatch and diffusion hypoxia. In any case, patient safety can only be assured by continuous observation and monitoring.

Whilst not wishing to be seen as biased, the advantage of using breathing system filters as oxygen delivery devices is their convenience. In this age where cost-effectiveness is an important consideration, re-using the filter (approximate cost £1.50) may result in significant savings. In our hospitals, T-pieces are used to provide supplementary oxygen through the laryngeal mask airway at an approximate cost of £1.10 per patient (the T-bag costs approximately £1.50). However, it is also important to realise that any adverse event that results from the use of the filter as an oxygen delivery device is the responsibility of the anaesthetist concerned since this contravenes the Medical Device Directive regulations, as highlighted by Wilkes & Vaughan [5]. Whether any manufacturer would wish to obtain approval for their filters to be used in this way remains to be seen, since the cost incurred would be significant.

R. M. L'E Orme
Kettering General Hospital,
Kettering NN16 8UZ, UK

M. Williams
Queen Alexandra Hospital,
Portsmouth PO6 3LY, UK.

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Supplementary oxygen with the laryngeal mask airway 2

We read with interest the article on oxygen supplementation with the laryngeal mask airway, comparing four devices (Peyton *et al. Anaesthesia* 2000; **55**: 992–9), and congratulate them on this study.

We feel that it would be worth raising two small points. Firstly, the potential for barotrauma when a facemask or circuit filter is used to administer oxygen through a laryngeal mask airway in an anaesthetised patient should the filter or facemask become obstructed. This theoretical risk has been raised previously [1, 2], although there have been no reports of barotrauma during anaesthesia with the laryngeal mask airway. This is thought to be due to the low-pressure seal, formed by the laryngeal mask airway and periglottic tissues protecting the patient from barotrauma [3].

The second point that we feel bound to mention is that of product liability regulations. It should be noted that anaesthetic circuit filters are not covered by product liability regulations for use as routes of oxygen supplementation as described in the paper by Peyton *et al.* The *Consumer Protection Act* sees this as a modification to a piece of equipment and that the 'designing' anaesthetist takes on the entire responsibility for the work done and any consequences [4]. By advertising your idea to colleagues in other hospitals, you become a manufacturer and the full rigour of regulations might apply. The *Medical Devices Regulations* that regulates every aspect of medical equipment development became law in January 1995 and was implemented in June 1998 [5].

W A. Marchant
The Middlesex Hospital,
London, UK

I. Calder
The Royal Free Hospital,
Hampstead, London, UK

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The self-regulating bag

At the end of a neurosurgical case, I had deepened anaesthesia and was allowing the patient to breathe spontaneously prior to extubation. The patient was breathing through a long extension attached to a circle breathing system. The reservoir bag was placed on top of the soda lime canister, in a position to allow easy

visual and tactile confirmation of spontaneous respiration (Fig. 1). Respiration was smooth, but I had to re-open the APL valve several times as the bag kept becoming 'tight' despite a low fresh gas flow; the valve was fully opened and no-one else was interfering with it. On close inspection, I readily discovered the cause of the problem – the bag itself was lying right next to the APL valve and with each inflation the movement of the bag rubbed against the valve and turned it in a clockwise direction (Fig. 1). This led to a stepwise increase in end-expiratory pressure, and eventually closed the valve off completely. No harm occurred to the patient. This is yet another example of why the anaesthetist must be attentive to detail at all times, especially at the busy period at the end of an operation.

N. Smith
The National Hospital for Neurology and Neurosurgery,
London WC1N 3BG, UK
E-mail: nataliesmith@btinternet.com

Figure 1

Herniation of tracheal tube cuffs: a simple teaching model

Herniation of tracheal tube cuffs through the glottis seems to be occurring with increasing frequency in intensive care; we have seen it on at least five occasions in the last year.

Typically, a leak is noticed when a tracheal tube with a large-volume, low-pressure cuff is being used. Usually, more air is injected into the cuff and the leak stops for a short time. With a recurrence of the leak, more air is injected and eventually it becomes clear that the cuff has herniated upwards through the glottis and re-intubation becomes necessary.

A simple teaching model demonstrates convincingly what happens and suggests how the problem can be avoided or managed. A tracheal tube with a large-volume, low-pressure cuff (such as the Portex Soft-Seal™) is placed in the empty barrel of a 20-ml syringe so that the proximal 3–4 mm of cuff are not in the barrel (Fig. 2). This simulates the position of a tracheal tube cuff that is not fully through the glottis. With successive 10-ml increments of air, the cuff can be seen herniating out of the barrel of the syringe, simulating herniation of the cuff through the glottis (Fig. 3 with 20 ml of air in the cuff; Fig. 4 with 30 ml of air).

Our experience suggests that where there are persistent cuff leaks, the possibility of cuff herniation should be considered. Early laryngoscopy allows re-positioning of the tube or re-intubation if herniation of the cuff is occurring.

We wrote to Portex to ask whether cuff herniation had been reported to them previously and were informed that it had not, although more than 1 million Soft-Seal™ tubes have been sold. Despite an absence of reports, however, we suggest that cuff herniation is a relatively common problem in clinical use and we hope that our letter draws attention to it.

D. Wright
M. Baruch
Western General Hospital,
Edinburgh EH4 2XU, UK

A new device to facilitate awake oral fibreoptic intubation

Fibreoptic-guided, orotracheal intubation is facilitated by use of airway aids such as the Ovassapian [1], Berman [2] and Bronchoscope intubating airways. These devices help to keep the tip of the fibrescope in the midline and act as a conduit for guiding it towards the larynx. They are also useful for awake, oral fibreoptic intubation where they have the additional advantage of acting as a bite block to prevent damage to the fibrescope [3]. They may, however, provoke coughing, retching and vomiting in the awake patient when fully inserted in the mouth, especially when the gag reflex is not fully obtunded. We wish to report the use of a device called 'Safe Bite'™ (AMS Ltd Milton Keynes, UK) for use as an airway aid for oral fibreoptic intubation. This device has recently been introduced for preventing damage to laryngeal mask airways in patients recovering from general anaesthesia.

A 51-year-old female patient was schedule for a right total maxillectomy

Figure 2

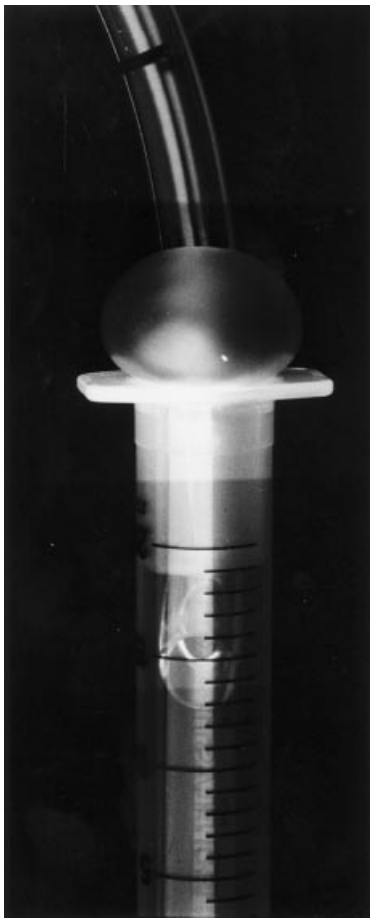

Figure 3

Figure 4

for excision of a non-Hodgkin lymphoma that had been treated unsuccessfully with radio and chemotherapy. She was predicted to have difficult laryngoscopy due to limited mouth opening and a Mallampati grade 3 airway. An awake, oral fibreoptic intubation was therefore planned. After a full explanation of the procedure the night before the operation, she was premedicated with morphine 10 mg and scopolamine 0.2 mg intramuscularly 1 h before the intubation was performed. Lidocaine 4% 4 ml was sprayed into the back of her throat to numb her tongue and oropharynx. She then received midazolam 1 mg and a target-controlled infusion of propofol 1% aiming for a concentration of 1 $\mu g.ml^{-1}$. A 'Safe Bite' was inserted into her mouth and she tolerated this without gagging or vomiting. An Olympus™ LF-2 tracheal intubating fibrescope was then inserted through the 'Safe Bit' and directed in the midline towards the epiglottis. Further topical anaesthesia to the supraglottic and infraglottic regions was achieved with the 'spray as you go' technique through the working channel of the fibrescope with 1.5-ml boluses of lidocaine 4%. Once the tip of the fibrescope entered the trachea, a 6.5-mm ID preformed oral RAE tube was successfully railroaded over the insertion cord of the fibrescope, its position checked fibreoptically and with end-tidal carbon dioxide monitoring. The patient was anaesthetised and paralysed and the 'Safe Bite' was then peeled from the tracheal tube, which was then secured.

The 'Safe Bite' can be opened from the side and peeled back from the fibrescope and the tracheal tube just like a Berman airway, but has the advantage of a reduced size (Fig. 5). This prevents it from touching the sensitive areas of the back of the throat and prevents gagging. Indeed, it is easily tolerated without local anaesthesia, as is seen in Fig. 6 with one of us (J.O.C.) demonstrating it. Our patient underwent two further awake oral fibreoptic intubations with the technique described.

J. Otero-Carrasco
K. Moaz
M. T. Popat
The John Radcliffe Hospital,
Oxford OX3 9DU, UK

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Transparent obstruction of RAE tube

A 12-kg, 23-month-old child was scheduled for tonsillectomy and adenoidectomy on a routine ENT list. He had a history of partial respiratory obstruction but no sleep apnoea. In the anaesthetic room with his mother, he appeared relaxed following an oral premed of midazolam 6 mg. After one failure to cannulate his rather mobile veins, he was induced with sevoflurane. A guedel airway was needed while he was deepened, then a 22-g cannula was inserted in his right foot. His trachea was intubated with a 4.5-mm RAE tracheal tube, which had been taken from a fresh packet and lubricated with KY jelly.

At this point, he became impossible to ventilate. The intubation had been straightforward and so the tube was withdrawn in case of malposition. After manual ventilation, he was again intubated and the same thing occurred except that a small amount of gas could be passed using higher inflation pressures. There was no audible wheeze or cutaneous signs of a reaction. The obstruction was so obviously related to the intubation that it was decided to extubate him again, and on *much* closer inspection of the tube a small clear foreign body was seen in the lumen. Figure 7 shows the whole tube and Fig. 8 shows the obstructed section magnified with and without KY lubricant. It was very easy to confuse the obstruction with surface lubricant. Another packet was opened and the next RAE tube worked perfectly and the anaesthetic continued uneventfully breathing isoflurane on a T-piece.

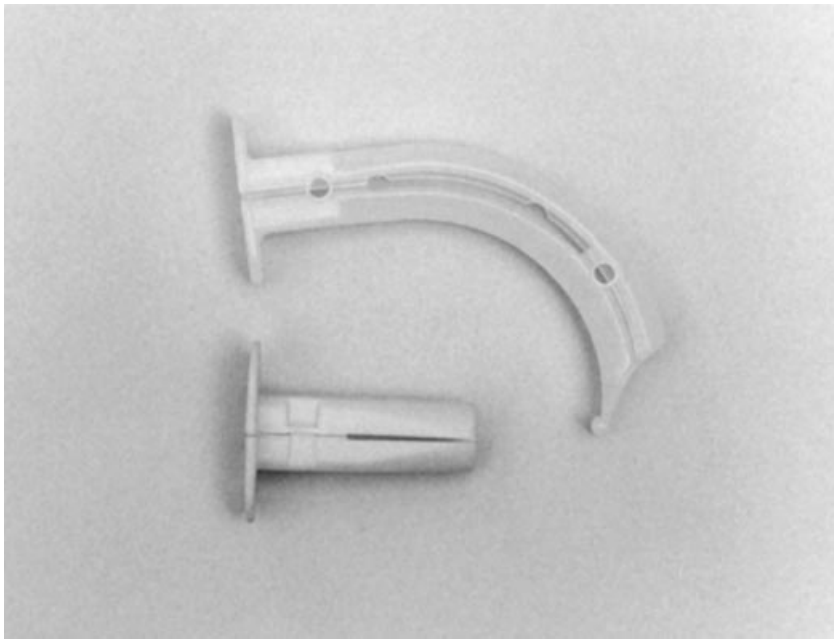

Figure 5

Because his adenoids were so large, no tonsillectomy was done, but he still oozed a little in recovery and needed some manual ventilation with a facemask. He was closely observed postoperatively for signs of any inhalation, either of blood or another foreign body, but he recovered well.

The tracheal tube was retained for further examination and the lot number noted. The obstruction was a very flexible clear membrane that blocked the whole lumen. It was quite tough, but after showing it to a

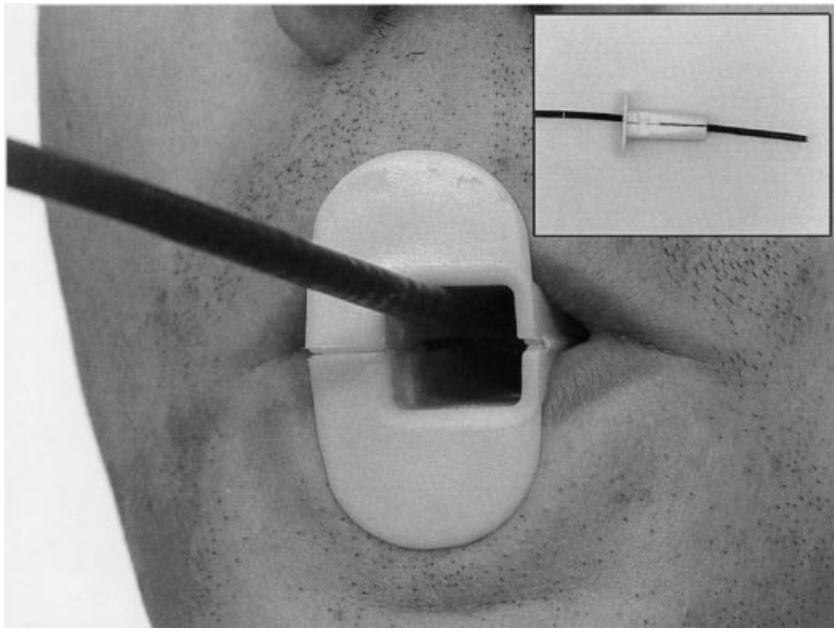

Figure 6

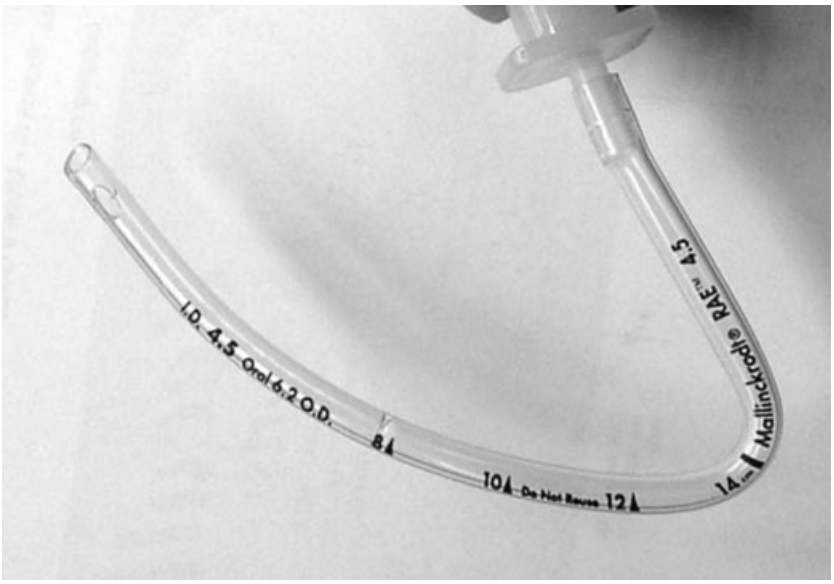

Figure 7 Whole RAE tube after cleaning, showing the obstruction at 8 cm.

number of colleagues, who bent and moved the tube, it became looser, though it was not removed. As no harm had come to the patient, the Medical Devices Agency recommended sending the tube back to the manufacturers, Mallinkrodt, for examination.

In due course, the report from Mallinkrodt stated that the sticky substance was alcohol based and was not used anywhere in their manufacturing process. The implication was that it may have been introduced locally. As far as we are concerned, this was impossible as the tube had been taken out of fresh packet and only come into contact with KY lubricant, which contains no alcohol. It looked more like a lump of glue, which might have been used to seal the packets.

No similar case has been reported in spite of 46 000 similar tubes being sold in the UK.

We are all aware of possible foreign bodies in tracheal tubes, and one of the advantages of transparent breathing circuits is meant to be the easier identification of obstructions [1]. This episode should draw attention to the extra care needed to identify transparent foreign bodies in reusable tubes.

R. Tackley
Torbay Hospital,
Torquay TQ2 7AA, UK

Reference

1 Foreman MJ, Moyes DG. Anaesthetic breathing circuit obstruction due to blockage of the tracheal tube connector by a foreign body. *Anaesthesia and Intensive Care* 1999; **27** : 73–5.

A reply

The reported complaint described an incident whereby the RAE Tracheal tube had an obstruction down the main lumen causing problems when ventilating the patient. In the above letter regarding this issue, the complainant stated that the contaminant 'looked more like a lump of glue which might have been used to seal the packets'. An extensive examination was carried out on the returned sample. The substance that was obstructing the lumen was found to be tacky and thick in nature. This substance is foreign to our production process. Uncuffed tracheal tubes do not come in contact with any glue-related substance, as there is no bonding required in their manufacture. The product is subsequently packed on a Multivac machine, which seals top and bottom webs of the pack by a heat process. It is important to note that there is no glue involved at this stage either.

When we received the sample in February of this year there was no indication on the Complaint Report that a lubricant had been used; however, in the above letter it is reported that KY jelly had been used.

We sent KY jelly to an outside test house for testing and a comparison was made between it and the contaminant found in the original RAE Tracheal Tube (Complaint Sample). The conclusion from the independent test house revealed that: 'Both samples were very similar and can be said to originate probably from a similar source' (i.e. – they are both hydrogels).

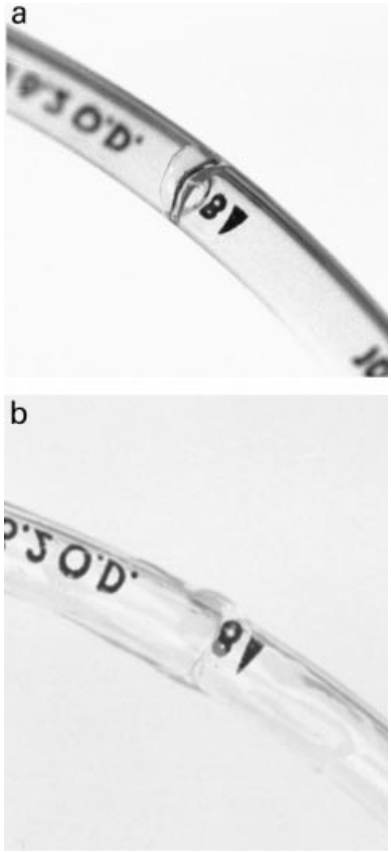

Figure 8 (a) Magnified section without lubricant; (b) with lubricant.

From the analysis it would seem likely that the contaminant involved in this complaint was a lubricant, e.g. KY Jelly.

On our Instructions For Use we supply with this product it states under the 'Warning Precautions (General)' section, that if the tracheal tube is lubricated prior to intubation, it is essential to verify that lubricant does not enter and occlude the tube lumen, thereby preventing ventilation.

We would like to highlight that fact that this is the first complaint of this nature that we have ever received with this product having marketed and distributed the RAE range of products for the past 15 years in Europe.

Mallinckrodt Medical
Cornamaddy, Athlone,
Co. Westmeath, Ireland

Administering ambient pressure oxygenation to the non-ventilated lung during thoracoscopic surgery

We are pleased to see that Baraka *et al.* have embraced the concept of attaching an ambient pressure oxygen source to the non-ventilated lung during thoracoscopic surgery (Baraka *et al. Anaesthesia* 2000; **55**: 602–3). The Mapleson E system oxygen source they describe, which utilises a 10-$l.min^{-1}$ oxygen supply, a T-piece and a length of wide-bore tubing, has obvious merit. It will achieve the desired exclusion of ambient air and therefore nitrogen from the non-ventilated lung. Second, being an 'open' T-piece system rather than a possibly 'closed' [1] reservoir-bag system [2], it will not carry the potential risk of negative pressure lung damage in the event that surgical suction is applied during thoracoscopic surgery being performed using airtight access ports [2]. Third, the 10-$l.min^{-1}$ oxygen flow will eliminate 're-breathing' of the tidal gas movement that occurs out of and back into the non-ventilated lung before the chest is opened and before the lung can collapse down [3]. Because ambient pressure oxygenation of the non-ventilated lung inevitably delays hypoxic pulmonary vasoconstriction (HPV), pulmonary blood flow through the lung is maintained and the tidal gas movement will result in either CO_2 re-breathing (in a 'closed' reservoir-bag system) or some CO_2 elimination (in an 'open' T-piece system). Recorded examples of both CO_2 re-breathing and elimination, from two patients prior to thoracoscopic surgery, are given in Fig. 9. Gas sampling ports were sited on the respective double-lumen tube (DLT) connectors, and tidal CO_2 and tidal O_2 (shown as percentages) were recorded via a single gas sampling line that was switched alternately from the ventilated to the non-ventilated lung.

In Patient A, an ambient pressure oxygen reservoir-bag was attached to the non-ventilated lung and the tidal CO_2% recorded from this lung increased progressively as the partial pressure of CO_2 in the reservoir-bag approached that of the mixed venous blood. At the same time, the tidal O_2% fell progressively as the partial pressure of N_2O in the reservoir-bag also approached that of the mixed venous blood. In Patient B, the non-ventilated lung was opened to air and the tidal O_2% recorded from this lung fell rapidly towards the percentage present in the ambient air, while the tracing of tidal CO_2% was similar to the end-tidal CO_2 tracing recorded from the ventilated lung. Thus, when a clamped-off DLT connector to a non-dependent lung is detached from the DLT itself before the thoracic cavity is opened, some on-going CO_2 elimination from that lung will result. This small component of CO_2 elimination is likely to be greater when using ambient pressure oxygenation, because, as already stated, HPV is delayed and pulmonary blood flow through the expanded, non-ventilated lung is therefore maintained.

For those anaesthetists wishing to experience the several practical clinical advantages of using an oxygen reservoir-bag system [2], we advise that until the non-ventilated lung is able to collapse down, ambient pressure oxygenation via this lung creates an atypical oxygenated shunt situation. This will result in a small further increase in the already relatively large arterial to end-tidal CO_2 gradient that is seen during thoracic surgery [4]. Therefore, in clinical situations where there is a delay in the lung collapsing away from the chest wall [2, 5], an arterial blood gas measurement may be required to guide the level of minute volume ventilation. In theory, the small increase in gradient will be marginally greater with a 're-breathing' system than with an 'open, non-rebreathing' system.

For anaesthetists who might be considering using a reservoir-bag system without the added safety of an incorporated in-let valve [1], of much greater importance is the need to ensure that thoracoscopic access ports are not airtight when surgical suction is being used [2].

Finally, it is worth noting that once the lung has collapsed, very likely to the point at which small airways closure occurs, the need for ambient pressure oxygenation no longer exists. This belief is based in part on the observation that once the thoracic cavity is opened to ambient air, and in the absence of pleural adhesions, air-flow obstruction or emphysema, the reservoir-bag is seen to expand over 30–60 s as the lung collapses down due to its inherent elastic recoil. Then, unlike the situation *before* the thoracic cavity is opened, there is no noticeable progressive reduction with time in the volume of gas in the reservoir-bag. Thus, most or all further collapse of the non-ventilated lung is a consequence of ongoing gaseous uptake from within the lung (absorption atelectasis). In spite of this, we still choose to leave the reservoir-bag attached while surgery is proceeding. We do this not in the belief that it will further prevent the entrainment of air and therefore nitrogen into the non-ventilated lung, but rather to enable immediate identification of any leakage of ventilating gases past the DLT bronchial cuff. Gas leakage past the bronchial cuff is seen when surgical traction on the lung or bronchus threatens to dislodge the DLT from the relevant main bronchus. Complete

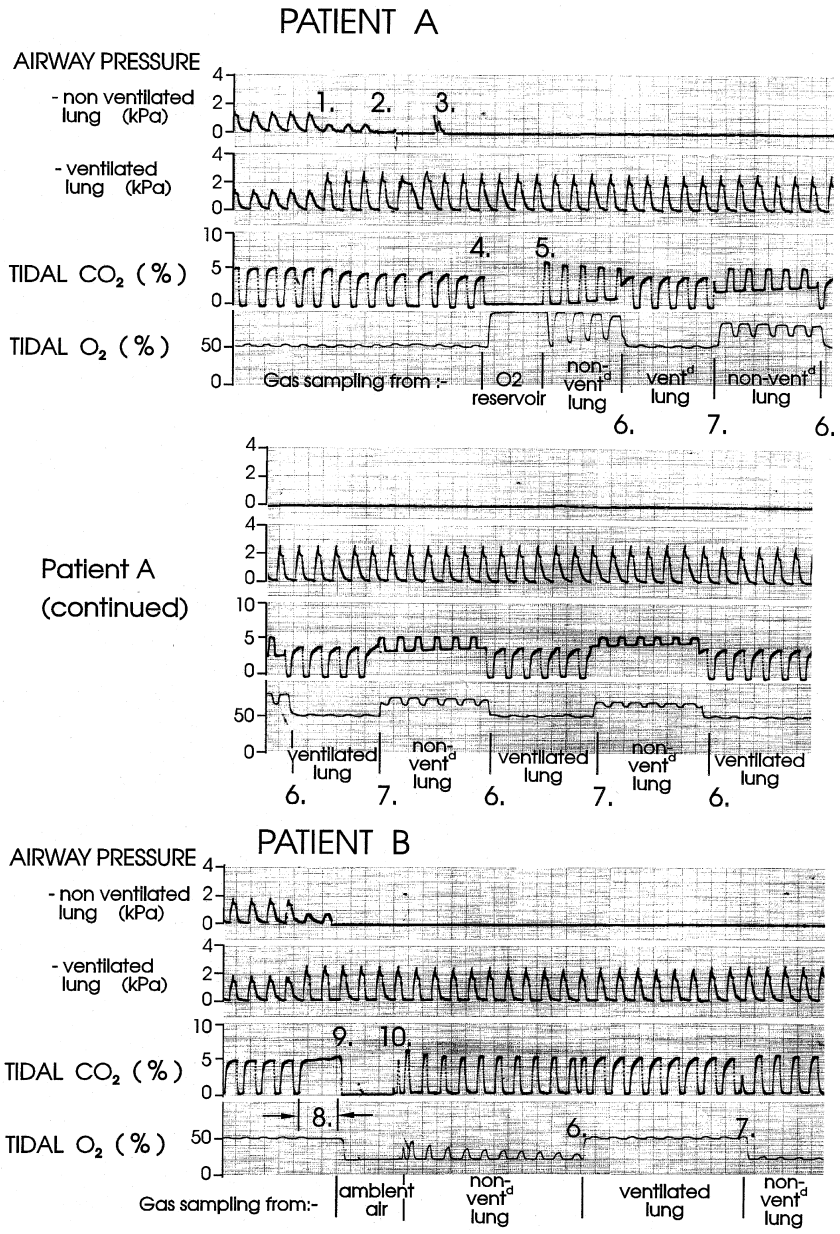

Figure 9 Tidal O_2 and CO_2 concentrations (percentages) recorded from the ventilated and non-ventilated lungs of two patients, one (Patient A) with an ambient pressure O_2 reservoir connected to the non-ventilated lung and the other (Patient B) with the non-ventilated lung opened to air. 1. Airway connector to non-dependent lung clamped-off at end-expiration, so initiating single-lung ventilation. 2. A second clamp placed across the DLT lumen itself, so isolating the non-ventilated lung. 3. (In Patient A only) Ambient pressure O_2 reservoir connected to the still-isolated (clamped-off) non-ventilated lung. 4. (In Patient A only) Gas sampling line switched from the ventilated lung to the O_2 reservoir. 5. (In Patient A only) Clamp removed from the DLT, so connecting the non-ventilated lung with the O_2 reservoir. 6. Gas sampling switched from the non-ventilated to the ventilated lung. 7. Gas sampling switched from the ventilated to the non-ventilated lung. 8. (In Patient B only) Period of gas sampling from the non-ventilated lung that is not yet opened to air. 9. (In Patient B only) DLT lumen to the non-ventilated lung clamped-off and the DLT connector detached, so opening gas sampling to ambient air. 10. (In Patient B only) Clamp removed from the DLT, so connecting the non-ventilated lung to the ambient air.

dislodgement in the course of thoracic surgery, although rarely reported, is well recognised and certainly best avoided.

J. Pfitzner
M. J. Peacock
B. W. Daniels
The Queen Elizabeth Hospital,
Adelaide, South Australia 5011

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4 Ip Yam PC, Innes PA, Jackson M, Snowdon SL, Russell GN. Variation in the arterial to end–tidal PCO_2 difference during one–lung anaesthesia. *British Journal of Anaesthesia* 1994; **72**: 21–4.

5 Pfitzner J, Peacock MJ, Tsirgiotis E, Walkley IH. Lobectomy for cavitating lung abscess with haemoptysis, strategy for protecting the contralateral lung and also the non–involved lobe of the ipsilateral lung. *British Journal of Anaesthesia* 2000; **85**: 791–4.

Light wand guided tracheal intubation through the intubating laryngeal mask

We read with interest the article comparing blind (BL) and lightwand-guided (LW) tracheal intubation through the intubating laryngeal mask (ILM) (Kihara *et al. Anaesthesia* 2000; **55:** 427–31). According to their results, the authors report that the ILM-LW intubating technique is superior to the ILM-BL technique, since it improves the success rate (100% vs. 93%) and diminishes either the number of adjusting manoeuvres required or the duration of the intubating procedure.

These results are in agreement with those reported in a similar randomised, double cross over comparative study [1], using a prototype flexible lightwand (FLW) [2]. However, Kihara *et al.* do not clarify whether in the ILM-LW group the lateral glow misplacement and/or the absence of glow in the neck was followed by specific LW adjusting manoeuvres, or did they apply the same sequence of BL adjusting manoeuvres as in the ILM-BL group. Additionally, we would like to ask whether the intubation time reported included not only the time for successful intubation but also the time required for proper ILM placement.

In a recent study [3], the ILM-LW intubating method was performed in a larger series of patients ($n = 400$). We found that the success rate was independent of factors usually predicting difficult laryngopscopy, since this was similar in patients with either normal (372/373, 99.7%) or potentially difficult airways (27/27, 100%) [3]. Absence of glow in the neck was noticed during the first intubating attempt in 19 of 400 patients (4.8%). In all these cases, accidental oesophageal intubation occurred and it was confirmed by capnography. However, 18 of 19 patients were finally intubated successfully during the second or third intubating attempt, resulting in a final incidence of persistent oesophageal intubation of 1/400 (0.25%). In the paper of Kihara and colleagues, it is reported that the incidence of oesophageal intubation in the ILM-LW group was zero. We wonder if this frequency concerns the first intubating attempt or the final outcome.

V. Dimitriou
G. S. Voyagis
Gennimatas and Sotiria Hospitals,
Athens, Greece

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3 Dimitriou V, Voyagis GS. Light-guided intubation via the intubating laryngeal mask using a prototype illuminated flexible catheter. Clinical experience in 400 patients. *Acta Anaesthesiologica Scandinavica* 2000; **44**: 1002–6.

A reply

We thank Drs Dimitriou and Voyagis for their valuable comments concerning our paper comparing intubation through the intubating laryngeal mask airway (ILM) with and without a lightwand. We will respond to each of their questions in turn.

First, if the light glow was seen in the lateral neck and/or was absent, we performed manoeuvres that were identical to the blind intubation group. For example, if a glow was seen initially in the lateral neck, we performed an extension manoeuvre (Dimitriou and Voyagis would have performed a twisting manoeuvre [1]). If this occurred a second time, we performed an 'up–down' manoeuvre (Dimitriou and Voyagis would have performed another twisting manoeuvre [1]). The sequence of adjusting manoeuvres proposed by Voyagis and Dimitriou is based on the concept of a probable cause and best correction tactic, whereas ours is based on the concept of starting with simple manoeuvres and avoiding size changes. Both these concepts have some merit. A comparative study is required to determine which of these concepts/manoeuvring sequences, if any, is best. Second, 'insertion time' was from commencement of insertion of the ILM to capnographic confirmation of tracheal intubation. If insertion failed, the time was excluded from the analysis of insertion time. Finally, the 0% incidence of oesophageal intubation in the lightwand group refers to the final outcome. Some initial oesophageal intubation events may have gone unrecorded since we only connected the anaesthesia circuit when the light glow was seen at the sternal notch and tracheal placement likely. Interestingly, on all occasions when the light glow was seen at the sternal notch, tracheal intubation was subsequently confirmed, suggesting that this is a reliable sign for successful tracheal intubation with the lightwand/ILM technique.

S. Kihara
S. Watanabe
Saiseikai General Hospital,
3 3-10 Futabadai, Mito,
Ibaraki 311-4198, Japan

J. Brimacombe
University of Queensland,
Cairns Base Hospital,
The Esplanade,
Cairns 4870, Australia

Reference

1 Dimitriou V, Voyagis GS. Light-guided intubation via the intubating laryngeal mask using a prototype illuminated catheter. *Acta Anaesthesiologica Scandanavica* 2000; **44**: 1002–6.

Full circle

I was interested to read the letter 'Iatrogenic foreign body in the bronchus' (Dutta *et al. Anaesthesia* 2000; **55:** 1036–7), and was reminded of a similar occurrence that I reported

in 1967 [1]. This involved the loss at laryngoscopy, into the oesophagus and subsequently to the colon, of the complete metal nozzle from a spray bottle of lidocaine. As a result of this mishap, the makers modified their spray by having the nozzle permanently fixed to the actuator valve.

In the same report, I also referred to another incident involving the use of a Rogers laryngeal spray a year earlier [2], and to my personal experience of losing the atomiser button of a Macintosh laryngeal spray into the depths of the bronchial tree.

Whilst it is somewhat disappointing to learn that similar problems can, and still do, occur over 30 years later, it is perhaps not all that surprising given that most things come round full circle if you wait long enough!

J. G. Francis
Exeter EX3 0QT, UK

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Anterior deviation of the trachea

I was interested to read the recent letter concerning difficult intubation in a patient with a tracheal diverticulum and 90° deviation of the trachea (Davies. *Anaesthesia* 2000; **55:** 923–5). The author states that he could find no previous reports in the literature of such marked tracheal deviation. I would like to add to the literature by reporting a case with which I was recently involved.

A 49-year-old woman with menorrhagia had been referred by her GP to one of our gynaecologists for further investigation and management. On examination, a large pelvic mass was discovered, and although ultrasound scan suggested that the mass was due to fibroids, there was concern that there might be an underlying malignancy. She was therefore listed for urgent admission and surgery. The consultant gynaecologist concerned discussed the case with me as the intended anaesthetist during the course of a routine list. He was anxious that she be operated on the following week in view of the possible malignancy. During the discussion, I learned that she was also due to undergo a total thyroidectomy in the near future for a multinodular goitre. Correspondence in her case notes confirmed this and indicated that she had no symptoms of mechanical obstruction. Otherwise she was 'fit and well'.

For a variety of reasons it was not possible to arrange to see the patient within the time frame proposed. We agreed that she would be admitted onto the ward on the day of surgery (as is the norm), but that she should have a plain chest X-ray as well as antero-posterior and lateral neck views performed before admission.

I met the patient the following week on the morning of surgery. She appeared fit and well and weighed 80 kg. She had an obvious goitre, but clinically appeared euthyroid, and indeed had no symptoms of oesophageal or tracheal obstruction. Examination of her head and neck revealed normal dentition, a slightly receding mandible, and a Mallampati grade 2 airway. It therefore came as a surprise to discover the appearances of her cervical spine X-rays. The AP film revealed some suprasternal narrowing of the trachea with deviation to the right (Fig. 10). The lateral view, however, showed marked anterior deviation of the pharynx and trachea with extensive soft tissue shadowing posterior to the larynx and trachea (Fig. 11).

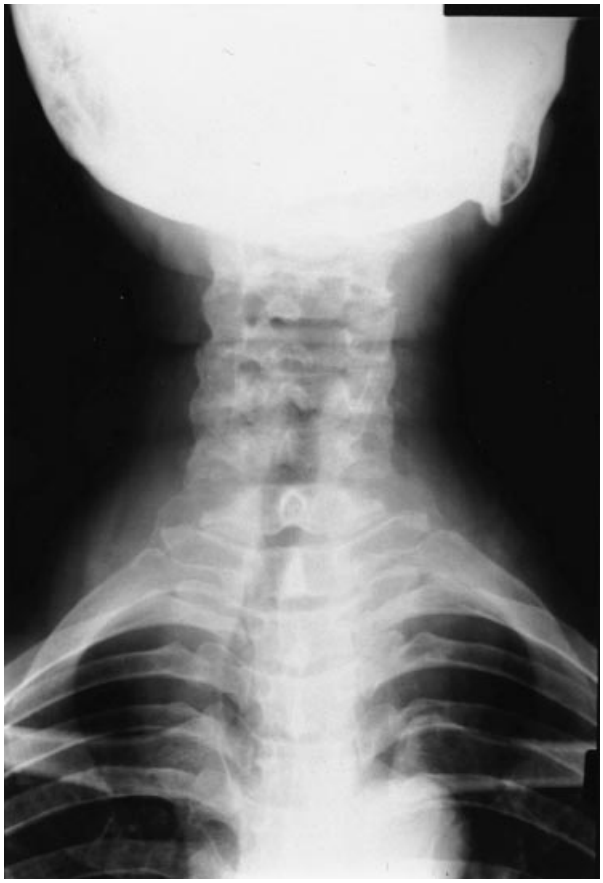

Figure 10

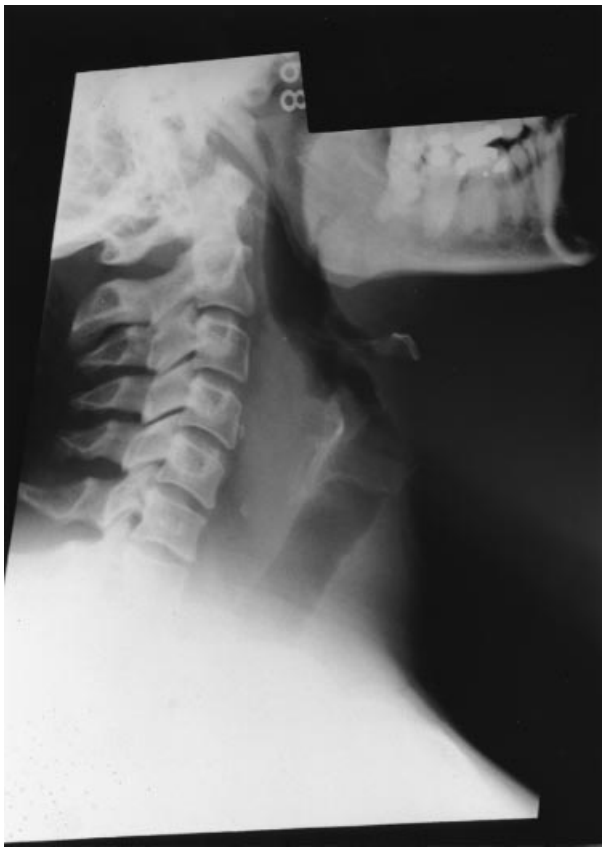

Figure 11

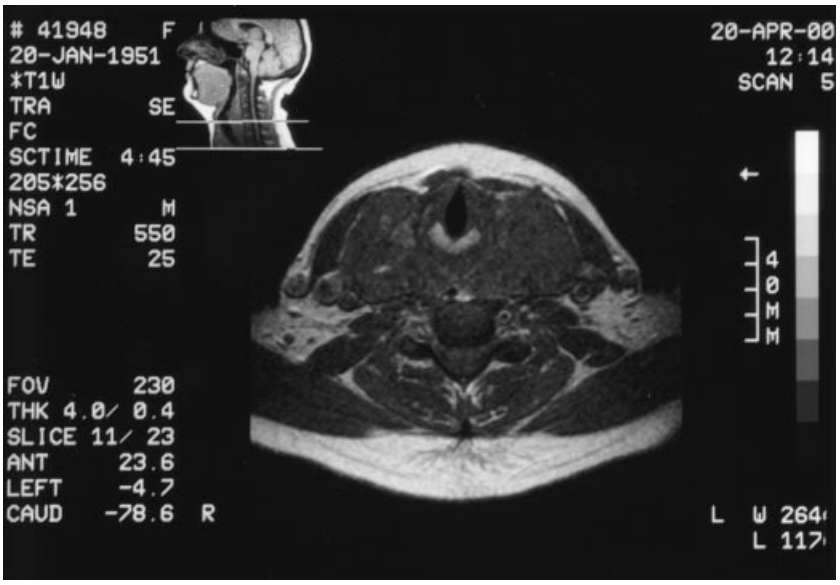

Figure 12

Because it is our policy not to undertake difficult or high-risk patients in our isolated gynaecology unit, we decided not to proceed with the case that day. She was rebooked to have her operation two weeks later, an awake fibre-optic intubation being the obvious choice for airway management.

In the meantime, she had an MRI scan of her neck performed, which revealed that the tracheal deviation was due to thyroid tissue extending behind the pharynx, larynx and upper part of the oesophagus, with associated narrowing of the trachea (Fig. 12). There was some discussion between the gynaecology and surgical teams regarding which operation should be performed first. It was even proposed that the two should be performed at the same time, since it was suggested that instrumentation of such a distorted airway could cause postextubation difficulties if the gynaecological surgery was undertaken alone. In the end, however, she underwent hysterectomy and bilateral salpingo-oophorectomy, and histology revealed only benign disease. Intubation was achieved with a 6-mm reinforced tracheal tube using an awake fibre-optic technique. Surgery and anaesthesia were both uneventful, and the patient made a problem-free recovery. At the time of writing, she is awaiting her total thyroidectomy.

I also have been unable to find any previous reports of marked anterior deviation of the larynx and trachea. In addition, I have been unable to find any literature about retro-tracheal spread of thyroid tissue, although retrosternal, spread and total intrathoracic thyroid is well recognised, as is ectopic retro-tracheal [1] and intratracheal [2] thyroid tissue.

The airway management of this case was fairly straightforward, armed with the X-ray information, but it serves to remind that patients with major airway distortion can still be symptom-free. Appropriate pre-operative investigation is required for all patients presenting for thyroid surgery to ensure that airway management presents no unpleasant surprises in the anaesthetic room.

I. J. Crabb
Gloucestershire Royal NHS Trust,
Gloucester GL1 3NN, UK

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Mishaps with a central venous cannula in the ICU

We wish to report an unusual case of disconnection from a central venous cannula (CVC) in a patient on the intensive care unit (ICU). A 66-year-old man had been transferred from the high-dependency area in a satellite hospital to our ICU for further management of respiratory failure secondary to fluid overload due to congestive cardiac failure (CCF) after a myocardial infarction 6 weeks previously.

The CCF had worsened when his cardiac rhythm changed to an atrial flutter tacchyarryhthmia. With management including intubation and ventilation, diuresis, anticoagulation and cardioversion, his condition began to improve. A trans-thoracic echocardiogram was performed to assess left ventricular function and for the presence of thrombus. On confirmation of the absence of intracardiac thrombus, M.W. went to instruct the patient's nurse to discontinue the intravenous heparin infusion. On entering the cubicle, it was noted that a streak of fresh blood was dripping down the patient's chest. On further investigation, it was found that the medial 'pigtail' of the four lumens to the right subclavian venous CVC (Arrow 8.5F 20 cm antibiotic-coated quad-lumen CVC) had become disconnected/unplugged from the base where the four lumens come together (Fig. 13). The patient was immediately laid head down whilst the heparin infusion was stopped and the faulty lumen was occluded to prevent entrainment of air. On balance, it was

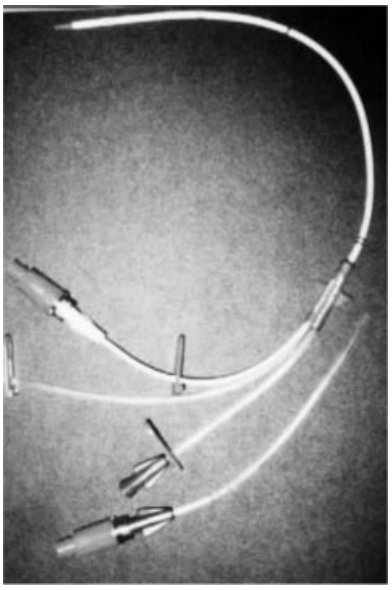

Figure 13

decided to leave the CVC in for 2 h to allow the clotting to normalise whilst nursing the patient supine. His condition remained stable throughout and he went on to be discharged back to the satellite hospital.

This case highlights the need to be vigilant to the possible hazards that may occur with multilumen CVCs and the various risks that we need to consider whilst utilizing them on the ICU. A literature search reveals no previous reports of similar mechanical failure during the short-term use of CVCs, although there are reports of fractures in the long-term use of implanted subcutaneous central venous catheters [1–3]. The exact nature of the mechanical failure is being investigated by the manufacturer; the results of this investigation will allow a more thorough risk assessment.

M. Williams
B. Siggers
Queen Alexandra Hospital,
Portsmouth PO6 3LY, UK

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Aortic cross-clamping in sickle cell disease

We read with interest the correspondence on the emergency repair of a large tender abdominal aortic aneurysm in a patient with sickle cell trait (Heames & Vincent. *Anaesthesia* 2000; **55**: 1034). We, too, recently anaesthetised a patient with sickle cell disease for a procedure requiring aortic cross-clamping.

Our case was slightly different in that it was a 68-year-old woman with haemoglobin SC disease (HbSC disease) who presented for coronary artery bypass graft surgery. Like the authors, we found very little published literature on major vascular surgery in patients with sickle cell disease, and none at all on coronary artery bypass grafting in sickle cell disease, although there was some on cardiac valve replacement in this patient population.

Although patients with HbSC disease have a milder clinical course than those with homozygous haemoglobin S disease (HbSS disease), with symptoms occurring later [1] and a longer median survival [2], we, too, were concerned about the potential problems of cardiopulmonary bypass and vascular stasis.

After reviewing the available literature, and having prolonged discussions with our haematologists, we decided on a management strategy for this patient. She was exchange transfused (to a total haemoglobin of 10.1 $g.dl^{-1}$, and HbA of 61%) pre-operatively and kept well hydrated. During anaesthesia, the inspired fractional concentration of oxygen was maintained at 0.5. Normothermia was maintained whilst on cardiopulmonary bypass, and an

intermittent cross-clamp fibrillation technique used with cross-clamp times of 9 min and 7 min, so minimizing blood stasis in the coronary vessels. The case proceeded uneventfully and the patient was discharged home well after 7 days.

In our opinion, the basic principles of anaesthesia in sickle cell disease, of keeping patients warm, well hydrated, well oxygenated and mildly alkalotic [3] to avoid conditions leading to relative regional hypoxia, increased sickling, and compromised oxygen delivery to tissues, should be extended to when these patients undergo surgery requiring cardiopulmonary bypass.

W. A. Marchant
S. Wright
The Middlesex Hospital,
London W1N 8AA, UK

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Close loop anaesthesia using bispectral index

I read the paper describing closed loop control of anaesthesia using bispectral index (Morley *et al*. *Anaesthesia* 2000; **55**: 953–9) with interest, and would like to comment on two points which concern me.

Bispectral index monitoring (BIS) is based upon EEG analysis and produces a dimensionless number from 0 to 100. As the authors correctly state, this is a score of hypnosis. It is not a measure of analgesia, although, to some extent, this can be derived from the trend of the BIS score. It therefore concerns me that they are using a score of hypnosis to titrate a mixture of hypnotic and analgesic agents in their infusion group and then drawing conclusions about the adequacy of hypnosis. In other words, as their infusion varies, the level of hypnosis and analgesia will vary. In contrast, their volatile group receive a measured amount of analgesia. Second, in their discussion they state 'episodes of light anaesthesia occurred in response to sudden surgical stimuli'. This would appear to be more due to inadequate analgesia than inadequate hypnosis. A sudden spike in the BIS score from a satisfactory, stable baseline, coincident with surgical stimulus, is suggestive of inadequate analgesia, while the level of hypnosis may be adequate. Hence, an increase in the analgesic component may have been required, while not necessarily increasing the hypnotic component, something they would have been unable to do with their infusion mixture. Alternatively, an infusion with a higher concentration of alfentanil may have been more suited to the type of surgery.

While closed-loop anaesthesia is an exciting prospect, it entails more than infusing a drug, or combination of drugs, to achieve a certain score on a monitor that is only monitoring one aspect of the anaesthetic, in this case hypnosis. Using such an approach, we run the risk of over-sedating patients while providing inadequate analgesia. I suggest their study would have been more meaningful if they had separated their infusions and been able to titrate each individually: using Propofol to achieve an adequate BIS score, and alfentanil, or another short-acting opioid, to provide adequate analgesia and maintain stability of the BIS score.

This study also serves as a reminder that the attending anaesthetist is still the single most important patient monitor in any operating theatre.

Finally, I would like to thank Dr Derrick for making his program 'Monitor' available for download over the internet. It is one of very few anaesthesia recording software programs available for the Apple Macintosh platform, and one that I have used successfully for my own anaesthetic recording purposes.

C. Stonell
Colchester General Hospital,
Colchester CO4 5JL, UK

A reply

Thank you for the opportunity to respond to the points raised in the above letter. In stating first that BIS is a score of hypnosis and not analgesia, and then that the stability of BIS may be maintained using alfentanil, an analgesic, Dr Stonell seems to want to have his cake and eat it. Confusion in this area has been partly resolved by recent work, summarised below and published while our manuscript was in preparation.

In volunteers, alfentanil does not appear to affect Cp50 or BIS50 (respectively, the propofol concentration and BIS associated with a 50% probability of loss of consciousness) [1]. The conclusion is that alfentanil does not exert a significant additional hypnotic effect under these circumstances. In another study, tracheal intubation caused a rise in BIS, which is attenuated in a dose-related fashion by pre-administration of remifentanil [2]. The authors of this study concluded that a change in bispectral index is as sensitive as haemodynamic responses after a painful stimulus for detecting deficits in the analgesic component of anaesthesia.

Dr Stonell's second point concerns episodes of light anaesthesia and the limitations of our intravenous technique. He quotes us incorrectly in his letter. Our discussion states: 'episodes of light anaesthesia occurred in all groups in response to sudden surgical stimuli'. In other words, these episodes were noted in patients receiving isoflurane/N_2O/morphine as well as propofol/alfentanil anaesthesia. The use of a measured amount of analgesia in the former group, rather than a variable one in the latter, is noted by Dr Stonell with apparent approval. However, the effect of a bolus dose of morphine, administered at induction, declines intra-operatively and is

no more titrated to analgesic requirements than is a variable rate alfentanil infusion.

Dr Stonell suggests that our study might have been more meaningful if we had been able to titrate a separate alfentanil infusion to provide adequate analgesia. He does not indicate how intra-operative analgesia might be measured in order to effect this titration. The fact is that intra-operative signs of light anaesthesia are often the same as those of inadequate analgesia – namely a rise in arterial pressure and heart rate, and increased BIS. That the two states are intimately associated is further indicated by evidence that painful stimuli at light levels of anaesthesia may enhance cognitive function [3]. This phenomenon is recognised, perhaps unwittingly, by the many anaesthetists who respond to cardiovascular 'evidence' of intra-operative light anaesthesia by increasing inspired volatile concentrations as well as administering a bolus of opiate analgesic.

The diagnosis of inadequate analgesia ultimately depends on observation by the anaesthetist of surgical events, information that is clearly unavailable to a closed-loop controller. Even with this advantage, the anaesthetists administering isoflurane in our study were unable to out-perform the controller in terms of number of episodes of light anaesthesia.

May I thank Dr Stonell on Dr Derrick's behalf for his kind comments regarding 'Monitor'.

A. P. Morley
St Thomas' Hospital,
London SE1 7EH, UK

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Are peri-operative peripheral nerve lesions preventable?

At first sight, the review of peripheral nerve injuries associated with anaesthesia (Sawyer *et al. Anaesthesia* 2000; **55:** 980–91) appears to be a useful account of an important condition, which often presents considerable medicolegal challenge. Unfortunately, I think that one of its main concluding statements is wrong, and potentially very damaging to our profession.

The authors conclude: 'Although they (peripheral nerve injuries) account for a small proportion of medicolegal claims, they are difficult to defend, being essentially avoidable'. However, throughout their article they quote individual cases, small series, and reviews which could not and do not support this contention. In most cases, clinicians are being wise after the event and reporting putative mechanisms. In many cases the mechanism is likely, but in others the mechanism suggested might be incorrect. In retrospect, much 'evidence' is necessarily suspect. Their citation of the relevant literature in this review is necessarily restricted, but the balance is unhelpful. In particular, I was surprised by the age and source of the references. Excluding the duplicate reference (citations 10 and 57), the article cites 66 sources, of which only 12 are less than 10 years old, and only seven of those are original reports. It does not cite two recent prospective studies [1, 2] and experimental data [3]. In an editorial comment on this latter study, Caplan wrote: '(it is) … a humbling reminder that we have a limited understanding of the relations between conventional peri-operative care and the genesis of peripheral nerve injury' [4]. This was also the conclusion of the article of which Caplan was a co-author in 1990 [5], which is in fact cited by Sawyer. That article called for 'prospective studies', and we now have two carefully conducted large prospective studies [1, 2] not cited by Sawyer (to be fair, one is a recent publication). A most important feature of this study of lower extremity neuropathies was that such neuropathies are sensory and present early, which is contrary to the catchall statement in the Sawyer review.

Sawyer and colleagues did cite an editorial by Stoelting [6] with a similar cautious sentiment: 'a conclusion that postoperative ulnar nerve palsy is always a preventable complication cannot be supported by available evidence and our current understanding of the problem'. I regret that they did not take this message from that editorial, rather than reproduce a figure that looks very much like Stoelting's figure 2, without acknowledgement.

I know we should avoid damage to our patient's peripheral nerves, by avoiding what could be harmful features such as excess pressure, and abnormal positions for long periods. It is perhaps even more important that we should avoid being shot in the foot by the assertion that all peri-operative nerve injuries are 'essentially avoidable'. This review has not provided any direct evidence that this is so: and scientifically the evidence that we have will rarely be able to do so. In fact, the review article has cited evidence that some injuries do 'just happen', presumably caused by effects that we do not realise, and probably cannot control. Does one of the authors, who is the Medical Director of the Medical Protection Society, truly wish to stand by the assertion that in essence all peri-operative nerve injuries are indefensible, particularly on the basis of the evidence adduced in this review?

G. B. Drummond
Royal Infirmary,
Edinburgh EH3 9YW, UK

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A reply

Thank you for the opportunity to reply to Dr Drummond's letter. We agree that in the genesis of nerve injuries, the mechanisms of injury are putative and unknown. It was to this end that our suggestions for avoiding possible predisposing factors, as described in our review article, were directed. This would be the safest course of action for an anaesthetist in clinical practice to follow until further research, such as that undertaken by Prielipp et al. [1], could be undertaken to elucidate a mechanism of neural injury. We have not stated that all peri-operative nerve injuries are avoidable, or that they can be wholly prevented, rather that an awareness of predisposing factors may reduce the frequency with which they occur. We have stated that nerve injuries are 'difficult to defend' rather than 'indefensible' and this is borne out by the fact that ' … payment was made in about half of the claims (for peri-operative ulnar neuropathy) where care was judged appropriate' [2].

The recent papers referred to by Dr Drummond [1, 3, 4], but unpublished at the time our artricle was submitted, support this view. Whilst we are cognisant of the length of time from submission to publication, we were unable to review these articles. Dr Drummond correctly takes us to task for failing to acknowledge the source of figure 4, for which we apologise. However, it was modified from an original article by Wadsworth [5] and not by Stoelting [6] as cited by Dr Drummond.

The time of onset of peri-operative peripheral neuropathy has been a subject for much debate. In the article quoted by Dr Drummond [3], in relation to lower extremity peri-operative peripheral neuropathy, Warner et al. stated that 'prolonged duration of time in lithotomy positions, especially for more than 2 hours, was strongly associated with these neuropathies'. Lower extremity neuropathies did indeed present early, within 4 h of completion of the anaesthetic and 14 out of 15 neuropathies resolved within 6 months. The same group of investigators prospectively studied ulnar neuropathy [7] with interesting results. They found that symptoms developed 2–7 days after surgery and in four patients symptoms resolved within 6 weeks; however, three patients still had residual symptoms 2 years later. These authors also observe: 'None of the neuropathies were present within the first 2 postoperative days'.

The experimental observations of Prielipp et al. [1] also showed that pressure on the ulnar nerve at the elbow was greatest with the forearm in pronation and least in supination. Referring to this last study, Caplan [4] in the same article quoted by Dr Drummond, wrote: 'Until we have a better ability to predict and monitor ulnar nerve injury, these findings can serve as guides for clinical decision making.' Our sentiments entirely!

R. J. Sawyer
M. N. Richmond
J. Jarratt
J. Hickey

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Peripheral nerve injuries

I read the recent review article on peripheral nerve injuries (Sawyer *et al*. *Anaesthesia* 2000; **55**: 980–91) with great interest. Most of the neurological damage described in the article resulted from poor surgical positioning, which caused either direct pressure or traction on the nerve trunk. I would like to report a case where significant, but thankfully temporary, damage was unwittingly inflicted on a major peripheral nerve by anaesthetic considerations alone.

A 52-year-old woman undergoing free-flap reconstruction of an intra-oral carcinoma was anaesthetised supine and both arms adequately padded with gel supports in the crucifix position. Direct arterial pressure was monitored via a 20-g cannula in the left radial artery. Shortly after

commencement of surgery, the arterial trace became markedly damped and a satisfactory waveform could only be re-established by extending the wrist. This position was maintained throughout the operation (some 5 h) by use of a roll of crepe bandage and adhesive tape (Fig. 14) without any further monitoring problems. On the following day, the patient complained of numbness and weakness in the left hand and examination revealed a complete median nerve palsy on that side. Fortunately, this gradually but completely resolved over the next few days.

In retrospect, it is clear that overextension of the wrist exerted traction on the median nerve and possibly compression of it within the carpal tunnel itself. In my experience, monitoring of the radial arterial waveform often requires a little wrist extension to produce a reliable trace but, in the light of the above case, I would urge caution as to both the degree of extension and the length of time for which it is applied.

P. J. Hilton
Morriston Hospital,
Swansea SA6 6NL, UK

Spinal cord damage

I would like to report two cases of damage to the spinal cord similar to those described by the editorial 'Logic in the safe practice of spinal anaesthesia' (Reynolds. *Anaesthesia* 2000; **55**: 1045–6).

Until April 1994, either spinal or epidural analgesia was used at Watford General Hospital for those obstetric patients requesting regional anaesthesia. In April 1994, the combined spinal epidural technique (CSE) was introduced using a Portex pack and the needle through needle technique. During the following 18 months, two patients suffered permanent severe damage to the spinal cord because the needles were inserted at too cephalad an interspace. The blocks were performed by anaesthetists with 8 or 10 years experience of siting epidural catheters but who were new to CSEs. Both patients were anxious and complained bitterly of pain when their skin was infiltrated with lidocaine. When they complained of severe pain as the spinal needle was introduced, it was in each case assumed that the pain they were experiencing was no worse than that of skin infiltration with local anaesthetic. The anaesthetists concerned believed they were using the L_{2-3} or L_{3-4} space but in each case a magnetic resonance imaging (MRI) scan carried out following evidence of nerve damage showed a lesion at T_{12}–L_1. It was concluded that the spinal needle had been inserted at an inappropriately high level and that clinical negligence had occurred resulting in permanent neurological damage. The patients continue to suffer pain, hypersensitivity and parathesia in one leg, and one of them also has greatly reduced power.

It is my opinion that the anaesthetists concerned had been using inappropriately high spaces for some time but as long as they were performing only epidural analgesia no problem occurred. Since these cases, we have been very careful not to use too cephalad a space. If there is any doubt we feel a CSE is contraindicated and use epidural analgesia alone. Thankfully, we have had no further similar cases, although we continue to use combined spinal epidural analgesia for many of our obstetric patients.

H. Parry
Watford General Hospital,
Watford WD18 0HB, UK

Epidural failures and trainees' performance

We read with interest the paper attempting to correlate obstetric epidural performance with psychomotor aptitude (Dashfield *et al. Anaesthesia* 2000; **55:** 744–9) and were shocked by the quoted 'failure rate' of 1 in 5 epidurals, which seemed to improve little with time. This seems much higher than the failure rate of trainees at Kingston Hospital, which for the last seven novices has been two failures for the first 10 epidurals, then 1.7, 0.85, 0.4 and 0.5 for each subsequent 10. We therefore wish to raise several points.

We judge success of epidural insertion as the establishment of good analgesia without technical problems. We question the validity of using success or failure of the epidural over the duration of labour as a measure of the trainee's performance of epidural anaesthesia. The authors hinted that good analgesia throughout labour was perhaps too 'stringent' a measure, and

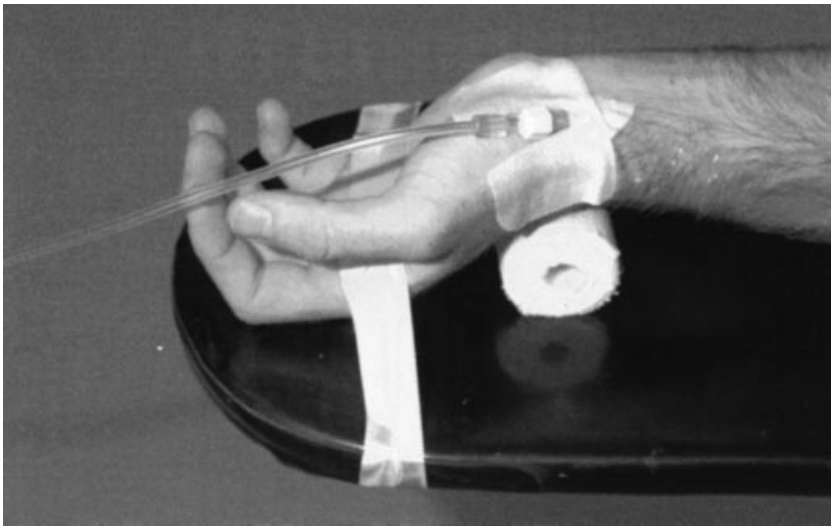

Figure 14 .

we feel that this is an important limitation of the study. It is a harsh measure and makes no allowance for the successfully inserted epidural that works well initially but subsequently provides insufficient analgesia. Audit data can shed some light on the size of this problem.

At Kingston Hospital over a 27-month period to June 2000, there were 3625 epidurals performed, of which 95.8% produced good initial analgesia. Of those that failed to achieve good initial analgesia, 35% were inserted too late or abandoned because of an urge to push, 50% had persistent perineal, one-sided or back pain, and possible failed epidural identification was responsible for 15%.

Of the 3473 women who achieved good analgesia at the initial insertion, 17.6% subsequently had analgesia problems. Expressed as a proportion of all epidurals inserted, the problems were: one-sided pain 6.8%, perineal pain 6.6%, catheter migration out of space 1.8%, other causes of abdominal pain 1.2% and back pain 1.1%. This population, if not correctly managed, would be unlikely to re-establish analgesia, and would count in the present study as failed epidurals despite the fact that a successful epidural had been performed. It is interesting to note that this figure of almost 20% is close to the failure rate of 1 in 5 found in this study. This begs the question about what contribution late failures made to the overall failure rate in the current study.

Postnatal assessment of all women with successfully established epidural analgesia revealed 6.9% who rated their analgesia as poor. Internal review of the reasons for these failures has shown withholding top ups in late labour and failure to get an anaesthetist to review a failing epidural to be the main causes. Clearly, it would be unfair to attribute these situations outlined above, where analgesia was initially good, as failures due to lack of technical proficiency.

The mean failure rate used in this study hides some interesting observations. The data in Table 2 reveal that three trainees performed worse in their final 20 epidurals than in their first 20, one trainee performed the same, two trainees performed marginally better, and the four remaining trainees who improved a lot seem to be responsible for the observed improvement in mean failure rate. We believe the fact that useful improvement was seen in less than half the trainees studied is either evidence of inadequate supervision (highly unlikely) or evidence that the measure of success or failure of an epidural over the course of a labour is not valid for assessing competence of insertion.

We would be very interested to learn the rate at which performance improved when measured against the establishment of analgesia, and any relationship between the psychomotor tests and this measure, which we feel is a far more relevant test of operator competence.

P. Dill-Russell
R. G. W. Stacey
Kingston Hospital,
Kingston-upon-Thames,
Surrey KT2 7QB, UK

A reply

Thank you for the interest in our study. As we pointed out in our paper, good analgesia throughout labour was a 'stringent' measure of success or failure of an epidural. The reason we chose this outcome measure was that it had previously been used and accepted in a peer reviewed journal [1]. We based the number of epidural procedures we studied for each trainee on Kestin's finding that 47 epidural procedures were required before an acceptable success rate was achieved; thus our end point had to be the same.

We have re-examined our data and correlated psychomotor test scores against the success rate of establishing analgesia. The correlation between the means of the first 25 and 50 consecutive epidurals and a eye-hand co-ordination or information management task was also not significant, suggesting that late failures of epidural analgesia were less important than Dill-Russell and Stacey suggest.

The conclusions of our study remain the same; psychomotor abilities appear to be poor determinants of trainees' initial proficiency at obstetric epidural anaesthesia or of trainees' rates of progress during early obstetric training.

A. Dashfield
J. Coghill
J. Langton
Derriford Hospital,
Plymouth PL6 8DH, UK

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Should epidurals be avoided in acupunctured patients?

Although acupuncture is becoming accepted among pain clinicians, an *in situ* needle technique called 'Oki-bari' in Japanese is not known well. In this technique, fine metallic needles are used to puncture the skin and left in the subcutaneous tissues. We report a rare case in which a massive number of needles were found on chest X-ray; with the result that epidural cannulation was abandoned.

An 82-year-old woman was scheduled for distal gastrectomy. She had received acupuncture for her backache for 20 years. Her chest and abdominal X-rays revealed hundreds of fine metallic needles around the vertebrae (Fig. 15). We feared that performing an epidural in this situation might lead to complications, such as spinal cord injury and pneumothorax. We therefore abandoned placement of an epidural catheter and we chose only general anaesthesia. Her intra- and postoperative courses were uneventful.

There were several complications associated with acupuncture, such as pneumothorax [1], abscess [2], haematoma with subarachnoid haemorrhage [3] and spinal cord injury [4]. If we had attempted an epidural puncture,

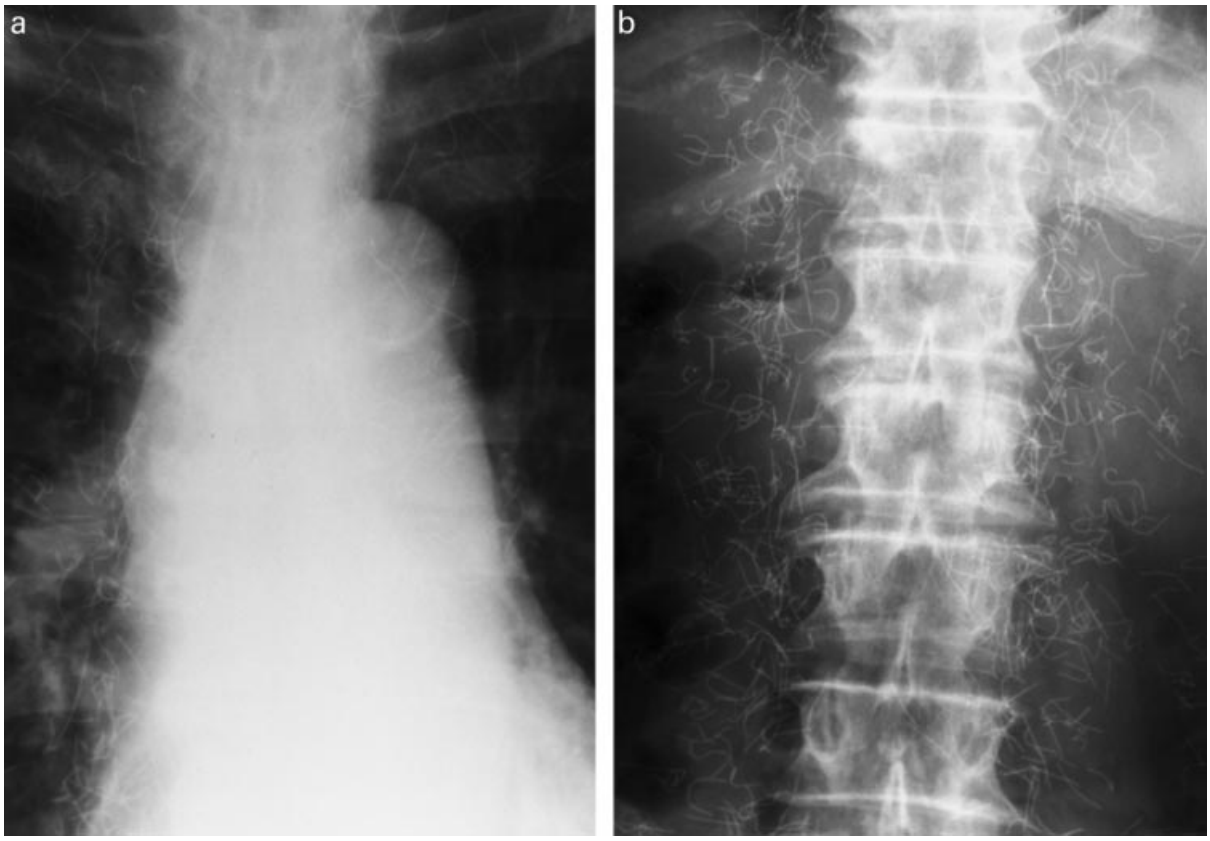

Figure 15 Chest (a) and abdominal (b) X-rays show hundreds of needles left around the vertebrae.

some of the needles in her back might have advanced and migrated deeply. In such a case, there is no guarantee of safety during the epidural procedure. We would like to stress the importance of pre-operative X-rays, especially in the patient who had received acupuncture to the back. If needles are left *in situ,* epidural puncture may be contraindicated.

K. Koga
A. Shigematsu
UOEH, Kitakyushu,
807-8555 Japan
E-mail: kkoga@med.uoeh-u.ac.jp

T. Noguchi
Y. Shiga
Chikuho Rosai Hospital,
820-088 Fukuoka, Japan
E-mail: nogutaka@dL.dion.ne.jp

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Total upper eyelid drop as an endpoint marker of peribulbar anaesthesia

I read with interest the recent article describing a novel approach to determine the optimal volume of local anaesthetic solution to produce satisfactory operating conditions for cataract surgery (Frow *et al. Anaesthesia* 2000; **55:** 750–5). In cases of peribulbar and subtenon anaesthesia, proptosis and lid fullness associated with the sensation of a full orbit have been used as end point markers for the injectate volume [1]. However, publication of any study reporting use of an objective criterion instead of a subjective one would be well received, especially by trainee anaesthetists. I would like to make the following comments.

Though complete ocular akinesia is not considered to be absolutely necessary for cataract surgery [2], it is used as the main index of the quality of anaesthesia such that supplemental injections are given to secure maximum eye immobility. In the present study, the reported rate of supplemental injection (10.2%) is similar to that observed by others [3, 4]. Hence, this technique does not seem to offer any additional

advantage with regard to supplemental injection.

It is a commonly observed fact that intra-ocular pressure rises acutely after injection and subsequently tends to decrease towards the pre-injection level [5, 6]. The authors report a mean rise of 6.9 mmHg after injection of a mean anaesthetic volume of 9.1 ml. Several authors who administered predetermined volumes of injectate have reported average rises of intra-ocular pressure of similar magnitude [7, 8].

The authors have emphasised the fact that in procedures involving injection of fixed volumes of local anaesthetic, the injected volume may be excessive for a particular patient and may dangerously increase the intra-ocular pressure. Although there is a tendency for transient intra-ocular pressure to rise with increase in injectate volume, no direct relationship has been reported. Moreover, the fears that a larger injection volume may cause a substantial increase in intra-ocular pressure jeopardising the retinal blood supply and impairing the surgical field have not been confirmed [9].

Inclusion of a qualitative explanation of the reported negative correlation between the injectate volume and the rise in intra-ocular pressure would have enhanced the significance of the observed relationship.

S. Verma
Queen Charlotte Hospital,
London W6 0XG, UK

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A reply

We thank Dr Verma for taking an interest in our paper and giving us the opportunity to reply to the points raised.

She is correct in her observation that our technique confers no additional advantage in terms of the level of akinesia achieved. Our aim was not to describe a superior technique for peribulbar block in this regard, but one which is at least as good as other techniques employed.

Concerning the mean rise in intra-ocular pressure of 6.9 mmHg immediately after injection without the use of external ocular compression techniques, we felt that this did compare favourably with the bulk of the literature we reviewed [1], although we do acknowledge the two studies she has quoted.

Her comments on the intra-ocular pressure increases are correct in that no proven link has been shown to exist between transient intra-ocular pressure rises and volumes injected. However, pain on injection, triggering of the oculocardiac reflex and ischaemic optic neuropathy have been linked to this by others [2–4].

The last point raised concerning the supposed lack of a qualitative explanation for the statistically significant negative correlation between injectate volume and intra-ocular pressure rise is invalid since a possible explanation was included in the discussion, based on our clinical experience. We do, however, feel this does warrant further attention to assess whether statistical significance can be linked to clinical significance and see if our result can be duplicated.

M. W. O. Frow
Central Middlesex Hospital,
London NW10 7NS, UK

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Anaesthetic menu: a step too far?

We read with interest the letter (Wood & Raghavan. *Anaesthesia* 2000; **55:** 1039)

concerning soya bean oil in propofol and could not understand their generalisation about genetically modified substances in propofol.

While it may be true that some of the soya bean oil source may have a genetically modified source, to brand propofol as genetically modified is stretching things a bit far in our opinion. While one can offer an alternative induction agent to most anaesthetic patients, it may be difficult to find a suitable short-acting sedative with a similar pharmacological profile in the case of intensive care use, where propofol is the first choice sedative for short-term sedation. What about the countless number of patients who have received propofol over the last 6 years, without consulting them? Taken a step further, what about the origins of soya bean oil in other lipid-based solutions such as diazemuls and intravenous lipid emulsions (TPN)? What about their alternatives? Imagine yourself offering (or being forced to offer!) this information and trying to suggest an alternative to diazemuls to the parents of a fitting child!

In this increasingly litigious and paranoid world such generalisation without a sound scientific basis can only make matters worse.

S. Dhileepan
A. Patel
Kingston Hospital,
Surrey KT2 1QB, UK

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A reply

The medical profession is frequently accused of arrogance and of withholding information regarding medical treatments and drugs from the public on the basis that doctors know best.

Conclusions on the safety of genetically modified products have yet to be reached and as yet there is no proven 'sound scientific basis' on this issue. However, there has been enough concern regarding the potential effects on health to warrant the introduction of legislation on labelling in September 1998. The safety of genetically modified products has been questioned so it would seem reasonable to exercise some caution in their use from now on.

Until firm conclusions are reached, perhaps it would be wiser for manufacturers to use soya bean oil in their products that is not from a genetically modified source as this would be quite possible. If the drugs were labelled accordingly, we would then be able to give accurate information to patients when required regarding the origins of the drugs we are using.

If in the future genetically modified substances are found to be harmful, an open approach to informing patients wherever possible now would surely prevent accusations of arrogance and the desire to litigate in the future.

C. C. Wood
V. Raghavan
Mayday University Hospital,
Thornton Heath CR7 7YE, UK

Physical incompatibility between atracurium and intravenous diclofenac

I should like to report the finding of physical imcompatibiliy between atracurium bresylate (Tracrium, Glaxo-Wellcome) and intravenous diclofenac sodium (Voltarol, Geigy).

Voltarol ampoules contain diclofenac sodium, mannitol, sodium metabisulphite (E223), benzyl alcohol, propylene glycol, sodium hydroxide and water, whilst atracurium exists solely in aqueous solution.

Inadvertent drawing up of intravenous diclofenac into a syringe previously used to administer atracurium bresylate results in the formation of a white precipitate.

Similarly, at room temperature, injection of atracurium bresylate via a three-way tap into the line of a giving set administering diclofenac sodium (75 mg/3 ml in 100 ml normal saline) also results in the formation of this white precipitate, regardless of whether normal saline is buffered with 0.5 ml of 8.4% sodium bicarbonate as suggested by the manufacturer's data sheet. This is a hitherto unreported reaction between two commonly used intravenous drugs. Whether the precipitate is harmful if it enters the systemic circulation is unclear.

G. Ackland
Barnet General Hospital, UK

Incidence of anaphylaxis under anaesthesia

The recent letter rightly pointed out the need for current UK epidemiological data on the incidence and outcome of anaphylactic and anaphylactoid reactions in patients under local and general anaesthesia (Watkins *et al. Anaesthesia* 2000; **55**: 1127–8). It is no longer satisfactory to extrapolate from data derived from publications in other countries, though comparison is of great interest. UK data need to be collected, collated and published as part of audit, possibly combining sources from the Committee on Safety of Medicines yellow card system, the Royal College of Anaesthetists Critical Incident System with the laboratory results and interpretation, NADRAS (National Adverse Drug Reaction Advisory Service). There is currently no feedback for clinicians though drug-specific information can be obtained from the Committee on Safety of Medicines (CSM).

In this hospital, there have been at least six major anaphylactic reactions in the past $2\frac{1}{2}$ years all confirmed with elevated blood tryptase levels, four with positive skin prick test positive. Four presented with sudden onset of pulselessness and/or cyanosis, one with a bradycardia and three with severe bronchospasm. Five patients were under general anaesthesia and one under

subarachnoid block. One died and one has short-term memory loss. Two were for Caesarean section under general anaesthesia and one manual removal of placenta under spinal anaesthesia. Drugs implicated were succinylcholine (four), atracurium (one), amoxycillin with Clavulanic acid (one). Is this incidence in one DGH representative of the UK?

It needs to be reiterated that patients experiencing an anaphylactic or anaphylactoid reaction under anaesthesia present with a variety of signs [1, 2] and that these may not all appear at once [3]. Whittington and Fisher in a study of 555 patients state that 'cardiovascular manifestations are the most common feature, that any system may be the only system involved and that the full constellation of systems does not occur in every patient' [4]. A high index of suspicion is required even if only signs from one system are noted, for early diagnosis with prompt and thorough treatment. In 10% of cases cardiovascular collapse is the sole presenting feature [1].

Treatment in patients presenting, for example, with sudden onset of severe hypotension (which as Watkins *et al*. point out may coincide with the time of patient transfer to the operating theatre) needs to be commenced rapidly. Cardiac massage will be of limited effect without immediate intravenous fluids and drug therapy. General and axial local anaesthesia will contribute to the effects of vasodilatation seen in anaphylaxis. In a recent report [5], 8 litres of crystalloid were administered over 2 h together, sequentially, with ephedrine, epinephrine, phenylephrine and metaraminol to a patient experiencing an anaphylactic reaction to rocuronium, in order to maintain the CVP > 0. Epinephrine, which is the basis of drug treatment for awake patients exhibiting the symptoms and signs of an anaphylactic reaction, may for some anaesthetised patients need to be supplemented by an alpha constrictor [6, 7].

The Association of Anaesthetists has recently announced that it will review their current report [1] and guidelines [8] on Suspected Anaphylactic Reactions Associated with Anaesthesia (Continuing Medical Education Day 7/9/2000). What is also needed, as Watkins *et al*. suggest, is a centre to co-ordinate all records of suspected anaphylactic and anaphylactoid reactions, together with laboratory and skin test results with a view to regular publication of results. Meanwhile, the College Audit Representative in each department might in 2001 collect data on all anaphylactic reactions observed under anaesthesia in their department for future reference.

R. M. Foxell
West Dorset General Hospital,
Dorchester DT1 2JY, UK

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How should we manage the electronically tagged patient?

A 25-year-old man recently presented as a day case for an elective plastic surgical procedure to a previous hand laceration. Medically he was a fit ASA I patient but he informed staff that he was wearing an electronic tagging device. Since this was due to be removed in approximately 1 month, it was decided to postpone the operation until after this date as little was known about these devices and possible complications.

Electronic monitoring is a system of home detention for offenders being brought into effect in the light of an ever-increasing prison population. An electronic tag radio frequency 'transmitter' the size of a wristwatch is worn around the ankle or wrist. This is the 'Personal Identification Device'. It is electronically linked to a receiver connected to the phone socket in the place of curfew. The continuous transmission of the tag is picked up by the receiver and checked intermittently by a central monitoring centre that dials the client's phone. The system therefore cannot 'track' a client but can detect whether a person is within range of the receiving unit at any time.

It is well known that interference with implanted medical devices such as pacemakers [1] and implantable defibrillators [2] can occur on exposure to magnetic fields emitted by electronic article surveillance systems (shoplifting gates) whose radiation can include radiofrequency, magnetic and pulsed electromagnetic fields. There is also evidence of interference by mobile communication devices in close proximity to medical devices [3], and cellular phones have been banned from operating theatres and intensive care units in the past.

The Home Office Electronic Monitoring Department states that

Figure 16

electronic monitoring equipment is not known to affect products such as pacemakers or communication systems. However, the Personal Identification Device is a radiofrequency transmitter and therefore could contribute to radiofrequency noise and possibly interfere with sophisticated electronic equipment, although there are no reports of this happening. Offenders are advised to contact the monitoring contractor prior to a hospital admission for removal of the equipment. Certification from medical staff is normally required to account for any curfew hours that have been missed. Once the offender returns home, officers would visit to re-install the personal identification device. In an emergency, it would be acceptable for the medical staff to cut the strap with a normal pair of scissors and the Control Centre would automatically record this.

Our patient should probably have contacted his monitoring department to have the device removed before the scheduled surgery but there was obviously some communication breakdown. As medical staff, we were also at fault due to a complete lack of knowledge of these devices but acted as we thought best in the circumstances.

I hope this information will be of interest to anaesthetists and medical personnel, allowing better management of this group of patients in the future. I am grateful to Stephen Crunkhorn at the Home Office for his help and advice.

J. Tye
University Hospital Birmingham NHS Trust,
Birmingham, UK

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An unusual method of diagnosing postoperative sore throat

A 32-year-old engineer presented for elective arthroscopy on a weekend. He was assessed pre-operatively and was noted to have a Mallampati 1 airway and was generally fit and well. He elected to receive a general anaesthetic. After induction with propofol, a size 5 laryngeal mask was placed uneventfully using the standard technique.

The operative course was uneventful and, immediately after the surgery, the patient was comfortable and did not complain of difficulty swallowing or a sore throat. However, 2 days after the surgery, he telephoned the anaesthetic department complaining that he had a sore throat that had not improved over the weekend and that 'the floppy thing at the back of my throat has turned yellow!' Despite his graphic description and worries, he was not keen to brave the Seattle traffic and come to the department. Instead, he reasoned he could take some pictures with his newly acquired digital camera and e-mail them to us. After a few minutes, we received the fruits of his labours. It appeared from the pictures (Fig. 16) that he had an ischaemic uvular. Over the next few days (and a series of pictures) the uvular demarcated and separated.

Uvular trauma has been reported after general anaesthesia with laryngeal masks [1]. The necrosis that our patient experienced presumably occurred as a result of compression of the uvular by the tip of the laryngeal mask being folded over, and remains an unusual complication. More unusual still was the method of diagnosis. Yet another useful use of e-mail!

M. Sanders
1722 NE 56th Street,
Seattle, WA 98105, USA
E-mail: mksander@u.washington.edu

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