Correspondence

Research for research's sake?

If Ruskin had been a scientist he would surely have approved of the study undertaken by Goodwin *et al.* (*Anaesthesia* 2003; **58**: 60–3) to investigate the effects of neuromuscular blockade on mask ventilation of the lungs. Other studies he may have enjoyed include:

- The effect of tourniquet application on the efficiency of aspiration of blood from a peripheral venous cannula.
- The effect of increasing F_iO_2 on oxygen saturation in patients with an oxygen saturation of 96% or above.

In each case one is left wondering where lies the important clinical question to be answered.

The question Goodwin *et al.* set out to try and answer is whether or not there is any value in withholding neuromuscular blocking drugs following induction until an attempt is made to ventilate the lungs. The experiment they designed was never going to answer this question, because the reason people do it is to detect the 'can't ventilate' patients before it is too late. Such patients are very rare, and certainly not studied by the investigators.

So what were they up to? If they had shown that administration of neuromuscular blockade to ASA 1 or 2 patients with no known or obvious airway problem reduced the efficiency of mask ventilation, what useful clinical conclusion would they have drawn? That we must stop doing it, or be careful if we do? If they had shown that the efficiency of mask ventilation was improved by administration of muscle relaxants, would they have suggested that this may be a useful technique in the group of patients that really interests us, namely those who are difficult to mask ventilate?

This is another example of a study that fails to address the question of genuine interest because of the numbers or ethics involved. In common with many published studies, the authors allude to just such a question in their final paragraph, clearly stating that their study was unable, and was never designed, to answer such a question, and that other, larger studies might shed more light.

Their suggestion that future studies might actually focus on the group of patients '… in whom mask ventilation is more likely to be difficult' teases the reader by rubbing in the fact that their study was of no use in addressing this, the crux of the issue. Furthermore, it is unlikely that any Ethics Committee would allow people to administer neuromuscular blocking agents to this interesting group of patients.

Although their study is of little clinical interest, it does throw up a few methodological points worth commenting on. The setting of alpha and beta errors at these levels seems unavoidable. We are told that the standard deviation of the tidal volume had been discovered previously to be 125 ml. It is not clear if this is expired (VTe) or inspired (VTi) tidal volume, or whether they mean the difference between the two, which is after all what they use to calculate sample size. What is then tested statistically is the change in VTe/VTi, which is different again.

With such eminent persons putting their names to it, one of whom is on the editorial board of *Anaesthesia*, one can't help being rather perplexed by it all.

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A reply

Thank you for an opportunity to reply to Dr Hutchinson's comments on our study. Perhaps if Hutchinson had read our Introduction, he might have been less perplexed as to our reasons for conducting the study. For his benefit, we are grateful for the opportunity to repeat our reasons.

Our study arose from the observation that trainees commonly give neuromuscular blocking drugs only after 'testing' the patient's airway with a few 'puffs' from the reservoir bag via a facemask. We wished to know whether this practice is justified or to be avoided. We did not know whether a previous study [1] suggesting that full neuromuscular blockade improves mask ventilation was correct, or whether the alternative suggestions of other work [2], that neuromuscular blockade might worsen mask ventilation, were correct. Hutchinson claims in his letter that he would already have known the answer

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Correspondence presented in any other style or format may be the subject of considerable delay and may be returned to the author for revision.

(but declines to state what this 'obvious' answer is). We were more ignorant than Hutchinson and it is because of our ignorance (or in more correct scientific parlance 'equipoise') that we conducted the study. Had we found that neuromuscular blockade worsens mask ventilation, we would have concluded that we must all be even more cautious about neuromuscular blockade in patients who might be difficult to ventilate by mask, and that the trainees' practice was justified. Had we found that neuromuscular blockade improves mask ventilation, our conclusion would be that trainees' practice was not justified because they were 'testing' the airway when ventilation was at its most difficult. Thus (unlike Hutchinson) we were entirely impartial in our approach to the results of this study.

Hutchinson also criticises the way we conducted our power calculation. Much has been written about the limitations of power calculations [3–6] and an extensive response to his points is beyond the scope of this letter. The power of a study is the probability that the study has to detect as statistically significant a real difference of a given magnitude [3]. If a study shows a 'negative' (i.e. non-significant) result, then this might mean one or more of the following. First, that there is a real difference but the study had insufficient power (i.e. was not large enough) to detect this. Second, that the real difference is much smaller than was provisionally predicted from the power calculation. Third, that there is no real difference. In most studies, we are not interested in very small differences (however real or statistically significant they might be), so the power analysis is at best a rough guide to the study size likely to detect only a relevant difference [5, 6]. This is what our study was designed to do. Power analysis is not, nor intended to be, a 'statistical straightjacket', which prevents all further intelligent thought or necessary sensible modifications of study design. The ultimate measure of 'power' of the study is the confidence interval of the actual result obtained [4].

We would suggest that if Hutchinson is truly interested in the scientific question we addressed, a more constructive approach for him would be to design and conduct the follow-up study we suggested in the conclusion of our paper.

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Paediatric intensive care training

Tomlinson summarises the problems surrounding the initial care of critically ill children in non-specialised hospitals (Tomlinson. *Anaesthesia* 2003; **58**: 309). However, although the suggestions he makes to enhance the abilities of trusts to provide the required high standards of care are sensible, we believe that a lack of 'joined-up thinking' in the training of new consultant anaesthetists may continue to undermine attempts to achieve these standards.

The clinical exposure of current trainees is now very different from that of existing consultants trained in the 'pre-Calman/New Deal' era. Gone are the endless nights and weekends working on-call, during which exposure to various paediatric emergencies could be anticipated, particularly if duty responsibilities involved simultaneous anaesthesia and ICU cover. Greater centralisation of paediatric intensive care to Lead Centres means that in many acute trusts, the care of sick children is now increasingly limited to a relatively brief time interval prior to the arrival of a retrieval team. The opportunities for trainees to learn from unplanned contact with sick children have therefore diminished significantly.

Had these changes been accompanied by modifications in training programmes to ensure that all anaesthesia trainees gained clinical experience in a Lead Centre paediatric intensive care unit (PICU), then their adverse effects might be minimised. Regrettably, however, it appears that the secondment of trainees to PICUs appears to be at best patchy, and in some regions such training is only facilitated for aspiring paediatric anaesthetists or intensivists. A further disincentive is that time spent in paediatric intensive care may not even be 'recognised' for the mandatory period of training in critical care.

Given that the majority of future district general hospital (DGH) consultants for any region are likely to be products of the School of Anaesthesia based in the regional tertiary centre, any failure to invest in their training carries important future implications for the local care of critically ill children. This raises important questions about the logic of training in anaesthesia. Although training in paediatric anaesthesia teaches core skills in airway management, assisted ventilation, pharmacology and fluid management, this does not necessarily confer confidence in the management of potentially unstable critically ill children, particularly if training has been limited to the minimum of 3 months in predominantly elective patients. Paradoxically, significant amounts of time are still allocated to training in elective and emergency

cardiac and neurosurgical procedures, yet most consultant anaesthetists working in non-specialist centres are very unlikely to provide anaesthesia for cardiopulmonary bypass surgery or for a craniotomy. By contrast, however, there is a high likelihood that these same consultants will be involved with the care of children either for elective procedures or for emergencies. As the long-term outcome for critically ill children (if managed well) is considerably better than for adult patients with cardiovascular disease or neurological trauma, why is there so little emphasis on training in paediatric intensive care?

Perhaps it is time to re-examine the priorities for training in anaesthesia. In order to give the future generation the best chance of survival, we must ensure that consultants have the opportunity first to acquire the necessary skills, as well as developing strategies to maintain them.

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Herbal medicines in the United Kingdom

I read with interest the recent correspondence concerning the use of herbal medications in the pre-operative population (McKenzie. *Anaesthesia* 2003; **58**: 597) and wish to add some information from a survey carried out at our hospital.

Dr McKenzie makes the point that a list of commonly sold licensed herbal substances obtained by him from the manufacturers differs from the list of medications chosen by Hodges & Kam in their Australian-based review [1]. A survey of 346 elective patients carried out over 3 months using a standard questionnaire administered by nursing staff in routine pre-operative assessment clinics for a variety of surgical specialties at the Royal Hallamshire Hospital, Sheffield, showed that the commonest substances taken were as shown in Table 1. These figures agree reasonably well with the prevalences reported in a larger survey by Skinner & Rangasami in Slough [2] and the 'top six' most frequently consumed substances are the same, with three patients in our survey taking kelp, ginger and aloe vera, and lesser numbers taking valerian, prickly ash and a plethora of other medications. They are still considerably less than the figures from the USA reported by Kaye *et al*. from Texas [3] and Tsen *et al*. from Harvard [4]. All of the medicines discussed by Hodges & Lam, as well as some of those on Dr McKenzie's list, appear. It is likely, however, that considerable local variation even within the UK exists.

Table 1 Most frequently used herbal medication.

Medication	% total patients using (Sheffield)	% patients using (Slough)
Echinacea	1.44	0.70
Garlic	4.60	1.58
Ginseng	1.15	1.25
Gingko	0.87	1.10
Primrose	3.47	0.40
Saint John's wort	1.15	0.99

I agree with Dr McKenzie's opinion that discontinuation of all herbal remedies 2–3 weeks before surgery, as recommended in ASA publications, and articles by Hodges & Kam and Leak [1, 5], is probably unnecessary in some cases, and might lead to unwanted withdrawal effects. However, I feel that continuation of, for example, valerian through the peri-operative period, as he suggests, might prove unworkable in practice for anything other than day-case surgery.

Ang-lee *et al*. [6] in Chicago, concentrating on the eight most commonly ingested substances in the USA, suggested a means of tackling this problem based on an extensive literature search of *Medline*, *Cochrane* and other databases and utilising knowledge from reviews concerning the pharmacokinetics of their active ingredients. They suggest discontinuing ephedra and kava more than 24 h before, gingko more than 36 h before, Saint John's wort 5 days or more before and garlic more than a week before elective surgery. It would seem reasonable to taper the doses of those herbal medicines with CNS activity, such as valerian, Saint John's wort and kava, to reduce the likelihood of withdrawal reactions. Clearly, to ensure potentially unnecessary cancellations of surgery are avoided and safe anaesthesia administered, the earlier that patients taking herbal medicines can be identified the better. This is a challenge for all departments of surgery and anaesthesia as well as general practitioners.

More studies extending the work of Ang-lee *et al*. to include more common herbs ingested in the UK are necessary to arrive at a consensus about the need and timescale for discontinuation of these substances in the peri-operative period. I would echo Dr McKenzie's call for a survey of current anaesthetic practice.

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Anorexia nervosa and the refeeding syndrome

In their review of the anaesthetic implications of anorexia nervosa, Drs Seller and Ravalia emphasise that patients with this illness can suffer a

wide range of problems resulting in multisystem abnormalities (Seller & Ravalia. *Anaesthesia* 2003; **58:** 437–43). We agree that: 'in extreme cases, the patient may have to be fed nutrients via a nasogastric tube or parenterally in order to sustain life'. Such treatments have important implications, which we think warrant mention.

Provision of nutrition to a severely malnourished patient (reported as less than 70% of ideal body weight [1]) carries the risk of the 'refeeding syndrome'. One definition is: 'severe electrolyte and fluid shifts associated with metabolic abnormalities in malnourished patients undergoing refeeding, whether orally, enterally or parenterally' [2]. Provision of substrate, nutrients and calories can provoke rapid transcellular shifts of electrolytes. The importance of serum hypophosphataemia resulting from refeeding has been stressed [3] and has recently been reviewed by Haglin [4]. In patients with anorexia nervosa, refeeding syndrome implicating hypophosphataemia has been associated with heart failure [1, 5] and neuropsychiatric events [1].

As anaesthetists may well be involved with these patients for critical care as well as in the peri-operative setting, it is wise to be aware of such important iatrogenic complications.

A scheme for treatment of 'moderate' serum hypophosphataemia in critical care patients (between 0.32 $mmol.l^{-1}$ and 0.65 $mmol.l^{-1}$), with due regard for refeeding syndrome, is described by Rosen *et al.* [6].

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Sleep disruption and performance

I read with interest the paper: 'The effect of sleep disruption on performance of anaesthetists – A pilot study' (Murray & Dodds. *Anaesthesia* 2003; **58**: 520–5). The authors suggest that poor performance in a driving simulation test after a disturbed night may indicate an increased risk of a fatigue-related error occurring during the conduct of anaesthesia. It occurs to me that impaired vigilance during the driving simulation test may also indicate an impaired ability to drive safely after a disturbed night on call.

Many anaesthetists after a very busy night on call want nothing more than to drive home. However, although it may be unwelcome news, this may not be the safest course of action. Dr Polly Davies has previously highlighted this citing the extreme case of Gary Hart [1]. On 28 February 2001, Mr Hart spent 5 h in telephone conversation during the night with a friend before driving his Land Rover and trailer along the M62. It appears that at 6:20 a.m., Mr Hart lost control of the vehicle, perhaps having fallen asleep. The Land Rover came to rest on the main East Coast railway line where it led to a train crash, which caused the death of 10 people and injuries to many others.

Doctors are not immune from the perils of fatigue whilst driving. I am aware of one trainee doctor who was awake for most of his weekend on call. After his duty was finished, he drove home but fell asleep at the wheel, sustaining fractures of both lower limbs in the ensuing accident.

Whilst these are extreme examples, Murray & Dodds' paper does raise an important question, not only about an anaesthetist's ability to work safely after sleep disturbance but possibly also about his/her ability to drive safely.

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Teaching airway skills

The recent paper (Kiyama *et al. Anaesthesia* 2003; **58**: 571–4) highlights some of the variations in anaesthetic practice and approach to difficult airway management between two countries. However, we do not believe that the authors have arrived at the appropriate conclusions.

First, it would be wrong to conclude that the absence of a formal module is indicative that airway training is a peripheral activity. Mason stated that 'airway management is the scaffolding upon which the whole practice of anaesthesia is built.' [1]. Therefore, should we not consider the entire anaesthetic time as a 7-year module? The question also remains as to whether or not a module always gives better training. Mason goes on to point out that 'difficult techniques cannot be mastered during short workshops'. Instead: 'Expertise in airway management can only be acquired gradually, over a long period of clinical experience'. The areas of anaesthesia that we have found to benefit from modular training are relatively peripheral to our everyday anaesthetic practice, but give regular, predictable training opportunities. Good examples would be intensive care, cardiac and chronic pain. Clearly, this is the very opposite to airway management training.

All would agree that, ultimately, airway training has to be experiential

and, owing to the nature of the difficult airway, haphazard and opportunistic. Therefore, should we not be offering advanced airway training earlier to give more time for trainees to come across these opportunities and consolidate their experience during their anaesthetic training?

Finally, unlike the authors, we firmly believe that the techniques taught should be based on local practice and personal experience. It is vital for all anaesthetists to have an individualised difficult airway plan, but based on established guidelines such as the ASA Practice Guidelines for the Difficult Airway [2]. However, even the ASA Task Force failed to find strong scientific data to justify recommending one alternative airway or intubation technique over any other [2]. Levitan *et al*. [3] point out that as the genuinely difficult airway is such a rare occurrence, 'assessment of alternative techniques in a randomised manner would be technically impractical and would require such a large number of patients that it is not feasible.' Furthermore, we believe that in any such study, the personal experience we alluded to, and preference of the operator would be a confounding factor for any conclusions reached. Rather than determining whether such a wide range of techniques could be taught, we need to ensure that all anaesthetists receive adequate training in one technique for each step of an airway algorithm, e.g. gum elastic bougie or intubating stylet. It is surely better to be taught to use one of these well, rather than both badly. This would be consistent with the aim of rationalising the contents of the difficult intubation trolley. Not only is it not feasible to stock equipment for every single technique, but worse, a cluttered trolley is confusing and distracting in the airway crisis.

To conclude, we wish to express our concern at the move towards the false promise of the 'difficult airway module' rather than emphasising the fundamental duty of all practising anaesthetists to train others or to improve our own airway skills at every opportunity throughout our anaesthetic careers.

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Confirming tracheal cannulation during percutaneous tracheostomy without endoscopic guidance

Percutaneous dilatational tracheostomy (PDT) continues to replace conventional surgical tracheostomy as the procedure of choice in adult intubated intensive care unit patients, a group which accounts for approximately two-thirds of all tracheostomies performed [1]. Endoscopic guidance has been advocated to enhance safety, with many of the complications previously associated with percutaneous tracheal cannulation being avoidable by the use of direct bronchoscopic visualisation [2]. However, use of a bronchoscope may impair ventilation [3], and endoscopy may not always be possible or indeed available for a variety of logistical reasons. Recently, ventilation via the laryngeal mask airway during PDT has been shown to be effective and safe in selected patients, with elimination of the various difficulties associated with tracheal tubes [4, 5]. We describe a simple, inexpensive technique to confirm tracheal cannulation during PDT without endoscopy, when performed in the operating theatre in a patient ventilated via a laryngeal mask using anaesthetic agent monitoring as an indicator of successful tracheal cannulation.

A 35-year-old female with a diagnosis of Guillain–Barré syndrome and progressive respiratory compromise was admitted to the intensive care unit (ICU) for ventilatory support. As she was likely to have a prolonged intensive care course, an early tracheostomy was scheduled following informed consent from next-of-kin. This was performed as a PDT in the operating theatre for logistical reasons. Bronchoscopy was not available at the time of surgery.

Whilst in the ICU, sedation had been maintained with midazolam and morphine infusions. However, following transfer to the operating theatre, this was replaced by sevoflurane 1% in oxygen, delivered via a circle circuit. Non-depolarising neuromuscular blockade was achieved with atracurium.

In order to eliminate possible complications such as tracheal tube (TT) cuff puncture, tube transection by needle, or accidental extubation during the procedure, we removed the patient's TT and replaced it with a laryngeal mask and continued ventilation with sevoflurane in oxygen via the latter. We attached the sampling tubing from an Ohmeda 5330 Agent Monitor to the side port of a luer-lock three-way-tap, and placed this in series between a syringe and an introducer needle (Fig. 1). We then determined the location of the optimal entry point on the patient's skin and inserted the needle through the skin. With the three-way tap initially turned 'on' to the agent monitor for several minutes to confirm a zero recording, the tap was turned 'off' to the monitor and the needle was advanced with the syringe aspirating in the usual manner. Tracheal puncture was suggested by entry of air into the syringe. After entering the trachea, we turned the three-way tap 'on' to the agent monitor, and paused to observe the monitor. After one breath, the monitor detected sevoflurane and displayed appropriate values and commencement of trend line (Fig. 2). We then removed the syringe and tap from the needle, advanced the guidewire through the needle into the trachea and continued uneventfully with Griggs' dilating forceps technique.

Endoscopic guidance for PDT would certainly be regarded by many as the 'gold standard' technique, but a bronchoscope may not always be available for a

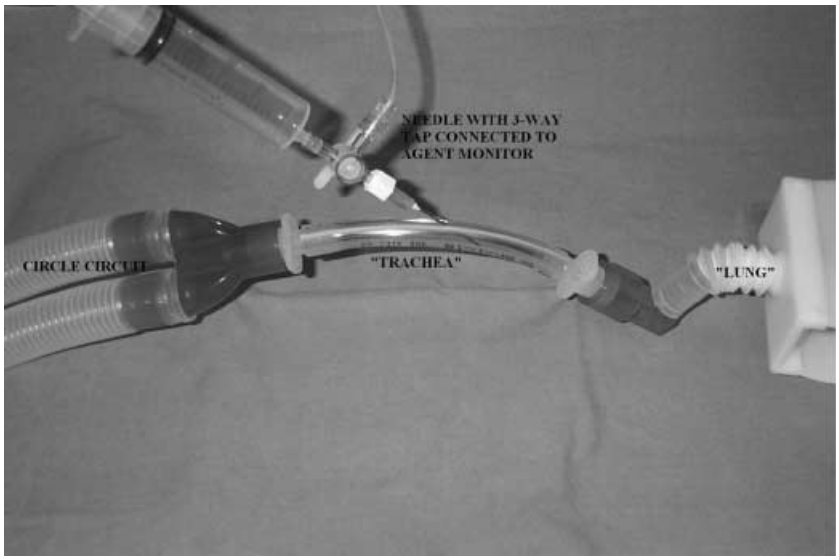

Figure 1 Model of three-way tap/agent-monitoring device to confirm tracheal cannulation (a test lung is ventilated with 1% sevoflurane in 100% oxygen).

Figure 2 Monitor showing agent detection after entry into 'trachea' as in Fig. 1.

variety of reasons. In such situations, it is useful to have an option other than just blind PDT with simple aspiration of air. By being able to detect anaesthetic agent in the aspirated air, this technique not only confirms entry to the trachea, but also excludes accidental entry to that other source of gas in the neck, the oesophagus.

Whilst limited initially to patients who can be ventilated with a detectable anaesthetic agent (which, in practice, usually comprises that subset of patients whose PDT is performed in the operating theatre), this simple technique may help to overcome the hazards associated with PDT insertion where endoscopy is not possible for logistical reasons, or for reasons of difficulty in ventilating the patient's lungs with the bronchoscope *in situ*. Once the trachea has been cannulated by the needle, anaesthetic agent may be detected via the three-way-tap after a very short time (15–20 s), giving early confirmation of correct placement. The technique is easy to perform and does not prolong PDT insertion by any significant duration. We suggest that this method be employed in all situations where PDT insertion occurs in the operating theatre without endoscopic guidance, where agent monitoring is available.

Furthermore, we would envisage that certain other anaesthetic monitors (such as the Datex AS-3) that employ a similar side-stream gas sampling method for routine detection of oxygen and carbon dioxide (in addition to anaesthetic vapour) may be able to detect increased levels of these two gases in a similar manner, if used with our PDT insertion technique. This, in turn, raises the possibility that a portable side-stream multigas analyser might be employed at the bedside in the ICU to confirm successful tracheal cannulation and thereby reduce the numbers of critically ill patients transported to theatre for PDT insertion. We plan to investigate this additional aspect shortly.

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Anaesthesia in an adolescent with Ohtahara syndrome

We would like to present the anaesthetic management of an adolescent with Ohtahara syndrome and acute testicular torsion. Ohtahara syndrome is an early-onset, epileptic encephalopathy, first reported in 1978 [1]. It manifests itself by early infantile onset, intractable seizures, suppression bursts in awake or sleeping state electroencephalography (EEG), severe psychomotor retardation and poor prognosis.

A 15-year-old male presented to our hospital with acute testicular torsion requiring orchidectomy. He was diagnosed with Ohtahara syndrome after generalised convulsions on day 1 after full-term normal delivery. Previously, attempts had been made to control his fits using phenobarbitone, nitrazepam, sodium valproate and thiopental, all of which were unsuccessful. On this admission, he was on long-term therapy with lamotrigine, vigabatrin, pyridoxine, lioresal and diazepam. A potential full stomach, a recurrent coryzal illness and fixed flexion deformity of the lower limbs, as well as severe mental retardation further complicated the case.

Intravenous access was aided by topical anaesthesia achieved with ametop cream and monitoring was applied as per AAGBI guidelines. Pre-oxygenation was difficult to perform, as the patient was uncooperative. General anaesthesia was induced with a rapid sequence induction using thiopental (7 $mg.kg^{-1}$) and succinylcholine (1 $mg.kg^{-1}$), and cricoid pressure was applied immediately after induction of anaesthesia. The patient's trachea was intubated easily with a 7.5-mm ID cuffed oral tracheal tube. Intra-operative opioid analgesia (fentanyl 1 $\mu g.kg^{-1}$) was supplemented with paracetamol and diclofenac suppositories and local anaesthetic to the wound site. Anaesthesia was maintained with a mixture of nitrous oxide, oxygen and sevoflurane. Atracurium (0.5 $mg.kg^{-1}$) was used with neuromuscular monitoring to aid intermittent positive pressure ventilation in order to maintain normal oxygenation and end-tidal carbon dioxide levels. A pain-free recovery was a planned necessity to reduce cerebral excitation on emergence from anaesthesia. Recovery from general anaesthesia was uncomplicated and the patient's trachea was extubated when he was fully awake to avoid the risks of regurgitation and aspiration. The patient was admitted to a paediatric high-dependency unit (HDU) for 24 h postoperatively. This immediate postoperative period progressed smoothly with no seizures or complications.

The patient was subsequently re-admitted to HDU following generalised tonic clonic convulsions on the paediatric ward on the second postoperative day. A partially successful attempt was made to treat the convulsions with lorazepam as per a previously agreed regimen. The patient was discharged the following day at his father's request as he was exhibiting his normal pattern of seizure activity.

To the best of our knowledge, this is the first case report of a general anaesthetic in a patient with Ohtahara syndrome. This syndrome presented difficulties with the anaesthetic through management of young age, mental retardation, history of frequent and intractable seizures and the potential effects of polypharmacy. A few interesting issues presented themselves during the management of this case. Pre-operatively, a literature search of standard textbooks and an online search (*Pubmed*) using the keywords 'Ohtahara syndrome' and 'anaesthesia*' failed to provide any information about the anaesthetic management of Ohtahara syndrome. A reference was located on a *Google* search [1]. The anaesthetic plan was finalised after discussion with the consultant anaesthetist on call.

During induction of anaesthesia, an additional dose of thiopental 2 $mg.kg^{-1}$ was required to produce adequate hypnosis. Minor neuroexcitatory side-effects of thiopental during induction [2] (nystagmus and fleeting twitches) were observed. The lack of formal EEG during anaesthesia meant that we were unaware of any seizure activity during the general anaesthetic. There was a normal response to neuromuscular blocking and opiate drugs. The lack of 'normal' seizure activity in the first 24 h after surgery is interesting; this was possibly a manifestation of the anticonvulsant effects of thiopental. Finally, as with all patients with recurrent seizure activity, the risk of neuroexcitation during an awake tracheal extubation had to be balanced against the risk of aspiration of gastric contents.

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The nasal intubation aid (NIA)

We read with interest the review article: 'Nasotracheal intubation for head and neck surgery' (Hall & Shutt. *Anaesthesia* 2003; **58**: 249–56) and want to remind you of the presentation of our 'nasal intubation aid' (NIA) – to our mind a tool that should not fall into oblivion.

As the authors pointed out, nasotracheal intubation can lead to certain complications. About 20 years ago, we published an article [1] describing a device protecting the nasal passage. For many years, this tool has been used in our department with respectable success. We examined this device in a clinical trial that proved a significantly reduced incidence of false passage, mucosal damage, erosion and bleeding.

Our NIA consists of a curved, flexible and distal closed catheter of 25 cm length and 0.4 cm diameter. At one end is cellophane wrapping open on the other side to pick up the distal end of a cuffed tube. The wrapping helps to protect the cuff from damage and prevents false passage, carriage of nasal flora into the trachea and abrasion of the nasal mucosa, provided the chosen tube is of an appropriate size (Fig. 3).

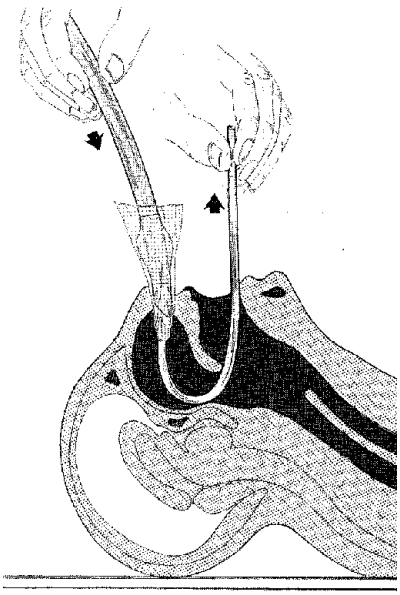

Figure 3 The 'Nasal Intubation Aid' *in situ*. By gently pulling and pushing (see arrows), the tube is guided through the nasal passage.

We found that trauma caused by nasal intubation was significantly reduced – an important gain. This was shown by lower rates of aching near the nasal root, blood-tinged mucous and impaired nasal breathing. Rare situations such as cuff damage, switching to the contralateral nostril, mucosal dissection or impossible nasal intubation did not occur when using the NIA. Both conventional and the intubation with NIA lead to typical complications such as erosions and bleeding. However, we found a significantly reduced risk of these complications in the NIA. For example, the frequency of bleeding was decreased from 58% in the conventional intubated tracheas to 11% when intubated with the NIA. We assume that, in addition, our tool is useful when there is abnormal coagulation because of its trauma-reducing qualities. Above all, it is safe, ready and easy to use and of negligible cost in comparison with its benefit.

We conclude by hoping that this useful tool will no longer remain overlooked and unmentioned.

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A potential hazard with Graseby 3000 syringe pumps

The Graseby 3000 series of syringe pumps is designed to accept different sizes of syringe, ranging from 5 ml to 50/60 ml. In addition, they can be programmed to recognise syringe sizes of varying brands, although this is usually a factory-preset function. The ability of the pump to recognise the syringe size is integral to the accurate rate of delivery from the syringe. It is achieved by a lever that springs down onto the side of the syringe and detects its diameter. In the 3100, 3150, 3200 and 3300 PCA models, the detected syringe size is displayed as a light-emitting diode (LED) on the control panel. In the 3400 and 3500 models, the detected syringe size is displayed in text on the display screen.

We would like to report an incident that occurred during an elective laparotomy in a 68-year-old patient under general anaesthesia with a thoracic epidural infusion, sited at T10/11. The epidural infusion was bupivacaine 0.25% in a 60-ml BD Plastipak syringe, running at 6 $ml.h^{-1}$ from a Graseby 3100 with factory settings to recognise BD Plastipak syringes. After about 90 min, it was noted that the Graseby syringe pump had already delivered 20 ml, which was more than double the expected amount of 9 ml. Immediately it was noted that the '20-ml' LED was displayed rather than the '50/60-ml' LED, and the pump was stopped and exchanged for another. The patient came to no harm.

The problem was caused by failure of the spring lever mechanism, causing the pump to default to the 20-ml syringe size selection, regardless of the position of the lever. The resulting infusion rate was therefore appropriate for a 20-ml BD Plastipak syringe at 6 $ml.h^{-1}$, which, in terms of linear scale, equates to about 13 $ml.h^{-1}$ from a 60-ml BD Plastipak syringe. It should be noted that there is no facility to override the syringe pump and manually to program the correct syringe size.

Although in this case there was no harm caused to the patient, we feel the potential risks from this equipment failure are great. The Graseby 3000 range of syringe pumps are used in a variety of situations, for example intravenous opiate and intravenous insulin infusions, where effectively doubling the infusion rate, as in this case, could be life threatening. In addition, if the syringe pump was to recognise incorrectly a 50/60-ml syringe size as a 5-ml syringe size, which would be the worst-case scenario, then the actual infusion rate could be as high as five times the set rate because the linear scale on a 5-ml BD Plastipak syringe is five times longer than a 60-ml BD Plastipak syringe.

We would therefore like to emphasise the importance of checking for correct syringe size recognition by the Graseby 3000 syringe pumps prior to use.

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A reply

Thank you for the opportunity to reply to Drs Wilkinson and Schofield's letter. Graseby Medical Ltd agrees that checking the syringe information displayed by the pump with that being used is important in ensuring the safe delivery of the infusion. The very first warning in the current Instruction Manual for the Graseby 3100 states 'WARNING: To avoid over- or under-infusion, always verify that the brand and size of the loaded syringe are the same as the brand and size displayed on the screen before starting the infusion. Failure to do so may result in an inaccurate delivery of medication, resulting in patient injury or death.' This warning is repeated in section 2.3 of the manual, where syringe loading is described. This warning appears in the same format in all the Instruction Manuals of Graseby syringe pumps. It is also emphasised in all training materials for these pumps.

The authors correctly note that there is no facility manually to override the pump and enter the correct syringe size in the case of a pump not measuring the

syringe barrel appropriately. Graseby Medical would not support this facility as it actually increases the risk of further problems because the user would not know the reason for the fault and the implications of manually overriding the pump. Current Graseby syringe pumps, e.g. Omnifuse, would not exhibit this fault as we have upgraded the design of the size sensing system such that the software is more intelligent and would detect the mismatch between the size registered and the size requested, and the pump asks the user to confirm the size and brand of the syringe.

Graseby Medical regrets the incident and apologises for the inconvenience caused.

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Clinical and economic choices in anaesthesia for day surgery

Publications designed to enable practitioners to make appropriate choices regarding anaesthesia for day surgery are welcome, and the recent article (Elliott *et al. Anaesthesia* 2003; **58**: 412–21) is a major contribution, which we found interesting, though somewhat difficult to fathom despite one of us (A.P.J.L.) having a qualification in health economics. The importance of avoiding postoperative nausea and vomiting (PONV) in association with day surgery, both in terms of delayed discharge and also adverse events at home, is well recognised by all practitioners, and simple, cost-effective manoeuvres to achieve this must be built into the anaesthetic technique and should include a prophylactic anti-emetic for high-risk cases [1]. The objective of the study was to assess relative value for money in terms of both the NHS and the patient, and not an examination of PONV as such. This would explain the absence of risk factors for PONV in the baseline data, and the randomisation between anaesthetic regimens stratified by hospital and sex only allowing a range of techniques. Low acceptance by adult patients and parents at recruitment to the study (23% and 25%, respectively), together with problems related to mask induction, might have led to bias, but with the large numbers overall and PONV not the primary outcome measure, this is acceptable.

The results of the study should come as no surprise, and a good demonstration that propofol is effective even when the sole anti-emetic, with only 14–18.4% of adults and 6% of paediatric patients experiencing PONV, which supports previous work [2, 3]. Omission of propofol led to a significant increase in PONV but no resultant overnight in-hospital stays, and the conclusion from the study has to be that propofol should be included in every day-surgery general anaesthetic technique for both its cost and clinical effectiveness.

We are not proponents of routine sevoflurane use for day-surgery as evinced by current evidence-based practice [4] but consider that future research might usefully be directed to examine possible benefits arising from inhalation induction and maintenance of anaesthesia at low flow when combined with an alternative cost-effective anti-emetic such as cyclizine [5, 6].

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A reply

We thank Drs Lake and Khater for their interest in, and support of, our study. We would like to respond to the issues they have raised regarding handling of risk factors for postoperative nausea and vomiting (PONV) in our study.

There are many factors that affect the incidence of PONV. The aim of the CESA randomised controlled trial was to maximise generalisability and thus to inform practical decision making for clinicians and funders. This requires studying sufficient numbers of patients using a design reflecting real-life anaesthesia practice. The large sample size and meticulous randomisation, further stratified by sex and by surgical specialty, resulted in recognised confounding factors being evenly distributed between groups [1].

Our recruitment rates were 77% of adults and 75% of parents approached, which is a relatively high acceptance and is unlikely to have led to serious bias in the results. Two hundred and twenty-six adults and 59 parents refused to enter the study because of the risk that they might be randomised to a mask induction, suggesting that the prospect of a sevoflurane induction may be unacceptable to many patients. Our study found low overall levels of PONV, despite the lack of anti-emetics, and little differences in overall costs to the NHS between the anaesthetic regimens investigated. This suggests that, where there are no other clinical considerations, patients' preferences should be considered when selecting an anaesthetic technique for day surgery.

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Reference

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Prolonged paralysis following sequential mivacurium administration

We wish to describe a case of acquired cholinesterase deficiency causing prolonged paralysis following sequential anaesthetics using mivacurium 2 days apart. The patient, who was not known to have the heterozygous atypical phenotype, had previously undergone an oesophagogastrectomy in 1988. She was cachexic and weighed 50 kg. Her past medical history was otherwise unremarkable. Routine pre-operative investigations were normal. Subsequent analysis of the pre-operative blood sample revealed evidence of mild liver dysfunction (elevated alkaline phosphatase and aspartate transaminase) and a low serum albumin concentration.

For the first procedure of upper airway panendoscopy, general anaesthesia was induced with fentanyl (75 μg), propofol (120 mg) and mivacurium (12 mg), and maintained with oxygen, air and isoflurane. The procedure lasted 40 min and revealed a large fungating pharyngeal tumour. Spontaneous respiration was re-established prior to extubation of the trachea and an uneventful recovery followed. Reversal of neuromuscular paralysis was not required.

Two days later, a feeding gastrostomy was required to be placed. Anaesthesia was conducted in a similar manner. On completion of the surgery after 45 min, there was no evidence of a return of spontaneous respiration. Supramaximal train-of-four (TOF) stimulation at 2 Hz of the ulnar nerve revealed no twitch. Post-tetanic twitch count in response to 50 Hz stimulation showed 10 weak twitches. Neostigmine 2.5 mg and glycopyrronium 0.5 mg were administered intravenously. TOF stimulation revealed four twitches with fade in amplitude, and as the patient was now able to breathe spontaneously and nod in acknowledgement to our voices, her trachea was extubated.

In recovery, it became apparent that, despite maintaining peripheral oxygen saturations of greater than 97%, spontaneous ventilation was inadequate. Therefore, her trachea was re-intubated following propofol (50 mg) and she was re-established on positive pressure ventilation with inhalational sedation.

Because of the possibility of cholinesterase deficiency, one unit of fresh frozen plasma was administered intravenously. Nearly 3 h after the end of surgery, the patient was able to obey commands, lift her head off the pillow and had bilateral grip strength in her hands of 3/5. Her trachea was therefore extubated, without event. Postoperative analysis of pre- and postoperative blood samples are shown in Table 2.

The patient's condition gradually deteriorated over the next few days, and she died peacefully 5 days after the second procedure. A serum sample referred to the Cholinesterase Investigation Unit in Leeds confirmed this patient's phenotype to be heterozygous for the usual and atypical cholinesterase variants (i.e. UA).

Mivacurium is metabolised predominantly by enzymatic hydrolysis by plasma cholinesterases [1]. The activity of this enzyme can be reduced by physiological, inherited or acquired causes (including malignancy, malnutrition and hepatic impairment, all of which were present in this patient [2]). The pre-operative results in this case are suggestive of a heterozygous phenotype, which can lead to a normal or slightly prolonged response to the administration of succinylcholine or mivacurium. The pronounced decline in cholinesterase activity cannot be accounted for by a dilutional effect, suggesting an acquired cause for the prolonged neuromuscular paralysis in addition to the genetic predisposition. However, the cause for the postoperative fall in dibucaine number is unclear.

Table 2 Patient's cholinesterase results (normal range).

	Pre-op.	Post-op.
Serum cholinesterase (54–125 U.ml^{-1})	61	13
Dibucaine number (76–82% inhibition)	61	17

The half-life of plasma cholinesterase is between 45 h and 16 days, depending on the method used to measure it [2]. Repeated use of drugs that utilise cholinesterases for metabolism within this time frame should be avoided, especially where there is evidence of impaired hepatic synthesis. The administration of fresh frozen plasma or a purified human form of cholinesterase can be used to hasten recovery of neuromuscular function but the benefit should be weighed against the risks of exposure to blood products [2].

Mivacurium is a short-acting agent that does not normally require reversal. It is not uncommon for anaesthetists to use neostigmine to reverse mivacurium-induced blockade. The manufacturer's data sheet for Mivacron (GlaxoSmith-Kline) does not, however, give specific advice for the use of neostigmine to assist reversal of neuromuscular blockade [3]. Neostigmine methylsulphate transfers a carbamate group on to the esteratic portion of the cholinesterase enzyme and is a potent reversible inhibitor of plasma cholinesterase. Neostigmine may therefore cause a paradoxical early but incomplete return of neuromuscular function.

Edrophonium is a less potent inhibitor of plasma cholinesterase, reversibly inhibiting acetylcholinesterase by binding to the anionic site and may be a more logical agent to antagonise the action of mivacurium. Antagonism with neostigmine in this case may have delayed the return of neuromuscular function despite the presence of four twitches in response to TOF stimulation.

In summary, we present a case of prolonged neuromuscular paralysis due to mivacurium of multifactorial origin. Despite evidence of a pharmacogenetic

predisposition to abnormal metabolism, the acquired factors of malnutrition, liver dysfunction and malignancy are likely to have been more significant in this case, particularly as the two procedures were so close together.

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Airway equipment failure due to a new staff member in Sterile Services

All new laryngeal mask airways in our hospital are labelled with a strip of coloured tape to identify the theatre complex that it is from (it is a large teaching hospital with eight separate operating theatre sites). This coloured tape is positioned at the proximal end of the laryngeal mask, just below the standard 15-mm connector (Fig. 4, left). During a routine pre-operative equipment check, a faulty laryngeal mask was identified (Fig. 4, right). The problematic laryngeal mask had its identification tape placed directly on the 15-mm connector rather than in the correct position just below the 15-mm connector (Fig. 4, left). The small increase in diameter caused by the tape then made it impossible to connect the laryngeal mask to a standard catheter mount, rendering it useless. Fortunately, this was discovered before the patient was anaesthetised. On investigation of the other anaesthetic rooms, five more laryngeal mask airways with the same problem were identified before anyone had tried to use them. The source of the problem was discovered to be a new member of staff in the Sterile Services Department who had not realised the clinical importance of the positioning of the tape.

Figure 4

This incident re-emphasises the importance of checking all ancillary equipment, as described in the Association of Anaesthetists Equipment Checklist [1]. Laryngeal masks should be included in this. In addition, it demonstrates that all staff working with equipment need to have some knowledge of its clinical function to prevent mistakes like this happening. There is currently no obligation for Sterile Services staff to spend any of their training in theatre. In our trust, Sterile Services staff do spend a day observing in theatre. However, this is limited to surgery and they do not get any specific training in the clinical uses of anaesthetic equipment. There are currently no national training programmes for Sterile Services Department staff but one is being developed and there will be a programme set up by the end of this year that should, we hope, address some of these training issues (Susan Meredith, Director of Education, The Institute of Sterile Services Management, personal communication).

Finally, Intavent (the company that manufacture the Laryngeal Mask Airway) oppose any alteration in their equipment and would not be liable if any problem had occurred due to this mistake. We have suggested that our Sterile Services Department stop this practice of adding tape to laryngeal mask airways.

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Reference

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Digital block improves pulse oximetry signal in vasoconstricted patients

Pulse oximetry is a cornerstone of minimum peri-operative anaesthetic monitoring and has undoubtedly improved patient safety since its introduction. It does, however, have a number of well-recognised limitations, including failure to detect a reading in peripherally vasoconstricted patients. This occurs if the monitor is unable to detect pulsatile blood flow, which is necessary to distinguish arterial blood flow from tissue, bone, capillary and venous signals. Peripheral vasoconstriction often occurs in patients who are critically unwell and in whom reliable pulse oximetry is highly desirable. The use of probes placed on the ear, buccal mucosa, or the nose, instead of a digit, can sometimes overcome this problem.

In situations where these options fail, I would propose performing a suitably placed digital block using plain 2% lidocaine. The vasodilatation this causes will produce a reliable pulse oximetry signal within 10 min, in my experience. Digital nerve blocks are extremely safe, the major potential complications being infection or the inadvertent use of a solution containing a vasoconstrictor, although the danger of this when using lidocaine has recently been questioned [1]. Potential benefits of having a pulse oximetry reading need to be weighed

against resorting to a more invasive strategy.

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Reference

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Gangrene of digits associated with radial artery cannulation

Arterial cannulation is the most common method of invasive blood pressure monitoring in the operating theatre and the intensive care unit. It allows continuous direct blood pressure measurement and blood sampling. The radial artery is often chosen, as it is superficial, allowing easy palpation with high successful cannulation rates, is conveniently accessible by the anaesthetist in most situations and is not an end artery. Several studies have demonstrated that vascular insufficiency from radial artery catheterisation is extremely rare, at least in patients without peripheral vascular disease. The risk of vascular insufficiency is, however, increased in patients with peripheral vascular disease and in those who have a haemodynamically unstable postoperative course. Staff need to be alert to such a complication. We present two cases of digital gangrene necessitating amputation after the radial artery had been cannulated in the operating theatre. Both patients had peripheral vascular disease and a very unstable postoperative course requiring high inotropic support.

An 80-year-old woman with a past medical history of atrial fibrillation and evidence of inferior myocardial infarction on ECG was admitted after collapsing at home. A CT scan and angiogram of her brain revealed a large subarachnoid haemorrhage from a ruptured cerebral aneurysm. An emergent craniotomy for clot evacuation was scheduled. In the operating theatre, an intra-arterial catheter was inserted into her left radial artery. The surgery lasted 4 h. During the anaesthetic course, boluses of ephedrine (total 27 mg) were given in order to maintain stable haemodynamics. Postoperatively, the patient became hypotensive in the neurosurgical intensive care unit. High infusion rates of dopamine and norepinephrine were needed to maintain an adequate blood pressure. There were episodes of fast atrial fibrillation. An echocardiogram of the heart showed no intracardiac thrombus. The left radial artery catheter was removed on the fifth postoperative day. On the seventh postoperative day, her left index finger was noticed to have turned purple in colour. A diagnosis of radial artery thrombosis was made. A conservative management approach was adopted as, owing to her intracranial pathology, anticoagulation was contraindicated and surgery deemed inappropriate. Over the following few weeks, the distal phalanx of her left index finger developed into a dry gangrene (Fig. 5), necessitating amputation.

An 84-year-old man with a history of ischaemic heart disease requiring several previous coronary angioplasty interventions was admitted with an intracerebral haemorrhage. An emergent craniectomy for clot evacuation was scheduled. In the operating theatre, an intra-arterial catheter was inserted into his left radial artery. The surgery and anaesthesia went smoothly and lasted around 1 h. Postoperatively, in the intensive care unit, he developed fast atrial fibrillation and septic shock, requiring a norepinephrine infusion. On the ninth postoperative day, the intra-arterial catheter was removed. Three days later, the patient's left thumb and index finger were noticed to have turned purple in colour, with reduced capillary return. Acute ischaemia affecting the thenar eminence, thumb and index finger was diagnosed. The affected area turned gangrenous. Four days later, the patient underwent amputation of his left thumb and index finger.

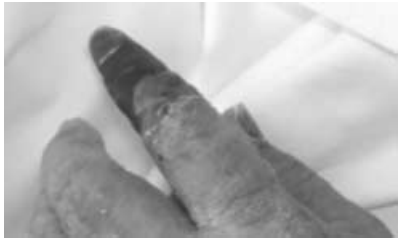

Figure 5

The most common complication of radial artery catheterisation is thrombosis. This, however, very rarely leads to ischaemic injury of the hand. The classic study on the safety of radial artery cannulation by Slogoff *et al.* [1] prospectively examined the radial artery flow by Doppler after decannulation in 1699 patients. They found partial or complete radial artery occlusion after decannulation in more than 25% of the patients, but no ischaemic damage or disability of the hand occurred in any patient. Only 411 (24%) patients had the modified Allen's test performed before cannulation. Abnormal results (greater than 15 s for the return of perfusion) were found in 16 patients. None of the 16 had abnormal flow or ischaemia on subsequent examination. They concluded that there was a high risk (more than 25%) of partial or complete occlusion of the radial artery after cannulation but there were no important clinical consequences of thrombosis, and that the Allen's test was not useful in predicting ischaemic damage or abnormal flow after radial artery cannulation.

There have been reports on ischaemic damage to the hand following radial artery cannulation. Such ischaemic damage developed a few days after the cannulation and all patients had exhibited the risk factors of postoperative hypotension and peripheral vascular disease. There have been sporadic reports on the ischaemic complication of arterial cannulation. Arthurs [2] described digital ischaemia on the seventh postoperative day in a 78-year-old man with bilateral femoral aneurysms scheduled for a femoral artery bypass graft. He had a modified Allen's test performed pre-operatively with normal results, indicating a patent ulnar artery. In the the operating theatre, his left radial artery was cannulated without difficulty. No hypotension occurred during the procedure and the cannula was left *in situ* for 4 h. On the seventh postoperative day, after another general anaesthesia that did not require direct

intra-arterial blood pressure monitoring, he became hypotensive with systolic blood pressure down to 75 mmHg in the ward. He then developed a blue discoloration and pain in his left thumb and index finger. No treatment was given and the discoloration and pain resolved spontaneously over the next 2 weeks. He recovered with no functional disability or symptoms in the left hand. The radial pulse was, however, still not palpable when the patient was followed up 1 year postoperatively. This case report is interesting in that no ischaemic complication relating to radial artery cannulation was noted until 7 days after decannulation and it developed following a period of hypotension after the second operation. In this patient, thrombosis in the cannulated radial artery was likely to have developed after the first operation. The thrombosis did not cause any ischaemic consequence until the patient developed hypotension. The normal Allen's test was not able to predict the thrombosis or the subsequent ischaemia.

Mangar *et al.* [3] presented a case of ischaemic hand injury in a 35-year-old patient scheduled for right femoral–tibial bypass under general anaesthesia, following an atraumatic placement of a left radial artery catheter. He had a complicated past medical history of insulin-dependent diabetes mellitus for 25 years, end-stage renal disease, coronary artery disease, silent myocardial infarction, peripheral vascular disease, hypertension and left hemispheric cerebrovascular accident. The Allen's test in him was equivocal: 12–15 s return of blood after ulnar artery release with palmar blush return to baseline. He had an uneventful general anaesthetic and accidentally pulled out the radial artery catheter on the following morning. On the 10th day, he complained of pain, coldness and discoloration in his left hand. Anticoagulation therapy was started. Unfortunately, 3 weeks later, he required hand amputation. The authors agreed that patients might develop ischaemic injury if adverse conditions are present for long enough, e.g. hypotension, use of vasopressors and multiple particulate emboli arising from within the heart. Again, in this case, Allen's test was not useful in predicting the ischaemic complication following the radial artery cannulation.

Cardiac surgeons have developed renewed interest in the circulation of the hand because of the use of radial artery grafts for coronary artery bypass. There are many studies on different techniques for assessing hand circulation, including the Allen's test, Doppler ultrasound, pulse oximetry, digit blood pressure and colour-flow Doppler. One case report described acute ischaemia of the hand after coronary artery bypass grafting with a radial artery conduit [4]. The modified Allen's test performed pre-operatively was normal. The ischaemia occurred 2 days after the operation at the time of onset of atrial fibrillation. The ischaemia improved after the patient had reverted into sinus rhythm and a revascularisation operation with a venous graft was subsequently performed.

Invasive monitoring during anaesthesia is relatively safe [5]. Radial artery catheterisation has a very low rate of serious complications. Although thrombosis following radial artery catheterisation is common, it very rarely leads to ischaemic complications. However, the ischaemic risk is enhanced in patients with peripheral vascular disease and hypotension in the postoperative period. The two cases we reported had vascular pathology and were hypotensive, requiring inotropic support in the postoperative period. It is therefore of paramount importance that the intensive care staff are aware of the risk and keep a high level of vigilance in vulnerable patients.

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Remifentanil infusion calculation made simple

The use of remifentanil by infusion should not be totally reliant on a flow rate calculated by an infusion device. Being able to calculate the flow rate oneself allows verification of the device settings, or the use of a simple syringe driver. Like your correspondent (Chelliah. *Anaesthesia* 2003; **58**: 104), I have had an occasion on which the flow rate set to be delivered by the infusion device (a Graseby 3500 syringe driver) did not correspond to the desired drug infusion rate. In this case, the flow rate was five times too fast. The infusion was not commenced, as the mentally calculated flow rate did not match the pump flow rate, which was five times too fast. The cause remains unknown as the printout retrieved from the pump confirmed the infusion rate in $\mu g.kg^{-1}.min^{-1}$ without reporting the rate in $ml.min^{-1}$. Even if the machine makes the correct calculation, it is dependent on the correct information being entered, from which to make the calculation, so human error cannot be avoided.

My calculation method is very simple indeed and thus reduces error.

- Prepare remifentanil to a concentration of 60 $\mu g.ml^{-1}$ (2 mg diluted to 33.3 ml).
- Divide the patient's body weight in kg by 10.
- This gives the rate in $ml.h^{-1}$ to deliver 0.1 $\mu g.kg^{-1}.min^{-1}$.

Once this basic rate is established, higher or lower rates can be calculated by simple multiplication or division of the rate. For example, a patient weighing 72 kg requires an infusion rate of 7.2 $ml.h^{-1}$ to deliver 0.1 $\mu g.kg^{-1}.min^{-1}$,

an infusion rate of 14.4 $ml.h^{-1}$ to deliver 0.2 $\mu g.kg^{-1}.min^{-1}$, or an infusion rate of 3.6 $ml.h^{-1}$ to deliver 0.05 $\mu g.kg^{-1}.min^{-1}$.

I have used this method for well over a year and apart from saving one potential critical incident it allows the calculating pumps to be available to colleagues who require them.

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Pre-operative psychosis following oral lorazepam

We report an unusual cause of acute psychosis and violent agitation following premedication with oral lorazepam. A 70-year-old, 68-kg male presented for transurethral evaporation of prostate. Past history revealed nothing significant in relation to major systemic illness, drug allergy or adverse effects, and psychosomatic disorder. Pre-operative investigation consisted of full blood count, and blood chemistry for urea-nitrogen, creatinine and liver enzymes, which were found to be within normal limits. The serum prostate-specific antigen titre (PSA) was 3.8 $ng.ml^{-1}$. American urological association symptom index for benign prostatic hypertrophy was 19/35. On pre-operative visit, the patient was calm, cooperative and relatively free from anxiety. The general condition was good along with normal higher mental function. The patient was not on any medications pre-operatively.

The patient received oral lorazepam 2 mg 90 min before the scheduled operation. Seventy-five minutes after oral lorazepam administration, the patient started showing grossly disorganised and agitated behaviour and became violently psychotic with irrelevant commentary. This led to the postponement of the surgical procedure. After ruling out other possibilities such as deranged renal function, electrolyte imbalance and hypoglycaemia, neurological consultation was sought, which revealed no abnormality. However, psychiatric consultation revealed that the patient's bizarre behaviour was most probably caused by the oral lorazepam. The patient's behaviour became normal after 8 h. Two days later, the patient was rescheduled for the same operation and was not given any premedication for obvious reason. Surgery was conducted under spinal anaesthesia uneventfully and lasted 80 min. The patient had an uneventful recovery.

Benzodiazepines are frequently used for premedication because of their hypnotic, sedative, anxiolytic and amnesic properties. Confusion, bizarre behaviour, amnesia, agitation and hallucinations are reported in the elderly after treatment with triazolam, a benzodiazepine [1]. Further, Ong *et al.* reported delirium, hallucination, confusion and agitation following intravenous administration of lorazepam (0.05 $mg.kg^{-1}$), in patients undergoing Caesarean section under epidural anaesthesia [2]. However, in the present case, the patient was elderly and received oral lorazepam that caused an adverse event that led to the postponement of surgery. The condition was self-limiting as was evident in the present case.

The pharmacokinetics of lorazepam is little affected by age, gender and renal disease [3], and hence is used frequently as a premedicant for different procedures. However, we suggest that following administration of oral lorazepam, especially in the elderly, the patient should be carefully watched for changes in behaviour during the pre-operative period. In the event of acute psychosis following lorazepam administration, the drug should not be re-administered. To avoid the recurrence of such an adverse event, this same should be reported on the anaesthesia record.

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Solvents in pregnancy: an emerging problem in obstetrics and obstetric anaesthesia

Although the use of 'major league' recreational drugs, such as cocaine and amphetamines, in pregnancy has received significant attention over the past two decades [1, 2], limited attention has been given to the consequences of maternal use of solvents, which are by far the most easily obtainable substances for illicit use. Additionally, although the deleterious effects of cocaine or amphetamines on the mother and the fetus are more pronounced and easier to detect, the addiction to solvents is usually subtle and more difficult to diagnose. As a result, recreational solvents use may continue undetected, significantly effecting pregnancy outcome and obstetric and anaesthetic management of these patients [3].

The (limited) literature concerning maternal and fetal implications of solvents use in pregnancy is focused exclusively on deleterious effects of chronic drug ingestion [4–6]. To date, no reports have addressed the management of obstetric and anaesthetic emergencies resulting from acute maternal solvents intake; I herein present such a case.

A 30-year-old, 167-cm, 64-kg, gravida 1, para 0, female with a history of bipolar disorder at 32 weeks gestation was admitted to the labour and delivery (L & D) suite for an emergency (crash) Caesarean section as a result of solvent-related maternal respiratory depression and fetal bradycardia. The patient had been found lying in her house with a plastic bag over her head sniffing 'household chemicals' from a soaked rag. Although she had nearly suffocated

herself, she remained haemodynamically stable.

The patient was taken to the operating room, positioned on the operating room table in the left-tilt position, anaesthesia monitors were applied, and the gravid abdomen was prepared for surgery with iodine solution. The lungs were denitrogenated with 100% oxygen (four vital capacity breaths). Rapid sequence induction of general anaesthesia was performed in a standard manner with intravenous etomidate 0.2 $mg.kg^{-1}$ followed by succinylcholine 1.5 $mg.kg^{-1}$ while cricoid pressure was continuously applied, and the trachea was intubated. A 1150-g female neonate (Apgar scores of 3 and 7 after 1 and 5 min, respectively) was delivered via the Caesarean incision. Surgery was promptly completed and the patient was taken to the postanaesthesia care unit intubated. There were no postoperative sequelae, she was extubated 50 min after the surgery end and her postpartum course was uneventful. Postoperatively, the neonate was diagnosed with growth restriction and the patient admitted to regular (recreational) household solvents sniffing throughout her pregnancy.

In the United States, nearly a million people, of whom 70% are young adults, misuse inhalants [2]. Inhalants include a chemically diversified group of substances such as organic solvents and volatile agents that affect the central nervous system. Toluene is a commonly used industrial solvent and a major component in many household paints and cleaning agents. Solvents may be sniffed from soaked rags, bags or open containers, as well as ingested orally for their narcotic effects.

Solvent inhalation causes intense central nervous system stimulation and disinhibition similar to alcohol ingestion. Toluene sniffing may lead to autonomic cardiac dysfunction, ventricular fibrillation and myocardial infarction. Glue sniffing can cause a unique distal and proximal tubular acidosis. Chronic exposure to toluene vapours has been reported to cause changes in the central nervous system such as diffuse brain atrophy and cerebellar degeneration. Increased airway resistance, pulmonary hypertension, acute respiratory distress syndrome and liver toxicity have all been reported in pregnancy with documented exposure to solvents [3–5].

Toluene sniffing for recreational purposes in pregnancy has been associated with an increased incidence of intrauterine fetal growth restriction, preterm delivery and prenatal mortality [3–5]. Although Hersh raised the possibility of toluene embryopathy and fetal solvent syndrome, the evidence was inconclusive and the fetal solvent syndrome was never defined [6]. Toluene may augment the manifestations of fetal alcohol syndrome in parturients abusing both substances simultaneously.

Optimal obstetric and anaesthetic management of solvent-misusing parturients requires a high level of suspicion and early diagnosis. Altered perception of sensory stimuli, loss of coordination, headache, nausea, vomiting and respiratory compromise may result from vapour sniffing. Careful physical examination including determination of possible sensory or motor deficits is indicated prior to induction of labour analgesia or surgical anaesthesia for abdominal delivery [2]. Both regional and general anaesthesia in the solvents-misusing parturient may be associated with intrapartum and/or intra-operative complications. When regional anaesthesia is selected, combative behaviour and altered pain perception may be encountered. Cardiac arrhythmias and labile blood pressures may be encountered under general anaesthesia. In acutely intoxicated parturients, administration of general anaesthesia for operative delivery may be necessary.

By definition, substance misuse is described as 'self-administration of various drugs that deviate from medically or socially accepted use, which if prolonged can lead to the development of physical and psychological dependence'. Psychological personality characteristics seem to predispose to, rather than result from, drug addiction. Because of the increasing prevalence of recreational drug use (including solvents) among young women (including in pregnancy), it is reasonable to expect that the incidence of solvent-related obstetric and obstetric anaesthesia emergencies will increase.

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Acute respiratory syndrome associated with extreme Superpruf aerosol

A 55-year-old male taxi driver was referred to us for intensive care and respiratory support. The patient had presented at the referring hospital's A & E department with a history of being found, by his mother, confused and drowsy. According to the mother who lives with the patient, he had gone to bed the previous night in good health. He had no previous physical illness or psychiatric illness, was taking no medication, was a lifelong smoker and drank very little alcohol. He did not have any pets and there was no history of any recent travel.

On examination at the referring hospital, he was found to be drowsy, confused, agitated and tachypnoeic and did not tolerate an oxygen mask or a

CPAP mask. His temperature was 36.4 °C, respiratory rate 38 breath.min^{-1}, pulse rate of 90 beat.min^{-1} in sinus rhythm and a normal blood pressure. Glasgow Coma Scale (GCS) was 13/15, there was no neck stiffness or photophobia and he was moving all limbs equally with normal reflexes. Fundoscopy was not possible owing to his agitated state. His heart sounds were normal with no murmurs, and his JVP was not raised. Chest auscultation revealed extensive bilateral crepitations and a chest radiograph showed bilateral pulmonary infiltrates in keeping with acute lung injury. Arterial blood gasses on 100% oxygen at 16 l.min^{-1} were as follows. P_aO_2 8.5 kPa, P_aCO_2 3.76 kPa and a pH of 7.39. Other blood results showed a raised urea of 12.2 mmol.l^{-1}, CRP 138, WCC 12.5 × 10^6.l^{-1}, normal electrolytes and blood glucose of 9.8 mmol.l^{-1}. The patient was given ceftriaxone 4 g and of clarythromycine 1 g and these antibiotics were to be continued.

At this stage, the patient was not tolerating CPAP and, with a deteriorating gas exchange, a decision was made to ventilate the patient. He was intubated and ventilated, sedated with propofol and fentanyl and a CT scan of the head was performed, which showed no abnormality. Owing to a local shortage of ICU beds, the patient was transferred to the intensive care unit at Crawley Hospital.

A lumbar puncture was performed, which showed an elevated opening pressure but was otherwise entirely normal. At this stage, the provisional working diagnoses were viral encephalitis and aspiration leading to acute lung injury (ALI/ARDS) and acyclovir and metronidazole were added to the current treatment.

The next morning, he remained ventilator-dependent with F_iO_2 of 0.55 and P_aO_2 of 10 kPa on 10 cm of PEEP. In line with our local sedation policy, sedation was stopped in order to assess neurological function. As the patient woke up, he tolerated the tracheal tube well and was able to communicate with the nursing and medical staff by writing. A big piece in the puzzle now fell into place. He gave a history of using a waterproof aerosol spray on his hat in a confined space. He either inhaled or ingested some of this agent, became sick, vomited and lost consciousness to be found by his mother some hours later. The patient was resedated and a bronchoscopy done later showed a markedly inflamed tracheobronchial tree extending to the limits of vision, associated with areas of mucosal sloughing.

The family later brought in the can of the spray that the patient had used, and the offending agent was identified. The product is marketed as EXTREME SUPERPRUF AEROSOL SPRAY, which is a blend of fluro copolymer in isoparaffinic hydrocarbons used as a waterproofing agent. We accordingly requested information from the manufacturers. On review of the product safety data sheet, we found that the agent can cause lung damage if swallowed and inhalation of the vapours in high concentration may also cause irritation of the respiratory system. Symptoms of inhalation may manifest with headache, dizziness, lethargy, nausea and vomiting.

We contacted the National Poisons Information Service at the Guy's and St Thomas's Hospital for any further information and guidance. They reported that they have recently become aware of a number of reports of poisoning after the use of waterproofing products. The information has come from poisons centres in Europe, particularly the Netherlands and Switzerland. Clinical effects vary from mild symptoms (coughing for several hours) to severe pulmonary reactions with oedema. In most of these cases, people used the sprays indoor often in small rooms, as was the case in our patient. The products involved contained fluorocarbon resins, butane and hydrocarbons.

It seems likely that it is the combination of various fluorocarbon compounds that causes the pulmonary problems, but the reason for this is unclear. It may be that because of the finer aerosols, the fluorocarbons are carried deeper into the pulmonary tract, especially if exposure is prolonged. The treatment of pulmonary (ARDS) and other problems is essentially supportive. The current position of the National Poisons Centre is that they have increased surveillance and vigilance of similar products and incidents.

We searched the medical literature for respiratory insufficiency and fluorocarbons, but were unable to trace any previous reports of ARDS following exposure to these products.

The patient developed progressive impairment of gas exchange and was referred to a tertiary centre for further care but unfortunately he died of respiratory failure and a post-mortem examination was not performed.

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An evaluation of safety of storage practices of laryngeal mask airways and tracheal tubes

In our hospital, the laryngeal mask airways were unpacked after sterilisation and stored in drawers and cupboards. The tracheal tubes were removed from their sterile packs before the start of the operating lists in readiness for an emergency. KY-Jelly-soaked green swabs were used to lubricate the tracheal tubes and laryngeal masks. We investigated whether contamination of this airway equipment was a possibility.

Tracheal tubes, laryngeal masks, KY-Jelly-soaked swabs and drawers and cupboards where these were stored were randomly sampled from four operating theatres and were subjected to appropriate microbiological cultures (Fig. 6).

Bacterial growth representing skin and environmental flora was obtained from all the laryngeal masks, tracheal tubes, drawers, shelves and green swab, except for one tracheal tube (Table 3, Fig. 7).

The manufacturers of laryngeal masks recommend that these should be sterilised after each use but there is no advice about their storage. Tracheal tubes come in sterile sealed packs and it is recommended that these should be unpacked just prior to use.

The Association of Anaesthetists of Great Britain and Ireland in their recent

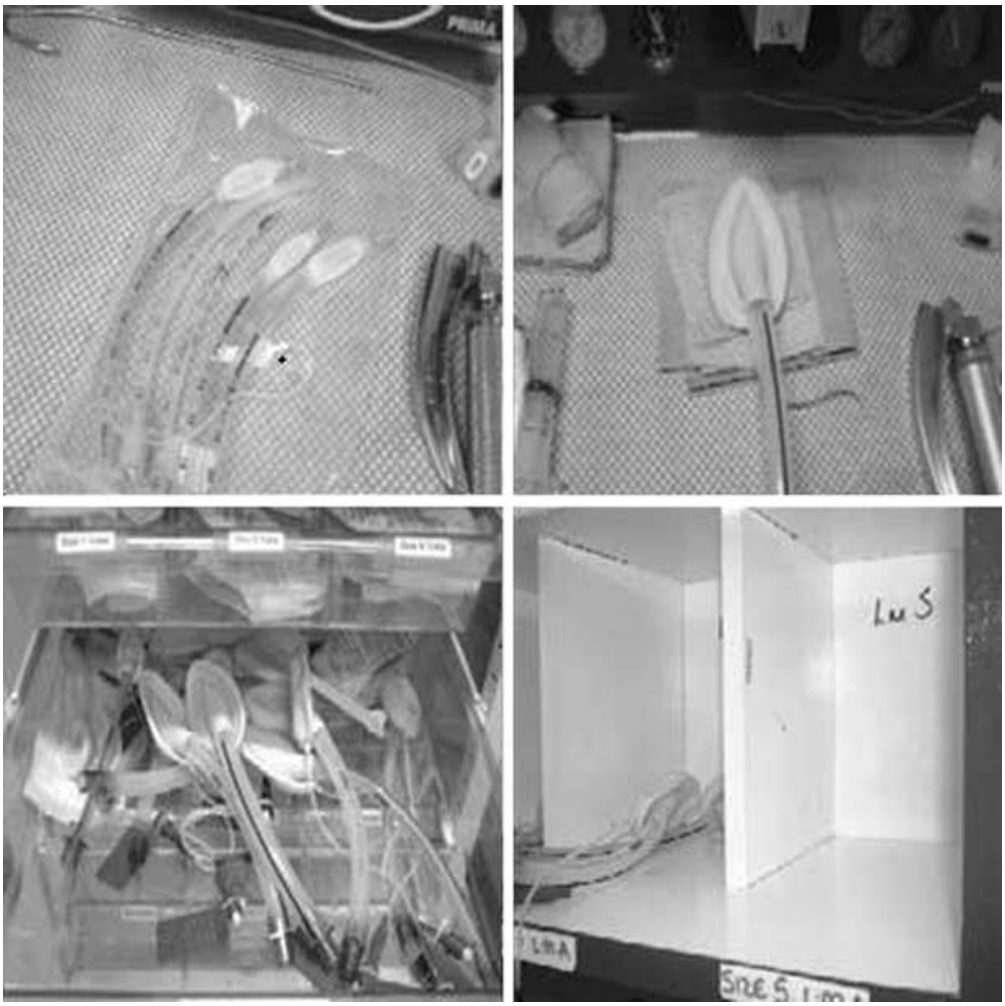

Figure 6 The airway equipment and the storage sites sampled.

booklet 'Infection Control in Anaesthesia' [1] does not mention storage practice after sterilisation of laryngeal masks. Similarly, the Royal College of Anaesthetists does not provide any such guidance. However, both the Australia and New Zealand College of Anaesthetists [2] and the Hong Kong College of Anaesthetists [3] clearly recommend that laryngeal masks as well as tracheal tubes and nasal and oropharyngeal airways should be kept sterile until used.

Stone *et al.* [4] have described transient bacteraemia after airway manipulation with clean laryngeal masks and tracheal tubes. In this study, the bacteria isolated represented patients' resident flora. Brimacombe *et al.* [5] have also studied bacteraemia following insertion of laryngeal mask airways in 100 fit patients, of which four blood cultures were positive.

These studies suggest that airway manipulation with laryngeal masks and tracheal tubes may lead to bacteria entering the bloodstream either from the patient's own flora or from the surface of the airway devices. There may be an increased risk of bacterial transmission if the devices are used un-sterile. Insertion of contaminated devices in the patient's airway could result in colonisation with new organisms.

We were unable to elucidate why the practice in our department involved removing laryngeal masks from their sterile packs for storage prior to use; perhaps it was because there are no clear recommendations in this area from within the United Kingdom. Removal of tracheal tubes from their packs in readiness for use in emergency situations, whilst understandable, resulted in a large number of tracheal tubes potentially being exposed to room air and the surfaces of anaesthetic machines. Our results show that our practice resulted in significant bacterial contamination. We have changed our practice; laryngeal masks are now sterilised and then packed individually. The packs of tracheal tubes and laryngeal masks are opened just prior to use. The use of green swabs to lubricate tubes and laryngeal mask airways has also been abandoned. In addition, we feel that there is a need for clear guidelines on storage practices of laryngeal masks and tracheal tubes in the UK.

Table 3 The sites sampled and the bacterial growth obtained.

No.	Samples	Results
1	Laryngeal masks from 4 sites	Light to heavy growth of *Bacillus, Staphylococcus epidermidis* and *Micrococci*
2	Tracheal tubes from 2 sites	*Bacillus, Staphylococcus epidermidis* and *Streptococcus viridans*
3	Green swab KY Jelly from 1 site	Light growth of *Bacillus, Staphylococcus epidermidis*
4	Laryngeal mask drawers and shelves from 3 sites	Light to moderate growth of *Bacillus, Staphylococcus epidermidis* and *Micrococci*
5	Tracheal tube from 1 site	No growth

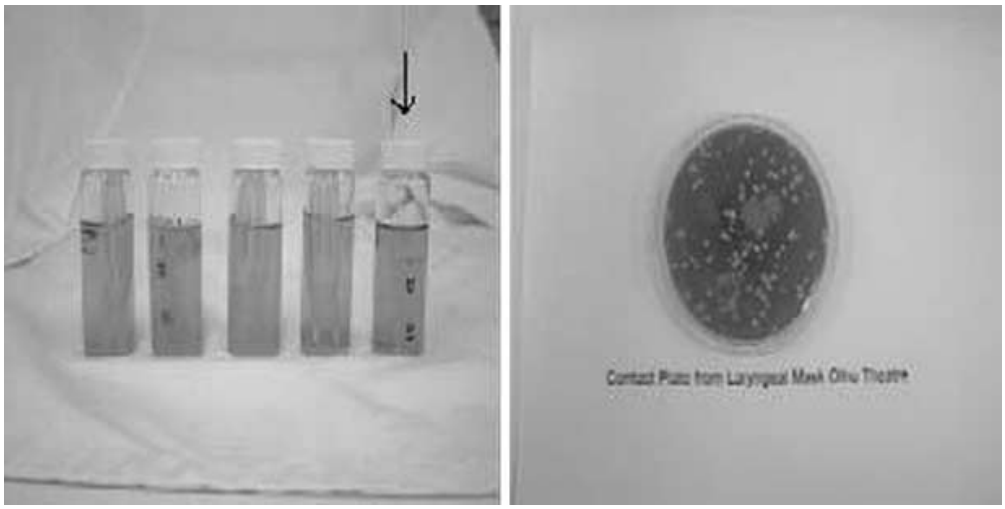

Figure 7 Broth bottles show turbidity due to bacterial growth from tracheal tubes, except the one marked by an arrow, and contact plate showing bacterial colonies from a laryngeal mask.

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Re-use of equipment between patients receiving total intravenous anaesthesia

I was astonished that Halkes & Snow found that only 46% of the consultants in their survey change all equipment for total intravenous anaesthesia (TIVA) between cases (Halkes & Snow. *Anaesthesia* 2003; **58:** 582–7).

When prefilled, electronically tagged, propofol syringes were introduced some years ago, their expense became a consideration. I decided to base my own practice on what I would do if I used TIVA equipment for a patient whom I knew to have an infection such as hepatitis A or B, or HIV. Would I be prepared to inject the remaining contents of any part of that equipment into another patient? No, certainly not, no matter how many lengths of tubing or one-way valves were changed. I certainly would not want it injected into me or a member of my family. I do not have evidence to confirm this prejudice, but gut feelings do not come any stronger, and I would not do to a patient what I would not accept for myself. As it is not generally possible to be sure that any patient is free from one of these infections, the only acceptable practice is to treat all patients as if they were infected, and I suggest that Halkes & Snow's assertion that all of the infusion equipment should be discarded between cases is indisputable.

I retired several years ago, and if I ever make another appearance in an operating theatre it will be as a patient,

not as an anaesthetist; I hope no-one re-uses any TIVA equipment on me.

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Transient hearing loss with labour epidural block

I read with interest the case of auditory dysfunction after epidural injection during labour (Rajaesekaran *et al. Anaesthesia* 2003; **58**: 613–14). Transient hearing loss after epidural injection has been reported previously [1, 2]. The mechanism of action is straightforward. The cochlear canaliculus connects the perilymphatic and subarachnoid spaces allowing passage of tracer from one to the other [3]. Perilymph in the inner ear is in continuity with cerebrospinal fluid so that any pressure wave within the subarachnoid space is conducted directly through to affect the basement membrane [4]. The surprising fact is that this phenomenon is not reported more often.

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Neurological sequelae following methylene blue injection for parathyroidectomy

Intra-operative staining of the parathyroid glands with intravenously administered methylene blue is well described and has been demonstrated as an effective and safe method to facilitate parathyroidectomy [1, 2]. However, we report a case of a 60-year-old woman who had an adverse neurological event following the use of intravenous methylene blue.

This patient, scheduled for a parathyroidectomy, had symptomatic hypercalcaemia secondary to a parathyroid adenoma, and a history of anxiety and depression. Her only medications were fluoxetine, coproxamol and beclamethasone nasal spray. She had no known allergies and had undergone one previous general anaesthetic for tonsillectomy with no adverse effects. Pre-operatively, she was administered a methylene blue infusion of 7.5 $mg.kg^{-1}$. She vomited once during its administration and, on arrival in the anaesthetic room, looked very blue but showed no abnormal behaviour. Surgery was carried out uneventfully under general anaesthesia during which all her vital signs remained within the normal range. At the end of the procedure, the patient was extubated and transferred to the recovery room.

Shortly after arrival in recovery, she was noted to have rotational nystagmus and dilated pupils that were unreactive to light. Thirty minutes later, she began to display rigid, jerky movements of all four limbs and remained very agitated over the subsequent 2 h with a fluctuating Glasgow Coma Scale (GCS) of 7–10. She had increased tone in all four limbs, bilateral up-going plantar reflexes, was noted to be sweating profusely and flushed above the shoulders, yet remained haemodynamically stable. An arterial blood gas revealed a respiratory acidosis with a P_{CO_2} of 60.1 mmHg, P_{CO_2} 103 mmHg and an ionised calcium level of 1.49 $mmol.l^{-1}$.

It was decided to transfer her to the intensive care unit for closer observation where she appeared to improve initially, but became more agitated and aggressive overnight. The maximum methaemoglobin level in arterial blood gases reached 0.7% on admission to the ICU. A methylene blue blood level obtained from blood taken 6 h postoperatively was reported as very low but the validity of the result could not be guaranteed. Information from the regional poisons unit suggested that plasma levels are not known to be clinically useful.

The patient was re-intubated the following morning in order to facilitate a CT head scan, which was normal. On the second postoperative morning, the sedation was stopped and the patient extubated without incident. She remained intensely blue-stained and had dark blue urine for 3 days. Her corrected calcium stayed within the normal range, and her haemoglobin dropped from a pre-operative value of 14.7 to 9.7 $g.dl^{-1}$, which was attributed to haemodilution. There was no evidence of haemolysis.

Over the following 2 days her speech and neurological status returned to normal. After a normal MRI scan on the fourth day, she made a full recovery, and was discharged to the ward before returning home.

Despite many years of apparent innocuous use of intravenous methylene blue, it has definite toxic effects. Nadler *et al.* [3] found that intravenous methylene blue excited the individual, and by its rapid elimination into the stomach and urine produced transitory gastrointestinal and urinary irritation. The most frequent toxic symptoms observed were restlessness, paraesthesia, burning sensation and chest pain, all of which subsided in 24–48 h. A number of individuals also complained of dizziness, headache and mental confusion, all of which completely resolved.

The UK National Poisons Information Service (Cardiff Centre) recommend an intravenous dose not exceeding 4 $mg.kg^{-1}$ of a 1% solution and list similar potential signs and symptoms of toxicity, namely nausea and vomiting, abdominal pain, precordial pain, headache, hypertension, profuse sweating, restlessness and confusion. They also state that methylene blue itself may cause methaemoglobinaemia (up to 7%) when given in very large doses, but that this amount of methaemoglobin (MHb) is clinically insignificant.

Another authority reports that if methylene blue intravenous dosage exceeds 7 $mg.kg^{-1}$, the same adverse symptoms may result [4]. Yet staining abnormal parathyroid glands with

7.5 mg.kg^{-1} of intravenously administered methylene blue is common practice among surgeons and we have been unable to find reports of toxicity in the literature associated with this dose.

The picture presented could be explained by the hypoxaemia caused by methaemoglobinaemia. However, it is unusual for MHb levels of less than 20% to be associated with hypoxic symptoms such as confusion, and in individuals with normal amounts of haemoglobin (Hb), an MHb level of less than 20% usually only causes cyanosis [5]. Our patient had a pre-operative Hb of 14.7 g.dl^{-1} and the maximum MHb present at the height of symptoms was only 0.7%. We are therefore led to conclude that the observed decrease in Hb was not proportional to the intensity of the symptoms, unless it could be argued that the reversibility of this reaction is almost immediate. Indeed, the reconversion rate of MHb to haemoglobin in normal individuals is about 15% per hour, assuming no ongoing MHb production [6].

Furthermore, if this clinical picture was brought about by acute hypoxaemia, we would have expected to see a more marked effect on respiration and circulation, whereas the primary effect seemed to be a neurological one. We are led to conclude that the harmful effects of methylene blue may not be associated primarily with methaemoglobin, but rather attributed to the direct action of the drug itself.

A further possible explanation might be a neuroexcitatory response to propofol. There are a number of reported cases of hypertonia, opisthotonus, jerky movements and seizures occurring in otherwise healthy individuals after an anaesthetic involving intravenous propofol [7]. Whilst some of the features seen in this case would be consistent with an adverse response to propofol, it would not explain the profuse sweating and sense of apprehension seen in this patient. Furthermore, the patient was sedated with propofol on the intensive care unit for 24 h, during which time her symptoms improved.

Thus, although we cannot give conclusive evidence that this patient's symptoms were wholly a result of the high dose of methylene blue, it remains the most likely cause. In summary, it is important to remember that no dye or drug given to a patient either intraoperatively or postoperatively should be regarded as inconsequential, and indiscriminate use of methylene blue in doses above those recommended may produce unpleasant results and may be dangerous to the patient.

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Caesarean section in a cocaine-intoxicated parturient: regional vs. general anaesthesia?

The illicit use of cocaine in pregnancy has been associated with obstetric and anaesthetic emergencies. I herein report a case of a parturient who presented with placental abruption and fetal distress following acute 'crack' cocaine intake and required emergency Caesarean section, which was conducted under uneventful single-dose spinal anaesthesia.

A 26-year-old, 167-cm, 66-kg, gravida 1, para 0, otherwise healthy female with a history of multiple-substance misuse at 30 weeks gestation required emergency Caesarean section as a result of crack cocaine-induced placental abruption and fetal distress. She had no known drug allergies and no prior anaesthesia. The course of her pregnancy had been 'uneventful'; specifically, there were no symptoms of pregnancy-induced hypertension; however, she admitted to recreational multiple-substance intake including cocaine, heroin and alcohol. Her admission blood pressure was 145/95 mmHg, heart rate 95 beat.min^{-1} and respiratory rate 20 breath.min^{-1}. Fetal heart rate was 130 beat.min^{-1} and non-reactive, with late and variable decelerations and no response to maternal oxygen administration. The maternal physical examination conducted by the obstetrician documented uterine tenderness, and the reading of the external pressure-sensitive tocodynamometer recorded uterine hypertonicity. Obstetric ultrasound documented a single fetus in cephalic presentation with intra-uterine growth restriction without any gross abnormalities. However, the presence of a hypoechoic area representing retroplacental bleeding (haematoma) causing separation of the placenta from decidua basalis was discovered. Laboratory coagulation studies were normal. The working diagnosis of acute placental abruption with fetal distress was established and emergency Caesarean delivery was indicated. Although the patient was slightly agitated, she cooperated with obstetric and anaesthetic recommendations. An intravenous fluid preload with crystalloid solution (Lactated Ringer's solution) was initiated and the patient was taken to the operating room. A spinal block, with 12 mg bupivacaine 0.75%, fentanyl 5 μg and morphine 0.2 mg at the L2–3 inter-

space, was induced with the patient in the sitting position. A T4 sensory level was established and surgery began. A 1250-g female fetus with Apgar scores of 5 and 9 after 1 and 5 min, respectively, was delivered via the Caesarean incision. No maternal and neonatal postoperative complications were reported. The diagnosis of eclampsia was ruled out by routine laboratory studies (liver and kidney function tests). However, urine and serum toxicology screening was positive for recent cocaine intake.

The prevalence of recreational drug abuse among young women (including in pregnancy) has increased markedly over the past two decades [1] and multiple substance misuse is common. 'Crack' is an almost pure form of cocaine. Pregnancy enhances the cardiovascular toxicity of cocaine. Acute cocaine intake has been associated with obstetric emergencies such as placental abruption and fetal distress. In most cases of placental abruption, delivery is the treatment of choice. The combination of hypertension, proteinuria and convulsions resulting from acute cocaine intake may be mistaken for eclampsia at presentation; consequently, laboratory studies (liver and kidney function tests) may be the key differential between the two disorders.

The diverse clinical manifestations of acute cocaine intake combined with the physiological changes of pregnancy and the pathophysiology of coexisting pregnancy-specific disease might result in life-threatening complications and significantly impact on the practice of obstetric anaesthesia. Both regional and general anaesthesia in the cocaine-misusing parturient may be associated with serious complications [2]. When regional anaesthesia is selected, combative behaviour, altered pain perception, cocaine-induced thrombocytopenia and ephedrine-resistant hypotension may be encountered. Cardiac arrhythmias, hypertension and myocardial ischaemia may be encountered under general anaesthesia. All inhaled anaesthetic agents may produce cardiac arrhythmias and increased systemic vascular resistance in cocaine-intoxicated parturients. The decision to perform spinal anaesthesia for Caesarean section in this case was based on maternal and fetal haemodynamic stability, normal maternal coagulation profile and a sufficient degree of patient cooperation (despite intoxication) with the anaesthetic management. Against general anaesthesia was the fact that maternal mortality rate is many times higher in parturients who undergo abdominal delivery under general anaesthesia as compared with those who receive regional anaesthesia [3].

In conclusion, this case is important because it shows the significance of individualising anaesthetic management in parturients presenting with diverse clinical manifestations of acute cocaine intake. For other patients with other maternal and/or fetal expressions of acute cocaine intake, other anaesthetic choices might be more appropriate.

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A method of blood conservation in Jehovah's witnesses

It is essential to make every effort to minimise the loss of blood during surgery, particularly in Jehovah's Witnesses. Although individuals' beliefs may differ, some Witnesses believe that it is acceptable to withdraw blood from the body and return blood as long as it remains in contact with the body.

Figure 8

When blood is taken from an arterial line for analysis, it would be usual to: attach a 5- or 10-ml syringe to withdraw heparinised liquid and diluted blood in the arterial line, remove the syringe, withdraw blood using another syringe for analysis and finally re-attach the first syringe to return the diluted blood to the body. This practice may not be acceptable to some Witnesses because, albeit for a short a period, blood in the first syringe had lost contact with the body.

We had occasion to anaesthetise a baby scheduled for cardiac surgery whose parents were Jehovah's Witnesses. We used a simple method to avoid wasting blood from the first syringe. We inserted two three-way stopcocks in an arterial line, attached a 5-ml syringe to the stopcock distal to the patient and attached a sampling syringe to the proximal stopcock (Fig. 8). Liquid in the arterial line and diluted blood were withdrawn into the 5-ml syringe, 0.3 ml of blood was withdrawn to the sampling syringe and diluted blood in the 5-ml syringe was then returned to the patient. The operation proceeded successfully and the baby was discharged from the hospital without any adverse effects.

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Labour-induced sickle cell crisis in a previously asymptomatic parturient with sickle cell disease

Faron *et al.* described the first sickle cell crisis triggered by induction of labour in a previously asymptomatic primigravida [1]. I herein report a similar case (and the first triggered by the spontaneous onset of labour) documenting the first lifetime occurrence of a vaso-occlusive crisis in a previously asymptomatic parturient with sickle cell disease.

A 17-year-old, 169-cm, 66-kg, gravida 1, para 0 African American female with sickle cell disease and poor prenatal care (run away from home, unwanted teenage pregnancy) was admitted to the labour and delivery suite at 37.5 weeks gestation in the active phase of labour. She was agitated, cold and thirsty, and complained of severe abdominal, back and chest (rib) pain. Although her past medical history was significant for sickle cell disease (haemoglobin SS), the patient denied any prior painful vaso-occlusive episodes. She was afebrile, had no known drug allergies and no history of prior anaesthesia. Her admission blood pressure was 135/70 mmHg, heart rate 74 beat.min^{-1} and respiratory rate 22 breath.min^{-1}. Pulse oximetry revealed an arterial blood oxygen saturation of 95% on room air. Her chest was clear to auscultation. Haemoglobin and haematocrit were 9.8 g.dl^{-1} and 30.0%, respectively. A peripheral blood smear demonstrated significant sickling of red blood cells and a working diagnosis of sickle cell crisis in a labouring parturient was established. Fetal heart rate was 145 beat.min^{-1} and reactive.

Oxygen via the facemask was administered, warm blankets were applied and the patient was hydrated. When optimised, she consented to combined spinal epidural analgesia (CSEA), which was performed in a standard manner with an 18-guage Tuohy–Schliff epidural needle and a 25-guage Whitacre spinal needle. An intrathecal induction dose of labour analgesia included ropivacaine 2 mg and fentanyl 20 μg. A 20-guage multi-orifice epidural catheter was inserted 4 cm into the epidural space and after negative catheter aspiration for CSF and blood, a continuous epidural infusion of ropivacaine 0.1% and fentanyl 2 μg.l^{-1} at a rate of 12 ml.h^{-1} was initiated. Shortly thereafter, the patient reported complete resolution of her symptoms. She remained haemodynamically stable and comfortable for the remainder of her labour. A 2350-g female infant was delivered vaginally (Apgar scores of 8 and 10 after 1 and 5 min, respectively) 3 h after induction of CSEA. The postpartum period was unremarkable.

Sickle cell disease is an autosomal recessive haematological disorder, which is caused by the single substitution of valine for glutamic acid in the sixth position of the beta chain of haemoglobin [2]. When de-oxygenated, the affected haemoglobin molecules polymerise, causing the sickled shape deformation of the erythrocyte cellular membrane. The sickled erythrocytes subsequently display diminished ability to traverse the capillary of the microcirculation, leading to microvascular occlusion of blood flow, tissue hypoxia, end-organ ischaemia and/or infarction. In the United States, sickle cell disease is most commonly seen in the African American population, with the estimated prevalence of 1 in 700 African American individuals. In patients with sickle cell disease, sickling occurs at a P_aO_2 of approximately 40 mmHg.

A sickle cell crisis typically becomes evident with abdominal and/or chest pain, fever, tachycardia and haematuria. Hypoxia, hypothermia, acidosis, dehydration, stress and/or intercurrent illness are all known precipitants of sickling, and should be avoided. Adequate hydration to maintain intravascular volume and ensure optimal tissue perfusion (preventing systemic acidosis) is necessary. Hypothermia should be avoided, because it produces sickling, probably as a result of vasoconstriction, increased blood viscosity and stasis. Parturients with sickle cell crisis should be well oxygenated, with close monitoring of oxygen saturation [3].

The physiological changes of pregnancy (anaemia, increased oxygen consumption, decreased activity and hypercoaguable state) may exacerbate the medical complications seen in patients with sickle cell disease. Patients with sickle cell disease have an increased incidence of preterm labour, placental abruption, placenta praevia and pregnancy-induced hypertension. It has been suggested that sickle cell crises are more frequent during pregnancy, particularly at term, and that these crises are most likely due to changes in clotting factors activities [4]. Effective management of labouring patients with sickle cell disease/crisis should include prompt administration of labour analgesia [5]. Finer *et al.* reported on a patient in active labour with superimposed sickle cell crisis and theorised that the termination of her crisis may have been the result of improved blood flow and decreased sludging through the affected area due to decreased sympathetic tone from the local anaesthetics used for her epidural labour analgesia [5]. Because of rapid onset of analgesia, the CSEA (as proven in this report) may be particularly suitable for labouring patients with symptoms of vaso-occlusive sickle cell crisis.

In conclusion, I believe that maternal peripartum stress (in this case homelessness, unwanted teenage pregnancy) in addition to dehydration, hypothermia and transient repetitive hypoxia (due to uterine contractions) all combined to play a causative role in triggering the first lifetime sickle cell crisis in our patient.

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Was Che an anaesthetist?

Commandancia de la Plata, Fidel Castro's base of operations during the Cuban revolution, lies at the end of a sweaty hike through misty forest that covers the hills of Sierra Maestre. I visited the camp earlier this year. Photography is forbidden and cameras must be deposited at the Ranger's hut before starting the trek. There is a command post, radio station (Radio Rebelde), a museum and, of greater interest to me, a thatched field hospital.

Here, I saw what was left of the anaesthetic machine. The head, mounted on a stand, had rotameter knobs for oxygen, cyclopropane and nitrous oxide. It resembled that of the Connell Stratosphere anaesthetic apparatus. (I had carefully studied 'Physics for the Anaesthetist' for my Primary all those many years ago.)

Ernesto 'Che' Guevara was a qualified doctor. He was holed up here with Fidel and 300 other rebels, when in 1958, the dictator Batista sent 10 000 mostly unwilling troops to flush them out. A carefully tended grave of one of the rebels lies alongside the path. According to the ranger, he died during surgery 2 days following a strafing attack. Was Che the surgeon or anaesthetist? (could have been both). Did the patient die of cyclopropane shock? Further musings later that evening fuelled by the heat, generously laced mojitos and the live music of the original Quintento Rebelde made me wonder whether I had actually twiddled the knobs of an anaesthetic machine used by Che, the campus icon of my years in medical school in the early sixties.

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