Correspondence

Noise in the MRI scanner

We welcome the paper by McBrien and colleagues (McBrien *et al*. *Anaesthesia* 2000; **55:** 737–43) in which they highlight concern over the exposure to occupational hazards such as magnetic fields, unscavenged anaesthetic gases and noise. We note with interest the attitude of anaesthetists towards their own personal safety, in particular, the group of 22 anaesthetists who 'always' or 'frequently' remained in the scanning room. Of this group, 19 of 22 identified no potential for harm from either magnetic field or acoustic noise, and none of the 22 identified acoustic noise specifically as potentially harmful. The authors comprehensively discuss the problem of occupational noise exposure and refer to regulations contained within the Health and Safety Executive (HSE) Noise at Work Regulations [1] requiring employers to take specific action to reduce the noise exposure of employees. Similarly, it is our duty as anaesthetists to ensure that we take action to minimise the risk of acoustic damage to our anaesthetised patients. This is perhaps easy to forget amongst the range of problems we instinctively recognise as unique to anaesthesia for MRI and are discussed in depth in this paper. However, if only 2 of 46 units (Table 7) identify the potential harm from noise with respect to their own personal safety, is this mirrored by a similar attitude towards the acoustic safety of their patients?

In 1989, Hurwitz *et al*. [2] measured noise in MRI systems from 0.35 to 1.5 Tesla. The measured noise ranged from 82 to 93 dB on an A-weighted scale. They concluded that the noise levels, although annoying, posed minimal threat of permanent hearing loss. However, with the trend towards stronger magnets and 'noisier' sequences, higher noise levels have been measured [3, 4]. Shellock *et al*. [5] measured the six 'worse case' sequences and found levels of 103 dB at the centre of an MRI using a gradient-echo pulse sequence. This figure, according to HSE regulations, would allow a maximum daily exposure of 15–30 min.

The acoustic noise can be a tapping, knocking or chirping and is produced by rapid alteration of currents within the gradient coils. The noise can frighten children (hence the need for sedation or general anaesthesia), can make existing tinnitus worse and can lead to temporary hearing loss. In a study of 14 patients without acoustic protection and 10 with acoustic protection, six patients (43%) of the group without protection suffered temporary hearing loss whilst only one in the group with protection suffered a temporary hearing loss [6]. It is worth noting that a maximum average daily exposure of 90 dBA is designed to protect only 85% of the exposed population from significant hearing loss [2]. Noise reduction is urged by the HSE to all employers and employees. The following active and passive noise reduction techniques may be employed.

Active: computer-generated low-frequency pulsatile sound waves that are at 180° out of phase with the anticipated noise. This can offer a reduction of approximately 10 dB through headsets.

Passive: foam earplugs, if sited correctly, may reduce a continuous non-impulsive noise of 92 dB to 63 dB (product data).

A snapshot survey of noise ablation techniques used in 20 MRI suites in the UK where general anaesthesia was provided for MRI is shown in Table 1. It is reassuring that while only two of 46 units identified potential harm to themselves from acoustic noise, in our small study only three out of 20 units (15%) did not use a noise reduction technique. We do, however, urge all anaesthetists involved in providing anaesthesia for MRI to be aware of the acoustic risk to anaesthetised patients, in particular those having prolonged or repeated scans, those with existing acoustic damage and those receiving aminoglycoside antibiotics.

We are currently attempting to be more scientific in our own approach to acoustic protection by involving our hospital's Health and Safety Department in measuring the noise emitted by our 1.5-Tesla MRI and the effectiveness of various noise ablation techniques. We intend to submit the results for publication in due course.

Finally, we endorse the comments of the authors of this paper with regard to the provision of recommendations for standards of anaesthesia for MRI from such a body as the Association of Anaesthetists.

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Table 1 Results of a survey of noise ablation techniques used in 20 MRI Units in the UK.

Noise ablation technique	No. of units (n = 20)
Nothing	3
Earplugs only	7
Foam packing around the head	5
Earplugs and foam packing	1
Construction ear mufflers (as available from DIY warehouses)	3
Home made wooden head box (insulated with foam known as 'the kennel')	1

MR Imaging. *Radiology* 1988; **169**: 539–40.

7 Goldman AM, Gossman WE, Friedlander PC. Reduction of sound levels with antinoise in MRI Imaging. *Radiology* 1989; **173**: 549–50.

Date-expired nitrous oxide cylinders

I was intrigued by Dr Ranasinghe's letter (Ranasinghe. *Anaesthesia* 2000; **55:** 819–20). What criteria determine the shelf life of a cylinder of nitrous oxide, and on what scientific basis? I ask because in the *Journal of Dental Science* 1877; **XX:** 662, Dr J. Ottley Atkinson reported having administered nitrous oxide on separate occasions to two patients, from Coxeter's 100-gallon cylinders 3 years and 7 days and 3 years and 4 months old, and found the effects satisfactory in every way. He commented that this spoke much for the retaining power of Messrs Coxeter's bottles. It is interesting that even in those very early days, cylinders were labelled with the filling date, one of which is quoted.

Twenty years later (*Journal of Dental Science* 1897; **XL:** 922) there was a report copied from an American journal, *Dental Cosmos,* of an SS White cylinder 19 years old, the contents of which worked perfectly. Considering that the dentists concerned were obviously very occasional anaesthetists, and that the nitrous oxide in the cylinders of those dates was much more likely to contain contaminants than now, one does wonder.

Certainly contamination of nitrous oxide has been a problem in the past. Visitors to the current exhibition in the Association's Museum will see that between 1927 and 1939 every cylinder of nitrous oxide supplied by BOC had a certificate of the purity of its contents attached. So my question is, once nitrous oxide of the requisite purity is in the cylinder, what goes wrong with it, and how long does it take to become unfit for use?

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Failure of Datex AS/3 anaesthesia delivery unit

We report the failure of a Datex AS/3 anaesthesia delivery unit. An ASA 1 patient was having a general anaesthetic for an orthopaedic manipulation under anaesthesia; surgery was progressing uneventfully. Suddenly, the oxygen and nitrous oxide electronic flow meters, isoflurane agent monitor and inspired oxygen concentration monitor screen went blank. An audible alarm sounded with a caution symbol displaying 'Fresh gas unit failure'. All other physiological parameters being monitored (heart rate, ECG, oxygen saturation, non-invasive blood pressure, end-tidal carbon dioxide) remained unchanged. The isoflurane and nitrous oxide were promptly turned off, reserve oxygen cylinder turned on, and the patient ventilated with what we thought was 100% oxygen. The oxygen cylinder gauge pressure indicated that the cylinder was full, gas was coming out of the fresh gas outlet, but with electronic flow meters (no rotameters) and no calculated oxygen concentration on the anaesthesia delivery unit screen, we were unsure of the exact composition of the fresh gas. In the meantime, a spare anaesthetic machine was brought into the operating theatre and the surgery completed using the replacement machine; the new machine's self-check procedure showed up no faults in the piped oxygen and nitrous oxide supplies. The patient never desaturated and made an uncomplicated recovery.

The AS/3 machine was promptly serviced and the problem rectified. Datex recommends calibration of its machines every 6 months. In our department, we calibrate our AS/3 units every 3 months and give them a full service once a year. This case illustrates that despite following the manufacturer's servicing guidelines, equipment failure does occur.

On discussing the problem with Datex, it appears that the likely problem was a leak in the vaporiser system. We had checked that the vaporiser cassette was correctly seated, but apparently leaks can still occur. This would mean that the oxygen concentration set on the electronic flow meters would differ from the concentration measured at the patient end of the delivery system. If this occurs, the AS/3 automatically switches itself into safe mode where 4 $l.min^{-1}$ of oxygen are delivered to the patient and the rotameters and vaporiser taken out of the circuit. The problem with the AS/3 is that it does not tell you that it has switched into safe mode, hence our confusion.

Two aspects of this case are worth highlighting:

1 The absence of rotameters in some modern anaesthetic machines means that you have no way of measuring fresh gas flow if there is an electronic failure.

2 When new anaesthetic machines are purchased, a lot of emphasis is placed on getting to know how they work normally. Perhaps we should also be learning how to deal them when they are faulty. Datex gives tutorials to staff members when they deliver a new anaesthetic machine to a hospital. These tutorials emphasise troubleshooting, as well as normal function, of the new machine. Unfortunately, with regular

staff rotations, most anaesthetists will not receive this training.

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A reply

The Datex-Ohmeda AS/3 Anaesthesia System includes a delivery unit (ADU), an anaesthesia monitor (AM) and an optional anaesthesia record keeper (ARK). The ADU must always be used together with an anaesthesia monitor and the design philosophy and risk analysis are based on the fact that the ADU and AM are totally independent modules of the system. Early this year we identified a reliability problem in the ADU fresh gas control unit (FGC). This problem could lead to a 'Fresh gas unit failure' and further put the FGC into a safe state. The reported problem from Medway Maritime Hospital is indicated to be of this kind. This problem was identified to the FGC software and upgraded software has been designed and implemented in all units with a corrective field action as from April this year.

In response to the two comments of Drs Aziz and Sanders:

1 The electronic measuring of fresh gas flows makes it possible to include a more advanced safety system of delivered flow composition. This design allows a hypoxic guard system in any O_2/N_2O/anaesthetic agent mixture (important when agents with high concentrations are used).

Our experience is that electronically displayed fresh gas flows show high reliability. In an event of display failure, it is most important to monitor and adjust the inspired gas concentrations, shown on the patient monitor. Especially when a low-flow rebreathing system is used, the fresh gas concentrations can differ significantly from the inspired gas concentrations.

2 We strongly support a regular training programme, including handling of emergency events and troubleshooting. This programme should be mandatory for all anaesthetists. A special troubleshooting section is included in the ADU users' reference manual.

Some further clarifications with reference to the letter from Drs Aziz and Sanders:

Anaesthetic agent and oxygen concentrations shown on the ADU display are set values. Monitored values are shown on the anaesthesia monitor (AM).

A suspected component failure in the FGC can lead to a 'Fresh gas unit failure' alarm and a safe state with N_2O and agent shut off. This is a safety system to eliminate any risk of delivery of too high N_2O or agent concentrations to the circuit. In such situations oxygen will continue to flow as set, and also air can be delivered if selected.

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Another case of anaesthetic machine failure

I should like to report another cause of anaesthetic machine failure. A patient was taken straight to the operating theatre from casualty, having presented with massive upper gastrointestinal haemorrhage. It had been decided that he required endoscopy and probable laparotomy. While a large-bore triple-lumen central venous line was being inserted in the left subclavian vein prior to induction of anaesthesia, the patient, whose head was turned to the left, complained of nausea. He was asked to turn his head to the right to keep the area clean, but suddenly vomited copious blood. This completely covered the anaesthetic machine (a Datex AS/3 ADU/AM). Anaesthesia was induced rapidly in order to protect the patient's airway. Invasive monitoring was then inserted, and it was found that it was not possible to obtain pressure readings because blood had entered the module housings causing short circuits. A portable monitor was obtained. It was then noticed that the electronic vaporiser was now malfunctioning and could not be used because of blood in the workings. The machine was changed while using an AMBU bag to ventilate the patient, and the laparotomy was completed uneventfully.

While it is difficult to blame the manufacturer for this failure, the consequences of this event need consideration. Cleaning the blood out of all areas that it reached required a 'strip down' of the machine, which took it out of service for several days. A spare machine was needed for this time.

On a personal note, one author is glad the patient's head was turned to the right, not the left.

The manufacturer has been informed.

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Expiratory dates on anaesthetic gas cylinders

I am writing further to my recent letter (Ranasinghe. *Anaesthesia* 2000; **55:** 819), which highlights the fact that gas expiry dates on cylinders attached to anaesthetic machines are not routinely checked as part of the 'cockpit drill'.

It is not possible to see the gas expiry date on a BOC cylinder (British Oxygen Company Gases) whilst it is attached to an anaesthetic machine. This is because BOC attaches the label carrying the expiry date to the side of the cylinder valve block and this is obscured by the anaesthetic machine pin-index yoke. Therefore, the only way to perform this check would be to remove each cylinder in turn and read the expiry date, which would be cumbersome.

Gas cylinders are currently supplied to our Hospital Trust by Linde Gas UK Ltd. The gas expiry date is marked in black on a white label placed on the cylinder shoulder, making it clearly visible, even with the cylinder attached to the anaesthetic machine.

Irrespective of the gas supplier, all cylinders attached to anaesthetic machines should have gas expiry dates that are clearly visible.

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Unusual pulse oximetry waveform

On transferring a patient from the intensive care unit to the operating theatre for the closure of a compound femoral fracture, the following unusual pulse oximetry trace was obtained (Fig. 1). The patient was at the time connected to a Nellcor Oxisensor II D-25 pulse oximeter probe that was being monitored by means of a Propak 100 for the transfer. As it was the intention to continue monitoring this patient on the return to the intensive care by means of this same adhesive oximeter probe, the original probe was left in place whilst other monitoring was connected to the patient. Unknown to the two accompanying anaesthetists, a further probe was connected to the adjacent finger by the operating department assistant (Fig. 2). A Datex AS/3 was monitoring this. The patient's blankets obscured the adjacent position of these two saturation probes from the two anaesthetists. The abnormal trace was first thought to be due to a problem with the Propak 100 machine, but this was ruled out when the abnormal trace was duplicated on the Datex AS/3 monitor attached to the second saturation probe.

Mechanical artefacts either voluntary or involuntary such as shivering, tremors or convulsions are known to produce such abnormal traces, but as the patient had just received a paralysing dose of vecuronium this did not seem a possibility. The patient also showed no signs of shivering, tremors or convulsions.

As with all problems with saturation probe readings or traces, the first place to look to solve a problem is the probe itself. On moving the patient's blankets it was noticed that the two probes were on adjacent fingers. When the position of the non-adhesive probe was changed so as not to be on adjacent fingers, a normal trace was obtained.

It is common knowledge that visible light has the ability to interfere with the reading or trace of such probes and that this is far worse if the light source is pulsatile. This can occur with ambient light, especially if it is fluorescent [1, 2].

Our abnormal trace can be explained by the fact that the two emitting diodes when placed on adjacent fingers were acting as a source of extraneous light for each other and were being picked up by their two semiconductor photo detectors; hence, the duplicity of the abnormal trace on the two monitors. The assembly of the probe should be such that it is protected from light over the range of wavelengths to which the detector is sensitive, typically between 660 nm (red) and 940 nm (near infrared). This was clearly not the situation when placed on adjacent fingers.

Knowledge and awareness of this abnormal trace could help save valuable time in searching for erroneous causes of the trace at a time when diversion of attention away from the patient could be dangerous.

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A modification to the Codman™ Microsensor™ Skull Bolt Kit

The use of intracranial pressure monitoring in the management of closed head injuries is increasing in intensive care units [1]. The placement of intracranial pressure monitoring devices has traditionally been the preserve of the neurosurgeons. However, more intensivists are now undertaking this procedure. We wish to report a modification to the Codman™ Microsensor™ Skull Bolt Kit with the aim of improving the safety of use of the above kit by non-neurosurgeons. Therefore, this modification is relevant to anaesthetic practice in the field of intensive care.

The human skull has great variability in thickness when measured at different points. Variation also exists between different sexes, ages and racial groups

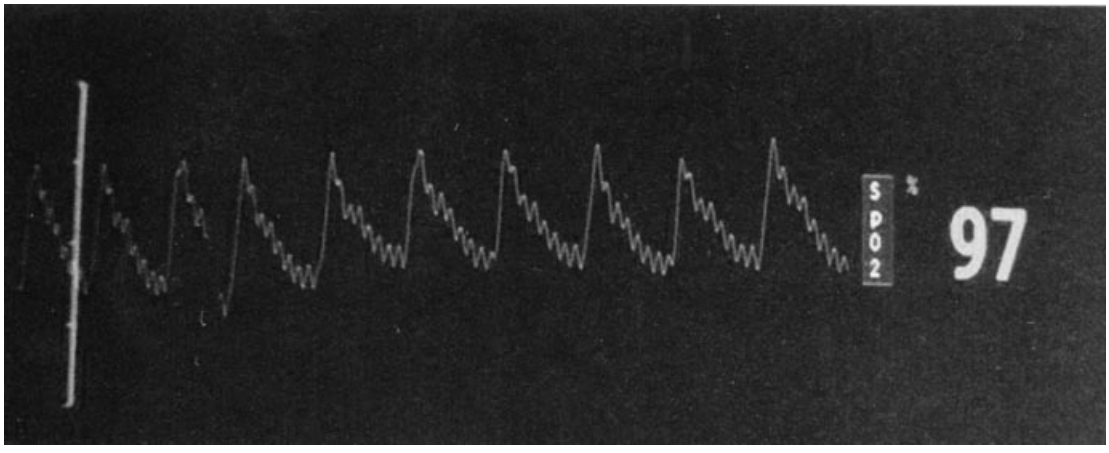

Figure 1

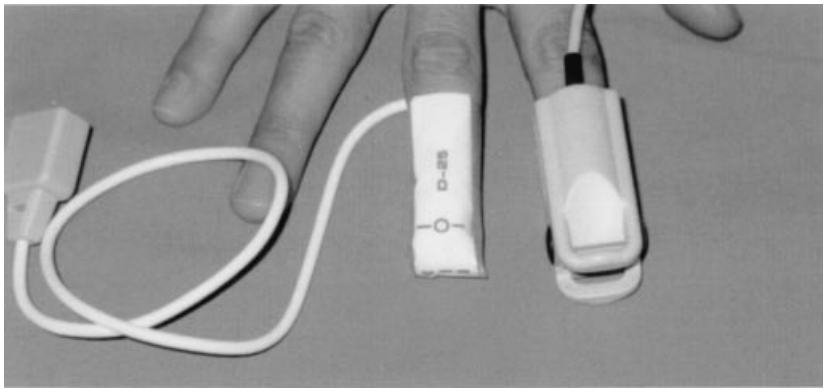

Figure 2

[2]. In a Korean study [3], the thickness of the frontal bone measured at a point approximating the point of insertion of an intracranial pressure monitor was 6.63 ± 1.77 mm.

In the Codman Microsensor Skull Bolt Kit we have noted that the grub screw on the depth guide collar does not easily tighten on to the drill bit if the depth guide collar is being set for a shallow depth. This is because the grub screw is trying to seat on the actual cutting portion of the drill, the first 15 mm (Fig. 3). The minimum depth that can be set where the grub screw is not mounted on the cutting portion of the drill using the Codman Microsensor Skull Bolt Kit is 14 mm. We therefore propose that the depth guide be elongated with the grub screw situated away from the cutting drill bit. This would surely make this piece of equipment easier to handle and would not prove expensive to modify. Two depth guides were manufactured to assess the change required. In the light of the Korean study [3] the longest depth guide would be most appropriate, allowing a minimum depth setting of 5 mm (Fig. 3). We have submitted our recommendation to Johnson & Johnson Medical Ltd, who manufacture the Codman Microsensor Skull Bolt Kit.

We would add the reminder that the use of modified equipment without the approval of the manufacturers transfers any product liability from the manufacturers to the medical personnel using the modified equipment. The modified equipment that we have made has not yet been used.

We would like to acknowledge the help of Mr I. M. Barker for engineering the new depth guides.

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Cricothyroidotomy comments

In describing modifications to a central venous cannulating needle to allow it to function as a needle for cricothyroidotomy,

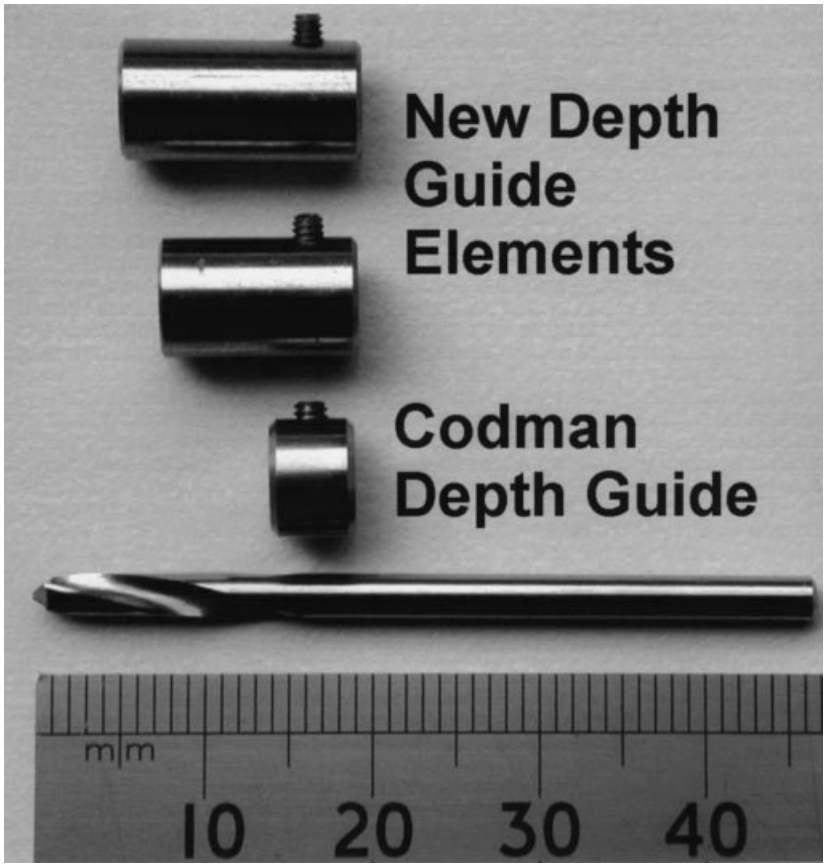

Figure 3 Orignal Codman drill bit and depth guide with new depth guide elements shown against millimetre scale.

Drs Dubey and Kumar (Dubey & Kumar. *Anaesthesia* 2000; **55:** 702–4) make points that deserve comment.

They mention that 'the side port of the needle works as a port for exhalation to facilitate carbon dioxide elimination.' Physics precludes this. Intratracheal pressure in the passive expiratory phase is less than 2.9 kPa (30 mmHg) and the diameter of the side port is too small to allow effective gas flow to contribute to carbon dioxide removal [1]. Safe expiration must therefore be accomplished through the patient's glottis.

Dubey and Kumar describe that the needle can function as a conduit for jet ventilation with an arrangement of needle – syringe barrel – cuffed tracheal tube 'connected to the anaesthetic machine to allow ventilation of the lungs using the flush valve as a jet injector.' They do not mention how the tracheal tube connects to the flush valve of the machine in a practical way. If connected to a machine with an interposed breathing system, sufficient driving pressure is not achieved [2]. The needle can simply be connected to the flush valve with a length of low-compliance oxygen tubing with some sort of control inserted along its length, such as a three-way tap. All connections must be robust enough to sustain pressures encountered in use. It is therefore important that this is previously prepared and immediately available.

The jet delivery system must connect directly to the flush valve of a suitable anaesthetic machine, which must be capable of generating the required pressure, and this depends on the limit of the overpressure valve [3]. This must be considered before approving or attempting to use a machine as an oxygen source for jet ventilation. If the pressure limit is too low, ventilation is ineffective as regards carbon dioxide elimination, and the system functions as an oxygen insufflator.

Despite these comments, we welcome Drs Dubey and Kumar's letter, which informs and reminds us of these important approaches to the difficult or desperate airway.

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Not all that wheezes is asthma

A 66-year-old woman was listed for a right total knee replacement. She had undergone uneventful general anaesthesia and tracheal intubation 1 year and 4 years previously and had also undergone a partial thyroidectomy over 20 years ago. She suffered from asthma for which she took three inhaled bronchodilators. Recent pulmonary function tests (with percentage predicted in parentheses) were FVC 2.3 l (106%), FEV, 2.11 l (118%) and PEFR 6.83 $1.min^{-1}$ (128%). They were unable to perform flow-volume loops due to patient difficulty.

On the morning of surgery, a chest X-ray was ordered because the surgical doctor felt that her chest sounded very wheezy (Fig. 4). However, the X-ray revealed significant tracheal narrowing, maximum at the level of T_2, with midline shift to the right. On listening to the woman describe how she has to crawl upstairs because she 'can't get her breath', it was clear that she had mild inspiratory stridor but no expiratory wheeze. Reviewing old X-rays revealed that the tracheal narrowing was present at least 5 years previously. The bronchodilators were clearly of no benefit to this patient. She had attended the hospital for pulmonary function tests but, unfortunately, a respiratory physician had not reviewed her. The most alarming fact was that she had undergone tracheal intubation on two recent occasions with no documented problems.

Obstructive lesions of the trachea typically produce dyspnoea and wheezing, and do not produce symptoms until the diameter of the trachea is reduced to less than 0.5 cm. The flow-volume loop is a reliable diagnostic test for differentiating fixed from variable obstruction and intrathoracic from extrathoracic obstruction. From the evidence available, it would appear that our patient probably had a fixed extrathoracic obstruction but with a symptomatic variable component. In this case, flow during inspiration is reduced (resulting from the Venturi effect) whilst expiratory flow is better maintained because the increase in airway pressure splints the trachea.

In the end, we decided to perform the surgery under a combined spinal–epidural technique. No problems were encountered. Unfortunately, despite numerous verbal and written reminders, the patient was discharged home without any attempt to investigate her neck problems. As highlighted by Rajesh *et al.* [1], she would clearly benefit from CT imaging with reconstruction to diagnose and plan any further management.

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Reference

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Swelling in the hypopharynx as a cause of difficult tracheal intubation

The laryngeal mask or the intubating laryngeal mask has a potential role in patients with difficult airways; nevertheless, its placement or tracheal intubation through it may fail [1–3]. It is important to elucidate possible causes for failures to establish its role in patients with difficult airways. We report failed

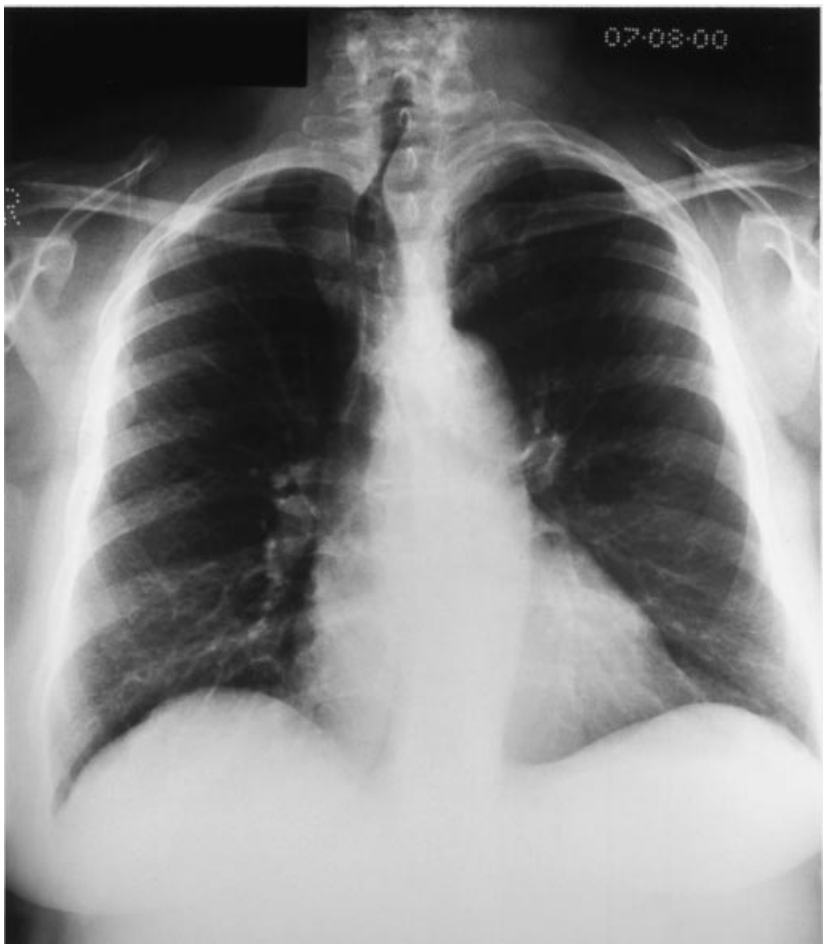

Figure 4

tracheal intubation through the intubating laryngeal mask in a patient in whom tracheal intubation using a laryngoscope or fibrescope was also difficult.

A 48-year-old woman with an unstable atlanto-axial joint was scheduled for posterior fixation of the cervical spine. Because of the unstable neck, we planned to intubate the trachea through the intubating laryngeal mask while manual in-line head and neck stabilisation was being applied. After conscious sedation was produced by midazolam 2 mg and fentanyl 50 μg, a size 4 intubating laryngeal mask was easily inserted with minimum discomfort to the patient. After inflation of the cuff with 15 ml of air, a clear airway was obtained. A 7.0-mm reinforced tracheal tube (Mallinckrodt, Athlone, Ireland) was passed through the mask so that the tip of the tube had just lifted the 'epiglottis elevating bar' at the aperture of the mask. A fibreoptic bronchoscope (diameter: 5 mm) was passed through these. Although it was possible to see the vocal cords, the arytenoid cartilages were tilted toward the glottis. The fibrescope was inserted into the trachea, but it was not possible to advance the tube over it. Since we felt that the distal part of the laryngeal mask might be folded over on itself, widening the hypopharyngeal space and thus tilting the laryngeal inlet, the mask was withdrawn for a few centimetres; however, there was no sign of folding over. The cuff was deflated completely to re-advance the mask into the hypopharynx, when we found that the arytenoids remained tilted anteriorly. Close examination of a lateral X-ray of the neck taken pre-operatively revealed swelling in the posterior wall of the hypopharynx, partially obstructing the laryngeal inlet (Fig. 5). The laryngeal mask was removed and laryngoscopy was attempted while the patient's head and neck were kept stabilised. Tracheal intubation was accomplished with great difficulty by the combined use of the laryngoscope and fibrescope.

Brain and colleagues have suggested that tracheal intubation through the intubating laryngeal mask may be easier in patients with difficult airways compared with those with normal airways [4]. However, the case described here indicates that this is not always so.

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Retrolaryngeal extension of goitre in a morbidly obese patient leading to a difficult airway

Patients with a large goitre are more likely to present difficulty at intubation [1]. We report a case of a difficult airway in a 68-year-old, short-statured, hypertensive, morbidly obese patient with a large goitre scheduled for elective

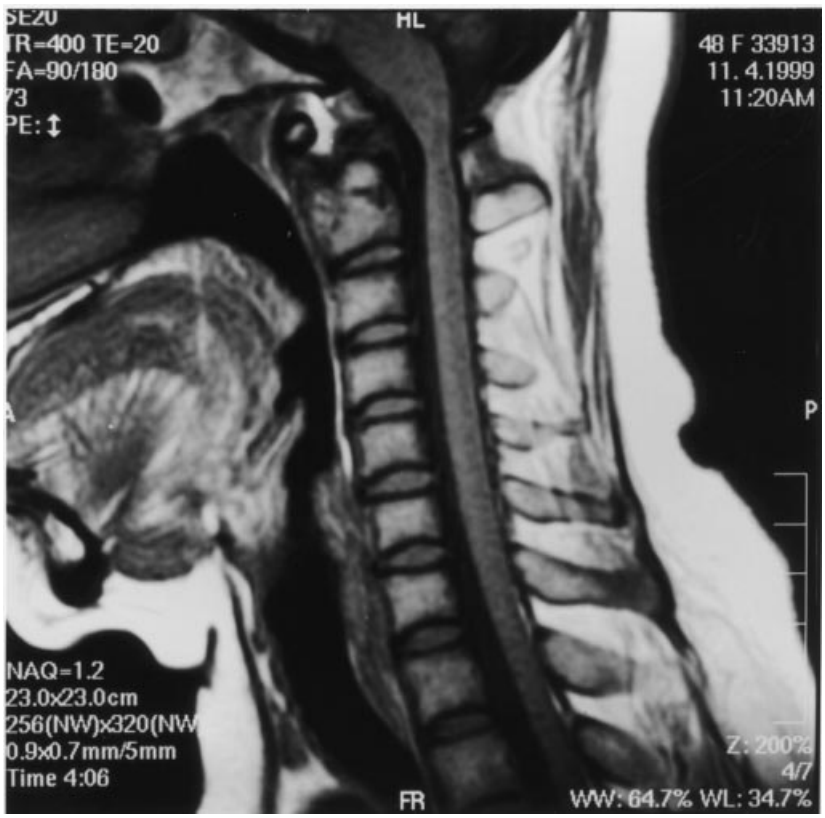

Figure 5 Swelling in the posterior wall of the hypopharynx, pushing the arytenoid cartilages toward the glottis and making tracheal intubation through the intubating laryngeal mask (and using a laryngoscope and fibrescope) difficult.

exploratory laparotomy for a malignant ovarian tumour. The goitre was progressively decreasing in size during the last 5 years without any treatment. She was dyspnoeic at rest and her daughter reported loud snoring during sleep. There was no history of nocturnal sleep awakenings, voice change, stridor or dysphagia. Pemberton manoeuvre [2] revealed absent superior vena cava obstruction. A cardiologist and chest physician ruled out any cardiac or pulmonary cause of dyspnoea. Pulmonary function test revealed mild airflow restriction and the ECG showed left ventricular hypertrophy. Evaluation of her airway showed a short neck with a large goitre, minimal neck extension, bucked teeth, Mallampati class III airway and a large tongue. A postero-anterior chest radiograph revealed normal cardiac outline and lung parenchyma with tracheal deviation to the left. The lateral neck X-ray revealed complex upper airway anatomy (Fig. 6). The trachea was compressed and posteriorly displaced at the supraglottic level, and an apparent retropharyngeal mass pushed it anteriorly at the glottic and subglottic level. A CT scan of the neck and upper thorax showed the tracheal compression, deviation and narrowing at and below the glottis (Fig. 7). The thyroid mass was limited to the neck, and revealed retrolaryngeal extension. Airway cartilages were normal. An ENT consultant noted mobile vocal cords and an adequate glottic orifice on indirect laryngoscopy. Laboratory investigations including thyroid function tests were within normal limits.

Airway control was approached by awake flexible fibreoptic bronchoscopy (FFB). Arterial blood gas analysis on room air showed P_aO_2 10.6 kPa, P_aCO_2 4.3 kPa, pH 7.40, HCO_3 21mmol.l^{-1}, base deficit of 2 and O_2 saturation 93%. Following premedication with aspiration prophylaxis, glycopyrronium 0.4 mg was given intravenously an hour before the procedure and 2% lidocaine by nebulisation, the patient was pre-oxygenated in a 15° head-up position. Routine monitoring was applied. The tip of epiglottis was visualised on FFB, but the view of the cords was obscured by a shiny, reddish mass. The procedure was abandoned when it was not possible to negotiate the fibrescope around this mass. On direct laryngoscopy under propofol sedation, the subjective impression of the laryngoscopist was of a ball-shaped mass (3.5 × 3.0 cm) protruding from the anterior wall of the oesophagus into the hypopharynx, having no connection with its posterior wall. A 6.0-mm I.D. red rubber tracheal tube was passed blindly between the mass and epiglottis. Tube position was checked and anaesthesia induced. Further intra-operative and postoperative courses were uneventful.

Despite the fact that the location of the glottis with FFB is likely to be more difficult when the larynx is deviated [3], awake fibreoptic-guided tracheal intubation may be the safest technique in such situations [4]. Laryngeal mask airway and a gum elastic bougie were not considered because of the impossible glottic inlet conformity and the risk of unwarranted bleeding, respectively. Probably, the application of forward traction on direct laryngoscopy separated the mass and epiglottis, thus enabling us to intubate the trachea.

It is known that nearly all long-standing goitres become multinodular, produce most extreme thyroid enlargements and extension, and are frequently mistaken for neoplastic growth [5]. The pressure effect of the retrolaryngeal extension of the goitre in our patient might have led to erosion of the anterior oesophageal wall, while keeping the mucosa intact. This may well represent itself as a reddish, fragile mass with a smooth and shiny surface.

To conclude, FFB is not the answer to every airway problem and may be less than ideal in certain situations [6]. This case sends a clear message to all physicians involved in the management of the difficult airway that with all the innovative and newly available investigative modalities and instruments for airway control, we still have a long way to go in regard to its various features and problems.

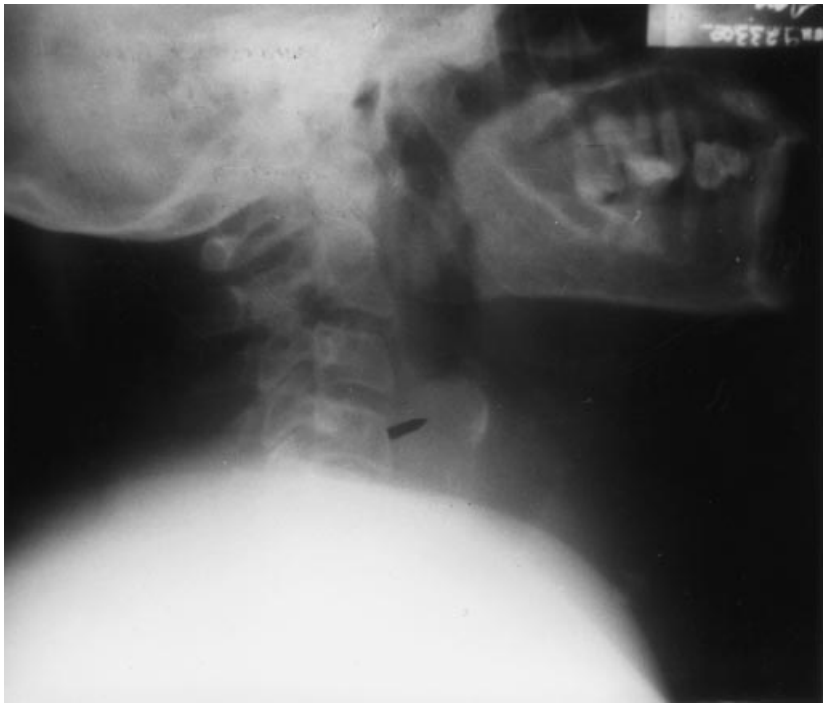

Figure 6 Lateral cervical radiograph showing an apparent retropharyngeal mass displacing the upper airway anteriorly at the level of the glottic inlet.

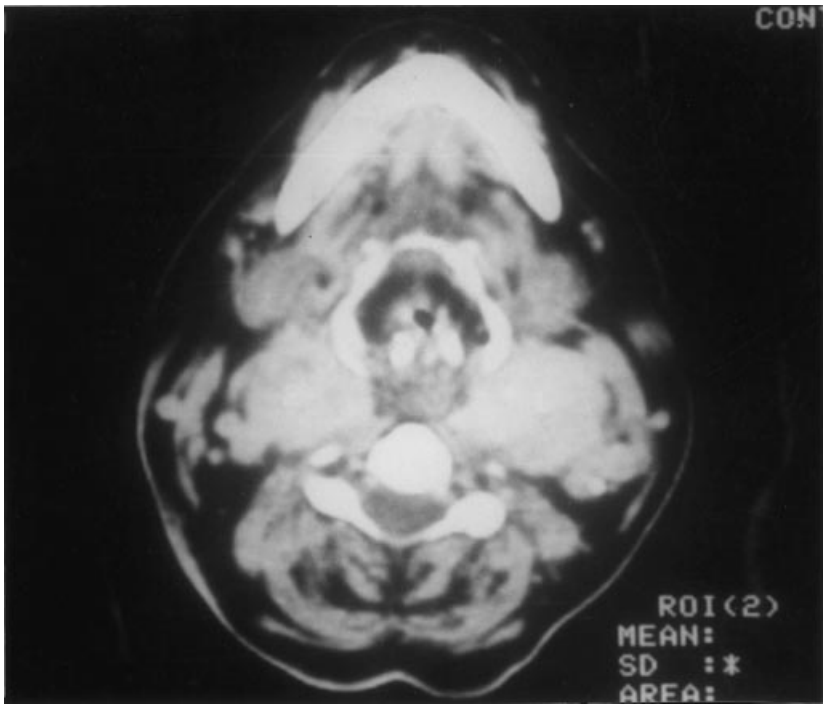

Figure 7 Non-enhanced axial computed tomogram section at the level of the glottis, showing a bilaterally enlarged thyroid gland insinuating between oesophagus and larynx. This led to compression and narrowing of the upper airway at the level of the glottis and subglottis.

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Acute respiratory failure secondary to severe hypokalaemia

A recent case report by Dunn *et al.* claimed to be the first reported case of acute, respiratory failure due to hypokalaemia requiring positive pressure ventilation [1]. We also have had a patient who developed acute respiratory failure requiring ventilation secondary to severe hypokalaemia.

A 57-year-old man with a 5-week history of deteriorating health and diarrhoea presented as an emergency with abdominal distension and shortness of breath. Prior to this episode, although a life-long smoker, he was active and had no previous chest problems. On examination,

he was distressed, peripherally cyanosed and his abdomen was grossly distended but soft. On admission, blood gas analysis showed a mixed picture of both metabolic alkalosis and respiratory acidosis: pH 7.42, P_{CO_2} 11.9 kPa, P_{O_2} 6.74 kPa, HCO_3 52.7 mmol.l^{-1}, BE + 27.5 on 100% oxygen. Serum electrolytes were potassium 1.26 mmol.l^{-1}, sodium 147 mmol.l^{-1} and chloride 87 mmol.l^{-1}. His ECG showed changes consistent with hypokalaemia. An abdominal X-ray showed a distended large bowel. A working diagnosis of sigmoid volvulus was made and his respiratory failure attributed to an exacerbation of chronic obstructive pulmonary disease (COPD).

A flatus tube was passed with a good result but his general condition and his respiratory function continued to deteriorate and he was admitted to the intensive care unit (ICU) for ventilation 3 h after arrival in hospital. Blood gases at that time were pH 7.1, P_{CO_2} 27.52 kPa, P_{O_2} 9.0 kPa, HCO_3 52.7 mmol.l^{-1}, BE + 24.1 mmol.l^{-1}.

He was resuscitated with a mixture of crystalloid and colloid intravenous infusions and initially required a small dose of epinephrine to maintain his blood pressure. He received 600 mmol of potassium over the following 24 h by which time the serum potassium had returned to the normal range. Following ventilation, the gas exchange was significantly improved and his respiratory acidosis corrected: pH 7.54, P_aCO_2 5.52 kPa, P_aO_2 12.22 kPa (F_iO_2 0.4) but a significant metabolic alkalosis persisted: BE + 13.6.

The patient continued to receive 200 mmol potassium daily. With this therapy, the metabolic alkalosis slowly resolved. He was extubated on the 4th day of admission. He remained a further 3 days on the intensive care unit. At discharge, his blood gases on 50% oxygen were: pH 7.43 P_{CO_2} 5.08 kPa, P_{O_2} 10.4 kPa, HCO_3 24.9 mmol.l^{-1}, BE 0.4. The patient's abdomen remained distended and he subsequently underwent a laparotomy and colostomy for a chronic sigmoid volvulus from which he made an unremarkable recovery.

We believe this man's respiratory failure was primarily due to hypokalaemic respiratory muscle weakness. The hypokalaemia was precipitated by his diarrhoea and may have been exacerbated by his lifestyle and diet. The combination of hypokalaemia and high plasma bicarbonate is more likely to have been due to potassium depletion, which is common, than due to a primary metabolic alkalosis, which is rare [2]. The metabolic respiratory muscle function and metabolic alkalosis both corrected following the administration of potassium. The original assumption that he had chronic respiratory failure with a compensatory alkalosis proved not to be correct as his blood gases returned to normal by the time of discharge from the ICU.

It is recognised that hypokalaemia can produce muscular weakness and respiratory insufficiency. Despite this widely held view, the case report of Dunn *et al.* appears to be the first that describes the use of positive pressure ventilation in such circumstances. We were also unable to identify any other reported cases. We were, however, able to find a case where negative pressure ventilation in an iron lung was used to treat respiratory failure precipitated by insulin therapy in the treatment of a patient in a diabetic coma. The patient was treated with potassium after which respiratory function improved. In this case, it was assumed, but not proven, that the respiratory failure was due to hypokalaemia [3].

Why has it not been reported previously? It may be that it is not appreciated that the hypokalaemia is the cause of respiratory failure and an alternative diagnosis made. In our case, respiratory failure was originally attributed to an exacerbation of COPD based upon a history of smoking and raised serum bicarbonate. Interestingly, the treatment given for this condition, furosemide and steroids, may have exacerbated the hypokalaemia. In Holler's case, the patient was moribund with severe diabetic ketoacidotic coma. Alternatively, it may require an additional factor, in our case a grossly distended abdomen, such that the muscle weakness is sufficient to precipitate respiratory failure.

Clearly, hypokalaemia may cause acute respiratory failure requiring mechanical ventilation. Conversely, hypokalaemic muscle weakness may be a contributory factor in other causes of respiratory failure so warrants prompt recognition and treatment in patients with incipient respiratory failure.

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Operative tumour handling and hyperkalaemia

Carvalho recently described a case of postoperative hyperkalaemia attributed to rapid blood transfusion [1]. The mechanism was contested by Horsey, but no alternative offered (Horsey. *Anaesthesia* 2000; **55:** 294). A possible cause suggested by Carvalho is operative tumour handling. We recently treated a patient for postoperative hyperkalaemia which we attributed to tumour handling.

The patient was a 70-year-old 96-kg male admitted to the intensive care unit (ICU) after an emergency splenectomy for intraperitoneal bleeding. He had a history of axillary nerve sheath sarcoma with secondary metastatic deposits in the liver, lungs and spleen. Whilst an inpatient undergoing investigations, he suddenly developed lower left chest pain, shortness of breath and dizziness. On examination he was hypotensive (70/45 mmHg) with a normal heart rate (72 beat.min^{-1}). Therapy included 500 ml of intravenous colloid, which raised his blood pressure back to his normal, and oxygen (6 l.min^{-1}) by face

mask. An arterial blood gas sample showed pH 7.34, P_aO_2 15.8 kPa, P_aCO_2 4.2 kPa, bicarbonate 17 mmol.l^{-1} and base excess − 7.5. Serum biochemistry revealed sodium 132 mmol.l^{-1}, potassium 4.1 mmol.l^{-1}, urea 4.9 mmol.l^{-1} and creatinine 79 μmol.l^{-1}. His haemoglobin had decreased from 10.2 g.dl^{-1} to 7.9 g.dl^{-1} over 6 days. Urgent abdominal ultrasound demonstrated an extensive collection of free fluid within the abdomen, which was found to be heavily bloodstained on aspiration. Findings at laparotomy included bleeding from a ruptured splenic metastasis and multiple friable intraperitoneal secondary deposits. He underwent splenectomy with debulking of peritoneal secondary deposits. Blood loss was estimated at 1950 ml, for which he received 2 l of 0.9% saline and three units (roughly 825 ml) of red cells over a period of 1.5 h. He remained haemodynamically stable throughout.

Postoperatively, he was transferred to the ICU, where he was stable, mechanically ventilated and producing adequate volumes of urine. Arterial blood gas sampling showed good gas exchange with a metabolic acidosis (base excess − 5.4) and a potassium of 7.8 mmol.l^{-1}. Laboratory analysis confirmed the hyperkalaemia of 7.5 mmol.l^{-1}, and showed normal sodium 134 mmol.l^{-1}, urea 6.3 mmol.l^{-1} and creatinine 135 μmol.l^{-1}. The hyperkalaemia was treated with intravenous 10% calcium gluconate (10 ml), two infusions of 50% dextrose (50 ml) each with 15 iu insulin, and per rectum calcium resonium (30 g). Serial potassium measurements showed a decrease to 6.4 mmol.l^{-1} after 6 h, then 4.7 mmol.l^{-1} 24 h after admission. Serum creatinine kinase, calcium, phosphate and urate were normal throughout. His renal function deteriorated over the first 48 h with peak values of urea 14 mmol.l^{-1} and creatinine 205 μmol.l^{-1}, subsequently returning to normal over the next 24 h. Extubation was delayed due to abdominal distension compromising respiratory function, but he was successfully extubated on the third day and discharged from the ICU on the fifth.

The reason for the hyperkalaemia remains unclear. He had not received succinylcholine, nor was the blood transfusion large or rapid enough to be a likely cause. He did show evidence of impairment of renal function, but insufficient to produce such profound hyperkalaemia. In addition, there was no history of previous, nor subsequent, electrolyte disturbances to suggest an endocrine cause. We postulated that manipulation of friable sarcomatous deposits may have released sufficient potassium to produce the hyperkalaemia. It is interesting to note that other reports of peri-operative hyperkalaemia have also followed resection of sarcomas [1–3]. This may simply be an association between vascular tumours where resection often leads to the requirement for rapid transfusion, or maybe that a subgroup of these tumours have the potential to cause sudden release of potassium when stimulated by drugs or manipulation.

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Anaphylaxis to rocuronium

We would like to report two cases of anaphylaxis to rocuronium bromide.

The first reaction occurred in a 66-year-old ASA III male smoker scheduled for mediastinoscopy for investigation of a left hilar mass. Previous general anaesthetics had been uneventful. Anaesthesia was induced with alfentanil 1 mg and propofol to a total dose of 200 mg. Following loss of eyelash reflex, rocuronium 30 mg was administered and manual ventilation commenced. Shortly after rocuronium administration, the patient developed bronchospasm, hypotension and tachycardia leading to electromechanical dissociation. The patient was resuscitated with epinephrine 0.3 mg given intravenously and intravenous fluids (1500 ml) and the total duration of circulatory arrest was approximately 2 min. Surgery was abandoned and, following the return of spontaneous circulation, the patient was transferred to the intensive care unit where he was extubated 1 h later. He made an uneventful recovery from this episode.

Blood and urine samples obtained at 1, 6 and 24 h after the reaction demonstrated elevations in mast cell tryptase and urinary methylhistamine in keeping with an anaphylactic/anaphylactoid reaction. Skin prick testing demonstrated a positive reaction to rocuronium (20 mm × 20 mm skin wheal) at a 1 : 10 dilution.

Our second case occurred in a 21-year-old ASA II female presenting for repair of an ostium secundum atrial septal defect. She had no other past medical history and had never had a general anaesthetic. Induction was achieved with remifentanil infusion at 1 μg.kg^{-1}.min^{-1} and a target-controlled infusion of propofol with a target concentration of 3 μg ml^{-1}. Following loss of consciousness, rocuronium 50 mg was administered. One minute later, the patient coughed and became difficult to ventilate manually, a narrow complex tachycardia of 165 beat.min^{-1} developed, the arterial pressure tracing became non-pulsatile and the patient developed florid generalised erythema with urticaria. The trachea was intubated and ventilation continued with 100% oxygen along with chest compressions. Epinephrine (total dose 2.5 mg), intravenous fluids (total volume of 2500 ml, a mixture of colloid and crystalloid) and incremental doses of norepinephrine up to 200 μg were given with no effect. Second-line management continued with hydrocortisone 500 mg, nebulised salbutamol 5 mg and ranitidine 50 mg.

In view of the lack of response to our initial therapy, we proceeded to sternotomy in order to perform internal cardiac massage. At no point prior to commencement of cardiopulmonary bypass (33 min after the arrest) was an external pulse palpable and the arterial line continued to register a non-pulsatile pressure. Surgery proceeded uneventfully and the patient was weaned from bypass with relative ease.

The postoperative period was complicated by coagulopathy, airway and facial oedema and haemothorax, all of which responded to appropriate therapy. She made a full recovery and was discharged 9 days later. Mast cell tryptase and urinary methylhistamine were elevated in keeping with an anaphylactic reaction. Skin prick testing to rocuronium 6 weeks later was strongly positive (30 mm × 30 mm) at a 1 : 10 dilution. In addition, skin prick testing for vecuronium produced a positive response (20 mm × 20 mm).

Both of these cases demonstrated grade IV anaphylaxis (Ring and Messmer Scale [1]) and also met Fisher and Baldo's criteria for an anaphylactic reaction [2]. We present these cases to highlight the fact that although rocuronium causes little or no direct histamine release, it can cause immunologically-mediated histamine release or anaphylaxis. The frequency with which these reactions are occurring is giving cause for concern. Neale *et al.* in their recent report suggest a crude incidence of reaction of approximately 1 : 3000 [3]. Based upon the number of reactions and the number of vials used, our 'hospital reaction rate' is approximately 1 : 6000. The manufacturers estimate that the incidence is approximately 1 : 200 000 worldwide (personal communication Organon-Teknika).

The obvious conclusion is that we do not have enough data to state definitively the incidence of reaction. In view of this, it is important that we monitor the use of this drug closely and ensure that any possible reactions are investigated appropriately and reported to the relevant agency to facilitate the gathering of accurate adverse event data.

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Isolated systolic hypertension and anaesthesia

Do we need to change the diagnostic criteria for hypertension in our practice? In two separate articles, Poulter [1] and Wilkinson [2] identified the importance of isolated systolic hypertension (ISH) as a significant predictor of future cardiac morbidity and mortality. The British Hypertension Society (BHS) also recommended, in recently published guidelines, drug therapy in people with sustained systolic blood pressure (SBP) greater than or equal to 160 mmHg, and the decision to treat sustained SBP between 140 and 159 mmHg to be made according to the presence or absence of other risk factors. In his article, Poulter referred to the Framingham data, which challenged the notion that diastolic blood pressure (DBP) alone is chiefly responsible for the cardiovascular sequelae of hypertension. Compared with DBP, SBP correlates more strongly with stroke, congestive heart failure, coronary artery disease, declining renal function and left ventricular hypertrophy. According to the latest BHS guidelines, target blood pressure in non-diabetic patients should be 140/85 mmHg, and in patients with diabetes $< 140/80$ mmHg.

In anaesthetic practice, however, the attitude towards cardiac disease in general, and hypertension in particular, has not changed over the last 50 years. Ischaemic heart disease and hypertension are considered to be associated with operative risk. Having said that, Goldman did not include hypertension as one of the independent risk factors in his famous pre-operative cardiac risk factors in non-cardiac surgery. Pre-operative management of hypertension has swung from one extreme to another. With the advent of antihypertensive drug therapy in the early 1950s and the introduction of potent antihypertensive drugs, such as methyl dopa and guanethidine, fears were expressed that cardiovascular homeostasis would be adversely affected during anaesthesia and surgery. Thus, it was commonly proposed that antihypertensive therapy should be withdrawn prior to elective surgery [3]. In fact, Goldman and Caldera [4] suggested that elective surgery in the absence of ideal antihypertensive control need not subject patients to an added clinical risk provided DBP is stable, not higher than 110 mmHg and perioperative blood pressures are closely monitored and treated to prevent hypertensive or hypotensive episodes.

The postponement of scheduled surgery because of incidentally discovered mild to moderate hypertension causes inconvenience to patients and doctors, as well as hospital management. Adequate blood pressure control may take weeks, if not months, to achieve. At the same time, the foregoing data, which suggest beyond doubt that ISH is a definitive risk factor for cardiac morbidity and mortality in the community, cannot be ignored. The million-pound question now is that should we, as perioperative physicians, take this as a gospel fact? Or should we wait for more prospective randomised trials of preoperative blood pressure control of hypertensive patients to establish the effect of anaesthesia and surgery on the outcome in these patients? The answer to the first question is that we should, since we live in the era of evidence-based medicine. Any inconvenience would certainly be justified if postponing surgical procedures until hypertension has been controlled decreases the risk of peri-operative complications. However, the answer is not as easy for the second question, as waiting may lead to more lives being put at risk.

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Microbial contamination of gum-elastic bougies

We read Cupitt's study on microbial contamination of gum elastic bougies (Cupitt. *Anaesthesia* 2000; **55:** 466–8) with interest and report a similar study with further evidence that bougies may be contaminated with potentially pathogenic bacteria.

Twenty-three single and multiple-use (autoclavable) gum elastic bougies from resuscitation trolleys on medical and surgical wards, the intensive care unit and operating theatres were investigated for bacterial contamination. The bougies were stored unwrapped either in upright uncovered holders attached to the side of the trolley or in one of the trolley drawers. After use, these bougies are usually washed with soap and water and dried before returning them to the trolley. The bougie tip was sampled by rubbing a moistened sterile swab across it and directly inoculating Columbia agar supplemented with horse blood. After incubation for 48 h in a carbon-dioxide-enriched atmosphere, the plates were examined and the number of bacterial colonies recorded as colony-forming units (cfu). Standard microbiological techniques were used to identify the bacteria to at least genus level [1].

Twenty-one of the 23 sampled bougies were contaminated, most (14/23) with less than 20 cfu, though one bougie yielded 91 cfu. The mean and median counts were 17 and 8, respectively, with an interquartile range of 5–24. The majority of bacteria were coagulase negative Staphylococci, Micrococci, diphtheroids and *Bacillus* sp. (Table 2). Of particular concern was the growth of *Proteus* sp. from one bougie tip and environmental gram-negative rods from another nine. The latter included members of the genus *Pseudomonas* and *Acinetobacter*, which are potential nosocomial pathogens and common causes of ventilator-associated pneumonia [2]. The different rates of contamination between our study and that of Cupitt may be because, in the latter, many bougies had been newly issued to the wards and probably had been used less often than the ones we sampled.

Ventilation and airway manipulation are recognised risk factors for hospital-acquired pneumonia [2]. The introduction of microbial contaminated bougies may be an additional risk for infection. The majority of bacteria identified were of low virulence and are rarely associated with infection but a significant minority were potential pathogens. If bougies are to be reused, autoclaving or efficient cleaning and disinfection of bougies are needed together with effective protection from recontamination during storage. As Cupitt notes, it may be safer and more cost effective to use single-use bougies only once, avoiding the need to decontaminate after use and protect from subsequent recontamination.

Table 2 Identities of bacteria contaminating bougie tips.

Bacteria isolated	No. of contaminated bougie tips
Coagulase negative staphylococcus/Micrococcus	4
Aerobic gram-negative rods	2
Mixed growth*	15
No growth	2

*Includes mixtures of coagulase negative staphylococci, diphtheroids, *Bacillus* sp. aerobic gram negative rods and *Proteus* sp.

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Ventilator trigger setting in an intensive care unit

We read with interest the recent case report 'Brainstem death and ventilator trigger settings' (Willatts & Drummond. *Anaesthesia* 2000; **55:** 676–7). We regularly face the same technical problem in setting ventilators in mechanically-ventilated patients (Puritan Bennett 7200 ventilator) with a left ventricular assist device (LVAD) in our cardiothoracic intensive care unit. The LVAD is implanted in patients with end-stage cardiac failure. In the LVAD system, a diaphragm pump is fixed intraperitoneally and is connected to left ventricular cavity and aorta beyond the aortic valve with preclotted, woven Dacron graft [1]. The pump maintains the cardiac output for the patient and it creates enough negative intrathoracic pressure to trigger the ventilator for synchronised intermittent mandatory ventilation (SIMV) or pressure support ventilation if the set sensitivity is at its most sensitive. The usual pressure below PEEP trigger has been found to be the cause of ventilator auto cycling, even at relatively insensitive levels of 4 or 5 cmH_2O; this with the patient apnoeic on disconnection of the circuit. Obviously, this will add to the work of

breathing and make weaning from IPPV difficult when the time comes.

We have found flow triggering to be the answer in these circumstances using the flow-by function on the Puritan Bennett 7200. An average base flow of 20 $l.min^{-1}$ and trigger setting of 3 $l.min^{-1}$ are generally suitable. We have found this demonstration of apnoea on disconnection of the ventilator circuit plus the elimination of 'auto-cycling' with 'flow-by' has reassured all intensive care staff of the true state of the patient regarding spontaneous inspiratory effort.

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Use of a carotid arterial line during organ harvest

To protect the function of organs harvested from patients who have satisfied the criteria for brainstem death, careful physiological support of the donor is required. Prolonged hypotension is associated with decreased allograft survival [1]. Organ harvest causes large insensible fluid loss and blood loss may be significant. To optimise fluid replacement and inotrope therapy, invasive blood pressure monitoring is mandatory.

Twice, I have anaesthetised inotrope-dependent patients for organ harvest, during which the radial artery line has failed. As access to the arms, groins and lower limbs was very difficult, on both occasions I placed an 18-g Jelco (Johnson and Johnson Medical) into the left carotid artery. This was straightforward and allowed transduction of arterial blood pressure.

Invasive arterial monitoring is traditionally performed in the radial, femoral, axillary or brachial arteries. The choice of site is determined by the profile of complications, which include haematoma, distal ischaemia, emboli, local infection and catheter-associated bacteraemia [2]. The carotid is not considered for arterial pressure measurement because the consequences of embolisation from the catheter or arterial occlusion may be devastating. Clearly, in the setting of brainstem death, cerebrovascular accident is no longer a concern and the carotid artery is an attractive alternative to more conventional sites.

In health, as one moves distally through the arterial tree, mean pressure remains constant, yet pulse pressure increases [3]. However, radial artery lines may yield a falsely low reading in patients receiving high doses of vasopressor [4, 5] and following CPB [6]. Therefore, in the setting of organ harvest, monitoring pressure in the carotid artery pressure may be superior to the radial artery.

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Preparation of anaesthetic drugs in the obstetric theatre

I read with interest the letter by J. M. Cupitt and C. Dunkley (Cupitt & Dunkley. *Anaesthesia* 2000; **55:** 721). Following a recent Health and Safety Review we also had to address the problem of providing obstetric emergency drugs safely.

We felt we would be putting patient safety at risk were we to stop having thiopental and succinylcholine immediately available. Genuine 'crash sections' are few and far between; however, when they occur, time is extremely precious and many urgent tasks need to be performed by the obstetric and anaesthetic team. Preparing anaesthetic drugs in this situation would either take up the anaesthetist's time when this can be ill afforded or might have to be delegated; the risk of preparation errors must be higher in these circumstances. Therefore, we continue to draw up thiopental and succinylcholine (no other drugs) daily, and keep them capped and labelled with date, time, concentration and initials in the refrigerator. A minijet syringe of atropine is kept with them.

To keep the drugs tamperproof in a locked refrigerator could of course prevent immediate availability. Since the chief purpose of the lock is to prevent tampering not theft, we now apply a numbered plastic lock, which is recorded in a logbook. The preparation time of the drugs is also logged. It only takes seconds to check the integrity of the tag and compare the number against the log, and we still have access to the essential drugs to induce general anaesthesia in an emergency section.

In time, we hope to obtain premade syringes of thiopental and succinylcholine from our pharmacy department, which would add a further degree of

safety. Meanwhile, we feel we have struck an acceptable risk–benefit balance.

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Epidural anaesthesia and splanchnic blood flow

Several points in the recent article about the effect of epidural bupivacaine on splanchnic blood flow (Mallinder *et al. Anaesthesia* 2000; **55:** 659–65) are worth highlighting. The initial power calculation estimated that 40 patients would be required but only 32 were analysed. All the patients lost from the study were from the epidural group (12 vs. 20) making comparisons difficult. There were also many more patients who were ASA 3 in the morphine group than in the epidural group (8 vs. 2). I note that 44% of the total group suffered complications, ranging from wound infection to death, which seems excessively high.

The authors acknowledged that it was possible that the epidural catheters were sited too low to achieve adequate sympathetic blockade, but countered this by suggesting that the large volumes of local anaesthetic used and the fact that patients were all comfortable post-operatively indicated an adequate dermatomal level of analgesia. The solution used in the study has not been validated to produce sympathetic blockade to T_5 with a lumbar epidural catheter [1]. Patients would only require a dermatomal analgesia level of T_8 to be comfortable intra- and postoperatively, far below the level required for sympathetic blockade of the mesenteric vessels [2]. At no point in the paper did they discuss blood pressure changes or the amount or type of intravenous fluid used to correct hypotension. It is well documented that epidural-induced hypotension can cause gut hypoperfusion [3]. If an adequate sympathetic blockade to T_5 was achieved with the initial bolus, especially in this group of surgical patients, some degree of hypotension must have occurred.

It would seem unlikely, given the combination of the above factors, that conclusions can be drawn about the effects of epidural analgesia as compared to systemic morphine on splanchnic perfusion.

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A reply

We thank Dr Low for his interest in our recent article. We acknowledged in our article that due to unforeseen losses of patient data the power of our study was decreased from the projected 0.9 to 0.7.

Although the solution used in Nydahl's experimental study [1] was 0.5% bupivacaine as opposed to the 0.25% used by us, it is well known that it is mainly the volume of anaesthetic and not the concentration that influences the height of sensory blockade. In Nydahl's study, the median maximal cephalad spread of analgesia after epidural injection of 20 ml of 0.5% bupivacaine in supine patients of a similar age group was T_4. It is therefore a reasonable assumption that the intra-operative spread of 20 ml of 0.25% bupivacaine would cover most of the splanchnic sympathetic outflow (T_5 to T_{11}). With regular top ups peroperatively, most patients had excellent analgesia in recovery with blocks from T_5 to T_8 (though not specifically stated in our article).

As regards Dr Low's comment on volume status, all patients had pre-operative fluid resuscitation and those receiving epidurals had a further 500–750 ml of crystalloid whilst the block was being established. Central venous and invasive arterial pressures were measured peroperatively in all patients. Comparison of mean arterial pressure between the epidural and opiate groups showed no significant differences over the course of the procedure (data yet to be published). We feel that adequate hydration was maintained peroperatively in both groups and that our conclusion that there was no tonometric difference in splanchnic perfusion is still valid.

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Pregnancy, anaesthesia and Guillain–Barre syndrome

I was interested to read the case report of Guillian Barre syndrome (GBS) in pregnancy (Brooks *et al. Anaesthesia* 2000; **55:** 894–8). I would like to add additional information arising from experience with a similar patient. This highlights the importance of gestational age during the illness, and also of attempting to define a precipitating cause for the illness.

A previously well 19-year-old presented with a rapidly evolving GBS 1 week after an episode of pharyngitis. Lumbar puncture and nerve conduction

studies confirmed the diagnosis. Serological testing was positive for CMV IgM antibodies in a dilution of 1 : 512, indicating recent Cytomegalovirus (CMV) infection. Other relevant serology was negative.

Treatment with intravenous immunoglobulin (IvIG) was started immediately on admission to hospital. By day 2 of her illness, she required mechanical ventilation for respiratory insufficiency. Despite marked autonomic dysequilibrium, a general anaesthetic was administered uneventfully on day 7 for insertion of a tracheostomy. Her illness reached a nadir at around 10 days when she exhibited a dense quadriparesis and retained only minimal motor function of her extraocular muscles. Three weeks following ICU admission, a pregnancy test was performed because of amenorrhoea. It was positive and ultrasonography suggested a fetal age of 9 weeks. In view of her severity of illness, gestational age and CMV status, she elected for termination of pregnancy. This was carried out under (another uneventful) general anaesthetic. After 86 days in the intensive care unit, the patient was discharged for further care. Her recovery was slow and complicated, but eventually of a good quality.

This case adds to the information provided by Brooks *et al.* CMV is causally implicated in 10–22% of GBS [1]. It is a serious infection in the first trimester: 5% of infected babies will have early multiple disabilities and a further 5% develop later handicaps [2]. These include microcephaly, mental retardation and motor disorders.

Other triggers associated with GBS (e.g. Epstein–Barr virus, *Varicella zoster* virus, HIV, *Campylobacter jejuni* [1]) may also have implications for the mother and fetus. These range from trivial to include congenital varicella, sepsis and fetal death [2–4]. Consequently, for all patients with GBS who may be pregnant, a thorough search for potential causes should be carried out, since the results may have significant consequences for the management of both patients.

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Von Willebrand's disease and neuroaxial anaesthesia

We would like to report the case of a 23-year-old primigravida woman with von Willebrand's disease (VWD). Our local consultant haematologist notified us of her when she was 36 weeks gestation. Apart from her von Willebrand's disease she was apparently fit and healthy and was enjoying an uncomplicated pregnancy. Her laboratory results were as follows: von Willebrand factor antigen (vWF:Ag) 28% and Ricof level 42%. On the basis of these results, the haematologist suggested that an experienced anaesthetist could perform epidural analgesia safely as her risk of bleeding into the epidural space was minimal. We, along with our colleagues, were not sure of the validity of this advice and were unfamiliar with these laboratory results. We therefore decided to determine the significance of the haematological investigations performed in patients with VWD and clarify the risk of bleeding associated with regional analgesia. We felt that anaesthetists need to be aware of this disease and have an understanding of how it is investigated in order to make valued judgements in the use of regional techniques in the presence of possible abnormal haemostasis.

Von Willebrand's disease is an inherited haematological disorder where there is a deficiency of von Willebrand's factor (vWF). In type I VWD there is a reduction in the amount of vWF in the blood, in type 2 VWD normal levels of vWF are found but it is functionally abnormal, and in type 3 VWD there is a severe deficiency in levels of vWF. VWF is required for platelet adhesion to damaged endothelium during the formation of the platelet plug. It is also the carrier protein for factor VIII. Deficiency of vWF leads to prolonged bleeding times and patients usually present with abnormal bleeding conditions such as menorrhagia or epistaxis

The haematological investigations are used in VWD to determine both the quantity and functional capacity of vWF in the patient's plasma. The amount of vWF is determined by measuring the vWF:Ag level, whereas the Ricof level is a measure of its functional activity. The vWF:Ag level in the plasma can be assayed directly and compared to a reference curve of standard dilutions so that a level $>50\%$ is normal. Ricof levels are determined by adding a sample of the patient's plasma to normal platelets and then adding the antibiotic ristocetin. Ristocetin reacts with vWF and the platelet membrane to induce platelet aggregation. This interaction requires the presence of a functional unit of the vWF known as the ristocetin cofactor. Ristocetin-induced aggregation follows a dose–response curve dependent upon the amount of functional vWF present. Thus, a 'normal' Ricof level is quoted as $>50\%$. Ristocetin does not agglutinate the platelets in many patients with VWD due to the absence of the ristocetin cofactor. Thus in VWD the Ricof level is often reduced. If the Ricof level is above 30% the risk of bleeding is not thought to be increased; however, when below this level, especially if the level is $<10\%$, there is a significant risk of bleeding.

There does not seem to be any consensus on the Ricof level that is safe for regional anaesthesia (Giangrande PLF, Oxford Haemophilia Centre, personal communication). We could only find one case report in the literature that described the use of an epidural in a patient with VWD. This obstetric patient had a Ricof level of 10% and

vWFAg level of 35%. The use of an epidural was uneventful [1].

Fortunately, our patient had an uneventful delivery and did not require our assistance. However, as a precaution we arranged for desmopressin to be available on the delivery suite in the event of a postpartum haemorrhage (PPH). Women with VWD are at increased risk of PPH as the level of vWF falls rapidly following delivery. Desmopressin is used therapeutically to boost the level of vWF production *in vivo*.

We would be interested to hear the experience of other anaesthetists in the management of patients with VWD and at what Ricof level they regard regional anaesthesia to be safe.

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Parenteral narcotic analgesic for labour pain

I read with interest the editorial 'Are we willing to change?' (Aly & Shilling. *Anaesthesia* 2000; **55:** 419–20), which discusses four narcotic analgesics administered parenterally for labour pain. I was surprised to read that pethidine does not act as an analgesic but works only as a sedative. It is well known that pethidine binds with μ-receptors, which modulate pain at spinal and supraspinal level and also induce sedation. Pethidine has been effectively used for other types of visceral pain such as that associated with gall bladder and renal colic. Conceivably, a patient is more likely to go to sleep under the analgesic effect of pethidine. The fact that a labouring mother who has received intramuscular pethidine wakes up due to labour pain does not mean that the drug has no analgesic effect. Probably, the pharmacokinetics of intramuscular administration are not ideal for the intermittent pain of labour. An opioid drug with a rapid onset and short half-life (such as fentanyl) delivered by PCA is better suited to labour pain. However, PCA is not available to millions of labouring women in developing countries, where pethidine may be the best available analgesic drug.

I agree with the authors that multicentre trials should be conducted to identify effective narcotics, but this should not be limited to only three drugs (morphine, diamorphine and fentanyl) and should include other narcotics which have unique pharmacokinetic (such as remifentanil) and pharmacodynamic (such as tramadol) properties.

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Parenteral narcotic analgesic for labour pain 2

We read with interest the editorial by Drs Aly and Shilling (Aly & Shilling. *Anaesthesia* 2000; **55:** 419–20) in which they discuss the reasons for the continuing use of intramuscular pethidine for relief of labour pain. However, we wish to challenge the suggestion that fentanyl administered as intravenous patient-controlled analgesia (PCA) should replace pethidine.

It has long been known that for up to half of parturients, pethidine provides either no or inadequate pain relief [1]. In current midwifery practice it is often administered in early labour to produce anxiolysis and sedation rather than analgesia. The reason that intramuscular opioids do not produce reliable analgesia is not related to the particular opioid chosen, but that an insufficient dose is given by an inefficient route. Scott, in his original description of PCA in labour [2], quoted a mean pethidine dose of 448 mg with a maximum dose of 1440 mg. These doses were very effective.

Aly and Shilling suggested that fentanyl could be administered via an intravenous PCA device but said that no studies had been done. We would like to draw attention to the study performed by Nikkola *et al.* [3] in which fentanyl PCA was compared to epidural analgesia. This study, whilst suffering from a number of design faults, showed that fentanyl PCA is a poor alternative to an epidural for labour analgesia.

The pharmacokinetic properties of remifentanil make it a far more ideal opioid for use in labour. Remifentanil is an opioid with an onset time equivalent to alfentanil but which is rapidly metabolised by blood and tissue nonspecific esterases. It is therefore noncumulative even when given over long periods of time [4]. In contrast, fentanyl has a context-sensitive half time that increases with the duration of administration causing it to accumulate. It will also cross the placenta and thus accumulate in the fetus, potentially causing neonatal respiratory depression or other neurobehavioural changes. Remifentanil, although it too crosses the placenta, is rapidly metabolised by the fetus [5].

In one of our institutions, we have used remifentanil PCA for certain parturients in whom epidural analgesia is contraindicated [6]. In the other, we have studied nine women who have received a remifentanil PCA with good reduction in visual analogue pain scores and no adverse effects to either mother or neonate (Volikas and Male, unpublished data). These women did not have any contraindications for an epidural other than a maternal wish to avoid an epidural if possible and would otherwise have received pethidine analgesia. Unlike the current use of intramuscular pethidine, which often provides sedation without analgesia, the women are able to use the PCA pump to attain a mixture of analgesia or sedation that they find acceptable.

We would accept that further studies should be performed formally to assess the efficacy of remifentanil PCA and its effects on the neonate. However, this drug would appear to offer the best way forward in developments for nonepidural labour analgesia.

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A reply

We are grateful for the comments of Dr Jones and colleagues. We were aware of one case report by Rosaeg *et al.* [1] as well as the study by Nikkola *et al.* [2] that involved six patients. Both the case report, as well as the study, showed some benefit of using fentanyl patient-controlled analgesia for pain relief in labour. We felt it was not appropriate to make a conclusion on the use of fentanyl PCA for labour analgesia on the basis of studies that included only seven patients, but we admit we should have included those studies in our editorial.

Regarding the use of fentanyl or remifentanil for labour analgesia, we think it is too early to suggest either is superior over the other and agree that further large studies are needed to establish the efficacy as well as the safety of these drugs.

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The safest prediction of epidural analgesia

Drs Singh and Kahn (Singh & Khan. *Anaesthesia* 2000; **55:** 830–2) report the use of a peripheral nerve stimulator after the injection of bupivacaine into the caudal epidural space as a predictor of successful caudal epidural analgesia. In their study, they performed whoosh test-1 [1] but they failed to perform whoosh test-2 [2]. Peripheral nerve stimulation is most safely used prior to the injection of local analgesic drugs [3], and not after the injection as the current required is much less. The use of a peripheral nerve stimulator with high current after the injection of local analgesic drugs is unsafe and could lead to neurological damage.

Whoosh test-2 confirmation of the correct position of the needle is safe and easy to perform and never produces neurological damage.

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Reducing the incidence of technical failures and paraesthesia in combined spinal–epidural techniques

Recent studies have shown that the combined spinal–epidural (CSE) technique is gaining in popularity for labour analgesia in the UK, with 24% of obstetric units offering CSEs in 1999 compared to none in 1991 [1]. The disadvantages of the single interspace needle-through-needle CSE technique include high spinal component failure rates due to spinal needle movement (10%) [2], and a high incidence of paraesthesia (29%) reported in certain studies [3].

During the CSE procedure, the removal of the inner spinal needle stillette to confirm successful dural puncture by visualisation of CSF at the hub of the needle as well as subsequent connection and injection of the intrathecal drug containing syringe are all potential points at which excessive spinal needle movement, and therefore failure of the technique, may occur. Paraesthesia, and potential neurological problems, on the other hand, can be elicited if the spinal needle is inserted too far through the dural sac [4]. Although it is well known that a maximum spinal needle protrusion distance of less than 15 mm (epidural needle tip to spinal needle tip) with a CSE can lead to increased failures by inability to puncture the dura, there is no consensus on the upper limits of the protrusion distance. An excessively long spinal needle such as the 26G, 124 mm Gertie Marx pencil point needle can

therefore produce an unacceptable incidence of paraesthesia (29%), presumably due to its long maximum protrusion distance, compared to the more commonly used shorter 27G, 119 mm Whitacre spinal needle [3].

We believe that the following two refinements of the existing CSE technique can potentially reduce the incidence of both spinal injection failure and paraesthesia and so lead to improvements in success rates and safety.

Firstly, the inner stillette of the spinal needle is removed [5], before slowly advancing the spinal needle within the epidural needle through the dura. The spinal needle is advanced until a 'dural' click is felt which, with modem spinal needles, is invariably followed by the instantaneous appearance of CSF at the hub. Although a dural click is usually felt in over 90% of CSEs [6], if a click does not occur during slow advancement of the spinal needle, the presence of CSF within the hub at the point of dural puncture indicates that further advancement should cease. Since the immediate appearance of CSF occurs on dural puncture without the presence of the stillette, the temptation to advance the needle further than necessary, causing potential paraesthesia and nerve damage, is avoided.

Second, during the intrathecal injection both the hub of the spinal needle and the epidural needle should be gripped firmly between the thumb and the index finger of the anaesthetist's hand while the back of the same hand is simultaneously stabilised against the patient's back.

By adopting a slow advancement technique and prior removal of the spinal stillette combined with adequate external stabilisation of both spinal and epidural needles with the operator's hand, our own spinal technical failure rates have improved in association with a paraesthesia incidence below 2%.

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Fixation of epidural catheters

We should like to add to recent discussions on 'migration' of epidural catheters and fixation devices. In their recent letter, Drs Tackaberry and Wadsworth describe two cases in which working labour epidurals could not be extended for operative delivery (Tackaberry & Wadsworth. *Anaesthesia* 1999; **54:** 914). Interestingly, both catheters had not moved relative to the skin, being fixed with the new Sims Portex Lockit® Epidural Catheter Clamp. The authors postulated that catheters were pulled out of the epidural space by relative movement of tissues superficial to the ligamentum flavum. Indeed, it has been demonstrated by Hamilton and colleagues that there is significant movement of catheters at the level of the skin as women change from the sitting flexed to the lateral position [1]. Therefore, if catheters are fixed firmly to the skin, the only movement of the catheter that can occur is outward through the ligamentum flavum and consequently out of the epidural space. This may occur immediately following placement if the woman changes position, or more gradually during the course of labour. It is reasonable to assume that this is more likely in obese women in whom greater movement of the skin relative to the ligamentum flavum is possible [1].

We should like to illustrate varying degrees of such catheter movement with cases investigated radiologically some years ago by one of the authors (L.E.S.C.). The X-rays were performed in an attempt to clarify why epidural blocks had failed to maintain effective pain relief throughout labour. In all cases, an epidural catheter had been sited using a 16-g Tuohy needle with the mother in the lateral position. A multihole catheter (side holes 0.5, 1 and 1.5 cm proximal to the catheter tip) had been placed between 3 and 5 cm in the epidural space and secured firmly to the skin. Following delivery and informed consent, a small volume of contrast medium (Conray 280) was injected through the epidural catheter and lateral views of the lumbar spines taken.

In the first case (Fig. 8), part of the catheter can be seen to have worked its way out of the epidural space and formed an almost semicircular loop between skin and ligamentum flavum. This epidural, although still functioning, was investigated for failing to provide adequate second-stage analgesia. X-ray measurement of this catheter revealed that at least half of the initial epidural portion was no longer in the epidural space. Although all the injected dye is shown in the epidural space, with a multihole catheter designed so that the most proximal hole was 1.5 cm from the tip, it is likely that further outward movement would have resulted in some loss of injected solution into superficial tissues. Such is the case in Fig. 9. Again, the catheter can be seen to loop in the superficial tissues where it has been pulled out of the epidural space. Dye can be seen in both the epidural space and the superficial tissues, indicating that the likely cause of increased local anaesthetic requirement was catheter holes both inside and outside the epidural space. In the third case, dye is

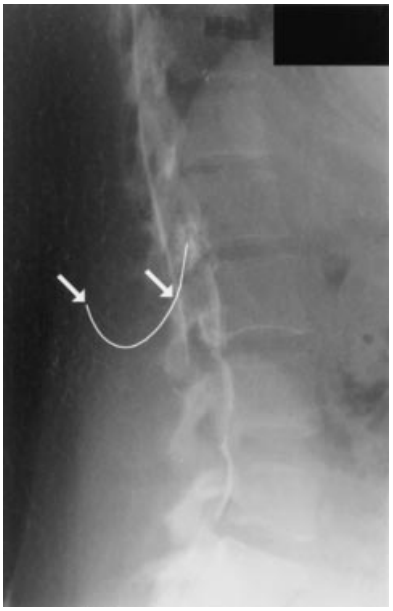

Figure 8 The epidural catheter has been highlighted to demonstrate looping in the superficial tissues although injection of dye produces a typical epidurogram. Arrows mark the site where the catheter is fixed to the skin and where it exits the ligamentum flavum.

seen only in superficial tissues as all three catheter holes have been pulled out of the epidural space (Fig. 10). In all three cases the position of the catheter had not moved relative to the skin.

Our cases provide further information on the behaviour of epidural catheters secured firmly to the skin. Securing catheters in this way may not be the most appropriate method of fixation as only outward movement of the catheter through the ligamentum flavum is possible, with the potential for inadequate analgesia as labour progresses. This possibility is increased with shorter lengths of catheter inserted into the epidural space and as some catheter movement is likely a sufficient length should be inserted, the length differing between single end-hole and multihole catheters. Should multihole catheters in which the proximal end-hole is 1.5 cm from the tip be placed less than 3 cm into the space, only 1.5 cm movement results in this proximal hole pulling out of the epidural space. Such movement is not uncommon [1]. This movement is facilitated by the passage of many 'midline' catheters being inevitably through the edge of the paraspinous muscle, rather than being secured more firmly in the narrow interspinous ligament (as demonstrated by the looping in Figs 9 and 10). Beillin and colleagues, using multihole catheters fixed firmly to the skin, found fewer problems when 5 cm was introduced into the epidural space when compared with 3 and 7 cm [2]. They suggested that failures occurred in the 3-cm group because catheters had been dislodged, whereas the 7-cm group had an increased risk of intravenous and paravertebral placement. Using end-hole catheters, D'Angelo and colleagues found that catheters inserted 2 cm into the epidural space were more likely to become dislodged than those inserted 4, 6 or 8 cm [3].

In order to place sufficient catheter in the epidural space to avoid dislodgement without threading excessive length and thus increasing the risk of intravenous and paravertebral placement, catheter design may be modified. The catheter described by Collier and Gatt with three side holes all within 4 mm of the tip was introduced to minimise the risk of multicompartment injection [4]. This design may coincidentally reduce the risk of dislodgement as, compared with standard wide spaced multihole catheters, a further 1 cm outward movement is necessary before any side hole leaves the epidural space.

Although often overlooked, the method by which epidural catheters are secured, in addition to the length of catheter insertion, is important in the maintenance of adequate analgesia. It is not the purpose of this letter to recommend lengths of catheter that should be left in the epidural space, but we believe it confirms that if such lengths are very short, the catheter is likely to move out of the epidural space no matter how effectively immobilised at skin level. Our X-ray studies suggest that the ideal fixation device (as yet undevised) would secure the catheter at or close to the skin puncture, but leave 2–3 cm between the fixation point and

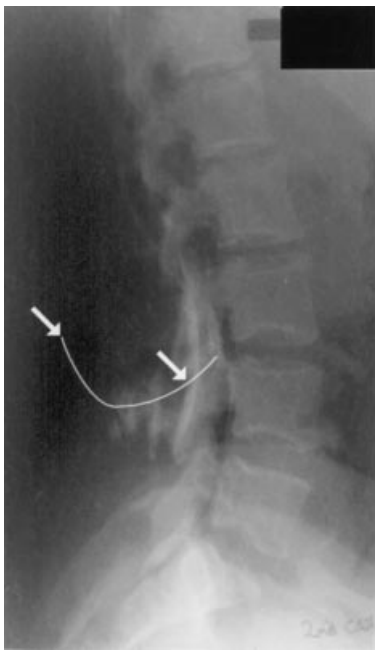

Figure 9 The epidural catheter has been highlighted and again loops in the superficial tissues. Dye is seen both in the epidural space and in the superficial tissues. Arrows mark the site where the catheter is fixed to the skin and where it exits the ligamentum flavum.

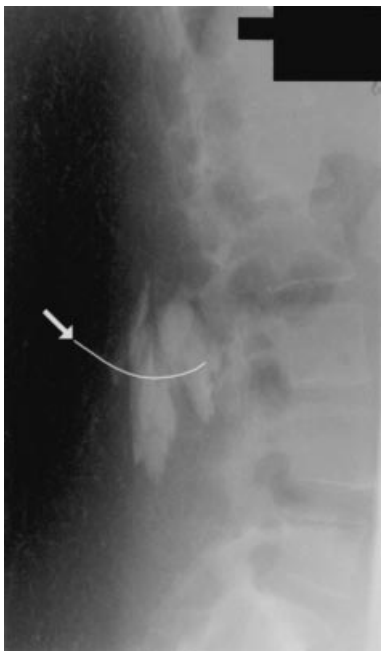

Figure 10 The epidural catheter has been highlighted and has pulled out of the epidural space with dye seen only in superficial tissues. An arrow marks the site where the catheter is fixed to the skin.

the skin puncture to allow for the increased length of catheter required for tissue movement between the skin and ligamentum flavum. However, as some degree of outward movement is likely during the course of labour, sufficient catheter length must be fed into the epidural space.

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Suturing epidural catheters

We read with interest the experiences of Drs Scawn and Pennefather (Scawn & Pennefather. *Anaesthesia* 2000; **55:** 304) in regard to fixing the epidural catheter too firmly at the level of the skin. In our own practice, we noticed that when the epidural catheter is fixed to the skin by a suture, it tends to be pulled away from the epidural space as the skin stretches with each movement of the patient. When the skin is unstretched, such as when a patient is upright, this excess epidural catheter coils under the skin. The excess coiled catheter, warmed to body temperatures, is softened and thus may kink under the surface of the skin.

When the epidural catheter is not sutured to the skin, but just secured with a transparent sterile dressing, it tends to migrate inward when the skin is stretched, and back outward when the skin is unstretched. In this situation, the catheter is usually anchored more to the ligamentum flavum than to the skin, therefore moving inward and outward with less tendency to coil under the skin.

In the past, when we used a low-quality 'Curity' epidural catheter, we occasionally had catheters kink under the surface of the skin. However, we now use better quality 'B. Braun' or 'Arrow' catheters, and have yet to see any sutured catheters kink under the skin as a result of coiling.

Suturing the epidural catheter increases the epidural success rate and reduces the incidence of catheter dislodgement, one-sided anaesthesia and epidural vessel puncture [1].

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Reference

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Epidural tunnelling techniques

Another aid to epidural tunnelling not mentioned in the recent editorial (Kumar & Chambers. Anaesthesia 2000; 55: pp.625–6) is to leave the Tuohy needle *in situ* following passage of the catheter until a subcutaneous tunnel has been created with a 16G intravenous catheter passed from lateral to medial exiting at the procedure site. Both needles can then be removed and the catheter fed down the cannula, which is then removed. A small scalpel incision may be necessary after the local anaesthetic prior to the initial passing of the Tuohy to ease delivery of the tunnelling device but the chances of damage to the epidural catheter or operator are minimised.

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A simple method for testing for the kinking epidural catheter

We would like to respond to the recent correspondence regarding 'kinking' epidural catheters (Roddin & Dancey. *Anaesthesia* 2000; **55:** 831) as our department has also experienced problems with the SIMS Portex 16G clear epidural catheter with three lateral eyes.

Over a 4-day period, there were at least six instances of epidural catheters becoming blocked in our hospital. The blockage could usually be relieved by altering the patients' position but often recurred. In one labouring patient, there were such frequent problems that the Mefix tape that was securing the epidural catheter to her back was taken down to investigate the problem further. On inspection, the catheter was found to have become completely kinked halfway down her back. When the catheter was straightened the blockage was relieved but the catheter appeared to be damaged and would easily re-occlude if the catheter was even slightly flexed again. In another patient, a similar kink occurred but the blockage was permanent, as the catheter walls appeared to have collapsed completely.

Figure 11 illustrates the problem with the catheter kinking in this defective supply batch. Figure 12 illustrates a SIMS Portex catheter from a different batch, which demonstrates the familiar resistance to kinking. Aware of this problem we have devised a simple test to check the catheters prior to use: if the catheter is bent back on itself as demonstrated in the photographs the catheters prone to kinking are easily identified.

Interestingly, the catheter in Fig. 12 comes from an individually packed epidural catheter pack, which had a bright green label on the front saying 'New Catheter material formulation'. In contrast to what Roddin and Dancey described in their letter, we do not appear to be have any problems with this supply. The batch that was affected

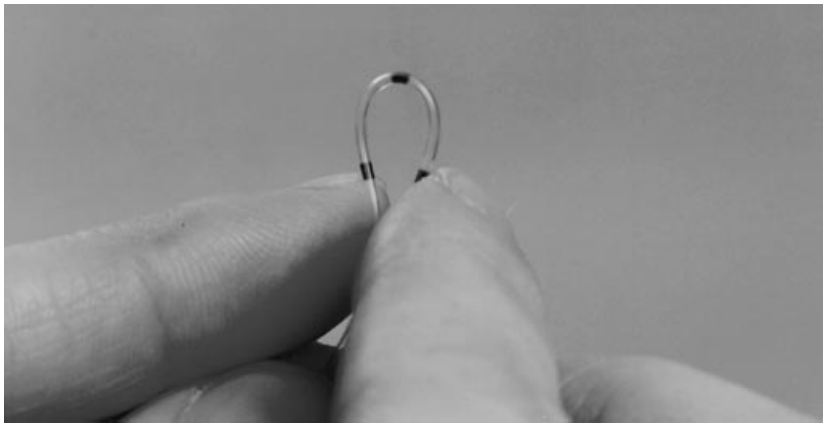

Figure 11

came from the custom-made epidural packs supplied to this hospital which had no mention of the new material formulation on them.

We too have reported this problem to SIMS Portex who are investigating it. The importance of this problem should not be underrated as at least six patients received less than adequate pain relief and some were subjected to a second epidural insertion with all the inherent risks.

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Value of attachments in A & E to intensive care training

Graham and Munro remind us of the overlap between accident and emergency (A & E) and anaesthesia with particular reference to airway management in a challenging patient population (Graham & Monro. *Anaesthesia* 2000; **55:** 814). I would add acute pain control, conscious sedation and perhaps even peripheral nerve blockade to the list of areas where practice in our specialties is similar. In the same issue, Dr Rooney provides a succinct account of the reciprocal benefits of anaesthetic attachments to other disciplines in terms of both practical and theoretical teaching (Rooney. *Anaesthesia* 2000; **55:** 813). Anaesthesia and A & E do indeed have much in common, but what of intensive care?

Before starting my anaesthetic career, I spent roughly equivalent time periods working in both general medicine and A & E. As I now progress through my Specialist Registrar ICU attachments, I am frequently struck by the obvious similarities between ICU and A & E practice. Indeed, I am certain that the experience I gained in A & E managing a huge variety of medical, surgical and paediatric emergencies as well as frequent resuscitation exposure is far more useful to me than that gained in medicine. Despite this, the Intercollegiate Board for training in ICM has stipulated that additional experience in medicine alone is required for dual CCST in anaesthesia and intensive care. I fear that by reducing time in A & E to an 'optional extra' the Board is encouraging prospective intensivists to miss critical care experience which A & E is uniquely able to provide.

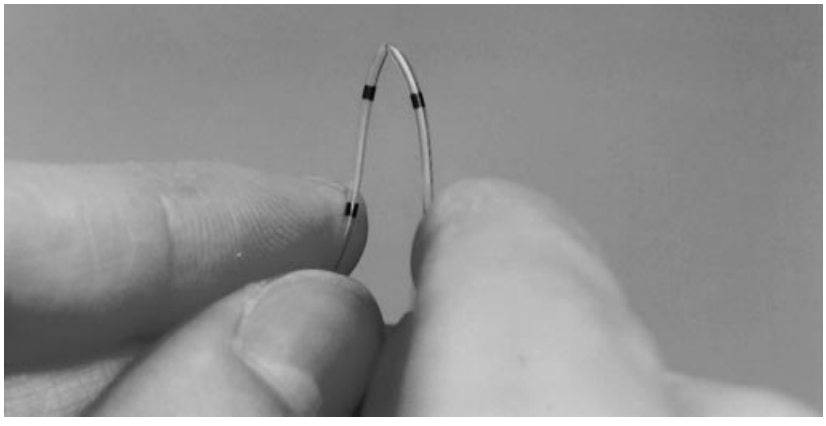

Figure 12

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From gasman to hospital saviour: how far should we go?

Professor Websters' recent editorial adds to the long-running correspondence on the issue of peri-operative physicians in this journal (Webster. *Anaesthesia* 2000; **55:** 839–40). I would like to add a few comments to the debate. Firstly, I would agree with Professor Websters' argument that adopting the title of peri-operative physician is perhaps a step too far at the moment. However, one aspect of this ongoing debate is that there are actually several related issues under discussion; they overlap somewhat, but also have some real differences worth exploring.

Firstly, the expansion of our role to participate in medical emergency teams (MET), patient at risk teams (PART), etc., and the development of 'Intensive Care without walls'.

Second, the expansion of our role to that of peri-operative physicians with responsibility for patient care from an undetermined point pre-operatively to another unidentified point postoperatively.

Third, the need to raise the status of anaesthetists in the eyes of the general public and other hospital staff.

Regarding the first issue, I fully support the introduction of 'Intensive

Care without walls'. The experiences of McQuillan *et al*. [1] are undoubtedly common in the UK and the results of introducing a MET speak for themselves. I feel that increasing the number of high-dependency units and improving identification of 'sick' patients on the wards can only be of benefit to patient care. We as anaesthetists are the logical choice to direct and lead such services.

Regarding the expansion of our role to become peri-operative physicians: many anaesthetists (perhaps the majority of those who entered the specialty in the last few years) have acute medical experience. Dual qualifications (FRCA & MRCP) are not uncommon. I would agree with Rooney [2] that all medical experience is beneficial (in fact, my feeling is that it should be a prerequisite for entry to the specialty). However, knowledge does advance rapidly, and it is unlikely that many of us will retain sufficient skill to provide a level of ongoing care equal to a specialist physician in all the specialties that we anaesthetise for. It seems paradoxical to aim for increased subspecialisation within anaesthesia (e.g. paediatric, cardiothoracic, etc.) while advocating the expansion of our role outside anaesthesia. Perhaps rather than taking full responsibility for the minutiae of patient care in such circumstances, we are better placed to co-ordinate appropriate care and referral when required. Discussion with parent medical and surgical teams is more likely to improve overall patient care than the unsolicited interference of someone armed with excess confidence, outdated facts and potentially rusty clinical skills. We should all be aware of the limits of our abilities. Some complex decisions are best discussed with the experts. As an aside, I recently spent a year working in a New Zealand ICU: I was impressed at how much interaction between specialties occurred. The surgical, medical and A & E registrars on rotation to ICU taught the anaesthetists useful skills and perspectives on management, and vice versa. Ward rounds with visiting medical specialists added much to our education and patient care. Becoming a peri-operative physician runs the risk of depriving us of such opportunities for ongoing learning.

On a practical (and lighter) note, to adopt the mantle of care for all patients who have an anaesthetic from before admission until discharge from hospital is rather like volunteering to join the Charge of the Light Brigade: noble, but not especially good for one's health. If you imagine you will achieve undying gratitude for such nobility, read a little Kipling [3]. There is an acknowledged and large, unmet need for adequate ward care [4], but few anaesthetic departments would be capable of undertaking all of this extra work without a great deal of additional resources. To attempt this without proper anticipation of the work involved runs the risk of discharging both old and new roles suboptimally, and of patients (as usual) suffering the consequences. Additionally, doing two jobs badly will do little to enhance our status with anyone.

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'For the times they are a-changing' – or are they?

I read the recent editorial (Harmer. Anaesthesia 2000; 55: pp.735–6) and was particularly interested in the comments concerning consultant office space. I work in a medium-size District General Hospital and as our consultant numbers have increased we have squeezed more consultants into the same space. The average office now has three desks and associated filing cabinets etc., leaving almost no floor area to move around.

Having an office with a personal desk is a status symbol that goes with the consultant position and we cram in another desk with each new consultant appointment. I carried out a casual survey of the consultant usage of their offices and found that our consultants use their office less than 10% of the working week. For the great majority of the time the offices are empty as our consultants spend most of their time in clinical areas.

I suggest that the time has come to change. I feel that we need to stop thinking of an office and desk as status symbols and think of them as functional working areas. If we were to have one desk per office and a personal filing cabinet for each consultant using the office then the office would be less cluttered. One good-quality computer could be shared between the occupants. Office time could either be booked or agreed at set times. As the offices are used so little it is unlikely that there would be a problem with congestion and the consultant using the office would have a better and more spacious environment. Can we justify having so much NHS real estate standing empty for so much time to provide office space to consultants that is so little used or should the times be a-changing?

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