Anaesthesia, 2001, **56**, pages 372–402

Correspondence

Opportunity knocks? – I think not

Reading Dr Willatts' editorial exhorting our specialty to even greater efforts in the future initially provoked the old familiar feeling of guilt that I (and presumably my colleagues) should be doing much, much more. Happily this emotion soon passed, to be replaced with one of incredulity and, rather surprisingly (since I think by nature I am a placid fellow), anger. Coming from the Prime Minister, the five P's sound, as one might expect, like yet more political sound bites. Coming from a senior and distinguished colleague who might be expected to represent the views from the 'coalface' they are rather surprising. As Clinical Director of a large department, I have just signed off the job plans of 37 consultant colleagues. All are working in excess of the European working time directive; so much for the 'rigid three and a half hour session'. All would agree that inadequate nursing levels on the wards and inadequate high dependency beds are a limiting factor in what they would like to achieve. The vast majority are still under 40 and are certainly not 'conservative and traditional'. They are certainly not 'protectionist' but imaginative and enthusiastic in doing their very best for patients with chronically limited resources in terms of staff and equipment. As for simply 'Passing Gas', the Association and Royal College recent snapshot of activity showed that at any one time 50% of us are engaged in other clinical activities anyway.

The Utopian solution that Dr Willatts proposes of greater working flexibility and the abandonment of normal working methods will not by themselves be enough to save the NHS. Indeed, the 'rock' of traditional medical practice may be all that has held it together for so long. Whatever the future holds, it is clear that Consultants cannot be expected to work any harder, or longer or with less support.

Time must be given for teaching, lecture preparation, appraisal, assessment, revalidation, CME, audit, risk management, research, CEPOD, complaints and the many other calls on their time. In essence, enough is enough. For those of us without a higher award and with years left to work before retirement, the prospect is bleak indeed and hardly one of 'Opportunity Knocks'.

Oh and I've just thought of one more 'P' … it's a decent Pay award.

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Problems with shared offices

I read the recent letter (Royle. *Anaesthesia* 2000; **55**: 1235) concerning consultant office space. Dr Royle is absolutely correct that use of office areas by Consultant Anaesthetists is less than 10%. He also encourages sharing of office space.

I used to use my office on a daily basis: in the morning before a theatre list and I would use the office at lunchtime when possible. I would return to the office at the end of a working day. The office is a safe place for my professional papers, it is a private place for reading and a space to talk with colleagues and trainees, especially those preparing for examinations.

I no longer use my office as illustrated above. The reason: it is now a shared office area.

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Pre-operative assessment clinics – the last word

I have followed with interest the recent correspondence regarding pre-anaesthetic assessment clinics [1–5], which discusses many of the merits and pitfalls of such a system. Within this discussion, however, I feel that one important aspect requires greater emphasis. For effective pre-operative assessment, anaesthetist-run clinics should not operate in isolation, but should be one component of a larger system that co-ordinates the various aspects of an elective surgical admission.

In our institution, pre-assessment begins with a written health questionnaire, which is sent to the patient at home as soon as they are listed for surgery. Completed questionnaires are screened by clerical staff using predetermined criteria and if necessary by an anaesthetist. The patient is then classified into one of the following categories:

1 requiring review in the Perioperative Clinic;

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2 no further review required (until the day of surgery).

In addition, clinic attendance can be arranged at the request of either patient or surgeon. As the majority of patients presenting for elective surgery neither wish for nor require a detailed anaesthetic consultation, this 'filtering' process means that only those patients who really need to be seen visit the clinic.

The benefits which Dr Cosgrove attributes to Preassessment Clinics [1] were actually conferred by the implementation of this peri-operative system [2]. This may seem overly pedantic, but I feel it is a point worth stressing. If a novel approach to pre-operative assessment is implemented in the UK, I feel that such a radical approach is warranted, not just a change in timing and venue of the standard pre-anaesthetic visit.

Like Dr Cosgrove, I am experiencing this system as a visitor to Australia. While it is by no means perfect, it is superior to the situation I have often experienced in UK hospitals where an unexpectedly complex patient presents on the day of surgery, resulting in a hurried (and often chaotic) 'workup'.

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So unlikely, yet so terrifying

I read your review article entitled 'The implications of HIV for the anaesthetist and the intensivist' (Avidan *et al*. *Anaesthesia* 2000; **55**: 344–54) with interest. I remember thinking at the time, probably like a lot of other readers of the article, that such an experience would be extremely frightening and so I must try to always remember to keep my practice safe. I then promptly placed it on my subconscious shelf of useful pointers. At that point, I had not thought in depth about the psychological impact of such an event, but now realise what a relevant aspect of the subject it is. The reality hits home hard. Having been a registrar for just over two months, approximately one week has now elapsed since I accidentally gave myself a potentially fatal inoculation. In view of the available statistics [1] regarding likelihood of seroconversion following needlestick injury with a sample from an HIV-positive patient (0.3%), this may sound a touch dramatic.

The needle in question was only a blue 23-gauge used to infiltrate local anaesthetic subcutaneously, and had already been outside the patient's body for a few minutes. According to protocol, I managed to squeeze a considerable amount of blood from my finger immediately afterwards, and within 2 h had been to Accident and Emergency and been started on a triple therapy of antiviral drugs; a protease inhibitor Nelfinavir, and Lamivudine and Stavudine which are both nucleoside analogue reverse transcriptase inhibitors, all twice daily.

Two days later, I had a consultation with an HIV consultant who explained to me the facts regarding risk, drug therapy and its possible adverse effects, future HIV testing, and also invited me to air any emotions that were worrying me. I found this episode relatively comforting. Various medical colleagues have been telling me about similar experiences of other professional acquaintances, which eventually resolved without any long-term consequences, and again I am reminded that my cause of concern is almost more a theoretical than a real one. and yet I feel a disconcerting uneasiness. It appears to me that the overall effect of such an anxiogenic event is a product of two variables; the likelihood of the worst-case scenario actually occurring (very small), and the repercussions should it actually happen. I think this second variable is immeasurably large. Hence, no matter how small the first variable, my overall feeling of worry is undeniable, and when I consciously remember it, disturbing despite its unlikelihood. Even friendly house officers, whom I barely know beyond a habitual polite nod of acknowledgement, were coming up to me with a slightly flushed and nervous facial expressions, to 'see if I was OK'. I don't know; am I OK?

Other reminders come along twice a day, morning and evening, in the form of my consuming seven tablets, five of which are the size of two paracetamol tablets rolled into a sausage – nauseating. For one careless error, it seems like harsh punishment to twice a day be made to feel as if I am taking an overdose. and these are not smarties. They can compromise liver and kidney function, cause gastrointestinal disturbance, hyperlipidaemia, anaemia, peripheral neuropathy, severe rash and, in my case to date, a general sense of nausea and malaise.

I know I have made several lucky escapes already in my life and, fingers crossed, mother probability will see me through this time once more. But my message to my fellow clinicians is clear and simple. Such is the psychological impact which I have tried to convey that taking too much care with sharps and body fluids of patients (irrespective of known or unknown infective status) is not possible. An accident is complete in a split second, yet the consequences cause months of worry on the scale of life, loved ones and the future. and it is this psychological angle of accidental needlestick injury that I wish to emphasise with respect to the original review article. Anxiety, sleep disturbance and low mood are added stresses to an already pressurised working role, and I feel could significantly compromise the everyday level of functioning of the anaesthetist – a potentially hazardous development (in a similar way to the over tired anaesthetist who has been awake working for too long).

I was fortunate in at least one respect to be referred to a supportive and

sympathetic HIV consultant who guided me skilfully. Perhaps these issues (including the option of supportive counselling) should also be considered and maybe even included in a further updated new post-exposure protocol design. I do believe that in six months time my HIV test result will be negative. I wish I could be as sure that these next six months will fly by.

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Pre-registration house officers in anaesthesia

I read with interest the correspondence concerning preregistration house officer (PRHO) training in resuscitation, fluid balance and analgesia (Meek. *Anaesthesia* 2000; **55**: 1128). I have just completed my first 4 months as a PRHO in Perioperative Care in Cheltenham. This new 1-year post is one of four in the South-West and was first introduced in Truro 3 years ago.

I became interested in the job after completing a 4-week attachment dedicated to anaesthetics and peri-operative care during my undergraduate training in Bristol. I also completed a compulsory 1-day certified ALS course before taking finals.

My job comprises sessions dedicated to anaesthetics, ITU/HDU and both acute and chronic pain. I have gained knowledge in the management of fluids, analgesia and postoperative complications and the recognition of the 'sick' patient. Practical skills learnt include intubation, arterial and central venous line insertion, spinal and intrathecal blocks, and the management of PCA and epidurals. I completed the ALS Provider course, and attend all cardiac arrest and trauma calls.

I would thoroughly recommend such a job to any final-year medical student or anaesthetic department. It has given me invaluable experience and confidence to deal with the problems highlighted in the letter. I hope this provides a more optimistic view of the training and education given to new doctors both in the undergraduate and PRHO setting.

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Inadequate pre-operative evaluation and preparation 1

The study (Kluger *et al. Anaesthesia* 2000; **55**: 1173–8) establishes an important link between poor pre-operative preparation and morbidity or mortality. I was interested to note that 'communication problems' and 'hospital processes' were frequent contributing factors to many incidents. Failings in systems of pre-operative care have resulted in some highly publicised deaths in recent years [1, 2]. Despite such incidents, there is little or no published data specifically examining how patients may suffer injury when pre-operative care pathways prove inadequate. NCEPOD reports continue to emphasise resuscitation prior to surgery [3] but have not scrutinised other aspects of pre-operative care in any detail. A Medline search back to 1960 did not reveal any other studies on this particular topic.

In a study of urgent and emergency surgery in a district general hospital [4], we found that over half of cases were affected by various failures of hospital processes. These included surgeon or anaesthetist unavailability, missing consent forms, inadequate pre-operative fasting and other staff shortages. Although none of the patients suffered any serious adverse event, a number of individual cases illustrated system failures that could easily result in injury. If this study were performed on a larger scale, it would undoubtedly reveal incidents similar to those in Dr Kluger's study.

The Australian Incident Monitoring Study has proved invaluable in directing us toward areas of care where standards are low. However, they are pointless unless followed up by a more detailed investigation. The government of the United Kingdom has set targets for the reduction of medical error [5]. Analysis and improvement of pre-operative care pathways is an obvious candidate for attention.

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Inadequate pre-operative evaluation and preparation 2

We welcome the recent paper on the important subject of pre-operative evaluation and preparation of patients (Kluger *et al. Anaesthesia* 2000; **55**: 1173–8). Inadequate pre-operative evaluation and preparation is known to affect intra-operative and postoperative morbidity and mortality. The authors have reminded us of some common reasons that lead to inadequate pre-operative evaluation, i.e. pressure of time, pressure from surgeons to proceed, late addition of patients to operating lists, changes in the order of operating lists. We agree with the authors that financial constraints over the last few years leading to more day case surgery and day of surgery admissions have compounded the problems and that implementation of any proposals would itself have

funding implications. We would like to make some general comments with regard to their findings in relation to pre-operative assessment of the airway and management of the difficult airway.

The authors have expressed concern that even in cases where there was clear evidence of difficult intubation in the past, the anaesthetist seemed reluctant to choose awake fibreoptic intubation as a method of choice when this would have been most appropriate. They wonder if this is inadequate training in the skill? An earlier study expressed similar concerns [1]. We like to suggest that while the skill of awake fibreoptic intubation is relatively easily acquired, e.g. by attending sessions of bronchoscopy with the chest physicians or attending one of the many courses run by anaesthetists with an interest in the subject, the problem is with keeping in practice: doing awake fibreoptic intubations often enough and building the numbers to acquire experience and confidence. In the normal population, the incidence of difficult intubation is quoted at between 1 : 200 and 1 : 1500. With the advent of the laryngeal mask airway, fewer patients are intubated, and an average anaesthetist may not meet a genuinely difficult airway for many years. How would one feel confident doing an awake fibreoptic intubation once every few years? There is no entirely satisfactory solution but some colleagues stay in practice by: (i) having a very low threshold for choosing awake fibreoptic intubation in their routine work; (ii) whenever there is a surgical indication for a nasal intubation they would elect to do this under direct vision with a fibrescope; (iii) use a fibrescope instead of a Macintosh laryngoscope in anaesthetised patients during elective surgery; (iv) visit the bronchoscopy unit every so often, and so on. Awake fibreoptic intubation is practised more frequently in North America than it is in the UK. Perhaps it reflects differences in training early in one's career. Furthermore, it seems time pressures are less of a problem in North America.

The authors also report that the majority of airway problems encountered were not anticipated by the anaesthetist. They suggest that this was either because the airway was not obviously difficult (such as difficult mouth opening) or that the airway was assessed poorly. We would like to propose an alternative explanation. We believe that airway problems, unless they are obvious, often surprise the anaesthetist even when pre-operative assessment was not poor, because they are uncommon and there is no satisfactory clinical observation or sign that the anaesthetist can use to predict difficult intubation. We would like to explain this, and hope it would encourage anticipation of difficulty and vigilance.

Many clinical and radiological features have been suggested [2–5] as useful in assessing potential difficulty with intubation, but they all only give an indication and in clinical practice are frequently unreliable. This unreliability is often explained as being due to individual variation. Cass *et al.* [2] analysed their experience with five cases of difficult direct laryngoscopy and suggested several features that in their view made visualisation of the glottis difficult. White and Kander [3] studied radiographs of the mandible, upper jaw and the cervical spine in 13 patients in whom direct laryngoscopy was difficult. Unlike the previous authors, they found that the most significant factor in the ease or difficulty of direct laryngoscopy was the posterior depth of the mandible. In all those patients in whom direct laryngoscopy was difficult, the posterior depth of the mandible was increased in proportion to the effective length of the mandible, such that if expressed as a ratio, it was less than 3.6 in all patients. The only other significant factor amongst their findings was a reduced gap between the occiput and the spinous process of the first cervical vertebra. They did not find many of the features suggested by Cass and colleagues significant. Lyons [4] reported his experience, over a 6-year period in a teaching maternity unit, of eight failed intubations. During investigations for establishing the aetiology of difficulty with intubation, five of the patients had radiological examinations. None of the five had a mandibular length to depth ratio of less than 3.6. Mallampati [5] proposed a very simple clinical sign to predict tracheal intubation and is widely used by anaesthetists during pre-operative assessment, but is not without limitations.

We would like to suggest that pre-operative assessment of airway is used only as a guide and not relied upon to predict difficulties with laryngoscopy. Instead, difficult intubation should be anticipated every time a laryngoscopy is performed and plan of action rehearsed, until otherwise proven. We also believe that awake fibreoptic intubation is not performed often enough in the UK. It would be interesting to know if colleagues share this opinion?

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Dentist anaesthetists

The letter (Masson & Payne. *Anaesthesia* 2000; **55**; 1130) was a gracious tribute to the part played by dentist anaesthetists in the development of anaesthesia in Edinburgh. It will have struck a chord with those of us who have lived through the rise and fall of general anaesthesia in the dental surgery during the past

40 years, with a number of innovations having been introduced from that source. One of the spurs for my own interest in the subject was the difficulty of obtaining the services of a medically qualified anaesthetist whose performance did not increase my stress level beyond bearing. It was firmly consolidated by the eye-opening opportunity of working in hospital with the local Consultant whose skill with intravenous pentothal and blind nasal intubation left me gasping enviously.

However, it should not be assumed that the administration of anaesthesia 'under the direction or in the presence of a qualified medical practitioner' necessarily improved safety, either in 1899 or subsequently. Many readers will remember that for many years it was the custom for the patient's own GP to be invited to administer the dental anaesthetic and I still vividly remember one (very rightly) much-respected holder of the Edinburgh M.D. who would approach my Walton II, set it to give a constant flow of 100% N_2O, and as he applied the face mask, turn to me with a charming smile and say, conspiratorially, 'I always like to start them off on a few breaths of pure oxygen!'

Incidentally, Messrs Masson and Payne cannot have checked the membership list of the History of Anaesthesia Society very carefully: there are many dental surgeons among the membership, some of whom were very early members – as, indeed, was Ian Brett, on whose letter they were commenting. Perhaps the confusion arises from the use of the courtesy title. It seems that we are all Doctors now – except for those who prefer to retain the surgical 'Mr' or just cannot be bothered to change!

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'All his anxiety resolved itself into a sigh and dissolved into apathy and drowsiness.' – Ivan Goncharov, Russian novelist, *Obolomov* (1859)

The induction of general anaesthesia with propofol in unanxious patients often seems to be accompanied by a yawning or sighing activity just before consciousness is lost. By anecdote, this is a relatively common phenomenon – although it does not appear to have been described in the literature. *The New Shorter Oxford English Dictionary* defines a sigh as a long, deep and audible exhalation [1]. It defines yawning as to open the mouth wide and inhale (silently or audibly) as an involuntary reflex when sleepy or bored [2]. In physiology and clinical medicine, various terms have been used in an inexact way when describing changes to the pattern of breathing. For instance, some ventilatory strategies are designed to give occasional larger breaths so as to recruit atelectatic lung – this is often called a 'sigh' function. In varying circumstances, defensive reflexes of the mammalian respiratory tract may be described as sniffs and gasps (inspiratory), coughs and sneezes (expiratory) or may just lead to apnoea. Yawning is a more stereotypical behaviour, occurring in animals from reptiles to mammals and associated with stretching and penile erection [3]. The function and mechanisms of yawning are still not clearly understood.

Forty ASA grade 1 and 2 adults undergoing day surgery for a variety of surgical specialties had a standard intravenous induction of 2 mg midazolam and 1 $\mu g.kg^{-1}$ fentanyl followed 2 min later by 2 $mg.kg^{-1}$ propofol. The presence or absence of one or more sighs before the loss of consciousness was noted. A sigh was defined as a breath that seemed clearly distinguishable in depth from the preceding few breaths. Twenty-three patients gave one or more sighs, and 16 became anaesthetised with no sudden change in their breathing pattern. One patient developed hiccoughs. A laryngeal mask was subsequently inserted without difficulty in all cases.

The causes of the change in breathing pattern are not clear, but may be associated with upper and/or lower airway reflexes. Pauses in breathing for any reason are associated with measurable decreases in functional residual capacity (FRC). Apnoeic pauses and spontaneous sighing activity are more common in infants than in adults. This sighing activity tends to restore FRC, recruit atelectatic lung volume and improve lung compliance [4, 5]. The apnoea associated with induction of anaesthesia may stimulate such a response. Alternatively, decreased muscle tone in the upper airways may cause a mechanical stimulation that initiates an inspiratory response [6]. The maintenance of ventilation and protection of the airway are some of the most powerful and primitive reflexes we possess. Perhaps it is not surprising that the effects of these reflexes may be elicited before we render people defenceless with anaesthesia.

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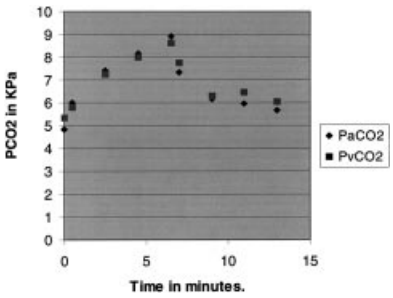

Figure 1

Carbon dioxide during and after the apnoea test – an illustration of the Haldane effect

We have investigated aspects of carbon dioxide kinetics in two patients having brain stem death tests performed. Arterial and mixed venous blood samples were obtained from arterial and the distal lumen of pulmonary artery catheters, respectively, at intervals during the apnoea test and upon recommencing ventilation. The samples were analysed immediately using an ABL 400 blood gas analyser. The change in arterial and mixed venous $P\text{CO}_2$ with time during apnoea and upon recommencing artificial ventilation is illustrated in Fig. 1. During apnoea the $P_a\text{CO}_2$ was seen to rise in a linear relationship with time (mean rate of increase with time was 0.63 kPa.min^{-1}). In both cases studied, the relationship between arterial and mixed venous $P\text{CO}_2$ were similar. Prior to apnoea, $P_v\text{CO}_2$ was greater than $P_a\text{CO}_2$. After the onset of apnoea, this relationship was reversed with $P_a\text{CO}_2$ becoming greater than $P_v\text{CO}_2$. The $P_v\text{CO}_2$ also continued to rise in a linear manner with time during apnoea at approximately the same rate. This reversal of the normal relationship between $P_a\text{CO}_2$ and $P_v\text{CO}_2$ during apnoea arises as a consequence of the Haldane effect [1]. In 1914, Haldane observed that the CO_2 dissociation curves are different for reduced and oxygenated haemoglobin (Fig. 2). This arises because reduced haemoglobin has the ability to carry more CO_2 as carbamino compounds due to a conformational change in the haem moiety facilitated by the absence of oxygen. Furthermore, from the geometry of the CO_2 dissociation curve we expect that the magnitude of the difference between arterial and mixed venous $P\text{CO}_2$ will increase with the duration of apnoea. In the two cases studied, this appeared to be true, as the rate of rise in mixed venous $P\text{CO}_2$ was less than that of arterial $P\text{CO}_2$.

After recommencing artificial ventilation, in both cases the usual relationship ($P_v\text{CO}_2 > P_a\text{CO}_2$) was restored and $P\text{CO}_2$ declined exponentially with time. This, together with the linear rise of $P\text{CO}_2$ in apnoea, suggests that a one compartmental model adequately describes acute changes in CO_2 homeostasis.

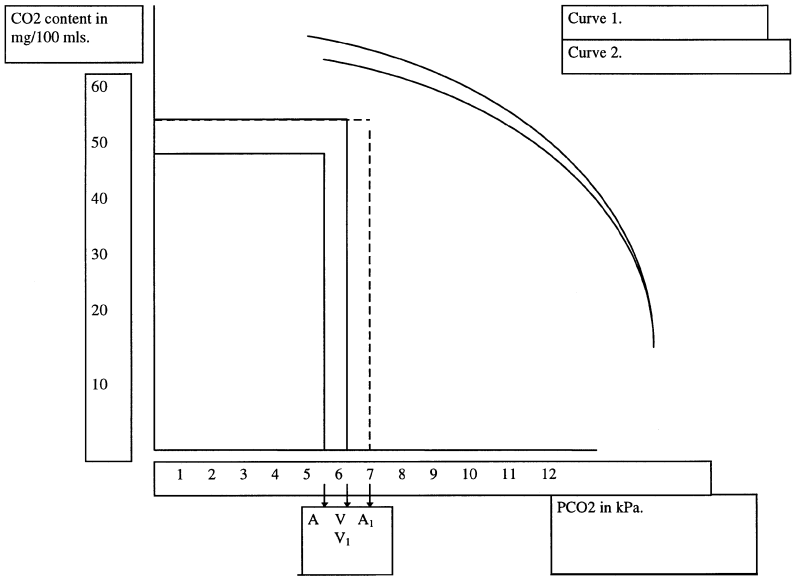

Figure 2

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Reducing unnecessary blood cross-matching

Mallett and colleagues' paper is an excellent example of the use of clinical audit to improve medical practice, in this case peri-operative transfusion practice for elective patients in a London Teaching Hospital (Mallett *et al. Anaesthesia* 2000; **55**: 1013–19). We have performed related audits of blood transfusion practice for patients undergoing urgent surgery for fractured neck of femur in two hospitals in the South-West. In both centres, we demonstrated problems in two areas – inappropriate clinical transfusion practices and poor utilisation of cross-matched blood. We were able to improve efficiency by introduction of transfusion guidelines based around a 'trigger' pre-operative haemoglobin concentration, generated from the initial audit. We should like to offer some comments based on our experiences.

Ninety-six consecutive patients undergoing hemiarthroplasty or insertion of dynamic hip screw were audited retrospectively. Sixty-six patients had blood cross-matched on admission (142 units), but only 26 patients were transfused, nine pre- or peri-operatively and 17 postoperatively (total 57 units). The transfusion to cross-match ratio (TCR) was 2.5, which we considered to

be very inefficient, although it compares favourably with the value of 3.3 for *elective* surgery given by Mallett *et al*. In total, 43 of the 96 patients (45%) were transfused during their admission. Single unit transfusions were infrequent (two patients), in agreement with the findings of Mallett *et al*.

At the time of the audit, 10 $g.dl^{-1}$ was chosen as the lower limit for an acceptable haemoglobin level in this patient group in line with the recommendations of the Committee of Experts on Blood Transfusion and Immunohaematology of the Council of Europe [1]. It was also clear that a value of less than 10 $g.dl^{-1}$ would be unacceptable to many of the clinicians involved. Our initial audit showed 47% of our patients had a haemoglobin level of less than 10 $g.dl^{-1}$ on the first or second postoperative day.

Receiver–operator curve analysis of our data determined that a pre-operative haemoglobin level less than 12.4 $g.dl^{-1}$ could predict a postoperative haemoglobin level of less than 10 $g.dl^{-1}$ (sensitivity = 0.73: specificity = 0.84). In practical terms: of the patients with a pre-operative haemoglobin of less than 12.4 $g.dl^{-1}$, 80% had a postoperative haemoglobin of less than 10 $g.dl^{-1}$. Guidelines were introduced establishing use of a 'trigger' pre-operative haemoglobin level of 12 $g.dl^{-1}$ for the cross-matching and peroperative transfusion of two units of blood and 52 consecutive patients were audited prospectively.

The guidelines were followed in 39 patients (75%): 17 patients (44%) were transfused peroperatively (2 units each). None of these 39 patients required a transfusion later in their admission. The TCR over the inpatient stay for those following the guidelines was greatly improved at 1.1 and only four of these patients had a postoperative haemoglobin level of less than 10 $g.dl^{-1}$ (mean (SD) 11.21 (1.24) $g.dl^{-1}$). The proportion of patients transfused remained at 45%. Cross-match and transfusion were more appropriately directed towards patients most likely to have a postoperative haemoglobin below 10 $g.dl^{-1}$. In Southmead Hospital, where about 220 patients undergo fixation of femoral neck fracture by DHS or hemiarthroplasty per year, these guidelines will reduce unneeded cross-matching of blood units from about 185 units to 20 units per year. At £10 per unit cross-match, this represents a saving of up to £1600.

One disappointing feature of our audit was the poor compliance with the guidelines, despite rigorous efforts to maintain awareness of their existence; we note that Mallett and colleagues required no formal policing of their guidelines. The two major reasons for breaching our guidelines were unexpected large peroperative blood loss or the anaesthetist forgetting to refer to the guidelines. None of the anaesthetists objected to them. Interestingly, an additional part of our guidelines encouraged use of a HemoCue® to check patients' haemoglobin level in recovery. In contrast to Mallett *et al*., we found that the HemoCue was used in only seven patients despite its availability.

The financial benefits we have demonstrated come primarily from making the cross-match process more directed. Mallett *et al*. improved practice in their hospital by targeting a later stage in the process, encouraging more frequent measurement of patients' haemoglobin pretransfusion. However, the downside of their changes was an increase in the TCR from 2.9 to 3.3. Calculations from Figure 3 in Mallet's paper show that in 1998, a total of 56 patients received at least 194 units. The TCR for this period was 3.3, implying that 640 units were cross-matched and therefore that 446 units were returned to the blood bank unused. At £10 per unit cross-match, this represents a cost of £4460, suggesting that further attention to the practice of cross-match requests, as shown in our audits, could further reduce spending from their annual blood bank budget. Their figures (Table 2, Mallett *et al*.) show particular patient groups to which audits similar to ours could be directed: total knee replacement (TCR 4.8) and total abdominal hysterectomy (TCR 9.0).

Our audit and the work of Mallet *et al*. demonstrate different but effective strategies by which audit and the introduction of guidelines may be used to improve the efficiency and appropriateness of transfusion without increasing the number of patients exposed to autologous blood.

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Universal precautions at cardiac arrests

Universal Precautions (UP) can be thought of as the steps taken to minimise the risk of transmission of infectious agents from patient to health care provider [1]. Their use can avoid 84–98% of mucocutaneous blood exposure [2]. However, healthcare workers take excessive risks when engaging in contact with patients [3–5]. Resuscitation is a high risk situation, with potential exposure of staff to the patient's blood or body fluids, as well as aerosol or droplet contamination. The first priority at an arrest should be to avoid risk to the rescuer [6]; however, if UP are not immediately to hand, they are either ignored or the search for them delays resuscitation. The anaesthetist is at particular risk at a cardiac arrest: not only are they at risk of mucocutaneous blood exposure, but also in dealing with the airway from droplet and airborne secretions [7]. Furthermore, the airway is often contaminated with vomitus, and potentially haematemesis. There is also the need speedily to establish oxygenation and ventilation to maximise the patient's prognosis.

We undertook a study in a large district general hospital, to assess UP

Table 1 The location of gloves, aprons and visors in 39 clinical areas.

	Gloves	Aprons	Visors
On arrest trolley	30	1	0
At bedside	5	5	3
In patient bay	3	6	5
In sluice	1	27	7
Not available	0	0	24

provisions for cardiac arrests in 39 clinical areas, namely: casualty, all general and specialist wards, all outpatient areas, and physiotherapy. Where equipment was not found on the cardiac arrest trolley, a member of the ward's nursing staff was asked to show its location.

Gloves were immediately accessible in all clinical areas; in the vast majority (30 sites), they were located on the cardiac arrest trolley. (Table 1). In 'frontline' clinical areas (i.e. Casualty, CCU, ITU/HDU), aprons were available in close proximity to the patient. However, in the 'non-frontline' areas, gowns were located remote from the patient. Facial protection could not be provided to a rescuer in 24 clinical areas. The current distribution of facilities reflects a selective approach to the provision of equipment to rescuers, rather than accepting that universal precautions imply a universal standard of provision of equipment.

This survey identifies a potential hazard to medical staff. We recognise that this study presents data from only one hospital, but feel it is representative of many institutions, and that it highlights an area of clinical risk. Hospitals owe a duty of care to staff for the provision of necessary equipment for the isolation of potential hazard [8].

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The fresh-gas flow sequence at the start of low-flow anaesthesia

In a theoretical study, Mapleson [1] used a multicompartmental physiological model of a patient and breathing system, to simulate the first 20 min of low-flow anaesthesia, using halothane, enflurane, isoflurane, sevoflurane and desflurane, in a standard man of 40 years and 70 kg body weight. The model was used to determine an 'ideal' sequence of fresh gas flow and vaporiser concentration which, for each anaesthetic, would raise the end-expired concentration to 1 MAC as quickly as practicable and then keep it within $\pm 5\%$ of that level for 20 min. We present a brief report of our experience in applying these ideal sequences in clinical practice.

The study was approved by the hospital ethics committee. Twenty-eight patients, aged 18–55 years, ASA grade 1 or 2, scheduled to undergo orthopaedic surgery, gave their informed consent. They were allocated to four groups for general anaesthesia with halothane, isoflurane, sevoflurane or desflurane, seven in each group. All patients received midazolam, 15 mg orally, as premedication 1 h before anaesthesia. Induction of anaesthesia was with propofol 2.5 $mg.kg^{-1}$, and fentanyl or remifentanil 1 $\mu g.kg^{-1}$, with vecuronium 0.1 $mg.kg^{-1}$ for muscle relaxation. After oral intubation of the trachea, the lungs were ventilated with a tidal volume of 10 $ml.kg^{-1}$ at 10 $breath.min^{-1}$. A circle breathing system was used with an internal volume of 4 l, including the ventilator bellows. The administration of the assigned inhalation anaesthetic commenced immediately and followed the corresponding Mapleson [1] sequence (Table 2), except that the maximum concentration for a desflurane vaporiser is 18% or 2.3 times the mean MAC of our desflurane patients.

In addition to routine monitoring of cardiovascular and respiratory variables, we measured the inspired and end-expired fractional concentrations of the inhaled anaesthetics with the gas monitor of the Ohmeda anaesthetic machine. Conversion to MAC units allowed for the age of each patient [2] and the mean ambient barometric pressure of 93 kPa.

The four groups of patients were similar in respect of age and body weight (Table 3). With all four anaesthetics, the mean end-expired concentration (Fig. 3) reached 1 MAC earlier than predicted by Mapleson (with a minor exception for desflurane) and remained above 1 MAC throughout

Table 2 Sequence of fresh-gas flows and vaporiser concentrations used for each anaesthetic [1]

	Time period (min)					
	0–1	**1–1.5**	**1.5–4**	**4–7**	**7–10**	**10–20**
Fresh-gas flow; l.min^{-1}						
Halothane	5	5	5	2.5	2.5	1.5
Isoflurane	5	5	1.5	1.5	1	1
Sevoflurane	5	1	1	1	1	1
Desflurane	3.5	1	1	1	1	1
Vaporiser concentration; MAC units						
Halothane	3	3	3	3	3	3
Isoflurane	3	3	3	3	2.5	2.5
Sevoflurane	3	2.5	2.5	2.5	1.8	1.8
Desflurane	2.3*	1.5	1.5	1.5	1.5	1.2

*3 in the original theory but limited to 2.3 by the vaporiser.

Table 3 Age and body weight, mean (SD), for the seven patients receiving each anaesthetic

	Halothane	**Isoflurane**	**Sevoflurane**	**Desflurane**
Age; years	31.1(11.9)	29.6(8.6)	30.9(12.9)	29.1(13.0)
Weight; kg	62.1(17.3)	65.7(9.2)	62.0(7.6)	61.1(16.4)

the remainder of the 20 min, significantly so on average (Table 4).

Although we followed the Mapleson sequence of fresh-gas flow and vaporiser concentration meticulously, our study differed systematically from the circumstances assumed by Mapleson: our patients were smaller, overall mean body weight 63 kg, not 70 kg; and ventilation was greater, 100 ml.kg^{-1}.min^{-1} not 79 ml.kg^{-1}.min^{-1} (5.5 l.min^{-1} for a 70-kg man). Both these differences help to explain why the mean measured end-expired concentrations were systematically and significantly greater than predicted (Table 4). However, from a clinical point of view, this might be regarded as an advantage since a concentration of 1.3 MAC may be more appropriate than 1 MAC for clinical anaesthesia [3].

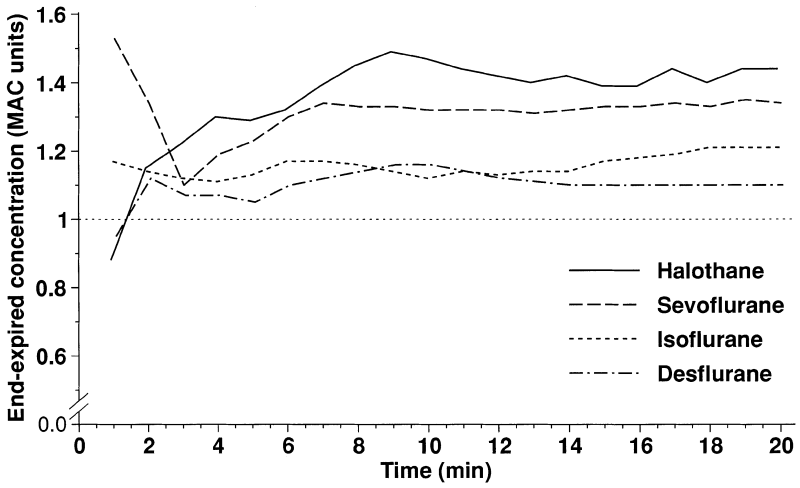

Figure 3 Mean measured end-expired concentrations of the four anaesthetics against time.

Our results confirm the clinical applicability of Mapleson's theoretical study that shows the way to minimise pollution of the operating theatre and the usage of volatile anaesthetics; we found it a simple and easy method with no complex calculation. However, in addition to routine monitoring, it is necessary to follow the end-expired concentration of the anaesthetic; this shows its relation to the patient's MAC value, which serves as a guide to the depth of anaesthesia and the clinical stability of the patient.

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Rate of change in gas concentrations in a charged circle system with absorber

During an operating list, a patient anaesthetised with isoflurane in the anaesthetic room breathed through a circle absorber in theatre that had been disconnected from the common gas outlet. The previous patient had breathed oxygen via the same outlet

Table 4 Predicted [1] and measured values of the time for the end-expired concentration of each anaesthetic to reach 1 MAC, and of the mean concentration after the predicted time: mean (95% confidence limits)

	Halothane	Isoflurane	Sevoflurane	Desflurane
Time to reach 1 MAC; min				
Predicted	4	1.5	1	1
Measured	1—2	<1	<1	1—2
Mean concentration after the predicted time; MAC units				
Predicted	1	1	1	1
Measured	1.40 (1.12—1.69)	1.16 (1.04—1.28)	1.32 (1.14—1.48)	1.10 (1.00—1.20)

Figure 4

during an awake spinal anaesthetic and the circle absorber circuit had not been reconnected. Vapour, nitrous oxide and oxygen monitors showed that a safe anaesthetic was given until the error was quickly noticed and rectified, because the circle had not been flushed with oxygen and was still charged with isoflurane, nitrous oxide and oxygen.

With Ethical Committee approval, a volunteer breathed Entonox mixture for 5 min in the anaesthetic room, was wheeled into theatre and breathed from an Entonox-charged 2-litre circle absorber that had been left disconnected for 30 min. Every 30 s, readings of oxygen and nitrous oxide concentrations were taken using a Datex Engstrom AS73 monitor. Results showed a fall in inspired oxygen concentration from 50% to 25% after 3 min. Inspired nitrous oxide fell from 29% to 17% over the same time.

To prevent a recurrence we now connect the machine end of the circle absorber to the patient end of the circle when oxygen is given via a Hudson mask so that anaesthetists remember to reconnect (Fig. 4).

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Training in difficult airway management

I read with interest the article (Smith and Jackson. *Anaesthesia* 2000; **55**: 1072–5) that began with 'Fibreoptic airway endoscopy has an important role in the management of patients who present difficulties in tracheal intubation and the acquisition of skills in this technique is an essential part of anaesthetic training.' This is a statement with which I wholeheartedly agree.

Having just attended the Difficult Airway Society Annual Scientific Meeting, I was struck by the number of presentations and questions that referred to the lack of training of specialist registrars not just in fibreoptic intubation but the whole approach to the difficult airway. As anaesthetists, our primary skills are in the maintenance of the airway. Managing a difficult airway should be a core skill.

Lack of fibreoptic endoscopic equipment availability was cited at the meeting as a reason for insufficient training. Yet it was also apparent that other techniques were not being taught to trainees. Training of specialist registrars is now unit- or modular-based so that each trainee can be guaranteed a comprehensive training programme. Is it not time for a module consisting of management of the difficult airway to be built into every anaesthetic training scheme? Skills such as mask ventilation, use of the laryngeal mask airway and direct laryngoscopy are developed by every anaesthetist. Equally, confidence with the fibreoptic endoscope used nasally, orally and through the laryngeal mask along with a means of transtracheal oxygenation should be found amongst those of us who spend our lives managing the airway.

I have been a trainee in anaesthesia for 10 years, pre and post Calman, mostly in centres of excellence with experience abroad. In spite of an active interest in the difficult airway, I have found problems obtaining both teaching and experience in this area. In my current base, I have been given the opportunity of training on a difficult airway module. This has involved a basic grounding in fibreoptic endoscopy, hands-on training in bronchoscopy clinics, blind nasal and fibreoptic intubation on anaesthetised patients plus awake fibreoptic intubation for those with difficult airways. I have just completed this module and feel it has enhanced my skills enormously. These skills are not a luxury but should be part of every anaesthetist's training programme.

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A complication of panendoscopy

I would like to report an incident that occurred recently, and had potentially serious consequences.

I had anaesthetised a 67-year-old man for an otherwise uneventful panendoscopy. The airway was secured with a Mallinckrodt 5.0-mm microlaryngeal tube (MLT). At the end of the procedure, the patient was waking up and pharyngeal suction had been performed prior to extubation. I was unable to find the pilot tube to deflate the tracheal tube cuff. The patient was sufficiently awake to self-extubate with the cuff still inflated. This was atraumatic and the patient suffered no ill effects. As the MLT tube was removed, it was evident that the pilot tube had been completely pushed down the oesophagus by the pharyngoscope.

On the Mallinckrodt MLT tube the pilot tube comes off the microlaryngeal tube 15 cm from the tip of the tube (Fig. 5) and would require Magills forceps to rescue it from the oesophagus. I now secure the pilot tube to the MLT before surgery commences. I suggest that it would be safer for the pilot tube to have a higher take-off on the MLT.

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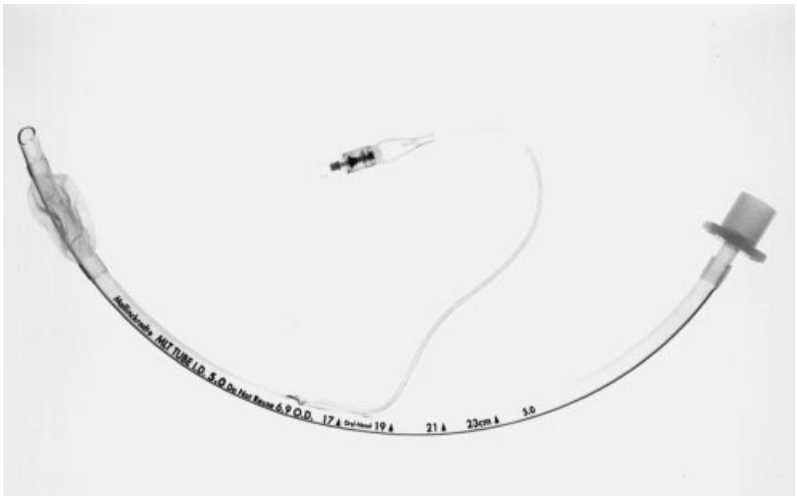

Figure 5

A new device has to be safe and reliable too

Asai and colleagues studied the efficacy of the laryngeal tube (VBM, Germany) during intermittent positive pressure ventilation (Asai *et al*. *Anaesthesia* 2000; **55**: 1099–1102). They found the device effective for this purpose on the basis of a study, which lasted for only 5 min. It is, however, equally important to look at the safety aspect of any new device, especially any new airway adjunct. The authors have failed to address this aspect.

I have had recent experience with a similar device called an Airway Management Device (AMD TM, Nagor Ltd, Isle of Man). This device has been marketed recently. As there was no published evidence of its efficacy and safety (except one case report [1]), a decision was made to undertake an audit project to address these issues. Fifty ASA 1 and 2 patients undergoing various surgical procedures under general anaesthesia were included in this project. Patients with anticipated difficult intubation and risk of regurgitation were not included in this audit.

The AMD TM device was found to be effective. However, significant problems including airway obstruction, congested tongue and regurgitation were also noted. Five of the six patients who had airway obstruction had to be managed with a laryngeal mask airway. Twelve patients had a congested tongue in spite of an intracuff pressure of less than 15 cmH_2O. Visible signs of regurgitation through the tube were seen in two patients. Fortunately, none of them had any signs or symptoms of aspiration. Based on these results, we recommended that a safety evaluation was made before its routine use in clinical practice.

I noted Asai *et al*. inflated the cuffs up to 60 cmH_2O. This huge intracuff pressure can cause congestion, oedema formation and swelling of the tongue secondary to venous and lymphatic obstruction. A swollen tongue can be a cause of postextubation airway obstruction. In addition, venous congestion over a period of time may cause venous thrombosis. A high intracuff pressure may compromise circulation of the tongue, especially in association with systemic hypotension, which is not uncommon under general anaesthesia. Concern has also been expressed over the possibility of lingual nerve damage [1]. Any one of these complications could be potentially serious and all of them are related to high intracuff pressure. Unfortunately, Asai and colleagues did not address these issues. Moreover, the efficacy and reliability of the device can only be assured if the device has been used over a period of time and in different clinical circumstances.

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Reference

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A reply

I am fully aware of the limitation of my report, and stated unambiguously why the efficacy of the device was accessed only for 5 min: 'Initially, we planned to study the laryngeal tube during surgery under general anaesthesia, but the Institutional Local Research Ethics

Committee, on granting approval, instructed us that in view of the lack of published data available for the efficacy of the device, its efficacy should first be assessed only during induction of anaesthesia, to eliminate possible airway obstruction during surgery.' [1]. I also believe that I carefully avoided overstating the efficacy and safety of the device. I stated at the end of the article that: 'In this study, because of ethical concerns, we only studied the efficacy of the laryngeal tube for approximately 5 min before the start of surgery. Therefore, it was not possible to see whether the efficacy of the device decreases over time, for example because of dislodgement of the device during surgery.' Also, I concluded in the summary that 'the laryngeal tube has a potential role in airway management during intermittent positive pressure ventilation for anaesthesia or cardiopulmonary resuscitation.

I adjusted the intracuff pressure to 60 cmH_2O, since it was the pressure recommended by the manufacturer. I cannot answer if this intracuff pressure can often cause ischaemia of the oropharynx. Dr Mandal claims that 60 cmH_2O is a huge pressure and reported that the Airway Management Device often caused tissue ischaemia at the intracuff pressure of less than 15 cmH_2O. However, another device, the laryngeal mask, rarely causes ischaemia at the intracuff pressure of 60 cmH_2O [2]. The intracuff pressure itself is often unrelated to the pressure exerted by the cuff on the oropharynx [2]; other factors, such as the shape, material and compliance of the device cuff, can be more important to increase or decrease the incidence of tissue ischaemia. I acknowledge that any new device has a potential risk of producing complications, but I believe that, until carefully performed studies have been performed, one should not overestimate or underestimate its efficacy and safety.

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A missing throat pack

Throat packs (pharyngeal packs) are commonly employed to prevent saliva or blood from tracking down into the pharynx and the respiratory tract during otorhinolaryngeal, dental and oral surgical procedures [1]. We present an unusual case of a pharyngeal throat pack inadvertently left behind at the end of an operation. The pack was subsequently identified by chest and abdominal radiographs and was retrieved successfully.

A 61-year-old woman with an infected mandibular titanium mesh tray underwent a removal operation under nasotracheal general anaesthesia. The operation was planned to remove the titanium mesh-tray extra-orally with a potential risk of opening up the oral mucosa. A piece of raytec gauze (5 cm × 20 cm) was placed in the oropharynx to serve as a pharyngeal pack. The titanium tray was removed uneventfully and general anaesthesia was reversed as usual. The patient was extubated without any complications. The vital signs were continuously monitored. The patient was breathing spontaneously and remained well oxygenated in the recovery room without any sign of respiratory distress. When it was confirmed that the throat pack was missing and had not been accounted for in the final swab count, an endoscopic examination of the airway down to the vocal cords was carried out in the recovery room. There was no sign of the throat pack or any foreign body impacted in the airway. A chest radiograph was taken to trace the radio-opaque Raytec gauze. A suspicious linear wavy shadow was found in the lower midline above the diaphragm (Fig. 6). A further abdominal radiograph confirmed that the retained

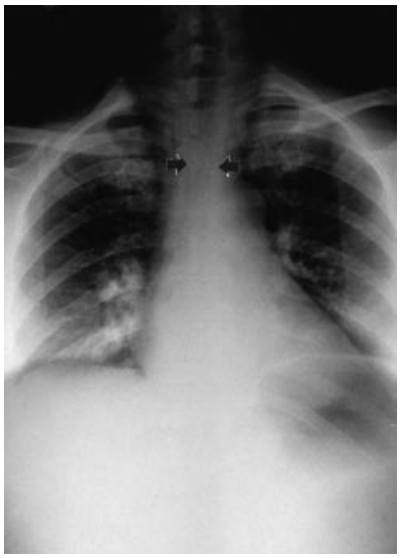

Figure 6 Postero-anterior chest radiograph showing an ambiguous wavy shadow in the midline obscured by the thoracic spine.

Raytec gauze had passed into the stomach (Fig. 7). While the patient was being prepared for endoscopic retrieval of the ingested pack, the patient vomited and the pack was regurgitated in the vomitus. The throat pack was easily identifiable. The rest of her recovery was unremarkable.

A retained surgical pack in the immediate postoperative extubation

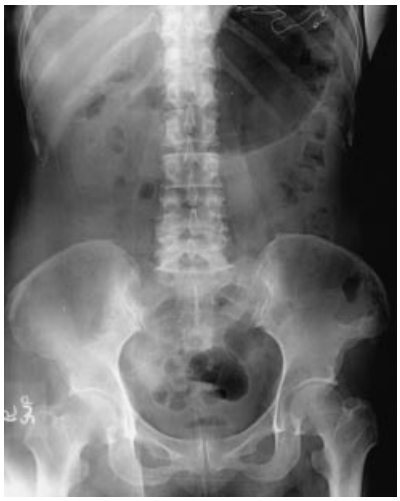

Figure 7 Abdominal radiograph demonstrating the radio-opaque marker of the throat pack in the upper left quadrant among the area of gastric bubbles.

phase is potentially catastrophic in obstructing the airway [2]. Fortunately, the pack was ingested into the gastrointestinal tract instead of being impacted or aspirated into the laryngo-trachealbronchial tree. In this case, the pack was expelled spontaneously by natural body mechanisms and no significant morbidity was inflicted upon the patient. The throat pack could have caused intestinal obstruction if not identified. The 'forgotten' throat pack was probably swallowed at the time of extubation while the patient was still in postanaesthetic stupor. The equivocal shadow in the chest radiograph was likely due to the underexposure of the film, superimposition with the thoracic spine and the reclining position of the patient in the recovery room. The abdominal radiograph with the correct radiation exposure and appropriate supine positioning of the patient allow definitive identification of the throat pack.

A variety of techniques have been proposed in the literature to prevent retention of throat pack [3, 4]. They include measures such as labelling the forehead of the patient, attaching a label at the end of the tracheal tube or fixing the pack onto the tracheal tube at a predetermined site. However, residual throat packs still occur from time to time, leading to complications in the immediate recovery period [5, 6]. A radio-opaque marker within the gauze pack (Raytec gauze which is incorporated with a radio-opaque strip) is essential for identification in case of the pack being accidentally left behind in the patient as illustrated in this case. The anaesthetist and scrub team must be kept well informed about the insertion and removal of the throat pack. Reversal of anaesthesia must not be started until the counting of swabs is confirmed. Alertness to the danger to the airway and a strict protocol in handling of surgical packs by the theatre staff, the surgical team and the anaesthetists are critically important for prevention of similar mishaps.

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Oxygenation during percutaneous tracheostomy

We were interested in your correspondents' description and experience with the Blue Rhino™ percutaneous tracheostomy technique (Scott & Leigh. *Anaesthesia* 2000; **55**: 917). Patients were ventilated with 50% oxygen in air for the procedure, with isoflurane and fentanyl used to provide anaesthesia. In the cases they described, the technique was safe and effective. However, the procedure does carry risks for critically ill patients; in particular impairment of oxygenation. Westphal *et al*. [1] found that the P_aO_2/F_iO_2 ratio fell in all 120 patients in their study comparing surgical, percutaneous and translaryngeal tracheostomy techniques. There is also the potential for catastrophic loss of the airway with any type of tracheostomy. We feel that these risks make the use of 100% oxygen essential during anaesthesia for tracheostomy, regardless of technique.

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Reference

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Improving success with the intubating laryngeal mask airway

The letter (Asai & Shingu. *Anaesthesia* 2000; **55**: 1218–19) describing failure to intubate through the intubating laryngeal mask (ILMA) attributes this failure to a 'swelling in the posterior wall of the hypopharynx' causing partial obstruction of the laryngeal inlet. However, the mid-line sagittal section magnetic resonance image (MRI) accompanying this report shows no obvious abnormality in the hypopharynx. Judging by comparison with 25 randomly selected adult MRI views taken from our Radiology Department, the visible parts of the arytenoids are in their normal position. Since the authors mention seeing the abnormal hypopharyngeal appearance on a pre-operative radiograph rather than an MRI scan, I wonder whether the abnormality was lateral to the mid-line?

Assuming abnormal hypopharyngeal anatomy was indeed present, it might have been worth attempting to intubate this patient using the silicone tracheal tube designed for use with the ILMA [1], particularly since it was found possible to pass a fibreoptic scope into the trachea. The distal tip of this tube is curved into the mid-line, making it less likely to catch on the arytenoids or the cords during attempted passage over a fibrescope.

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Reference

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Airway management/failed intubation drill

We read the correspondence on the subject of difficult intubation (James. *Anaesthesia* 2000; **55**: 1133) with interest. The brevity of the correspondence scarcely did justice to the very important questions it raised. We would like to congratulate Dr James for raising these questions. Generations of anaesthetists have been brought up learning and then teaching personal convictions, expressed by senior colleagues at various meetings or published in the form of an abstract [1].

Tunstall, in 1976, suggested a failed intubation drill for Caesarean section, which was the first of its kind, and was based on what was recommended practice in Aberdeen at the time. Despite little evidence for his convictions, this was seen as a significant step in the right direction, in view of the role of anaesthesia in maternal mortality. Tunstall's drill became enormously popular. Every final FFARCS examination candidate was expected to repeat Tunstall's drill parrot fashion. Dr James has rightly questioned some aspects of Tunstall's drill. Today every obstetric unit in the country has a failed intubation drill of their own, agreed locally. Are they helpful in times of crises? Evidence [2] shows that in nine out of 26 cases the drill was not adhered to and the outcome happily was favourable.

What do we learn from that? Benumof [3, 4] suggests that a dogmatic approach to the various possible scenarios an anaesthetist is likely to find him/herself in is not possible. It might be better to concentrate on the teaching of the basic principles involved in the causes of difficult intubation and their management. Use simulators to rehearse these, including awake fibreoptic intubation and encourage better preparation to deal with the situation that one finds him/herself in. Tunstall also stressed the importance of prior preparation in his drill. Armed thus, we should be left to use our initiative as the circumstances dictate. Evidence [2] shows that pre-operative assessment and therefore preparation was often missing.

To date, many studies have reported the incidence of failed intubation in obstetrics [2], their management and drawn conclusions. They have all been retrospective studies with many limitations, not least the poor quality of records they had to deal with [2]. Moreover, differences of opinion amongst anaesthetists as to the best way to manage failed intubation in obstetrics, including use of the laryngeal mask airway, has been highlighted [2]. There is a large body of anaesthetists who feel, like Dr James, that this issue should be tackled nationally with an OAA initiative, perhaps by setting up a nationwide database and appropriate guidance given. Perhaps one could follow the example of the American Society of Anesthetists and produce a difficult airway management algorithm, which would also take into consideration the place of the laryngeal mask airway under these circumstances.

May I make a suggestion? Extrapolating the data available regarding the number of deliveries per year nationally and the number of Caesarean sections under general anaesthesia, one might expect nearly 100 failed tracheal intubations. This should be an adequate number to draw conclusions from if a prospective study is organised and data are collected as problems occur and are dealt with. Perhaps Benumof [3] is right and there is no room for a dogmatic approach.

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Placement of nasogastric tubes

The use of a split rubber tube passed through the nose into the oesophagus to facilitate the placement of a nasogastric tube has been described [1]. However, this technique may be associated with nasal trauma and bleeding due to the size of the rubber tube, especially if a large nasotracheal tube is required. We would suggest that rather than passing the tracheal tube through the nose, it is passed orally into the oesophagus. The nasogatric tube can then be passed nasally, retrieved in the oropharynx and then passed down the rubber tube. Once the nasogastric tube is placed, the split rubber tube can be removed and the nasogastric tube withdrawn to take up the slack in the oropharynx.

This modification of the technique should reduce the incidence of trauma to the nasal passages.

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Reference

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Cocaine-excited delirium and severe acidosis

We would like to report a case of cocaine-excited delirium in which the patient survived despite extreme acidosis.

A 25-year-old male patient jumped from a first-floor window to escape his

pursuers who were allegedly chasing him with swords. Whilst giving a statement to the police, he suddenly ran off and was apprehended. Although initially conversant with the paramedics, he became drowsy, and in the ambulance had a clonic seizure lasting 1 min.

On arrival in casualty at 13:30 h, there was no eye opening, he was flexing to pain and making incomprehensible sounds. His pulse was 116 beats.min^{-1}, blood pressure 100/40 mmHg, respiratory rate 28 breaths.min^{-1} and temperature 38.3 °C. The initial blood gases revealed a hydrogen ion concentration of 292 nmol.l^{-1} (pH 6.53), P_aCO_2 of 13.13 kPa, base deficit of 35.6 mmol.l^{-1} and a P_aO_2 of 25.61 kPa on 10 l of oxygen via a trauma mask. Because the 12-lead ECG demonstrated tall, peaked T waves, he was assumed to be hyperkalaemic. He was intubated with a rapid sequence induction and manual in-line stabilisation of the head with 2 mg of alfentanil and 100 mg of propofol. Hyperventilation was instituted; he was given 10 ml of 10% calcium gluconate and 50 ml of 8.4% sodium bicarbonate. He had 1.5 l of 0.9% saline over 90 min. The initial plasma potassium level was 7 mmol.l^{-1}, sodium 153 mmol.l^{-1}, bicarbonate 12 mmol.l^{-1}, anion gap 44 mmol.l^{-1}, urea 8.4 mmol.l^{-1}, creatinine 202 μmol.l^{-1}.

By 14:00 h, his temperature had risen to 39.7 °C. He was given a total of 2 mg.kg^{-1} of dantrolene in two separate aliquots within 20 min. Ice packs and a fan were employed as cooling measures. Blood gases taken at 14:47 h revealed a hydrogen ion concentration of 40.1 nmol.l^{-1} (pH 7.4), P_aCO_2 of 4.28 kPa, base deficit of 3.8 mmol.l^{-1} and a P_aO_2 of 42.92 kPa on an F_iO_2 of 0.6. Repeat electrolytes showed a potassium of 5.1 mmol.l^{-1} and an anion gap of 30 mmol.l^{-1}. By 15:00 h, his temperature had decreased to 37.6 °C. X-rays of pelvis, cervical spine, chest and CT scan of head were normal. Bacteriological and biochemical analysis of the CSF was unremarkable.

He was admitted to the ICU and extubated at 20:00 h. Clotting studies were normal and the creatinine phosphokinase level peaked at 8460 μmol.l^{-1} the following day. He was discharged from the ICU the day after admission and from the hospital on the succeeding day with normal renal function. On further questioning, he admitted drinking heavily on the night preceding hospital admission, and to taking cocaine. Toxicological analysis of his urine showed no trace of opioids, benzodiazepines or amphetamines. Cocaine was omitted from the toxicological screen.

The paranoia, agitation and rapidly progressive pyrexia with which this patient presented are features of cocaine-excited delirium [1]. This condition occurs within 24 h of cocaine ingestion in habitual users. Coma and death result without intervention. The prompt administration of hyperventilation, passive cooling, sodium bicarbonate and dantrolene led to a remarkably swift correction of the acidosis and a successful outcome in this case. Survival after such a severe acidosis illustrates that the arterial hydrogen ion concentration gives a restricted view of what is happening at the intracellular and mitochondrial level.

We are reporting this case to increase awareness of cocaine-excited delirium and to suggest one potential management strategy for this potentially fatal syndrome.

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Reference

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Lingual thyroid – another potential airway threat

Drs Buckland and Pedley report effective pre-operative evaluation and subsequent airway management of a patient with lingual thyroid presenting for orthopaedic surgery (Buckland and Pedley. *Anaesthesia* 2000; **55**: 1103–5). They correctly report that whilst the condition is rare, haemorrhage into the airway following instrumentation threatens this group of patients. To the risk of haemorrhage should be added hypoxia following obstruction due to application of cricoid pressure. Georgescu *et al.* reported the management of a parturient with undiagnosed and asymptomatic lingual thyroid who required urgent Caesarean section. Following induction of anaesthesia and application of cricoid pressure, intubation was attempted but was impossible, as was mask ventilation. Severe hypoxia ensued and 'no intermediate level of cricoid pressure would permit oxygen flow' [1]. Only complete release of cricoid pressure allowed salvage of the situation and the survival of mother and child. Cricoid pressure is now a subject of renewed interest [2, 3]. We must be aware that in the silently abnormal airway, mechanisms other than those under investigation can be the cause of airway obstruction during cricoid pressure and that the patient with lingual thyroid faces a dual risk of haemorrhage and obstruction.

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Torticollis following induction of anaesthesia

A 48-year-old woman was admitted for a vaginal hysterectomy. She had no significant past medical history and was

taking no regular medication. Pre-operative physical examination was unremarkable. Anaesthesia was induced with propofol 150 mg and fentanyl 100 μg, and neuromuscular blockade was achieved using vecuronium 8 mg. Following the administration of these drugs, the patient's head twisted towards the right, first noticeable during ventilation with a facemask and which progressively worsened over the following 15 min. Laryngoscopy revealed a grade 1 view and the trachea was intubated without difficulty. We were unable to alter the position of the head due to intense spasm of the right sternocleidomastoid, which felt hard and contracted.

The patient was apyrexial, cardiovascularly stable and capnography readings remained satisfactory. The degree of neuromuscular blockade was assessed using a peripheral nerve stimulator and showed no response to the train-of-four stimulus.

At the end of surgery, residual neuromuscular blockade was reversed with neostigmine 2.5 mg and glycopyrronium 0.5 mg. There was immediate visible resolution of the head deviation such that by the time the patient entered the recovery room, the head was in the neutral position. The postoperative recovery was uneventful.

The following day, the patient complained of minor tenderness along the right sternocleidomastoid. The muscle felt hard but she had a full range of head movements. On questioning, the patient admitted to having a 'lopsided' head (to the right) from an early age for which she had learnt to compensate and correct. It was of such little relevance to her that she had not thought to mention it during the pre-operative assessment.

Muscle spasm during anaesthesia often results from the action of a drug in a specific disease, e.g. succinylcholine and malignant hyperpyrexia, or from the direct action of the drug itself, e.g. fentanyl and muscle rigidity. The clinical picture in our case appears to been due to a drug revealing a latent problem. We presume that neuromuscular blockade antagonised the voluntary muscle action on the left side and resulted in the unopposed contraction on the right, giving rise to torticollis under anaesthesia. An isolated muscle spasm under anaesthesia is very unusual but often has a simple explanation.

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Liver rupture after cardiopulmonary resuscitation during peri-operative cardiac arrest

Liver rupture is an uncommon complication of cardiopulmonary resuscitation (CPR). We wish to report the case of a patient who arrested in theatre, prior to induction of anaesthesia and sustained traumatic liver rupture as a result of CPR.

A 52-year-old schizophrenic man was admitted to our A & E Department with a history of lower back and abdominal pain. There was no history of trauma. He had undergone cataract surgery some years previously but had no other medical history and he was on no medication. On examination, he was found to have a distended and moderately tender abdomen with absent bowel sounds. Pulse rate was 108 $beat.min^{-1}$ and blood pressure was 90/50 mmHg. Blood tests were sent to the lab and revealed a haemoglobin of 9.4 $g.dl^{-1}$ and a white cell count (WCC) of $1.55 \times 10^{9}.l^{-1}$. Urea was 19.1 $mmol.l^{-1}$ and creatinine 289 $\mu mol.l^{-1}$. The patient was catheterised and 10 ml of urine was returned.

While awaiting surgical review, his blood pressure dropped to 60/40 mmHg and this was treated vigorously with 3 l crystalloid and 500 ml Gelofusine®. This increased the blood pressure back to 90/60 mmHg. The patient was reviewed by a senior surgical SpR, who made a diagnosis of a leaking abdominal aortic aneurysm and a decision was then made to proceed to theatre with resuscitation in progress. The patient was seen by the anaesthetic team, which consisted of a consultant, an SpR1 and a SHO, and was transferred directly to theatre. In theatre, a further two 14G cannulae were sited and preparations were made for a rapid sequence induction.

As the patient was being pre-oxygenated, he became asystolic and, after checking ECG leads and gain, CPR was commenced. The patient was intubated with a 9.0 cuffed oral tracheal tube. Atropine 3 mg and epinephrine 1 mg were given intravenously followed by 1 min of CPR in accordance with Resuscitation Council (UK) guidelines. Re-evaluation revealed that the patient was in pulseless electrical activity (PEA). The surgeons performed a laparotomy immediately and a perforated 2-cm gastric ulcer was found on the lesser curve of the stomach, thought to be at least 24–48 h old. The precipitating cause for the patient's asystolic arrest appeared more likely to be severe acidosis secondary to prolonged hypovolaemia than due to acute blood loss, although no arterial blood gases had been performed. Sepsis secondary to a prolonged visceral perforation was thought to be the cause of his low WCC.

The wound was covered and left by the surgeons while resuscitation was in progress. A further 8 mg of epinephrine, 200 ml of 8.4% sodium bicarbonate, Gelofusine® 5 l and fentanyl 250 μg were given. CPR continued for a further 15 min, during which time pulseless electrical activity was present.

An anaesthetic SHO and an anaesthetic nurse, both of whom were ALS trained, performed chest compressions. At this point, the abdomen was re-examined by the surgical registrar and a large volume of blood was noted in the peritoneum, which had not previously been present. Further examination of the abdomen revealed a large 17-cm hepatic split under the diaphragm, which was bleeding profusely. A total of 8 units of blood plus 1200 ml of fresh frozen plasma were given and finally an output was restored. The total time taken to restore an output was approximately 35 min.

The liver was packed with 10 large abdominal packs and the gastric ulcer was oversewn. Anaesthesia was maintained with 0.5% isoflurane in air/O_2 mix and central venous and

arterial cannulation were performed. Epinephrine was commenced at 50 $\mu g.min^{-1}$ to maintain a blood pressure of 120/80 and heart rate of 109 $beat.min^{-1}$ despite a CVP of 18 mmHg. The estimated total blood loss was 6 l.

Surgery was concluded about 30 min after the restoration of an output and the patient returned to ITU postoperatively, where he was fully sedated and ventilated overnight. Here bloods were rechecked revealing a haemoglobin of 10.1 $g.dl^{-1}$, WCC $0.97 \times 10^9.l^{-1}$ and platelet count of $99 \times 10^9.l^{-1}$. His clotting was deranged with an INR of 2.6 and APTT ratio of 2.01. A chest X-ray was performed but no rib fractures were seen then or indeed on subsequent X-rays.

Overnight, he was given a further 2 units of blood to transfuse him to a haemoglobin of 13.0 $g.dl^{-1}$ and 1200 ml of fresh frozen plasma to improve his coagulation. However, he remained in oliguric renal failure. The following day, he was transferred to our regional liver unit where he underwent a pack change 2 days later. A further two laparotomies were carried out over the following 2 weeks but the patient was eventually discharged from intensive care, albeit with some mild cognitive deficit, and eventually returned to Greenwich Hospital about 8 weeks after the original incident.

Damage to visceral structures is a rare but documented complication of cardiopulmonary resuscitation, usually with devastating consequences. Damage to spleen, stomach and liver have been reported. We conducted a *Medline* search from 1966 to 2000 and are aware of two reports of liver rupture following CPR; however, both of these were in association with the administration of thrombolysis [1, 2]. One of these cases had a fatal outcome. We also found a further two reports of liver laceration [3, 4], one of which was in a pregnant woman who also died. We found no instances of this condition occurring during an intra-operative cardiac arrest. In cases where the abdomen is not open, diagnosis is much more difficult and the condition is probably under reported. Two of the above cases were diagnosed on clinical grounds. Of the remaining two, one was diagnosed during thoracotomy for a pulmonary embolectomy and one at autopsy. It is important to use the correct technique while performing CPR to avoid damage to adjacent structures. In this case, it is worth noting that the injury to this patient's liver was not associated with overlying rib fractures. The possibility of visceral damage due to CPR should be considered in cases of prolonged PEA. In this case, the blood loss associated with this patient's liver injury was probably the cause of his persistent PEA and it was only when it was discovered that the patient had profuse bleeding from a ruptured liver that effective therapy was given and an output restored. Finally, in the case of intra-operative cardiac arrest when the abdomen is open, some authorities have recommended the early use of internal cardiac massage and also of thoracotomy and cardiopulmonary bypass (if available) even in non-traumatic intra-operative cardiac arrest [5]. This may have been beneficial in his case and certainly may have prevented the liver rupture.

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The use of sevoflurane in acute intermittent porphyria

Drugs have been classified as unsafe in porphyrias because they have been shown to be porphyrinogenic in animals or *in vitro*, or have been associated with acute attacks in humans. Of the halogenated inhalation agents currently available, halothane and probably isoflurane are thought to be safe with enflurane labelled as 'use with caution only'. The safe use of sevoflurane has never been described in acute intermittent porphyria (AIP) [1]. We describe two patients in whom there was no adverse reaction to the use of sevoflurane on clinical grounds.

Case 1
A 44-year-old female patient with a documented history of AIP, but who was otherwise well, underwent an excision biopsy and split skin graft of a lesion on her left leg lasting 40 min. Anaesthesia was induced with propofol 200 mg and fentanyl 100 μg, the airway was secured with a size 3 laryngeal mask and anaesthesia was maintained with sevoflurane 2–3% in 50% N_2O. The surgery and anaesthesia were uneventful. Following surgery, she was prescribed intramuscular morphine and prochorperazine to provide analgesia and anti-emesis. The patient made an uneventful recovery from surgery and at no time exhibited symptoms or signs associated with an acute attack of AIP. Follow up in clinic was unremarkable.

Case 2
The second patient, a 33-year-old female, also had a diagnosis of AIP. She had endured years of acute porphyric crises with severe abdominal pain requiring referral to pain clinic, hypertension, neuropsychiatric problems and a peripheral neuropathy, which had necessitated the use of a wheelchair. Two months prior to her elective admission for surgery, she had been admitted for abdominal pain control under the physicians with input from

the hospital pain service. Her regular medications were amitriptyline 25 mg once daily and fentanyl patch releasing 50 μg hourly.

The patient suffered long-standing menorrhagia and dysmenorrhoea, and was listed for total abdominal hysterectomy and bilateral salpingo-oophorectomy. After securing intravenous access and instituting routine monitoring, anaesthesia was induced with fentanyl 100 μg, propofol 200 mg and atracurium 40 mg. The trachea was intubated and anaesthesia was maintained by sevoflurane at an end-tidal concentration of 1.3–1.8% in 50% N_2O supplemented by morphine 10 mg during the 50 min procedure. Postoperative analgesia was provided by morphine PCA and anti-emesis by cyclizine. In the postoperative period, the patient complained of no symptoms and exhibited no signs of an acute porphyric crisis and was discharged home as per routine. At 8-week follow up, the patient remained well with no further attacks.

The frequency of acute attacks in AIP is greater amongst women. Attacks rarely occur before puberty or after the menopause, with the highest frequency in the 3rd and 4th decades [2]. Both patients belonged to this sex and age group and suffered no reported ill effects. In case 2, the lack of postoperative exacerbation of AIP may have been due to 'surgical menopause' and it remains to be seen whether the use of hormone replacement therapy influences the future frequency of attacks.

With rapid elimination and minimal metabolism, it would appear that modern inhalation agents, with the exception of enflurane, should be safe. However, James and Hift suggest that repeated or prolonged exposure may prove to be dangerous [1]. More detailed reports, especially involving longer or repeated exposure to sevoflurane, are obviously needed before sevoflurane could be considered truly safe and caution is advised.

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Prediction of degree of hypomagnesaemia during general anaesthesia

Sasaki and colleagues have demonstrated a reduction in serum magnesium associated with the administration of magnesium-free crystalloid, and suggest that magnesium supplementation should be considered when large amounts of magnesium-free fluid is administered (Sasaki *et al*. *Anaesthesia* 2000; **55**: 1137–8). They do not tell us whether they measured total or ionised magnesium. From the quoted serum levels, it has to be assumed that they measured ionised magnesium. They tell us that magnesium levels fell from a mean of 0.51 $mmol.l^{-1}$ to 0.45 and 0.41 $mmol.l^{-1}$ following the administration of 1500 ml and 3000 ml of fluid, respectively. Unfortunately, they do not inform the reader of the lower limit of the reference range for ionised magnesium. This has been quoted as 0.44 $mmol.l^{-1}$ [1].

Previous studies have documented changes in serum magnesium in relation to anaesthesia. In a study comparing magnesium-free and magnesium-containing crystalloids, the serum magnesium level fell significantly in both groups [2]. General anaesthetic agents have been suggested as a cause of significant falls in ionised magnesium intra-operatively [3]. The relevance of these changes in serum magnesium is not clear.

We feel that it is important to make the distinction between statistically significant and clinically significant changes, and the suggestion that this fall in ionised magnesium may warrant treatment requires comment. Ninety-nine per cent of magnesium is intracellular. Serum magnesium represents only 0.3% of the total body magnesium and serum magnesium may not reflect levels in other body stores [4]. Using a magnesium-loading test as a reference, it has been suggested that ionised magnesium is an insensitive marker of functional hypomagnesaemia [5]. Thus a low ionised magnesium does not necessarily equate with magnesium deficiency.

In acutely ill patients, a low serum magnesium has been associated with an increase in mortality [6], but this cannot be extrapolated to well patients undergoing elective surgery. The development of ion selective electrodes allows us to measure the physiologically active component of minerals, but the clinical relevance of these measurements is unclear and requires clarification before we can accept the suggestion of magnesium supplementation in this setting.

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A reply

We are grateful for the comment. We measured whole blood ionised magnesium concentration (mmol.l^{-1}) in our report using the NOVA-8 (Nova biomedical, Waltham, MA, USA), and the lower limit of clinical normal range has been considered as 0.45 mmol.l^{-1}. Although the clinical relevance of measurements of ionised magnesium was argued in the comment, it is generally accepted that physiological processes are dependent on ionised cationic activity (i.e. ionised calcium, ionised magnesium), not the fraction of total cation which is bound to protein. Since cell membrane is permeable to ionised magnesium, the gradients between extracellular and intracellular ionised magnesium could approach equilibrium within minutes to hours without active transport systems. Measurements of whole blood (extracellular) ionised magnesium not only give the physiological magnesium fraction in extracellular space, but also reflect the intracellular ionic magnesium [1]. Thus, ionic magnesium is a better discriminator of a patient's magnesium status than total magnesium.

Zuccala *et al.* [2] measured the intracellular magnesium concentrations in surgical patients under general anaesthesia using atomic absorption spectrophotometry. They found that intracellular magnesium depletion correlated well with the extracellular (serum) magnesium concentration, and that decreases (approximately 10%) in both intracellular and extracellular magnesium concentration predicted postoperative worsening of ventricular arrhythmias. Consequently, we still suggest that magnesium supplementation should be considered when a large amount of fluid is infused.

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A complication of non-invasive positive pressure ventilation

The use of non-invasive positive pressure ventilation (NIPPV) is becoming increasingly common. We wish to report a patient with a hiatus hernia who was treated with NIPPV in whom a massive intrathoracic dilatation of the stomach was clinically identical to a tension pneumothorax.

An 82-year-old man underwent an elective right hemicolectomy for villous adenoma. Pre-operative investigations included a chest X-ray (Fig. 8) which was thought to be within normal limits. Three years previously he had undergone diagnostic endoscopy for dyspeptic symptoms of 25 years' duration. Erythematous gastritis and a hiatus hernia were diagnosed and he received *Helicobacter pylori* eradication therapy. He still took occasional cimetidine.

Surgery and anaesthesia were uneventful and he returned to the surgical wards. On the fourth day postoperatively, he deteriorated with increasing dyspnoea and confusion. His temperature rose to 38.0 °C and his respiratory rate to 40 breaths.min^{-1}. He was wheezy with diminished breath sounds on the left. Oxygen saturation (S_pO_2) decreased to 80% breathing from a facemask supplied with 5 l.min^{-1} oxygen. A chest X-ray (Fig. 9) was interpreted to show a collapsed left lower lobe with elevated left hemidiaphragm.

He was transferred to the intensive therapy unit where he was treated with chest physiotherapy, antibiotics and

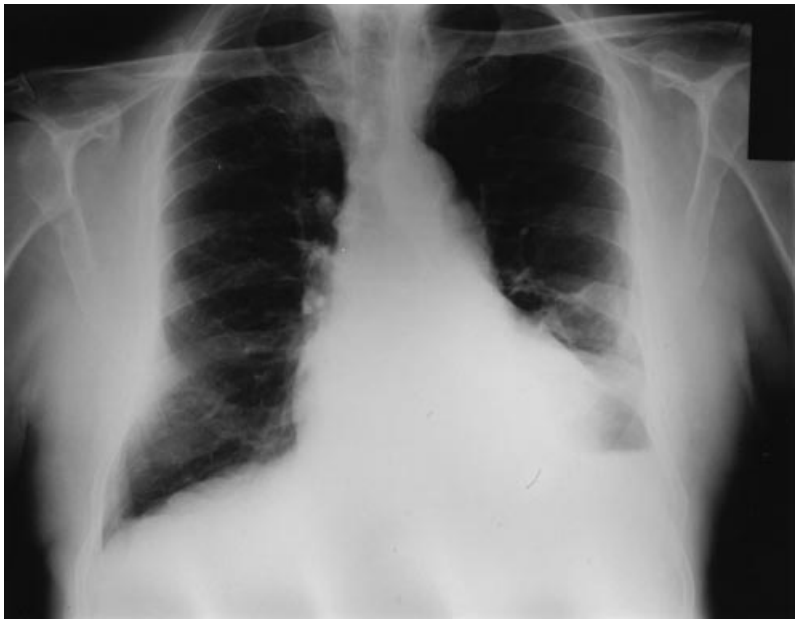

Figure 8

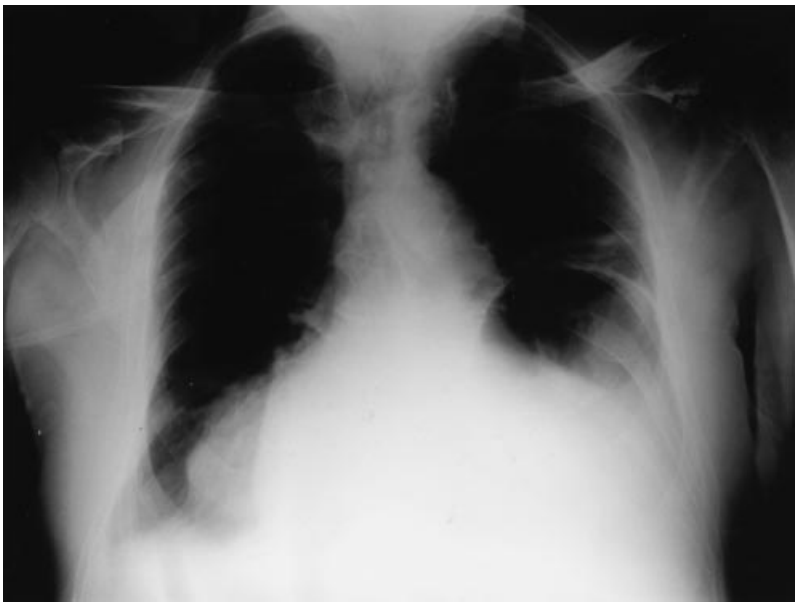

Figure 9

bronchodilators. NIPPV via a facemask was commenced. Over a period of 36 h his condition improved significantly.

However, in the night his condition suddenly deteriorated. He became increasingly dyspnoeic, tachycardic and hypotensive. Clinical examination revealed a tracheal shift to the right. On the left side there was hyperexpansion with fullness of the supraclavicular fossa, chest movement was reduced and breath sounds were absent. The apex beat was displaced to the right and the heart sounds were distant. S_pO_2 dropped to 80%. The clinical diagnosis of a tension pneumothorax secondary to NIPPV was made and a 24 gauge intercostal drain was inserted into the fourth intercostal space in the midaxillary line. His condition did not improve significantly and he required intubation and ventilation. A nasogastric tube was inserted resulting in a small improvement in his condition. The chest X-rays were reviewed by the radiologists the next morning when the diagnosis of a grossly distended hiatus hernia was made. The patient subsequently underwent a thoraco-abdominal exploration when a very large rolling hiatus hernia was repaired.

To our knowledge, this complication of NIPPV in a patient with a hiatus hernia has not been previously described. We would like to emphasise caution in the use of this therapy in such a patient in the future.

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ECG dots and regional blocks

A 38-year-old man presented for a right shoulder arthroscopy and acromoplasty as a day case procedure. An interscalene block is routinely performed for these cases for analgesia and to prevent muscle spasm postoperatively. For the block, a Braun Stimuplex DIG nerve stimulator with a 22-g short-bevel, insulated, 50-mm needle was used with an extension lead connected to an ECG electrode placed on the chest wall. Medicotest (Blue Sensor disposable electrodes) manufactured the ECG electrode. Unexpected difficulty was encountered with obtaining any muscle twitches and so the equipment was checked and the needle changed, all to no avail. It was only when the ECG electrode was replaced that it was noticed there was no conductive gel in the central area, although it was firmly adherent to the skin. Once this was replaced, the block was performed easily.

This illustrates that when using ECG electrodes for regional techniques, the conductive gel area should be checked to ensure that a good contact is made and potential neural damage avoided from unnecessary attempts. It is also of note that this particular nerve stimulator does not indicate if there is poor electrode contact as with some more modern stimulators.

We contacted Medicotest who said this use of their electrode was reasonable and the problem lay with the incorrect storage of the electrodes. In dry air they would expect the electrodes to dry up within 1–2 days but if correctly stored within the foil sachet they would not expect this problem to occur. In our centre, the ECG electrodes are all stored in open containers following opening of the packet.

We should be interested to hear if anyone else has encountered a similar problem, either with these or other ECG electrodes.

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A faulty PCA device

In 1992, in common with many other UK hospitals, we responded to the Royal College of Surgeons and College of Anaesthetists joint report on pain after surgery by setting up an Acute Pain Service. The backbone of this service was the provision of PCA and ward-based epidural infusion analgesia systems. At the time, the most appropriate device for our needs and expenditure was the Abbott Provider 5500 pump. These pumps have given us 8 good years of service. We carefully audit any errors that occur in the usage of these pumps to enable us to deal with any training matters that may arise from a ‘near miss’. We had our most serious near miss today; one that I feel may be of

interest to other departments using such pumps.

It is our standard practice to use morphine 1 $mg.ml^{-1}$ in 100-ml saline bags. With this particular patient, it was programmed to administer a 1-mg bolus every 5 min. This patient was receiving a 1-$mg.h^{-1}$ background infusion because of pre-operative use of high doses of morphine. The patient underwent his operation and the initial 12-h period was uneventful. His pain control was sufficient for his needs and observations were stable.

Following surgery on our routine acute pain ward round, it was noted that the pump had delivered 12 mg of morphine in the previous hour and the pump was continuously bleeping. The patient stipulates he did not activate the button within this hour as he was pain free, but was indeed distressed by the continuous noise. Fortunately, the patient came to no harm from this and the pump was replaced for a functioning one. This pump and patient button has been returned to Abbott laboratories for investigation. We are assuming the problem lies with the wiring of the button.

A bleeping Abbott provider 5500 is not a particularly discreet device, but this failed to be registered as anything other than a nuisance by the ward staff. This case highlights the importance of good nurse training and accurate patient observation.

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Pressure for success

I read with interest the recent letter (MacKenzie. *Anaesthesia* 2000; **55**: 1136–7) describing a novel high-pressure flush device for arterial and venous pressure transducers. The device is elegant in its simplicity and very effective for the purpose described. In the interest of safety and to prevent inadvertent dislodgement of the pressurised syringe during patient transfer, it would be prudent to replace the syringe shown in the photograph with a 10-ml luer lock syringe. This would be firmly attached to the three-way tap and less likely to be dislodged leading to blood bleed-back and catheter clotting. Furthermore, the limited 10-ml volume pressure reservoir will be rapidly exhausted if the fast flush mechanism is activated during transfer. Users of the device should therefore remember to recharge it with heparinised saline following fast flushing in order to re-establish pressurisation and prevent catheter clotting. Replacing the glove fingers with an elastic or rubber mechanism of known and reproducible properties would allow the syringe to be calibrated in terms of both driving pressure and volume delivered. I hope Dr MacKenzie's idea is taken up commercially.

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Ventilator failure due to non-ISO-standard components

Anaesthesia was provided for emergency exploration of a bleeding tracheostomy wound using a Modulus SE anaesthetic machine and 7900 SmartVent ventilator (Datex-Ohmeda, Hatfield UK). Thirty-five minutes into the procedure, the ventilator bellows collapsed and alarms sounded indicating disconnection of the breathing system. Manual ventilation was immediately instituted, whereupon the patient could be ventilated easily, and the monitors demonstrated no disconnection. Careful examination of the bellows and its connections revealed the source of the leak.

The bellows were connected to the absorber assembly of the circle system via three components: 120 cm of corrugated tubing, a breathing system filter, and a 6-cm length of tapered, flexible tubing. The latter is supplied with the breathing system for this purpose, and is described as a 'DABC non-conductive single use anaesthesia ventilator tube' (Part no. 225 317 5800; Intertech, FL, USA). The tube has a non-ISO standard taper, with an internal diameter of 3.4 cm tapering to 2.5 cm, and is designed to connect the female 22-mm fitting of the ventilator filter to the male 22-mm fitting of the absorber assembly by fitting over the external core of both.

Figure 10. View of the absorber assembly illustrating the segment of tapered tubing and site of gas leak (arrow).

The height of the absorber assembly may be adjusted by sliding it up and down a vertical rod. If the assembly is near the top of its excursion, and is pushed towards the anaesthetic machine by nearby persons or equipment, a shearing force is exerted on the tapered tubing and filter, causing a leak to occur at the wide end of the taper (Fig. 10).

Adjusting the position of the absorber and securing the connections rapidly resolved the problem, and there were no adverse consequences; however, the design of the apparatus was a contributing factor to a potentially critical incident. A safer alternative would be to replace the tubing with two standard ISO-compatible 22-mm connectors joined by a short length of flexible 'concertina' tubing.

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A reply

Thank you for giving me the opportunity to reply to this letter. The text and photographs show a Datex-Ohmeda

Mk5a absorber with attached bag/vent switch. Fitted between the bag/vent switch and the bellows is a tube and filter assembly that seems to be the cause of the problem. All of the Datex-Ohmeda fittings on this equipment are ISO 22-mm standard taper. The Flexible hose assembly described in the letter is made by the USA-based Intertech Company and marketed in the UK by Sims-Portex. Datex-Ohmeda are unable to comment on the designs of a competitor product.

Our advice is that the bellows are connected to the absorber/vent-bag switch by an ISO-standard 22-mm flex tubing without a filter.

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Internal jugular vein cannulation doesn't have to be a pain in the neck

Cervical plexus blocks are widely used by vascular anaesthetists to produce regional anaesthesia for carotid endarterectomy surgery [1]; however, it can have wider applications. Owing to recent increase in demand on the anaesthetic service in our hospital for central venous access in awake surgical patients on the ward, we have been using superficial cervical plexus block with great success to provide anaesthesia for internal jugular vein puncture.

It is simple to perform [2], reliable, safe and produces anaesthesia over the neck and shoulder providing increased comfort for the patient and improved conditions for successful cannulation. We would like to outline how it is done and the possible complications in the hope that this block will become more widely used.

The block is performed with the patient lying supine with the head extended and turned to the opposite side. Having cleaned the skin, the midpoint of the posterior border of sternocleidomastoid is located and 2–3 ml of lidocaine 1% is injected in the subcutaneous plane. Further local anaesthetic is infiltrated in the cranial and caudal direction along the posterior border of sternocleidomastoid to a total of 10–15 ml. This blocks the superficial branches of the ventral rami of the first four cervical vertebra and produces a field of anaesthesia running diagonally from the occiput through the lower ear to the tip of the chin. The inferior border runs from the sternoclavicular joint along the inferior boarder of the clavicle and then down the lateral side of the shoulder. This allows the operator a large area of anaesthesia in which to locate the internal jugular vein using a high or low approach and then to stitch the line securely in place.

With a superficial block, there are very few complications as the injection is only subcutaneous. The main ones are intravascular injection of local anaesthetic into the external jugular vein and haematoma formation. Should the injections go deeper, epidural, subarachnoid, intravenous and intra-arterial injection have all been reported. Phrenic, vagus, glossopharyngeal and cervical sympathetic chain nerve involvement is also known although these are associated more with a deep cervical plexus block.

We hope by publicising this block it may become more widely used as we have found it useful, easy to do and a consistent improvement in anaesthesia on local infiltration at the point of skin puncture for every needle or cannulation attempt.

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Peri-operative peripheral nerve injury

The review of peripheral nerve injuries associated with anaesthesia (Sawyer *et al.* *Anaesthesia* 2000; **55**: 980–91) does not mention the major contributions to this subject by Dr Warner and colleagues at the Mayo Clinic. In a series of papers over the last decade, Warner *et al.* have used meticulous archival information contained in the medical records of the Mayo Clinic. These have been kept for decades, and their utility is a salutary reminder that culling of medical records is not helpful.

Their papers included retrospective and prospective studies, with large patient populations. A retrospective study of 1129 692 patient records covering non-cardiac procedures has been performed [1], which identified that 1 in 2729 patients sustained an ulnar nerve deficit, sensory or motor, with 9% bilateral. Men at extremes of body habitus with prolonged hospital stay were susceptible. Recorded patient position or anaesthetic technique were not identified as risk factors.

A more recent prospective study has been performed evaluating ulnar neuropathy in 1502 patients [2]. This study showed that seven patients developed a deficit, primarily in men between the ages of 50 and 75 years. They speculate that gender-dependent anatomic differences may account for these findings. Other workers have shown that hypermobility of the ulnar nerve is a predisposing factor, but not the radiological configuration of the postcondylar groove [3].

Warner's group has also investigated the development of postoperative lower limb nerve deficits. Again, the size of the studies is impressive. A retrospective study of the postoperative progress of 198 461 patients undergoing one of 56 surgical procedures performed in lithotomy position showed that prolonged surgery in that position for over 4 h was a risk factor for lower limb motor deficit, occurring at a rate of 1 in 3608 [4]. Other risk factors identified were patients with a low body mass index (20 or less) and peri-operative smoking habit.

A prospective study of 991 patients again concluded that prolonged time in lithotomy position (more than 2 h in this study) was a 'major risk factor' with 15 patients demonstrating lower limb

nerve deficit [5]. Time in 'pathological position' should therefore be added to the long list of aetiological factors mentioned in the review. Other factors may contribute. Anaesthesia using nitrous oxide may provoke neurological deficit [6].

There is an array of causative and contributory factors interacting in the development of a particular lesion in a particular patient. Factors may be causal or confounding.

Aspects of the peri-operative period during which complications develop, evolve or resolve can be reviewed as components of a non-linear system ('chaotic') with a variety of potential outcomes exquisitely sensitive to initial conditions [7]. Identifying them, with their relative contribution to a particular case, is and remains a great challenge.

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Pressure sores and epidurals – blame the sheets?

In 1992, seven unilateral, full-thickness, heel sores, which took weeks to heal, occurred following lumbar epidural analgesia for postoperative pain in my patients having total abdominal hysterectomies. The sores occurred in patients at the local private hospital but not in my hysterectomy patients at the National Health Service hospital with the same anaesthetic technique and excellent nursing care in both centres.

I had changed from an epidural infusion of 0.25% bupivacaine to a combination of bupivacaine 0.1% and fentanyl 2 $\mu g.ml^{-1}$; patients still had numb legs but were now able to move them in the postoperative period and were encouraged to do so.

Polyester cotton sheets were used at the private hospital, pure cotton in the National Health Service. To see if this could be the reason, Leicester medical students were given a project and rubbed their heels up and down both sheets without knowing which was which. Polyester cotton made the heels hotter than the cotton with equal 'rubs'. Attempts to measure temperature failed; nothing was written up.

The problem was solved not by changing sheets but by inserting a low thoracic instead of lumbar epidural at the T_{10-11} space and using a continuous infusion of 6 $ml.h^{-1}$ with a bolus facility of 6 ml, 25 min lock-out; foot and heel sensation is maintained.

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Logic in the safe practice of spinal anaesthesia – 1

I should like to congratulate Professor Reynolds on her editorial on the safe practice of spinal anaesthesia and the timely warning (Reynolds. *Anaesthesia* 2000; **55**: 1045).

I first became interested in the neurological consequences of spinal anaesthesia in 1993. I wrote a letter to *Anaesthesia* in which I drew attention to the high incidence of paraesthesia associated with spinal anaesthesia using atraumatic needles [1]. I also asked for respondents who had had similar experience of paraesthesia to correspond with me. In the intervening years, I have had numerous reports of paraesthesia associated with and without neurological damage from all over the world. The high incidence of paraesthesia related to a CSE technique using the $L_{2/3}$ interspace I reported in 1995 [2]. I postulated that the needle may be the explanation for this phenomenon. I reiterated my concern in a letter in your columns in 1998 [3].

Subsequent tragic case reports presented to the annual meeting of the Obstetric Anaesthetists Association, Winchester, UK, 2000, by Professor Reynolds have done nothing to reassure me that spinal anaesthesia is being performed safely. It is salutary that the explanation for the paraesthesia and nerve damage may be related to the apparent difficulty of correctly locating at what level spinal anaesthesia is being performed [4]. It has been my intention to promote the safe practice of spinal anaesthesia, a technique of inestimable value to anaesthetists. I would hope not to see Woolley and Rowe revisited.

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R. Ability of anaesthetists to identify a marked lumbar interspace. *Anaesthesia* 2000; **55**: 1122–6.

Logic in the safe practice of spinal anaesthesia – 2

I am rarely moved to comment on the content of your normally excellent journal's editorials. However, despite the fact that I take such editorials at face value (an academic perspective on issues raised by a paper or papers that appear in that edition), I feel obliged to protest at Professor Felicity Reynolds' recent editorial on 'logic' in spinal anaesthesia (Reynolds. *Anaesthesia* 2000; **55**: 1045).

Essentially, she sounded surprised that anaesthetists cannot identify a given intervertebral space [1] and implied that if we didn't all employ the services of a radiographer and an image intensifier, we could look forward to a future of litigation consequent to damage to the spinal cord during spinal anaesthesia. Personally, I have never been certain where I'm putting my spinal needle, or indeed my epidural needle for that matter. If that sounds cavalier then perhaps I should qualify things a little: I know roughly where the needle is but not exactly. Whenever I insert sharp instruments into patients I do so carefully. When I nominate a space to write on the anaesthetic record, I use the 'nearly equal' symbol to indicate that the level is approximate.

Professor Reynolds quotes two references [2, 3], both case reports, to support her assertion that there are innumerable disasters out there waiting for compensation. How many spinal anaesthetics are given in the UK annually? I'm reasonably certain that the figure will be in the hundreds of thousands. Now, bearing in mind that those inserting said spinals are probably worse than professional darts players (in terms of accuracy anyway), am I worried? No, I am reassured.

The editorial implies that because we don't really know where we are putting our needles, we should worry more. Surely, the evidently low morbidity of the technique despite such ignorance should have the opposite effect? She suggests that several textbooks should be rewritten. Perhaps she is right, but if so, I sincerely hope that the new authors maintain a rather more realistic perspective.

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A reply

I thank Dr Turner for his kind remarks and am grateful to many colleagues who have emailed in support of my editorial. I am glad to have the opportunity to respond to Dr Flatt, whose letter will serve a useful purpose if it continues to focus attention on the possibility of conus damage. I am unsure why he thought I was surprised that identifying interspaces was difficult. I have long been familiar with the problem, which is compounded by uncertainty about the length of the spinal cord, and I therefore always taught trainees to choose a lower lumbar interspace when conducting spinal or combined spinal epidural anaesthesia.

The important message is that both Van Gessell *et al.* [1] and Broadbent and her colleagues [2] found the commonest error of identification lay in believing a selected space to be lower than it actually was. Figure 11 is derived from table 1 of the latter article. Readers will note that the distribution of the actual space is not centred on the assumed space, but the mean, the median and the mode are all one space higher than assumed. This cannot be due to chance, but rather to a systematic error that

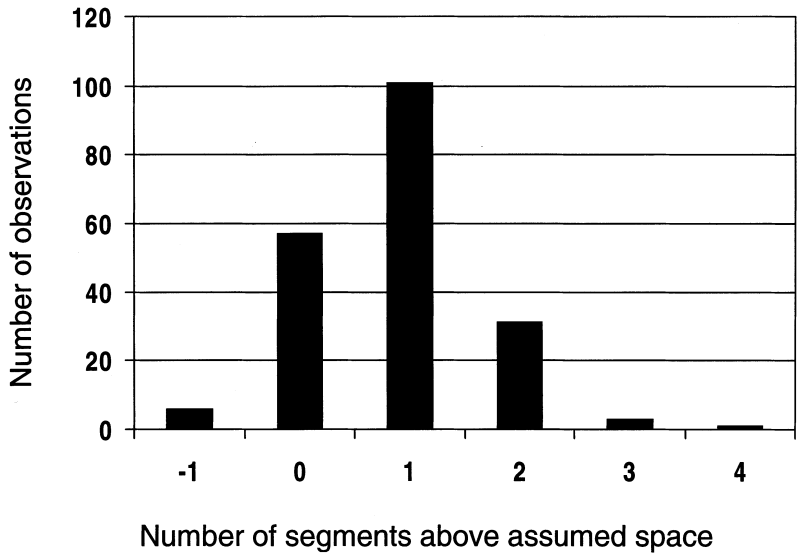

Figure 11. Identification of lumbar interspaces by Oxford anaesthetists. The horizontal axis shows the position of the actual interspace, identified by MRI, relative to the assumed space, in 200 observations. Data from Broadbent *et al.* [2]

explains why dangerous mistakes may occur.

I myself have encountered seven women, from all over the country, who suffered damage to the conus medullaris following spinal anaesthesia that was intended to be sited at $L_{2/3}$ or thereabouts [3]. Six of these cases were described in an abstract cited as reference 16 of my editorial. Since publication of my editorial and presentation of these cases, I have been told of others. Moreover, I am certainly not the only anaesthetist who does this type of medical negligence work, and not all cases are published or become the subject of litigation, so it may be assumed that more exist. This being so, it is surely inappropriate that the authors of some texts appear to condone the selection of an upper lumbar interspace ($L_{2/3}$ or even $L_{1/2}$) for spinal needle insertion, particularly in women, in whom the cord extends on average lower than in men [4].

I agree with Dr Turner that spinal anaesthesia is too valuable a technique to be consigned to limbo once more. I assume it may not be possible to avoid the nerve root paraesthesiae that he has described, and would not wish to banish atraumatic needles, whose ability to reduce headache has revolutionised the practice of obstetric anaesthesia. I do feel, however, that the risk of conus damage can be reduced. As imaging equipment is unsuitable for use in the hurly-burly of routine anaesthesia we cannot be certain of our selected interspace, but we can at least aim low.

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Prolonged epidural catheterisation – a multidisciplinary technique

We read with interest the article on methods of epidural catheterisation (Aida & Okada. *Anaesthesia* 2000; **55**: 499–500). Although the authors are to be congratulated on their methods to minimise infection, prolonged catheter survival seems an elusive goal as this group and others find an average catheter survival of only 20 days [1]. In the field of chronic pain management, this time period is disappointing. We recently audited our domiciliary epidural catheter service for chronic non-malignant pain management and found considerably longer catheter duration averaging 20.5 weeks without serious adverse effects and would like to share a report of our data and techniques.

Patients at the Dudley Pain Centre with non-malignant chronic pain who had had domiciliary epidural catheters inserted between July 1996 and April 2000 were recruited into a retrospective study and provided 65 catheter insertion episodes. A third party outside the pain service (K.V.) was invited to review the case notes on these patients. Mean (range) age was 49 (24–76) years; 31 were female, 34 male; 51 had mechanical low back pain and 10 the failed back syndrome. The commonest drug used was morphine (46); others used were bupivacaine (20), clonidine (9), baclofen (2), fentanyl (2) and buprenorphine (1). Catheter duration ranged from 2 days to 2.5 years with a mean of 20.5 weeks. The commonest reason for catheter loss was migration (27); other reasons were inadequate pain relief (8), blocked catheter (6), suspected infection (5), pain on injection (4), leaking catheter (3). Eight were removed electively as patients proceeded to intrathecal drug implantation. The five clinical infections were all superficial, the organisms identified were *Staphyloccocus aureus* (2) and *S. epidermidis* (2) and none had long-term sequelae.

All catheters were inserted by pain consultants using similar technique and undertaken together with theatre teams experienced in this procedure. All patients were screened for carriage of MRSA prior to the procedure by dedicated pain nurse practitioners. Strict asepsis was used but without antibiotic prophylaxis. Under local anaesthetic infiltration, a Portex epidural catheter was passed from the lumbar spine to between 5 and 10 cm into the epidural space. The catheter was then tunnelled subcutaneously towards the flank using three sequential entries of a Tuohy needle. The catheter was sutured to the skin with non-absorbable material to minimise migration and an epidural filter was applied. Skin puncture sites were dressed with Mefix and the catheter port taped to the skin over the side of the abdomen.

Patients were then admitted to a designated area of a pain ward in which all patients admitted had screened negative for MRSA. Using strict aseptic technique, trained pain ward nurses trialled the patients with the variety of epidural medications described above administered as a bolus in a single blind fashion with patients not informed of the type of medication and without access to their drug chart. If a medication or combination produced clinically significant relief, deemed as 50% reduction in pain without unacceptable side-effects, then the patient was taught by the nurses how to administer safely the medication epidurally themselves using aseptic techniques over several days. They were instructed to refrain from bathing and only to shower after applying cling film to the mefix dressing. They were educated on signs of infection.

Patients were reviewed by the pain nurse practitioners at two weekly intervals. At this review, the satisfaction with treatment and any problems were identified and any symptoms or signs of infection were looked for. The filter was changed and medications for the next

two weeks dispensed. The nurse also assessed the impact of the catheter upon patients pain and quality of life. Patients had access to the pain nurse at any time if there were problems.

Our data and experience suggest that a reasonably long duration of catheter survival can be achieved without significant complications by adhering to strict aseptic conditions and working in a multidisciplinary team.

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Reference

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Neuroaxial block for von Willebrand's disease

We read with interest the letter regarding neuroaxial block for von Willebrand's disease (vWD) (Stedeford and Pittman. *Anaesthesia* 2000; **55**: 1228–9) and would like to describe our experience with parturients having vWD. We have provided an epidural block for two cases of parturients with vWD for labour and delivery. The first case cited was at another institution [1] in 1989 and the second one documented in 1997 involved one of our obstetric floor nurses. In both cases, platelet counts, PT, PTT and fibrinogen levels were normal. Elevation of factor VIII activity has been demonstrated in normal pregnancy as well as during pregnancy in patients with vWD [2]. In our patients, factor VIII activity levels went up from 40% and 45% in the first trimester to 200% and 106% in the third trimester for the first and the second case, respectively. However, the second case had factor VIIIc (AHG) of 46; below the normal range of 57–157. Bleeding times were less than 10 min before an epidural catheter was placed for both patients. The first parturient received desmopressin after an epidural catheter placement but before delivery, and the second one received factor VIII concentrate (Humate-p, Centeon) before receiving an epidural block and 12 h later. Both patients had excellent, uneventful labour epidural analgesia.

We suggest that the decision to provide epidural analgesia and care for a parturient with vWD should be individualised. The risk of epidural haematoma and the benefits of excellent labour analgesia with a potential for an extended use of a neuroaxial block for a possible Caesarean delivery should be explained to the patient. All coagulation tests are required (not just one diagnostic test), and they should be normal or close to normal. Patient's treatment of clotting abnormality prior to a neuroaxial block or delivery should be individualised and determined by a haematologist. The patient should be positioned preferably in a lateral decubitus position. The epidural needle should be placed in the midline by an experienced anaesthetist. Motor deficit from the block may be avoided by adding an opioid to a diluted local anaesthetic solution. Administering a local anaesthetic solution via the needle before inserting the catheter [3] and applying a soft catheter (e.g. Arrow International Inc.) [4] can further reduce the chance of an epidural vessel puncture.

We conclude that a parturient with vWD may still benefit by having a neuroaxial block for labour pain. The decision to perform the block should be individualised, based on coagulation tests.

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Identification of epidural space using air and normal saline

I read with interest recent letters (Kale and Oosthuysen. *Anaesthesia* 2000; **55**: 615–16. da Silva. *Anaesthesia* 2000; **55**: 1041), which describe and criticise, respectively, an alternative method for the identification of the epidural space using both air and saline. I agree with the concerns of Kale and Oosthuysen regarding the safety of using air as the medium for loss of resistance and agree with the suggestion that there is extra 'feel' with air. However, I also agree with da Silva in that the contraption Kale and Oosthuysen suggest is complicated, cumbersome and unwieldy.

I have been using a technique that involves the introduction of a small (less than 0.5-ml) bubble of air into the saline-filled loss of resistance syringe. The bubble can be manipulated close to the plunger where it is easily observed and is in little danger of entering the Tuohy needle. With this technique, the bubble can be compressed when resistance is present but when the epidural space is encountered and resistance is lost the bubble will tend to re-expand. In this way, the epidural space can be identified with the sensitivity of air and the safety of saline. This technique achieves everything that Kale and Oosthuysen's suggestion promises but

in an easy and simple way that requires no extra equipment or connections.

I routinely use this technique and have found it to be very useful. This system appears to have been in use for many years so I cannot claim the credit for its discovery as during my research for this letter I encountered a paper describing the exact same technique for use in children [1]. Barros also alluded to a similar technique in a letter in 1995 [2]. It is interesting to note that Brown [3] supplies a diagram demonstrating a compressed air bubble in saline while identifying the epidural space using a loss of resistance technique. He does not, however, justify the reasons behind the use of this technique. In addition, I uncovered a paper regarding the EpIdent syringe [4]. This is a dual chambered syringe and is described using air in the proximal chamber and saline in the distal chamber to successfully identify the epidural space.

With this letter, I hope I have highlighted a relatively unknown but safe, simple and effective technique to identify the epidural space that uses the advantages of saline and air while avoiding their disadvantages.

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Simultaneous infusions of parenteral opioids with epidural local anaesthetics

It is my usual practice to place a thoracic epidural after induction of anaesthesia but prior to start of surgery in major colorectal (T_6) operations using ropivacaine. Following a number of unsatisfactory postoperative analgesia scores using a narcotic/local anaesthetic epidural infusion, despite a perfectly functional and effective epidural intra-operatively and immediately postoperatively, I have resorted to separating the administration of the two agents in the postoperative period with good analgesic effect.

The ropivacaine is continued per epidural at a dose of 50–250 $\mu g.kg^{-1}.h^{-1}$, and the narcotic papaveretum, which I find effective for visceral pain relief, is given by intravenous infusion at a rate of 20–100 $\mu g.kg^{-1}.h^{-1}$. This circumvents the problem often faced when the traditional epidural infusion of narcotic anaesthetic proves less effective than hoped for, precluding the use of a parenteral narcotic injection for 6 h following cessation of the epidural infusion. Many patients are inclined to have just the epidural infusion on day 3, and are perfectly comfortable without the papaveretum.

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Should Mendelson's syndrome be renamed?

Dr Ravalia (Ravalia. *Anaesthesia* 2000; **55**: 1040) suggests renaming Mendelson's syndrome by pointing out that Hall identified 15 deaths in parturient women caused by pulmonary aspiration during anaesthesia [1], and reported it 6 years before (1940) Mendelson's report in 1946 [2]. There were already reports in the mid 19th century of death due to pulmonary aspiration during anaesthesia [3, 4]. However, it was late in the 1930s when the danger of pulmonary aspiration during anaesthesia was recognised: a report in 1937 stated that '… under the influence of general anaesthesia, flooding of the respiratory tract with vomitus leads to acute alterations that have not been described and recognised anatomically and clinically to a degree proportional to the frequency of occurrence … Clinically, dilatation of the heart, pulmonary embolism, coronary thrombosis and acute pulmonary oedema are the usual interpretations' [5]. Since then, several committees on maternal death have pointed out the danger of aspiration during obstetric anaesthesia before Hall's [6, 7] or Mendelson's [8–10] report. Hall claimed that 'a careful search of all the medical literature failed to yield a single detailed report of a similar case.' [1]. In fact, Kaye made a detailed report of such a case (although non-obstetric) [11] well before 1936 when Hall made his claim.

Hall reported cases, but did not elucidate the mechanisms [1]. It is generally considered that Mendelson [2] was the first to show pathological changes caused by aspiration – but it is not so. Winternitz and colleagues reported in 1920 the effect of hydrochloric acid infused into the bronchi of rabbits [12], although they were not thinking of aspiration of gastric acid but of war gases liberating free chlorine. Apfelbach and Christianson [5] were probably the first to report (as a proceeding in 1937) pathological changes caused by aspiration through animal research. In a subsequent article, they stated that animal gastric fluid containing normal amounts of hydrochloric acid did not usually produce death, but a temporary hyperaemia of the lungs [13]. Mendelson's contribution was that he established the aetiology of pulmonary aspiration of gastric acid and solids through extensive clinical observations and comprehensive animal research, but there had been a general awareness of the danger of intra-operative pulmonary aspiration since which his name has been attached to the syndrome.

We agree with Dr Ravalia that Hall's report has been forgotten and his work should be acknowledged as late 1930s. There is also no need to rename Mendelson's syndrome as this term is now used less frequently and is

increasingly replaced by 'aspiration pneumonia'.

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A disposable plastic sub-Tenon cannula

Sub-tenon anaesthesia for ocular surgery is becoming increasingly popular following its re-introduction in 1992 [1, 2]. Anaesthesia is achieved by delivering the local anaesthetic into the posterior sub-Tenon space. This may be by the use of a commercially available metal cannula [1], ophthalmic irrigation cannula [A. P. Rubin, personal communication] or by an intravenous cannula sheath [3]. The administration into the anterior sub-Tenon's space is usually by a flexible polyethylene cannula [2]. Most metal cannulae are 19 gauge and are approximately 25 mm long with a blunt end designed to administer the local anaesthetic agent into the posterior sub-Tenon's space. Although the onset of anaesthesia is rapid, 15% of patients complain of pain during the injection [1] and complications related to cannula trauma have been reported [4, 5]. The commercially available polyethylene plastic cannula (Greenbaum) [2] is 14 gauge and approximately 10 mm long, and is designed to deliver local anaesthetic agent into the anterior sub-Tenon's space. The incidences of conjunctival haemorrhage and chemosis are higher particularly if diathermy is not used [6]. A cannula that is tough but flexible and of sufficient length may deliver the local anaesthetic into the mid sub-Tenon's space and might be a better alternative.

We believe that an ideal sub-Tenon cannula should be cheap, simple, disposable, flexible, non-traumatic and the length should be sufficient to deliver the local anaesthetic agent into the mid sub-Tenon's space, thus avoiding any trauma to major structures and to a reduced incidence of chemosis. While we were planning to develop a prototype cannula for this purpose, we accidentally came across a plastic cannula being used in our plastic surgical unit. This cannula appears to have all the above features. The cannula is marketed by Semco, Scotland, and is called a disposable taper tip (Part No. CT22LL). It is produced from moulded polyethylene and has a universal luer lock lug fitting enabling it to fit all popular syringe designs. The cannula is 22 gauge, internal diameter 0.41 mm and with a total length of 40 mm. The cannula has a proximal hub portion (10 mm), a middle taper portion (12 mm) and a distal straight portion (18 mm) and round blunt tip (Fig. 12).

We have used this cannula for sub-Tenon anaesthesia delivery in 87 patients undergoing cataract surgery. The sub-Tenon's space is accessed in the inferonasal quadrant, as described by Stevens [1] and Greenbaum [2], using scissors and forceps without using a diathermy. The cannula is attached firmly on to the syringe. The conjunctiva is lifted with the forceps and the cannula is introduced into the sub-Tenon space until the hub of the cannula fits snugly into the conjunctival initial incision hole. After an initial 1-ml injection, the desired volume of the local anaesthetic is injected.

We believe that the length of the above cannula is just enough to deliver the anaesthetic agent into the mid sub-Tenon's space. This is supported by our initial clinical experience. Anaesthesia and akinesia are achieved with 4–5 ml of lidocaine 2% with 1 : 200 000 epinephrine and 150 units of hyaluronidase. Pain on injection and chemosis are not as frequent as associated with the metal and Greenbaum cannulae. No other complications have been seen. The cost of this disposable plastic cannula is £0.50 as compared with the commercial cannulae, which range from £2.00 to £2.50. The only disadvantage is that it is

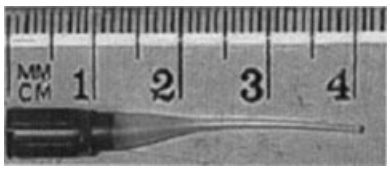

Figure 12

not supplied in a sterile pack and hence autoclaving is required. After autoclaving, the distal portion of the cannula becomes slightly curved and this further facilitates the entry of the cannula into the sub-Tenon space. We believe that the described plastic cannula is a simple, non-traumatic and cost-effective alternative to commercially available cannulae for sub-Tenon's anaesthesia delivery.

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Useful advice when using Trilene

In a recent attempt to quench my thirst for the history of anaesthesia, I glanced at a book written by E. H. Seward & R. Bryce-Smith, entitled, *Inhalation Analgesia in Childbirth*. I thought the following advice that I found there would be of particular interest both to experienced and to aspiring 'Trilene anaesthetists' alike.

'In contact with red-hot surfaces Trilene is broken down into phosgene gas and hydrochloric acid vapour. It is unlikely that a series of circumstances could arise in which a patient would inhale these decomposition products. Nevertheless, it may be worthwhile warning the practitioner or midwife not to smoke a cigarette in the presence of Trilene vapour! The whole of an inspiration is drawn into the lungs through the red-hot tip and one or two puffs will suffice to make the smoker feel distinctly uncomfortable.'

Interestingly, although trichlorethylene was first discovered in 1864 by the German chemist Emil Fischer, the above advice and book were published rather more recently than that, in 1957.

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Olanzapine overdose

We are writing to comment on the recent letter reporting olanzapine overdose (Johal & Shelly. *Anaesthesia* 2000; **55:** 929). The letter describes a typical case of olanzapine poisoning, but although there had been over 10 case reports already published at the time the letter would have been submitted, the authors have not referred to any of these cases and state that the effects of toxicity are not well defined. This is clearly not the case with both considerable data on the pharmacology of olanzapine and other similar antipsychotic drugs, and a significant number of cases of overdose reported to the manufacturer of the drug [1]. The toxic effects that Johal and Shelly suggest can be extrapolated from those of other similar drugs are incorrect, with only central nervous system (CNS) depression and coma being reported.

In the case that the authors report, they describe the typical features of olanzapine overdose, with CNS depression, miosis and tachycardia. These have been reported in numerous case reports and case series already and do not add to our current understanding of the drug in overdose [1–3]. In the light of this understanding, any of the investigations done were unnecessary, including the clotting screen, creatine kinase and urine myoglobin. Although deaths have been reported [4], supportive care is usually all that is required in olanzapine overdoses.

It is unclear in the authors' statement about the possible toxic effects of olanzapine what data these features were extrapolated from. The clinical effects can be extrapolated from the known receptor effects of olanzapine, past case reports of olanzapine overdose and clinical effects of other agents of its class (clozapine, quetiapine). Olanzapine acts at a number of receptors, which predict its effects in overdose quite accurately [5]. It has antimuscarinic activity causing tachycardia without arrhythmia, reduced bowel sounds, agitation and altered mental status; antihistaminic activity causing sedation; alpha-adrenergic effects causing miosis, orthostatic hypotension and tachycardia, and agitation [3]. This is supported by previous case reports and case series of olanzapine overdoses [1–3] and also by clozapine overdoses [6], a similar drug that has been used for many years. This differs markedly from the effects listed by the authors, which are actually more typical of the older antipsychotic agents. Importantly, convulsions, QT-prolongation and AV block have not been reported with olanzapine and related antipsychotic drugs, and are not likely to occur.

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Trivial pursuit: which drug is in the syringe, nitroglycerin or epinephrine?

Recently, a situation of doubt arose regarding the accuracy of labelling of the drugs within two syringes used in cardiac anaesthesia. In this centre, an infusion of epinephrine is prepared by drawing 2 mg of the drug into a 50-ml syringe containing 48 ml of 0.9% saline; while that of nitroglycerin is prepared by diluting 50 mg (10 ml) with 40 ml of 0.9% saline. During one particular cardiac surgical list, the blood pressure of one patient was found to be especially difficult to control during sternotomy with a nitroglycerin infusion. Despite confirming the correct connections and settings of the syringe pumps and solutions, the consultant anaesthetist thought that the anaesthetic nurse preparing the drugs may have mistakenly labelled the syringe containing epinephrine as nitroglycerin and vice versa. At that time, a decision was made to prepare fresh syringes of epinephrine and nitroglycerin. Fortunately, the existing ones were put aside so that the verification could be done later.

Later in the afternoon, I heard about this apparent mix-up, and I wish to share, from observation, how the nitroglycerin-containing syringe can easily be distinguished. No matter how meticulously the air bubbles are expelled from a solution of nitroglycerin, many small bubbles will appear on the inside of the syringe soon after preparation. However, this phenomenon usually does not repeat itself when these small bubbles have deliberately been made to coalesce and then expelled from the syringe. This phenomenon does not occur with the epinephrine solution.

The nursing officer in charge of anaesthetic services performed a simple test to distinguish the two drugs, which I thought was ingenious and therefore want to share it with all anaesthetic colleagues. Since the carrier agents in the commercial preparation of the individual drugs were different, their absorbency by paper might be different. One drop of solution from each syringe was expelled onto a sheet of cardboard paper taken from the backing of A4 size foolscap paper. The drop of 'nitroglycerin' was absorbed very rapidly by the cardboard paper while the drop of 'epinephrine' was not. For comparison, two correctly labelled syringes of nitroglycerin and epinephrine prepared for another cardiac theatre were used and the same procedure was repeated. The nitroglycerin solution spread very rapidly along the paper while epinephrine was absorbed very slowly. The simple comparison test showed that the 'dubiously' marked syringes were in fact correctly labelled. The anaesthetic nurse was exonerated.

As the syringes of the two drugs were to be discarded, a further observation test was done to convince the anaesthetist involved. This time, some water was drawn into the syringes of drugs. As the viscosity of nitroglycerin is greater than that of epinephrine, turbulence could be observed as water is drawn into the syringe marked as containing nitroglycerin.

Of the three observations, the most feasible and rapid way to determine the probability of correctness in the preparation and labelling of the syringe containing nitroglycerin is by checking for the presence of small bubbles on the inside of the syringe. This simple observation can help the anaesthetist to quickly determine and manage other causes of hypertension during sternotomy without delay, such as inadequate depth of anaesthesia or analgesia.

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Sodium citrate bottles

We wish to report a 'near miss' that occurred on our delivery suite recently. A bottle of 5% acetic acid, used by the gynaecologists to identify the cervix during colposcopy, had found its way into the anaesthetic cupboard in the delivery suite theatre. It was placed in amongst bottles of sodium citrate, which unfortunately are packaged in a similar way in our hospital, with both the bottle and the cap being identical to those of acetic acid. In both cases, the label consisted of black writing on a white background, except that the acetic acid is marked in red letters 'for external use only' (Fig. 13). The presence of the bottle in the anaesthetic cupboard was not noticed until sodium citrate was needed prior to an emergency Caesarean section, when the error was then picked up, and no drug was administered.

Department of Health Guidelines produced in 1988 require that there should be separate lockable cupboards for internal and external medicines [1]. Previous recommendations to reduce the likelihood of anaesthetic drug administration error have included emphasis on reading the label and training, distinctive drug packaging, an effective system of drug error reporting, standardised drug location in anaesthetic cupboards, and a check list for correct location of

Figure 13

drugs in anaesthetic cupboards [2]. The topic of ampoule/drug labelling was the subject of an editorial in this journal [3], which discussed requirements for labelling and colour coding to minimise drug error, but concluded that the only way of being certain of knowing what drug is being given is by reading the label. This case yet again demonstrates the importance of checking and double-checking all drugs prior to administration. The incident was reported to the Hospital Risk Management Team and to the Pharmacy Department, who are changing the packaging of acetic acid, so that it appears in a different bottle in future.

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A first left-handed intravenous cannula

With reference to the letter concerning the few reports of defects in intravenous cannulae (Bannister and Stacey. *Anaesthesia* 2000: **55**: 937) I would like to report a different defect I recently noticed in an Optiva 16g intravenous cannula. When the needle was fully advanced into the cannula the bevel of the needle was found to be 90° to the left of the normal position (Fig. 14). I felt that this may constitute a breakthrough for those of us who are left handed and when siting intravenous cannulae puncture the skin first prior to entering the vein lateral to the site of puncture. The bevel would thus end up in the correct alignment for entering the vein.

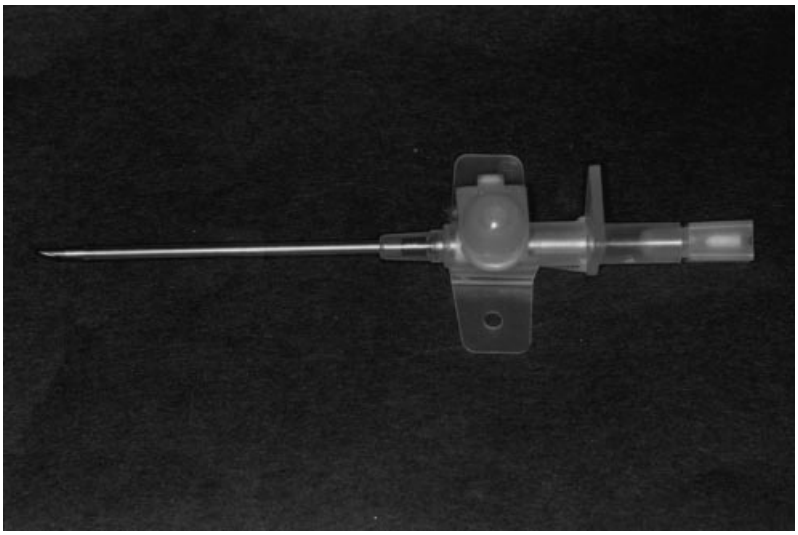

Figure 14

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An unusual ECG!

A middle-aged man presented to hospital for the removal of a large battery-powered foreign body from his rectum. Examination of his abdomen was unremarkable except for a buzzing sound audible on auscultation. An ECG recorded on admission showed an irregular baseline variation in leads I, II and III (Fig. 15). Unfortunately, we were unable to determine the exact PR interval! We wonder whether this should be reported as a case of anal fibrillation!

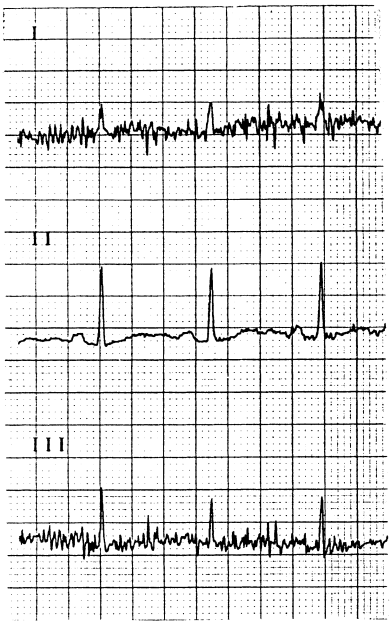

Figure 15

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