Correspondence

Flow through disposable alternatives to the laryngeal mask

There are now various disposable alternatives to the laryngeal mask, including the Portex Soft-Seal laryngeal mask airway, the Streamlined Pharynx Airway Liner (SLIPA) and the disposable laryngeal mask airway Unique.

The SLIPA has been designed to provide an inexpensive single-use alternative to the laryngeal mask [1]. It is boot shaped with a toe, bridge and heel (Fig. 1). The distal boot shaped part of the SLIPA is connected to a proximal 'shaft', which in cross-section is rectangular. This shaft is connected to the breathing system proximally by a round connection with an inner diameter of 7 mm. The cross-section area of the 'shaft' varies from 8 mm × 29 mm for the smallest SLIPA to 8 mm × 33 mm for the largest. It is not known to what extent flow through the shaft is aided by the large inner diameter (8 mm at a minimum) or impeded by possible turbulent flow through the rectangular 'shaft'.

We therefore conducted a study to compare the resistance to gas flow through the SLIPA, the standard laryngeal mask, the Portex Soft Seal laryngeal mask airway and a Portex tracheal tube (TT). The airways and TT were chosen to represent comparable sises (a size 3 laryngeal mask, a size 3 Portex Soft Seal laryngeal mask airway, a size 53 SLIPA and an 8-mm TT). If the relationship between flow and pressure is not linear, it would imply turbulent flow. A flow of 30 $l.min^{-1}$ was generated through these airways, and there was no significant drop in pressure between the distal and proximal openings, suggesting that the airways pose minimal resistance at 30 $l.min^{-1}$ and therefore turbulent flow could not be a factor at these flow rates.

In order to compare the resistance to gas flow through the different airways we used a mechanical lung model (Dräger LS 800) in which the compliance (C) and resistance (R) was set to low values in order to allow for a high flow rate on expiration, and to mimic a clinical scenario where an additional increased resistance imposed by an airway may have clinical implications. A maximum expiratory flow rate of 122 $l.min^{-1}$ was thus achieved. By using a calibrated 30 L syringe, the true compliance of the mechanical lung was calculated (22 $ml.cmH_2O^{-1}$). In this mechanical lung model, the 'tracheal' pressure is measured, digitised at 300 Hz and recorded using a computer program (LungSense, University of Stellenbosch). It was thus possible to plot the airway pressures against time during deflation (Fig. 2). Since the mechanical lung model does not have a natural exponential drop during the terminal phase

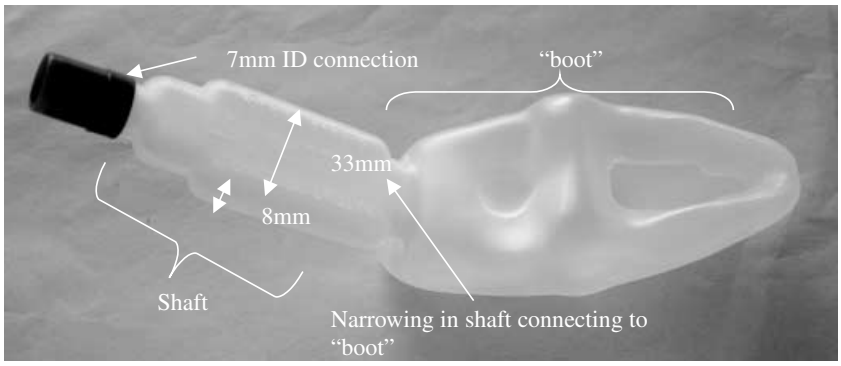

Figure 1

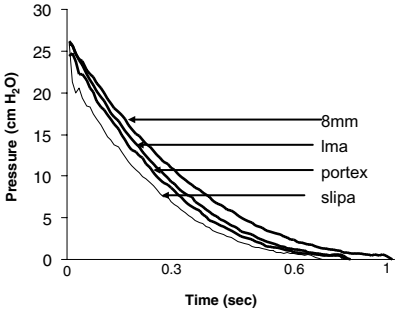

Figure 2

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

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Table 1 Time constant and calculated resistance for the different airways.

Equation from linear regression of a pressure drop from 20 cm H_2O to 10 cm H_2O			τ (s)	Resistance; cm $H_2O.ml^{-1}.s$
SLIPA	$p = 3.176 - (11.90 * t)$		0.084	0.038
Portex Soft Seal	$p = 3.241 - (11.36 * t)$		0.088	0.040
Standard laryngeal mask airway	$p = 3.278 - (10.20 * t)$		0.098	0.045
8 mm ETT	$p = 3.276 - (8.69 * t)$		0.115	0.052
R^2 for all the gradients was > 0.98	p: pressure	t: time	τ: time constant	

of deflation due to the properties of a metal spring, the time constant (τ) for each airway was calculated for a decrease from 20 cm H_2O to 10 cm H_2O by plotting the natural logarithm of pressure against time and performing a linear regression to obtain the slope of the regression line.

Regression yielded the equations for the different airways as shown in Table 1. The time constant (τ) is the inverse of the slope. From the equation $\tau = C \times R$ the resistance (R) for each airway using this model could then be calculated.

Comparing the four curves in Fig. 2 using One Way Repeated Measures Analysis of Variance, there was a statistically significant difference ($p < 0.001$) among the airways, and using Tukey's Test it was shown that there was a significant difference between all airways ($p < 0.001$).

In conclusion, despite the irregular shape of the SLIPA, it does not pose a greater resistance to airflow in a mechanical lung model set at 'worst scenario' clinical setting than the laryngeal mask airway, the Portex Soft Seal laryngeal mask airway and a 8-mm TT. The difference in resistance among the different airways is statistically, but not clinically significant.

G. J. Coetzee
University of Stellenbosch,
Tygerberg 7505,
South Africa
E-mail: gertcoetzee@telkomsa.net

Reference

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General principles of consent

I read with interest the editorial 'Consent and ethics in postoperative pain management (Harmer. *Anaesthesia* 2002; **57**: 1153–4). Prof Harmer is incorrect in saying that the General Medical Council's advice [1] on obtaining consent is intended primarily for surgical procedures. Our legal responsibilities as anaesthetists regarding consent have not changed; however, clinicians' perceptions of consent has. Consent derives from the principle of autonomy; in law this was stated by Justice Cardozo in Schloendorff v Society of New York Hospital [2], 'Every human being of adult years and sound mind has a right to determine what shall be done with his own body'. This principle of autonomy is not limited to surgery and therefore anaesthetists have a duty to gain consent before performing any procedure, be it a general, regional or local anaesthetic. We do this every day in our practice when we explain to patients what is going to occur in the anaesthetic room. Without valid consent, we are assaulting our patients and we leave ourselves open to a civil action based in trespass. This does not occur for the simple reason that consent forms are not the process of consent. This is clearly stated by Bristow J in Chatterton v Gerson [3], "… that getting the patient to sign a pro forma expressing consent …" should be a valuable reminder to everyone of the need for explanation and consent. But it would be no defence to an action based on trespass to the person if no explanation had in fact been given. The consent would have been in form only, not in reality… The duty of a doctor is to explain what he intends to do, and its implications'. Thus, the role of a consent form for postoperative analgesia is limited to that of providing evidence of the process of consent taking place. Similar evidence is provided by making a written note of the proposed anaesthetic technique and the possible complications discussed with the patient. This record could be written on the anaesthetic chart without the need for an extra form.

Considering Prof Harmer's fictitious patient who refused an epidural because of the possible associated insertion of a urinary catheter, the patient is perfectly at liberty to do so; in addition, it is clear from case-law that he does not have to explain his reasons for refusing any option that he has been offered [4].

Finally, I would partly concur with Prof Harmer's suggestion of pre-operative anaesthetic assessment clinics. As I have discussed, consent is a process not a piece of paper and needs time for the patient to appreciate the options available. I feel that providing patients with a large amount of anaesthetic information on the day of surgery may pressure them into accepting the option suggested by the anaesthetist. This is an example of medical paternalism and if it continues, then I feel that a case involving inadequate anaesthetic information disclosure is inevitable. Therefore, we must adopt methods of providing patients with adequate information about our techniques prior to the final pre-operative visit of the anaesthetist. Solutions include both pre-operative anaesthetic assessment clinics and pre-operative information leaflets.

C. M. Danbury
Royal Berkshire Hospital,
Reading RG1 5AN, UK
E-mail: chris.danbury@rbbh-tr.nhs.uk

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Editor's reply
I am delighted that my editorial has led to a heightening of awareness and would agree with Dr Danbury's observations. I would correct his initial assertion that I stated the advice 'is intended primarily for surgical procedures' as the precise words were 'whilst the advice might be intended' because that is the main slant of most consent documents. I would not disagree with Dr Danbury that any procedure performed on a patient should be done with consent.

M. Harmer
Editor-in-Chief

In defence of traditional Chinese herbal medicine

Kam and Liew have reviewed the side-effects and complications of traditional Chinese herbal medicine (TCHM) (Kam & Liew. *Anaesthesia* 2002; **57**: 1083–9). As members of the German taskforce on quality control of Chinese herbs of the German Scientific Society of Traditional Chinese Medicine (DWGTCM e.V.) we would like to clarify some points in this review article.

First, we want to thank our dear colleagues P.C.A. Kam and S. Liew for pointing out the importance of herbal traditional Chinese medicine (TCM) in so many different countries. The impressive numbers support our estimation that more than 1.5 billion people all over the world are trusting in the efficacy and safety of TCM. Taking into account this widespread use of TCM, the number of reported severe side-effects is relatively low. This is even more impressive when compared with the number of side-effects seen with conventional medications. For example 1 in 200 patients treated with NSAIDs for longer than 2 months die of gastrointestinal bleeding [1]. The new Cox-2-inhibitors are not much better: only 1 out of 12 patients treated with these very expensive drugs benefits in terms of prevented fatal gastrointestinal bleeding compared to conventional NSAIDs [2]. Both non-selective and selective Cox-2-inhibitors can cause renal failure [3]. Nevertheless, it should be the goal of all national and international societies in the field of TCM to make the TCM-therapies as safe as possible. The focus should be on two important points: proper education of therapists and good quality of drugs.

Most reported cases of severe side-effects of TCHM are caused by incorrect treatment. Typical examples are the two cases of nephropathy induced by Aristolochia in Great Britain in 1999. Both patients were treated over a period of 2–6 years with the same formula by general practitioners with no education in TCM or TCHM [4]. Such a long period of TCHM-treatment with the same formula is very uncommon. Combined with the lack of proper knowledge by the therapist, this long period of intake could be responsible for the nephropathies rather than the Aristolochia itself. Only therapists with proper training should be allowed to treat patients. Unfortunately, political pressure has forced German officials to halve the time of TCM-training in conflict with the suggestions of almost all German TCM-societies.

We are grateful to Kam and Liew in highlighting the problems of getting herbs of good quality. Most of these difficulties could be easily solved in the few countries where the herbs originate (especially China): toxic contamination, blended herbs, non-proper preparation, incorrect handling of herbs and incorrect labelling. The major way of applying Chinese herbs is still the herbal decoction (in China > 80%), not the patent remedy that was described by Kam and Liew as the main form of application. The quoted report from Clinical Pharmacology and Drug Safety checked without exception only patent remedies, in which 1.53% (32 of 2080) had contaminants of conventional medications and 2% heavy metal contaminants. This can be attributed to illegal drug companies before 1997 located in Hong Kong. For this reason, the Chinese FDA has not only closed some of these factories, but also requires from any herbal pharmaceutical company proof of GMP (Good Manufacturing Practice) by the end of 2003 and will close any company that fails to do so.

Second, we want to correct some misunderstandings of Kam and Liew. The raw ingredients quoted in the review cannot cause toxic reaction in the ordinary patient for the following reasons: Aconitum species are not allowed to be sold unprepared except to pharmacies and thus can only reach a patient in illegal ways. Datura was only added to the Chinese materia medica in the last century, when it still was used in Europe for asthma. Although it is now also listed in the Chinese pharmacopoeia, it is no part of any traditionally applied prescriptions. As for Venenum Bufonis and Podophyllum (correctly spelled 'Gui Jiu'), they are also rarely used remedies, which are only applied externally. An accidental ingestion, as suggested by the authors, is less likely than a mistaken ingestion of amphetamine instead of aspirin because of the similar spelling.

The review tends towards exaggeration, especially if single case reports with nearly no evidential value are quoted, such as the 87-year-old-patient who had a possible adverse effect due to the consumption of too much garlic.

We want to correct the authors' link between TCHM and the 5-element-theory. The prescription of Chinese herbs is usually made with other theories but not the 5-element-theory.

The reported side-effects of TCHM are not specific. Every therapy has its own 'pros and cons'. Dealing with problems of 'alternative medicine' should avoid both 'uncritical enthusiasm… (and) uninformed scepticism' [5].

To focus more on valid data of TCHM interactions with general anaesthesia should be the centre of our attention. In the opinion of the anaesthetists in our group, the interactions of TCHM with anaesthesia are more commonly due to reduced coagulation (especially after heart-surgery) and deeper sedation during regional anaesthesia. For example, salvia milthiorriza and ginkgo biloba do have real effects on blood coagulation and thus should not be taken prior to surgery as with other platelet-inhibitors. But the lowering of blood viscosity is the desired therapeutic effect, not an adverse or side-effect of these drugs. As for the 'Ginseng abuse

Syndrome' with mood elevation and nervousness, it can be said that Ginsengoids Rg1, Rc, etc. have a known interaction with caffeine, which is why prescribers of Ginseng with a proper education in their field, warn their patients not to take caffeine-containing beverages during the treatment period. Our group recommends discontinuing TCHM, especially if taken with western drugs, before anaesthesia.

To evaluate these clinical impressions we invite all anaesthetists to report their experiences to our homepage *http://www.dwgtcm.com/English/english.html*. Hospitals should look to cooperate with patients taking herbal remedies and their therapists. As Kam and Liew have stated, there is only a tiny amount of research into Chinese herbs, as no major western pharmaceutical companies are lobbying or financing this kind of research. In the Peoples Republic of China, 30 years of scientific research has been done, some with impressive results, but remains mostly untranslated and thus ignored.

Herbal prescriptions are medications and any medication that has a main effect may have undesired side-effects too. It is to the credit of Kam and Liew that their review will encourage anaesthetists world-wide to focus on potential herb anaesthesia interactions.

I. Hosbach
G. Neeb
S. Hager
S. Kirchhoff
B. Kirschbaum
German Scientific Society of Traditional Chinese Medicine, Osnabrueck, Germany
E-mail: hosbach@dwgtcm.com

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A reply

We thank Dr Hosbach and colleagues for their interest in our review article and for the opportunity to respond.

It is clear from their comments that traditional Chinese herbal medicines (THCM) are widely used throughout the world. The aim of our article was to increase the awareness of anaesthetists to the adverse effects and the contaminants in THCM. The main problem is that the prescription and preparation of THCM is not controlled in many countries. Because of the absence of strict international and national regulations, patients have access to various traditional medicines that have not been evaluated for safety by good preclinical animal studies, controlled clinical studies and post marketing surveillance. This is compounded by the failure or reluctance of many patients to disclose the THCM they take, and frequently the exact herb (as a dried herb or as a tablet) that is dispensed by the traditional Chinese physician is not revealed to the patient.

Although western literature on THCM is restricted to case reports, we suggest that the adverse effects of THCM are probably under-reported. So far, literature from the Peoples Republic of China that suggests impressive results from THCM are not evidence based and/or poorly controlled. Given that healthcare professionals and patients are ignorant of the possibility of interactions between Chinese herbal remedies and anaesthesia, and also the possibility of added contaminants, the most important step is to increase the awareness of these potential adverse effects to all concerned. We support the views of Drs A Sehgal and J E Hall that 'questioning regarding any herbal medicines should be included in any pre-operative assessment', and this includes traditional Chinese and Indian herbal medications [1].

We agree that THCM is linked to many theories as suggested in our paper, and not only to the 5-element theory.

We hope that we have increased the awareness of anaesthetists to the potential drug interactions and adverse effects of traditional Chinese herbal medications, and call for international and national regulation of all herbal medications (natural remedies, and traditional Chinese, Indian (ayurvedic) and other cultural herbal medicines).

P. C. A. Kam
S. Liew
University of New South Wales, Kogarah, NSW, Australia
E-mail: p.kam@unsw.edu.au

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Transoesophageal echocardiography during surgery for intra-atrial masses

We would like to report the following case of difficulty with diagnosis of a right atrial mass. An 80-year-old-lady was referred to our cardiothoracic unit for urgent resection of a right atrial myxoma. She had recently been seen in the medical outpatient clinic complaining of increasing shortness of breath during the preceding 3 months. On examination, she was noted to have a systolic cardiac murmur radiating to the carotids and bilateral pitting oedema at the ankles. An ECG showed sinus rhythm and her full blood count showed a microcytic hypochromic anaemia. A transthoracic echocardiogram (TTE) was performed. This was reported by a consultant cardiologist as showing moderate mitral regurgitation and a mass in the right atrium that appeared to be pedunculated and attached to the intra-atrial septum (IAS) (Fig. 3). The diagnosis of a right atrial myxoma was made and she was transferred for urgent surgery.

The surgical plan was to use bicaval venous bypass lines to facilitate a right atrial incision and use transoesphageal echocardiography (TOE) intraoperatively to assess the surgical result.

General anaesthesia was induced and the TOE probe inserted by the anaesthetist whilst the patient was prepared and draped. As part of the standard TOE examination [1] a bicaval view was obtained (Fig. 4) demonstrating both atria, the IAS and the inferior and superior caval veins. In this view, the right atrial mass was clearly seen to be arising from the inferior vena cava (IVC) and to not be attached to the IAS. This contradicted the diagnosis of atrial myxoma and surgery was abandoned prior to incision. A CT scan performed the following day showed a renal hypernephroma with extension into IVC.

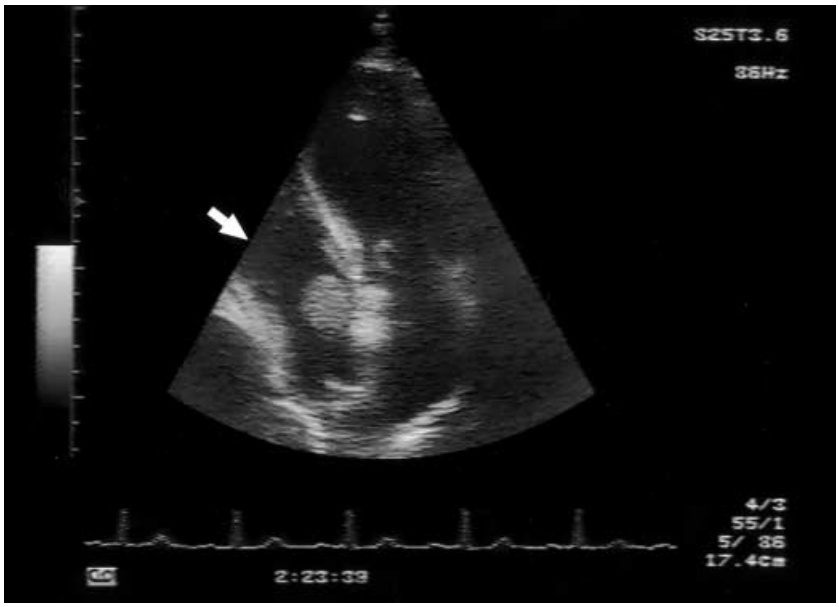

Figure 3 A transthoracic image showing a mass in the right atrium that appears to be pedunculated and attached to the intra-atrial septum.

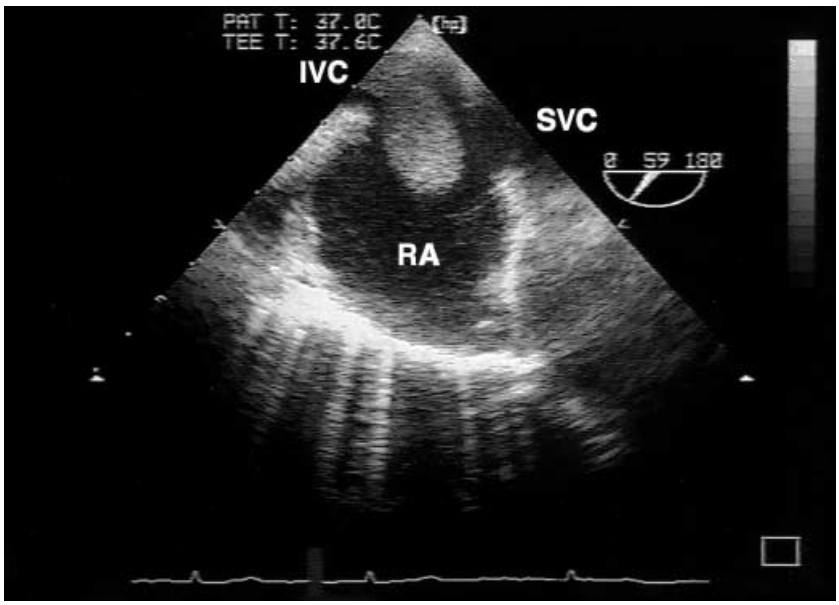

Figure 4 A transoesophageal bicaval view demonstrating both atria and the inferior and superior caval veins. In this view the right atrial mass is clearly seen to be arising from the inferior vena cava.

Intraoperative TOE has been recommended for the evaluation of removal of intracardiac tumours [2]. In this case, TOE prevented an unnecessary operation and potentially averted technical difficulties and morbidity that may have occurred had the IVC been cannulated as planned. However, Leibowitz *et al.* suggest that TOE should be considered in the diagnostic assessment of patients with right atrial masses, even when these masses have been demonstrated with TTE [3]. On the basis of this case, we believe that TOE should be considered for all cases of suspected right atrial tumour prior to surgical referral. In our patient, such a policy would have avoided an unnecessary anaesthetic.

A. Mellor
J. Clark
N. J. Morgan-Hughes
P. C. Braidley
Northern General Hospital,
Sheffield S7 5UA, UK

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A sore throat in a dialysis patient

I would like to present my recent experience with a tragic case of epiglottitis.

A 34-year-old-man presented to the emergency department having been referred by his GP. He gave a 24-h history of sore throat, malaise, nausea, vomiting and sweating. His nausea had improved and he had eaten following his arrival in the emergency department. He was short of breath on exertion but not at rest with no cough or sputum. The patient suffered from chronic renal failure and was currently treated with peritoneal dialysis. He had not dialysed that day but the previous day his peritoneal fluid was clear. He was suffering from chronic renal failure secondary to IgA nephropathy. In the past, he had undergone two failed renal transplants, a parathyroidectomy and had been on haemodialysis. He was currently treated with lisinopril and amlodipine for hypertension.

Supplementary history was obtained from his dialysis unit. He had suffered recurrent episodes of peritonitis and vancomycin-resistant enterococci infection. He had been advised to recommence haemodialysis but had not followed this advice.

On examination, he was sat by his bed, apyrexial, alert and orientated, with an oxygen saturation of 96% in room air. His respiratory rate was 18.min^{-1}, talking freely without distress, pulse 110 beats.min^{-1}, blood pressure 110/60 mlHg, and was noted to be cool peripherally. Examination of his chest was unremarkable. The abdomen was noted to be soft with multiple striae, a dialysis catheter *in situ* and was non-tender to palpation. Examination of his throat showed it to be mildly erythematous but nil else.

His biochemistry revealed: Na 137 mmol.l^{-1}, K 3.7 mmol.l^{-1}, Urea 20.8 mmol.l^{-1}, Creatinine 1.13 μmol.l^{-1}, Bicarbonate 18 mmol.l^{-1}, Alkaline Phosphate 133, ALT 35, Albumin 30 g.l^{-1} and CRP of 21. Full blood count revealed: Haemoglobin 113 g.l^{-1}, WBC 2.6 and Platelets 135. The patient was noted to be lymphopaenic.

His chest X-ray was unremarkable and blood cultures later produced no growth.

He was treated empirically with intravenous fluids and timentin 3.1 g, although the diagnosis was not clear at this time. Approximately 1 h after the initial assessment, the patient developed a hoarse voice and complained of increased difficulty in breathing.

Senior assistance was sought from the anaesthetic department, intensive care unit and the on-call general surgeon. The patient was given nebulised epinephrine 5 mg repeatedly and dexamethasone 8 mg intravenously. The patient's condition continued to deteriorate with increasing respiratory distress and stridor.

An inhalational induction was performed with sevoflurane and the trachea intubated with a size 7 tracheal tube with the aid of a bougie. The epiglottis and surrounding structures were noted to be swollen at laryngoscopy. The patient became hypotensive post intubation requiring inotropes and fluids. He was transferred to the intensive care unit where he was treated with vancomycin 1 g, gentamicin 320 mg and ceftriaxone 2 g. Infusions of epinephrine, norepinephrine and vasopressin were commenced sequentially. The patient was commenced on renal replacement therapy with haemodialafiltration. However, he failed to respond to any of the interventions and died of refractory septic shock approximately 12 h after his arrival in the emergency department.

A swab was taken from the epiglottis, culture of which showed group A Streptococci sensitive to penicillin. A post mortem examination was in keeping with the diagnosis of epiglottitis.

Adult epiglottitis typically presents with severe sore throat and difficulty swallowing usually in a pyrexial patient [1,2]. The presence of respiratory distress at presentation is more variable than in children but is considered along with stridor and the inability to lie flat a strong predictor of the need for airway intervention [1,2]. Even enthusiasts of conservative management acknowledge that airway intervention can be expected to be required in around 15–20% of adult patients [1,2]. Patients can often be managed conservatively but there are other cases where unexpected airway loss has occurred [3]. I believe it is important that such cases are reported so that the literature accurately reflects the incidence of this complication.

This patient was undoubtedly immunosuppressed as a result of his renal failure not being optimally treated. It is likely this contributed to his demise. Overwhelming sepsis complicated this case resulting in acute lung injury and septic shock. I hope to increase awareness that epiglottitis may prove fatal even after the airway is secured.

S. Williams
Nambour General Hospital,
Queensland 4560,
Australia
E-mail: steven85@doctors.org.uk

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Cocaine and pulmonary oedema

Dr Symons has drawn attention to the anaesthetic problems that may be associated with illicit drug abuse (Symons. *Anaesthesia* 2002; **57**: 1142). This problem may be more widespread than hitherto suspected. Recent correspondents have described cases of acute pulmonary oedema in healthy adults at

the time of anaesthesia without obvious cause (Norman. *Anaesthesia* 2002; **57**: 206, Dodd. *Anaesthesia* 2002; **57**: 726). It has been overlooked that cocaine smokers are at risk of acute pulmonary oedema [1, 2] and that this may present during anaesthesia in the manner described by Norman and Dodd [3, 4].

Acute pulmonary oedema in a young otherwise healthy adult without predisposing risk factors must alert the anaesthetist to the possible diagnosis of cocaine abuse. This possibility must always be investigated fully. Experience shows that when such a complication arises, unless a non-negligent cause can be demonstrated, lawyers and their medical expert advisers may attempt to identify or surmise some relevant error or minor misjudgement by the anaesthetist, and claim compensation.

C. Hutter
City Hospital
Nottingham NG5 1PB, UK

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Magnesium sulphate as a first line therapy in the management of tetanus

We were delighted to read the paper (Attygalle & Rodrigo. *Anaesthesia* 2002; **57**: 811–17) indicating renewed interest in the treatment of tetanus, a disease that still affects up to 1000 000 people a year. Most tetanus occurs where facilities for ventilation are limited, and there is a desperate need for new treatments that diminish the need for ventilation, improve cardiovascular stability and reduce mortality. Magnesium offers the possibility of all three and its use has been described in a small number of case reports [1] and uncontrolled case series [2,3]. The authors of this recent series report remarkable success with its use, and conclude by recommending its use as a first line agent in tetanus.

Magnesium poses real risks including muscle weakness, paralysis and cardiovascular side-effects. Reservations have already been expressed in this journal regarding its safety, particularly in settings where facilities for ventilation are not available [4]. Given these very real concerns, it is vital that the drug is carefully evaluated before recommending its widespread use. Our own experience with magnesium is that it may be helpful in controlling spasms and cardiovascular parameters, but the results are by no means clear and we remain very concerned about the risks of profound muscle weakness and hypotension.

Drs Attygalle and Rodrigo state that when designing the study they considered it 'unfair to subject patients to conventional therapy'. We, however, argue that with the available evidence, magnesium remains a potentially exciting treatment for tetanus, but one that carries a substantial risk of causing more harm. A carefully designed double blind randomised controlled clinical trial of sufficient sample size is still required to establish the evidence for magnesium's efficacy and its safety profile. Until the results from such a trial are available, we believe magnesium cannot be advocated as a first line treatment in tetanus.

C. L. Thwaites
J. J. Farrar
The Hospital for Tropical Diseases,
Ho Chi Minh City,
Viet Nam
E-mail: lthwaites@hcm.vnn.vn,
jeremyjf@hcm.vnn.vn

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Minor or major?

We read with interest the paper (Fikkers *et al. Anaesthesia* 2002; **57**: 1094–7), looking at the results of performing percutaneous tracheostomy using the Ciaglia Blue Rhino^{TM} technique. In 100 patients, they managed to achieve a headline complication rate of 36.7% (36/98), of which 6.1% were classified as major. We agree with them that it is important to distinguish between major and minor complications as many of the complications reported in previous papers about percutaneous tracheostomy are of minimal or no clinical consequence to the patient; for example, minor bleeding from the skin edges. We do disagree, however, with their classification of what constitutes a major or minor complication. To classify posterior tracheal wall puncture as a minor complication is wrong – it is this very complication which is most feared during percutaneous tracheostomy, as it may lead to posterior tracheal wall tear, pneumothorax, pneumomediastinum or tracheo-oesophageal fistula formation [1–3]. It is for this very reason that many authors have advocated the use of fibre-optic bronchoscopy during the procedure to minimise the risk of this occurring [4]. We would also contend that accidental extubation, subcutaneous emphysema, puncture of the tracheal tube, difficult tube placement and air leakage constitute major complications. Recalculating their major complication rate raises it from the original 6.1% to 21%. Despite the use of a bronchoscope, the authors still report a very high complication rate, far in excess of the rate seen in our ICU where bronchoscopy is not routinely used – our complication rate in 220 patients (up to the end of 2001) with the Blue Rhino^{TM} technique is 6.4%

(14/220) – 8 bleeds, 1 subcutaneous emphysema, 1 posterior tracheal wall injury, 2 difficult insertions, 1 cuff leak, 1 procedure abandoned.

We agree with the authors that procedure time is not important in the performance of a percutaneous tracheostomy – safety is paramount, not speed. We would therefore ask why they, like several others, have timed their procedures? [5]. They also state that the use of a Crile's forceps for blunt dissection shortened their procedure time. Is this either statistically significant or clinically relevant? The use of blunt dissection increases tissue trauma, increasing the incidence of bleeding and wound infection and so is not part of the procedure in our ICU. For easier passage of the dilator, we recommend maintaining the wet conditions on the hydrophilic coating during insertion and an adequate initial incision. We wish the authors continued success with the Blue Rhino™ technique of percutaneous tracheostomy, and I expect that their complication rate will fall as they gain experience. We strongly advise them, however, to abandon blunt dissection and learn to push a little harder with the dilator!

P. Morgan
R.G. Roberts
University Hospital of Wales,
Cardiff CF14 4XW, UK

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A reply

We thank Drs Morgan and Roberts for their interest in our study. Dealing with the points raised sequentially:

Minor and major complications. We carefully registered all the problems we encountered, even those problems that were easily overcome. We defined major complications as those requiring surgical or medical intervention. Posterior wall puncture, seen at bronchoscopy, in this context constitutes a minor event. The use of bronchoscopy creates a direct view of the posterior tracheal wall. In cases of imminent puncture of the posterior tracheal wall, puncture can be avoided. This is the main reason why we routinely advocate bronchoscopy [1]. We choose to speak of 'puncture' and not of 'perforation', which can be devastating [2]. It is important to note that this would remain unrecognised without performing bronchoscopy. Pneumothorax only can happen if the puncture is off the midline and perforates the posterior tracheal wall. Understandably, we always take precautions to avoid accidental extubation, but it is only a real problem in cases of difficult intubation. The two patients where accidental extubation has happened were easily re-intubated. The only case where subcutaneous emphysema was impressive was related to the use of a fenestrated tube [3]. With appropriate measures, the emphysema disappeared within several hours, so we decided to define this as an annoying, but minor, complication because there were no residual problems for the patient. Difficulties in introducing the cannula were encountered twice and were related to the transition from obturator to cannula-tip. As discussed, the diameter of a Shiley-cannula is relatively large, making introduction sometimes difficult, but not impossible. Puncture of the tracheal tube is a well known problem, which is diagnosed easily by rotating and oscillating the tube to ensure that the needle has not impaled the tube. In our opinion, it does not constitute a major problem either.

Timing of the procedure. We agree that this is usually not very important and is influenced, for example, by the procedure being performed by less experienced colleagues under supervision, as is often the case in a teaching hospital like ours. But sometimes the duration of the procedure, and particularly the time that the patient is not adequately ventilated, may be critical in patients with severe respiratory difficulties. Also in emergency situations, percutaneous techniques may be used [4,5]. In those circumstances, the fastest procedure may be desirable [6]. Therefore, timing of the procedure was and is of interest.

Blunt dissection. This is not always necessary. However, the use of the Crile's forceps was in many cases clinically important. We always perform blunt dissection in the midline, avoiding blood vessels lying more laterally. Identification of the trachea, by digital palpation, was a lot easier, so the puncture was easier and the time of compromised ventilation was therefore shorter than before the use of the Crile's forceps. Many other authors using the multiple dilators technique, and even Ciagla in his original paper, advocate the use of some blunt dissection and so do several authors using the Blue Rhino technique [7–10].

We appreciate the good wishes of Drs Morgan and Roberts, but we want to stress that the meticulous way we prospectively registered our peri-operative complications makes our survey difficult to compare with registrations not done in the context of research.

B. G. Fikkers
J. M. M. Verwiel
F. J. A. van den Hoogen
University Medical Center Nijmegen,
6500 HB Nijmegen, The Netherlands
E-mail: b.fikkers@ic.umcn.nl

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A simple leak detection device for TIVA

Leakage from total intravenous anaesthesia (TIVA) devices is a potential cause of inadequate anaesthesia and awareness. Loose connections between syringes, taps, extension tubing and intravenous cannulae can result in loss of anaesthetic agent. Pooling of propofol and clear opiate solutions on the syringe driver and sheets is not always obvious. We present a simple, low-cost device for detecting leakage in a TIVA system.

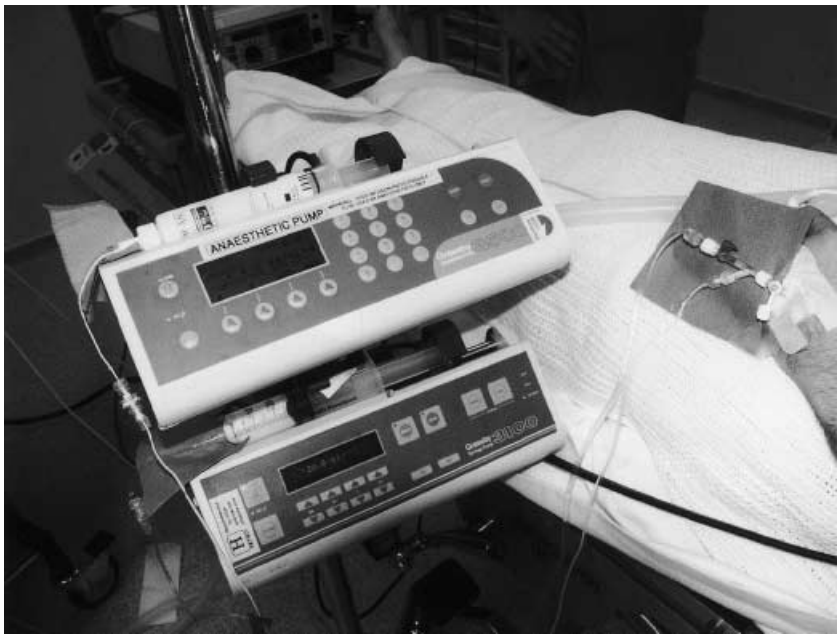

Figure 5

A coloured paper towel, as routinely used in the anaesthetic room, is folded and placed under system connections at the syringe and at the patient cannula (Fig. 5). Fluid leaking onto the paper towel results in a dramatic darkening of the coloured paper, which is appreciable at a glance and from a distance. This simple device adds to patient safety at negligible cost.

A. J. Matthews
E. Rawlings
Derriford Hospital,
Plymouth L6 8DH, UK

Phenylephrine in obstetric regional anaesthesia

We wish to raise awareness of potentially serious drug administration errors that may occur when changing to the use of phenylephrine from ephedrine as the first line vasopressor for obstetric regional anaesthesia.

Recent evidence supports the use of alpha adreno-receptor agonists as an alternative, or in addition, to ephedrine for the treatment or prevention of hypotension during spinal anaesthesia for elective Caesarean section [1–4]. We are concerned, however, about the risks of changing practice *en masse* towards the use of potent agents that may be relatively unfamiliar to many anaesthetists. We suggest the following measures to minimise the risk:

Policy change should be on a unit basis rather than an individual basis.

Units should have a clear, written policy for the dilution and administration of their chosen vasopressor. We append our local regimen as an example.

For phenylephrine, there may be advantages in arranging for the hospital pharmacy to supply premixed solutions.

In addition, we wish to draw attention to the existence of different formulations of phenylephrine. An initial order for the supply of phenylephrine to the delivery suite from the hospital pharmacy at one of our units led to the supply of ampoules containing 50 mg of phenylephrine in 5 ml.

Clearly this represented a hazard, and others should be aware of this.

V. E. Bythell
Royal Victoria Infirmary,
Newcastle-upon Tyne,
E1 4LP, UK
E-mail: vbythell@aol.com

P. Mowbray
University Hospital of North Tees,
Stockton TS19 8PE, UK

D. W. Cooper
James Cooke University Hospital,
Middlesborough TS4 3BW, UK

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Appendix

Protocol for phenylephrine (100 μg.ml^{-1}) infusion during spinal anaesthesia for Caesarean section

IMPORTANT: Dilution of Phenylephrine to 100 μg.ml^{-1}:

Add 10 mg of phenylephrine (1 ml of 10 mg.ml^{-1} solution) to a 100-ml bag of normal saline.

Clearly label as phenylephrine 100 μg.ml^{-1}, date, sign and add red phenylephrine sticker.

Exclude patients with PIH.

Apparatus/preparation

Use a 'Graseby 3400 anaesthesia pump'.

Draw 30 ml of the diluted phenylephrine solution (100 μg.ml^{-1}) into a 50-ml luer lock syringe.

Before attaching to the patient, prime a fine bore infusion line using the bolus facility on the syringe driver.

Connect the infusion line and the i.v. fluid line, via a Y-connector, to an i.v. cannula. Use an anti reflux valve to prevent reflux of vasopressor into the i.v. giving set. Ensure that the i.v. fluid infusion runs continuously during the phenylephrine infusion to flush the dead space in the Y-connector. Alternatively use a dedicated i.v. cannula for the phenylephrine infusion.

Infusion protocol

Baseline maternal systolic blood pressure (SBP) should be measured before coming to theatre. NB the SBP measured in the anaesthetic room immediately prior to spinal anaesthesia is often 10–20% greater than true baseline.

Immediately following intrathecal injection of local anaesthetic:

Start the phenylephrine infusion at 20 ml.h^{-1} (33 μg.min^{-1}).

Measure SBP every minute.

Measure heart rate continuously.

Aim to keep systolic arterial pressure at baseline.

Alter infusion rate by doubling or halving (range 2.5–40 ml.h^{-1}).

If the SBP decreases below baseline, double the set infusion rate (maximum rate 40 ml.h^{-1}). If SBP continues to decrease give 1 ml boluses as required from the syringe driver.

Maternal hypotension (SBP < 75% of baseline) not responding to the maximum infusion rate of 40 ml.h^{-1} and 1 ml boluses: give 3 mg boluses of ephedrine and/or put the patient in the full left lateral position. Treat any associated bradycardia with glycopyrronium.

If the SBP increases above baseline, keep halving the infusion rate, and stop the infusion if < 2.5 ml.h^{-1} is required.

Maternal hypertension (SBP > 125% of baseline): stop the infusion and restart it at half the rate once it has fallen below this threshold.

If heart rate < 60 bpm and the patient is hypotensive, or if heart rate < 45 bpm: give glycopyrronium 200 μg i.v.

NB treating relative bradycardia (heart rate 50–60 beat.min^{-1}) with glycopyrronium when the SBP is normal, or increased, can lead to moderate/severe hypertension.

Discontinue infusion following delivery.

Problems performing a sciatic nerve block in an amputee

We would like to highlight an interesting clinical conundrum that we faced recently. An 81-year-old-male diabetic patient had undergone a below knee amputation for a gangrenous right foot under spinal anaesthesia. He presented on the emergency list one week later for an above knee amputation of the same limb due to infection. This time he was toxaemic and uncooperative. It was decided that a 'light' general anaesthetic with a combined femoral and sciatic block would be the best technique in this circumstance, giving good intraoperative and postoperative analgesia.

Anaesthesia was induced with midazolam, fentanyl and propofol. A laryngeal mask airway was inserted and anaesthesia maintained with oxygen, air and sevoflurane. Under aseptic conditions the femoral nerve was located using a 20G short bevelled polar needle with a nerve locator set at 1Hz with a current of 0.4 mA. Following a negative aspiration test, the block was instituted using 20 ml of bupivacaine 0.375%. However, when the sciatic block was attempted via the lithotomy approach, we realised that we would not be able to elicit contractions of the foot. After careful consideration, 20 ml bupivacaine 0.375% was injected in the groove between biceps femoris and semitendinous muscles at the midpoint between the greater trochanter and the ischial tuberosity at a depth of 6 cm as described by Raj *et al.* [1]. Surgery proceeded uneventfully and the patient emerged from anaesthesia pain free.

Whilst we are aware of the controversies of putting regional blocks in patients who are anaesthetised [2,3], even had the sciatic block been performed awake, we could not have relied on the patient to describe paraesthesia over the sciatic distribution. We would

welcome any comments or suggestions on this case.

C. Lok
P. Kirk
North Manchester General Hospital,
Manchester M8 5RB, UK

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Volume and colour coding for syringes

We read with interest the article: 'Standardised colour coding for syringe drug labels: a national survey' (Christe & Hill. *Anaesthesia* 2002; **57**: 773–98) and thought of reporting our hospital policy regarding syringe labels.

In our hospital, hand-written white syringe drug labels are used but only on volume-coded syringes of specific capacity. For an adult patient, it is a convention to load the drugs in appropriate volume-coded syringe, label and keep them on the anaesthesia machine as follows:

Induction agents	20 ml
Neuromuscular blockers	
Depolariser	2 ml
Non-depolariser	5 ml
Narcotics	1 ml
Anticholinesterase + Anticholinergic	10 ml
Others	3 ml (labelled and kept on side trolley)

We fully agree with the authors that syringe labels should have an international colour coding, but along with it we strongly recommend that the syringes should also be volume coded of specific capacity for specific drugs so that only looking at the volume of the syringe immediately tells the anaesthetist the class of drug it contains.

D. Sood
M. Rupinder Singh
A. Grewal
S. Saini
I. Singh
D.M.C. & Hospital,
Ludhiana-141001,
Punjab, India
E-mail: drgurdeep@yahoo.com

Stylet for reinforced laryngeal mask airway

I read with interest the recent correspondence 'Stylet for reinforced laryngeal mask airway' (Shimoda & Yoshitake. *Anaesthesia* 2002; **57**: 1140–1). We have been using similar introducers for some nine years for safe and simple insertion of the reinforced laryngeal mask airway (RLMA) without the need to guide it by hand inside the mouth. Our original device (Fig. 6) incorporated a metal intubating stylet fixed into a Portex female plastic tracheal connector with cold acrylic glue, and an uncuffed paediatric red rubber tracheal tube was railroaded over the stylet to ensure a 'snug fit'.

A second version was devised shortly after (Fig. 7) that dispensed with the red rubber tube as it was found to be more straightforward to use, particularly when removing the introducer from

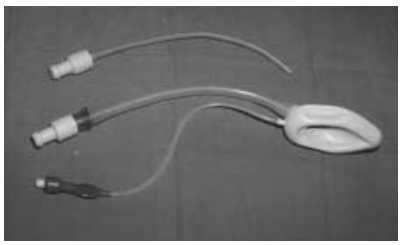

Figure 6

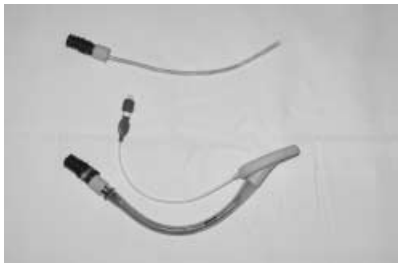

Figure 7

the RLMA when *in situ*. In this device, the tracheal connector stops the RLMA from spinning on the stylet and yet leaves it easy to withdraw once positioned in the patient.

Whilst interesting to learn of a broadly similar solution to this problem, it is suggested that the need to inflate a cuff (to prevent the outer tube rotating) as an alternative to the simpler East Grinstead design, is perhaps an overcomplication and, in use, more time consuming.

P. Andrews
Queen Victoria Hospital,
East Grinstead, RH19 3DZ, UK
E-mail: paul.andrews@qvh.nhs.uk

Measurement of oxygen consumption during low-flow anaesthesia

A recent paper (Leonard *et al. Anaesthesia* 2002; **57**: 654–8), evaluated elaborate techniques for the estimation of oxygen consumption, but it does seem rather perverse to use invasive measurement of cardiac output and the reverse Fick method to do so. Also your recent correspondent (Shankar. *Anaesthesia* 2002; **57**: 1136) does not need to do all those sums to estimate the oxygen consumption of his patients. If he sticks to his first principle of adding enough oxygen to keep a closed circuit full, then it follows that the patient's requirements will always be met. But if the bellows are at the top of the ventilator 'bottle', he will not know how much gas is being dumped from the circuit into the scavenging system. All that is required is for the bellows to be below the top of the bottle and for enough oxygen to be supplied to keep it at a constant level at the end of each expiration. The oxygen added is then equal to the patient's consumption, assuming nitrous oxide is not being used.

In any case, Dr Shankar's sums are a little suspect. He first asserts that the oxygen consumption is limited by the amount of oxygen flowing into the system, and that the patient cannot take up more oxygen than is given to him. This may be true when the oxygen in the circuit is running out, but oxygen consumption is dependent on the

metabolic rate, not on the oxygen supply. Even if the oxygen supply is turned off, oxygen continues to be consumed at the same rate and all that happens to begin with is that the volume in the circuit decreases. If the oxygen concentration in a 6-l circuit is 33%, then it contains 2 l of oxygen. At a consumption of 250 $ml.min^{-1}$, this would be halved after 4 min, and the oxygen concentration would then be 20% (1 l in a total of 5 l).

He then omits to say where the machine is sampling gas. Most machines measuring 'inspired-expired' oxygen levels sample from the patient end of the circle, so the oxygen measured is end-expired, not mixed-expired. This means that his calculation of inspired-expired oxygen difference needs to be multiplied by the alveolar, not the total minute volume, to derive oxygen consumption. He is over-estimating the oxygen consumption considerably. The best place to measure mixed-expired gas concentrations is from the expiratory limb of the circle, as far away from the patient as possible, just before the gases go into the soda-lime canister. The mixed-expired oxygen concentration measured is usually 2–3% higher than end-expired, due to 'enrichment' with dead-space gas (containing inspired oxygen concentrations). The inspired-expired difference is thus only about 2–3%, and multiplying it by the total minute volume will give a fair estimate of oxygen consumption. It is also possible to measure mixed-expired carbon dioxide concentration here, and using end-tidal carbon dioxide and the Bohr equation, to derive dead-space and alveolar ventilation. You have to be very keen on doing sums, however, to favour this method.

D. M. Jackson
Swindon, SW6 6PB, UK
E-mail: dmjackson@doctors.org.uk

Diffusion of nitrous oxide into the cuff of the laryngeal tube

We read with great interest the recent correspondence (Giatini *et al. Anaesthesia* 2002; **57**: 506) in which the authors reported a rise of 15 cm H_2O in the intracuff pressure of the laryngeal tube (LT) 30 min after its insertion, rightly expressing their concerns of mucosal damage that may occur [1,2]. With these concerns in mind and after approval from our ethics committee, and consent from the patients, we conducted a study in which 40 ASA grade I and II patients, undergoing elective surgery had their airways maintained with a LT size 4 (0.3 mm cuff). We assessed the rise in intracuff pressure of the LT with the progression of surgery.

After induction of anaesthesia and muscle relaxation, a size 4 LT was inserted. The cuff of the LT in group A ($n = 20$) was inflated with air to a 60-cm H_2O pressure using a manometer, while in group B ($n = 20$), the cuff was inflated with Entonox to a pressure of 60 cm H_2O. Anaesthesia was maintained with halothane in Entonox. The intra cuff pressure was monitored using a monometer at 10 min interval for a period of 30 min.

We found that in group A, the mean (sd) rise in intracuff pressure was 12 (3) cm H_2O while in group B the rise was 4 (2) cm H_2O. Our results confirm the earlier study of Giatini *et al.* and possibly provide a cheap and easy method to solve the problem of nitrous oxide diffusion into the LT cuff.

S. A. Khan
M. M. H. Siddiqi
R. M. Khan
J.N. Medical College Hospital,
A.M.U. Aligarh, India
E-mail: drshariqalikhan@hotmail.com

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Anaesthesia for cardioversion

We conducted a postal survey of anaesthetic departments which enquired about anaesthetic practice for cardioversion. A questionnaire was sent to consultant anaesthetists at 150 randomly selected hospitals throughout the UK. Questions were designed to establish basic data about the site and timing of the procedure and the personnel, drugs and equipment available.

Of the questionnaires sent, 107 (71.3%) were returned. The results showed that 86% of cardioversions were performed either in theatre or a critical care area. A consultant anaesthetist provided anaesthesia in 79.5% of cases whilst less than 5% were administered by an SHO. This can be compared to the 46% and 18.5% of cardioversions performed by consultant physicians and SHO's, respectively. Anaesthesia for cardioversion was provided by on-call staff in 53% of cases whilst 40% were done during a specifically allocated session. Ninety-eight percent of cardioversions were done during normal working hours and skilled anaesthetic assistance was available for 86% of cases. Recent electrolyte and coagulation results were available in 79% and 69% of cases, respectively. Adequate emergency airway equipment, suction and drugs were available for 90% of cardioversions, whilst full monitoring was available in 85%, and an anaesthetic machine was used in 69% of cases. Propofol was the anaesthetic agent of choice in 90% of hospitals whilst 9% used etomidate; 43% of departments used short-acting opiates to provide analgesia during cardioversion. A written protocol for anaesthesia for cardioversion existed in 14% of anaesthetic departments, but only 7% actively audited their results.

The Royal College of Anaesthetists has published recommendations regarding the safe conduct of anaesthesia for cardioversion [1]. Concerns have been expressed that cardioversion is often performed out of hours, in remote sites, by inexperienced anaesthetists, with suboptimal assistance and equipment [2]. The results of our survey show that, in general, the standards of equipment and staffing are high with the vast majority of cardioversions being performed by consultant anaesthetists in an

appropriate setting during normal working hours. However, few departments have written protocols for, or actively audit the results of cardioversion.

S. James
I. J. Broome
Falkirk & District General Hospital,
Falkirk, UK
E-mail: ian.broome@fvah.scot.nhs.uk

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Let there be light

Fibre-light laryngoscope blades can be dismantled and the bulbs sterilised and re-used. We report a critical incident arising from incorrect assembly.

Prior to an emergency intubation, a laryngoscope was tested and a bright light was seen from the tip of the blade. On inserting the laryngoscope into the patients mouth, no light was apparent. Direct laryngoscopy was therefore not possible.

On further inspection of the laryngoscope, a size 4 MacIntosh blade had been assembled with a size 3 fibre-light bulb. The size three bulb does not extend sufficiently through the flange of the blade (Figs 8 and 9). Consequently, the patient's tongue occluded the light source. We believe that this represents a unique problem to the fibre-light bulb system with the potential for patient harm.

Figure 8 Above: a Mac 4 blade with a size 3 fibre-light bulb: it does not extend through the flange. Below: correctly assembled.

Figure 9 The size 3 bulb can be occluded by the patients tongue.

R. J. Price
D. Russell
M. A. Garrioch
Southern General Hospital,
Glasgow G51 4TF, UK
E-mail: rjp@doctors.org.uk

Air in the epidural space leading to a neurological deficit

We report a case of transient neurological impairment following the use of air to detect loss of resistance during placement of an epidural catheter for labour analgesia.

A 29-year-old Asian female, primagravida presented to the delivery suite in labour at 39 weeks gestation having had an uneventful antenatal period. Her uterine contractions were of moderate intensity, palpable every 4 min, with a foetal heart rate of 140 beat.min^{-1} without any decelerations. Pelvic examination revealed an effaced cervix, 2–3 cm dilated, with a cephalic presentation at minus 2 station. Intramuscular pethidine was used for relief of labour pain after artificial rupture of the membranes. After the start of an oxytocin infusion, the patient requested epidural analgesia.

Following a full explanation of the risks and benefits, the epidural was attempted with the patient in the sitting position using a 16G Tuohy needle at the L3–4 space and an air-filled syringe to detect the loss of resistance. The first attempt was unsuccessful so a second attempt was carried out using the same space. Despite difficulties, the epidural space was eventually located using approximately 10 ml of air. The catheter was then advanced about 4 cm into the epidural space. Neither cerebrospinal fluid nor blood could be aspirated from the epidural catheter. Following a test dose of 2 ml of lidocaine 2% with 1 : 200 000 epinephrine, a bolus of 20 ml of a mixture bupivacaine 0.1% and fentanyl 2 μg.ml^{-1} was injected into the catheter. This was followed by a continuous infusion at 10 ml.h^{-1}. After 20 min, pain relief was unsatisfactory despite withdrawal of the catheter by 1 cm and repeated bolus doses of 10 ml bupivacaine 0.25%.

The epidural catheter was therefore re-sited for the third time at the L1–2 space using 3 ml of air from a 10-ml air filled syringe to detect loss of resistance. Good analgesia was achieved with this attempt providing a sensory level of block up to T7. Normal delivery occurred 6 h later. Following this, the epidural catheter was removed prior to discharge to the postnatal ward.

The following day, the patient complained of numbness in her right buttock and foot. Neurological examination revealed multiple areas of altered sensation over the right buttock and lateral aspect of the right calf corresponding to dermatomes L4/5. There was no motor weakness with intact deep tendon reflexes. Perineal sensations were present with a normal rectal sphincter tone. Computed tomograph (CT) scans showed a low absorption image consistent with the presence of gas in the epidural space. EMG revealed no conduction disturbance.

On day 4, sensations started to return on the lateral aspect of the patient's right leg and there was complete neurological recovery over the right buttock and thigh within the proceeding 5 days.

We consider that the air injected into the epidural space may have been responsible for the neurological symptoms experienced by the patient. The return of sensation after 4 days suggests that air pockets in the epidural space take a few days to absorb into the blood stream. There was no other

apparent cause for this neurological deficit. This iatrogenic problem could have been potentially avoided by using saline instead of air to detect loss of resistance.

Various neurological deficits caused by air in the epidural space have been reported including acute nerve root compression [1], chronic radiculopathy [2] and cauda equina syndrome [3]. There is therefore a risk of inflicting a neurological deficit following the use of air to detect the epidural space, which can potentially lead to protracted investigations to exclude any neural damage and a prolonged hospital stay. This sort of complication might be occurring more frequently than is reported and it is our firm belief that air should no longer be used during epidural catheter placement. However, anaesthetists who continue the practice of injecting air for the identification of the epidural space should be aware of this and associated complications.

Krishnam
A. Mallick
Leeds General Infirmary,
Leeds, LS1 3EX, UK

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Treating ECG changes during Caesarean section: is it worth the headache?

ECG changes are not uncommon during Caesarean section [1,2]. In the vast majority, these changes are innocuous and do not require any intervention. The most disconcerting are ST segment depression. However, intervening can literally result in a severe headache for the mother as we found out.

Our patient was a 27-year-old-mother, a doctor, who required operative delivery for failure to progress. An epidural was placed at the L 3–4 level during labour, which was topped up for the Caesarean section. The block was effective up to the T4 dermatome level. Shortly after, the systolic blood pressure dropped from 105 mmHg to 70 mmHg, and as she complained of nausea, a 6-mg bolus of ephedrine was administered. Over the next 10 min, further boluses of ephedrine totalling to 24 mg was given to restore the blood pressure. In the meantime, surgery had commenced and a healthy male infant was delivered. A syntocinon-bolus of 5 units was administered and an infusion of 20 units in 500 ml of saline commenced. About 30 min into the operation, the ECG trace showed marked ST depression (2 mm) but the mother did not complain of any symptoms. The heart rate was around 75–80 beat.min^{-1} and the systolic blood pressure 85 mmHg. The syntocinon infusion was discontinued and metaraminol 0.25 mg administered. It was at this stage that the ECG changed from sinus rhythm with ST segment depression to junctional rhythm with a rate of 55 beat.min^{-1}. The mother remained symptomless, even as the systolic pressure dropped to 75 mmHg. Surgery was stopped temporarily to rule out a vagal reflex to handling of the uterus, but it did not restore the rhythm. A bolus of atropine 0.3 mg was given to correct the dysrrhythmia, but within a minute following the injection, she complained of a severe headache, so severe that she became acutely distressed. The heart rate increased to 150 beat.min^{-1} and the systolic blood pressure to 160 mmHg. Fentanyl 100 μg and midazolam 2 mg was given to ease the pain and reduce the anxiety, but the headache remained. Surgery was allowed to proceed as further increments of fentanyl (25 μg) were given to ease the intensity of her headache. The mother was transferred to the recovery room an hour after the start of the operation where she was fully monitored. The heart rate was down to 120 beat.min^{-1} and systolic blood pressure 110 mmHg. A 12-lead ECG was recorded, which showed a normal sinus rhythm with no ST depression. The headache persisted and lasted over 2 h. She made a full recovery and serum troponin I levels taken 12 h later proved to be normal.

Headaches are reported adverse effects of ephedrine, metaraminol and atropine. These are usually brief and of mild to moderate intensity. Our patient experienced such an intense headache and for over 2 h that we had to consider pre eclampsia in our differential diagnoses. The question remains: should we have intervened as we did? We were concerned as the ECG progressively changed from sinus rhythm to 'significant' ST depression, then to bradycardia with ST depression and through to a junctional rhythm. Throughout these changes, the mother remained symptomless despite the hypotension. There is at least one study that showed that ST segment changes are not necessarily innocuous [3]. Syntocinon has been implicated as one of the causes [4]. However, in view of the presence of a dysrrhythmia and hypotension, we felt it appropriate to intervene. Would glycopyronium have been a better alternative? Was this a case of treating the patient and not the ECG? We would like to know what others might do if confronted with a similar situation. We feel that if faced with the same problem again we would be compelled to intervene, but probably use glycopyronium.

K. Ramachandran
Srirangadarshan
V. Kapoor
North Manchester General Hospital,
Manchester, M8 5RB, UK

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The use of nitrous oxide in anaesthetic practice: a second questionnaire survey

Following our questionnaire survey among consultant anaesthetists (Henderson *et al*. *Anaesthesia* 2002; **57**: 1155–8), it was felt that a further survey should be carried out among junior anaesthetists to ascertain their views on the use of nitrous oxide in anaesthetic practice.

Whilst we achieved a 75% response rate amongst consultants, we only had a 42% response rate amongst the trainee grades. This may reflect either the increased mobility of trainees so that fewer of the questionnaires were received, or that the trainee population are less interested in the topic of this questionnaire. However, a total number of 206 questionnaires were returned, which is not an insubstantial number. Of the valid questionnaires, 52% were male, 41% female and 7% did not state. Thus the sample had a much greater proportion of female anaesthetists than the original consultant questionnaire. The minimum number of years in clinical anaesthesia was 3 months; the maximum was 28 years, with a mean of 4.96 years.

Generally the responses followed that of the consultant body, with 81% stating that they used nitrous oxide frequently and 19% occasionally. Over the last 3 years, 42% said that their use had stayed the same, with 55% stating it had decreased and 3% saying it had increased. An overwhelming majority (96%) stated that they avoided using nitrous oxide for certain operations, 4% stated they wouldn't avoid using nitrous oxide.

Most trainees (74%) did not feel that pollution from nitrous oxide was a problem, but 79% did not feel that their use of nitrous oxide had been influenced by the effect of pollution on staff or other health implications. A small number of trainees (5%) felt that it would be acceptable to make nitrous oxide unavailable, 32% felt that it should be available on request and 62% felt that it should remain freely available. Thus, 38% felt that there should be some restriction in nitrous oxide availability, whereas only 20% of consultants felt there should be some restriction in its use.

Half the trainees (50%) stated that all their anaesthetic machines had the provision of medical air, 48% said that some machines have medical air and 2% that none had provision. As to when medical air is used, 68% stated frequently, 30% occasionally and 2% never.

When asked about their use of total intravenous anaesthesia (TIVA), 57% stated that over the last 3 years their use of TIVA has increased. When TIVA is used, 80% supplement with oxygen/air and 20% with oxygen/nitrous oxide.

Generally the responses indicated that the majority of trainees who took part felt that nitrous oxide pollution in operating theatres was not a problem. A total of 18% have decreased their use as a result of health and pollution concerns; however, 14% considered nitrous oxide to be a problem but had not altered their practice.

Further analysis was carried out on the female responses to determine whether there was a greater concern for health and pollution issues among this group. A total of 26% stated *that this was the case*, whereas 14% of the male responders felt there was a problem. A minority of females (18%) had changed their practice as a result of health and pollution issues and 13% felt that it was a problem but had not changed their practice.

Despite the poorer response rate from the trainee grades to this questionnaire, the absolute number of responses was not negligible and there appears to be a greater concern about the effects of health and pollution generated from nitrous oxide amongst this group. A higher percentage of trainees than consultants (38% vs 20%) would like to see a restriction on the use of nitrous, again raising the question as to whether nitrous oxide should still be readily available on the anaesthetic machine?

K. A. Henderson
N. Raj
J. E. Hall
University Hospital of Wales,
Cardiff, CF14 4XW, UK
E-mail: hallje@cf.ac.uk

Concealed airway complication during LeFort I osteotomy

We would like to report an unusual but potentially serious airway complication of Lefort I osteotomy. A 34-year-old-female, weighing 80 kg, presented for LeFort I osteotomy for the correction of class III malocclusion. The pre-operative physical examination and the routine blood tests were normal. She was anaesthetised with propofol, remifentanil infusion, rocuronium, and sevoflurane in oxygen and air. The trachea was easily intubated with a preformed nasal tracheal tube (Portex 6.5 mm internal diameter) via her left naris. The surgical procedure was completed in 2 h and the intra-operative anaesthetic management was uneventful. Ventilatory parameters including peak airway pressure, expired minute volume and end-tidal carbon dioxide were continuously monitored throughout the procedure. On completion of the operation and after attaining spontaneous breathing and regaining consciousness, the trachea was extubated. At the time of extubation a cut was noticed on the lateral aspect of the tracheal tube (Fig. 10), which was 45 mm away from the black mark. It was an oblique cut in the longitudinal plane of the tracheal tube and it measured 12 mm in length.

On closer examination of the cut edges (Fig. 11), it was confirmed that the pneumatic saw that was used on the maxilla had made the cut on the tube. The cut involved the full thickness of the wall of the tracheal tube. It had very narrowly missed the pilot tube hence the cuff was intact. Surprisingly and fortunately, this cut did not produce a gas leak. Also, no changes either in the peak airway pressure or in the expired minute volume were noted during the

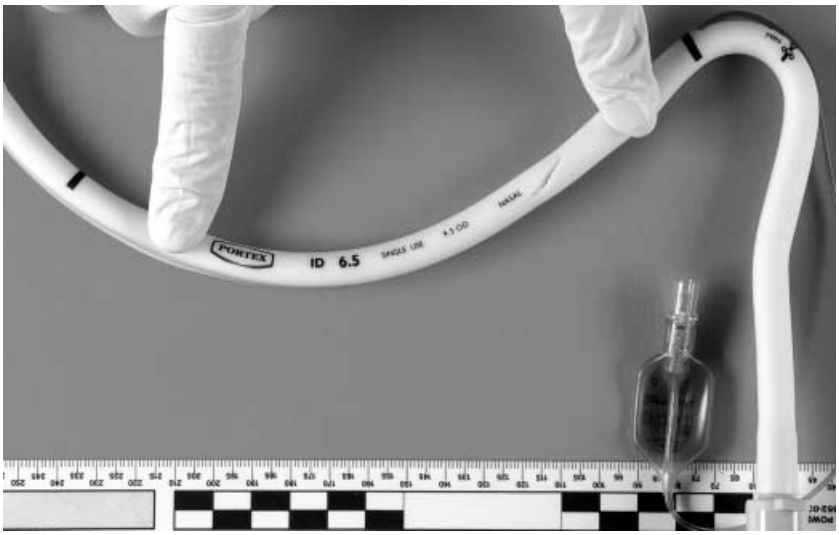

Figure 10

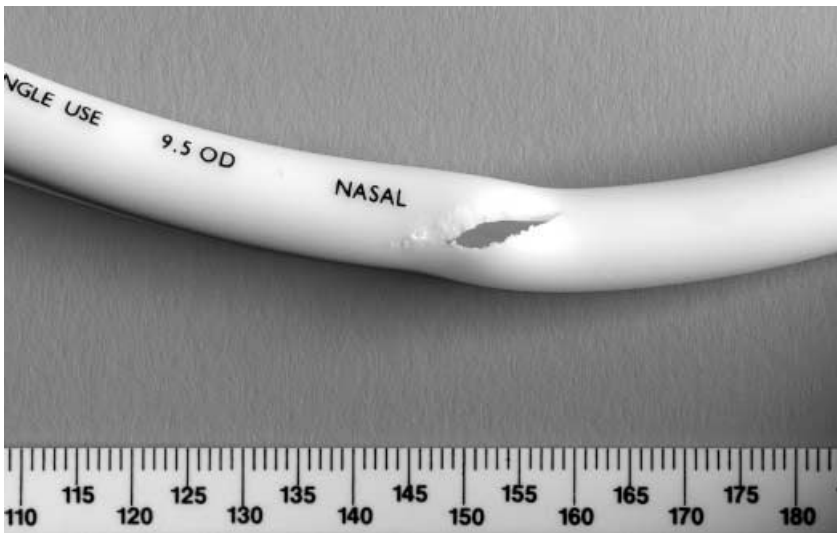

Figure 11

procedure. A fresh gas flow of 1.5 $l.min^{-1}$ was used during the maintenance phase of anaesthesia.

Our aim of reporting this case is to increase awareness of a possible serious airway complication during maxillary osteotomy. The nature of the surgical procedure and the intermaxillary fixation at the end of the procedure requires nasotracheal intubation. The surgical procedure involves mobilisation of the maxilla from the lateral nasal wall, nasal spine and the nasal septum. During these surgical steps, the nasal tracheal tube is vulnerable to the surgical instruments. There are few reports of inadvertent severing of the nasal tracheal tube during maxillary osteotomy [1–4]. Several options for managing the airway have been described. Packing around the tracheal tube may seal the air leak in certain situations [1], whereas on some occasions, there may be a need to change the tracheal tube during the middle of the surgical procedure [4]. Bypassing the tear in the tracheal tube by inserting a smaller uncuffed tracheal tube through the lumen of the original tube may help to ventilate the patient [2]. The Cook airway exchange catheter would also be a useful tool for changing the tracheal tube. Hought *et al*. have described the use of a metallic tube protector to avoid accidental damage to the nasal tracheal tube [5].

In conclusion, during the maxillary osteotomy and similar orthognathic surgical procedures, one should be very vigilant for the immediate recognition of any damage to the nasal tracheal tube. An alternative plan for managing the airway and appropriate equipments should be readily available.

M. Adke
C. Mendonca
University Hospitals of Coventry and Warwickshire,
Coventry CV2 2DX, UK
E-mail: mendoc@doctors.org.uk

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Not NICE advice

In September 2002, the National Institute for Clinical Excellence (NICE) published a document entitled: Guidance on the use of ultrasound locating devices for placing central venous

catheters [1], which recommended that two-dimensional (2-D) imaging ultrasound guidance should be the preferred method for elective internal jugular vein catheterisation in adults and children. One could make any number of criticisms of this document: for instance, references for the 20 randomised controlled trials on which the guidelines was founded are not quoted, there appears to have been a general lack of consultation with anaesthetists (who site the majority of central lines in hospitals), there is an apparent lack of clinical experience of central line insertion of 36/41 of the appraisal committee members, data that did not support the proposed guidelines has been suppressed (paragraph 4.1.6), none of the trials reviewed involved elective non-cardiac surgical patients, and clinically irrelevant endpoints were used (does it really matter that 2-D ultrasound guidance quickens central venous catheterisation by 69s?).

Several important points need to be made about this guidance. Firstly, there is an issue of cost [2]. Lacking evidence of cost effectiveness from the trials reviewed, NICE based their analysis on a theoretical model, using a set of unjustified assumptions. Amongst other variables, their analysis suggested that cost savings were made because the ultrasound method 'avoided 90 arterial punctures for every 1000 patients treated'. My logbook tells me that in 7 years of anaesthesia, I have punctured the carotid artery five times during 1095 otherwise successful attempts at central line placement (all involving a 19G locator needle; two of the five cases related to misinterpretation of a 2-D ultrasound image), which tends to indicate that the cost effectiveness model used may not be terribly accurate. In addition, the initial equipment expenditure for hospitals would be significant (at our hospital, 15 machines costing at least £6500 each – Site Rite™ II).

Second, there is an issue of training. I would suggest that, if my own experience is anything to go by, the reason for a significantly higher complication rate using the landmark method is that operators have never been trained properly in its performance – the 'see one, do one, teach one' approach that forms the usual teaching of the landmark technique will always fare poorly in terms of outcome when compared to an ultrasound guided technique that involves a 2–3 h initial training session.

Finally, significant legal implications may result from this pronouncement. Guidelines are not law, but conduct that departs from accepted practice may lead to liability in negligence [3], in this instance both for the doctor (for failing to use ultrasound guidance) and vicariously for the hospital (for failing to provide the equipment/enforce the code of practice). It would be for the court to decide, however, whether non-compliance with the guidelines was an unreasonable departure from accepted practice. Interestingly, the court could be asked to consider whether the guidelines are reasonable or rational in the first instance.

Until the logistical and medicolegal ramifications of these guidelines are resolved, it would seem prudent for anaesthetists to inform patients that the new guidelines exist but that equipment and expertise are not yet available, whilst continuing to inform the patient about the risks and consequences of central line insertion via the landmark method.

S. M. White
St.Thomas' Hospital,
London, SE1 7EH, UK
E-mail: igasbest@hotmail.com

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Sevoflurane conscious sedation for MRI scanning

While undergoing conservative treatment for a gastrointestinal complaint as an inpatient, a 48-year-old-lady developed symptoms suggestive of a cauda equina syndrome. An urgent magnetic resonance imaging (MRI) scan was arranged, and while in the MRI suite, the patient expressed concerns about going into the scan room. The orthopaedic SHO administered diazepam 20 mg. This had minimal effect and the patient still felt unable to cooperate. Therefore the anaesthetic team was asked for support. In the patient's past history, an endoscopic retrograde cholangiopancreatography (ERCP) had to be postponed and later to be performed under sedation as the patient suffered from a fear from enclosed spaces. Her BMI was 41 $kg.m^{-2}$ and she had a full meal less than 6 h ago.

We offered a trial of conscious sedation to the patient, explained the technique to her and showed her the facemask to be used. Following reassurance, the patient felt able to enter the scan room and was asked to hold the facemask as close to her face as she could possibly manage. She was given 50% nitrous oxide in oxygen and sevoflurane 0.5% to breathe. Verbal contact was maintained with the patient throughout the procedure, and the patient's heart rate and oxygen saturation were monitored continuously. Furthermore, she was given an alarm button that she could press should she experience any form of distress and which would lead to the procedure being stopped and the patient being taken out of the scanner. The procedure was completed without problems, and the patient was pleased with this form of treatment. As the investigation confirmed a cauda equina syndrome, an emergency operation was arranged.

About a year later, the patient was admitted to our hospital again for an elective operation to be anaesthetised by one of the authors. On this occasion, she reconfirmed that the conscious sedation had been much appreciated by her, and that her neurological

symptoms had improved dramatically following the operation.

Sevoflurane conscious sedation as described by Lahoud *et al.* [1] has become an established part of anxiety management in community dentistry. The technique described by Lahoud *et al.* employs the use of nitrous oxide 40% in oxygen, supplemented by sevoflurane 0.1–0.3%, administered through a nasal mask. Lahoud *et al.* report a success rate of over 90% for effective treatment of anxiety during dental procedures, which is a major improvement of the results that can be achieved with nitrous oxide in oxygen alone (40% failure rate). The advantage of inhalational sedation over intravenous sedation is its ease of controlling the depth of sedation as gases are quickly exhaled again, whereas metabolism of intravenous drugs takes considerably longer.

We are not aware of the routine use of conscious sedation in the MRI setting. For this patient, the technique allowed us to alleviate her fears and enabled her to cooperate with the procedure, and avoided general anaesthesia in a patient with a full stomach. A slightly higher concentration of sevoflurane was chosen for our patient, to compensate for the dilution of anaesthetic gases as the patient felt unable to hold the mask directly onto her face.

Sevoflurane conscious sedation proved to be a valuable alternative to a general anaesthetic in this case, especially as intravenous sedation had already failed. The patient was comfortable, maintained her own airway and retained verbal contact with the treating team at all times.

Scavenging remains a problem, especially if a patient is not able to tolerate a tight fitting mask. Further studies are needed to evaluate the possible use of conscious sedation for elective MRI procedures on a regular basis.

S. Jürgens
Queen's Medical Centre,
Nottingham, NG7 2UH, UK

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To premed or not to premed

National Anaesthesia Day is our opportunity as a profession to make contact with the public and provide them with a better understanding of what exactly it is that we do. Equally, it is also a valuable time to explore the public's perceptions and views on various aspects of our day-to-day practices. During the last National Anaesthesia Day, we conducted a survey at the Oxford Radcliffe Hospitals to look at people's experiences and understanding of the use of premedication.

Members of the general public who viewed the National Anaesthesia day displays were asked to complete a 4-point questionnaire about premedication. The questions asked included.

When you have an anaesthetic for an operation do you expect to be given a premed before you go to the operating theatre? What do you think premeds are given for? Have you ever been given a premed before an operation and wished you had not? Have you ever had an operation without being given a premed when you wished one had been prescribed?

We had 92 responses to our questionnaire. These were completed by a cross section of the public including patients, outpatients, visiting relatives and friends and members of staff (non-theatre staff). The majority (68%) indicated that they expected to be given a premed before an operation; 20% were undecided and only 12% did not expect to have a premed. The public were asked to indicate from a list of options the possible reasons for having a premed. The responses to this question are provided in Table 2. Of those people who had previously had an operation, only 13% who were given a premed wished they had not been; while 18% of those who had been to theatre without being prescribed a premed indicated that they would like to have had one. There are a wide variety of indications for various drugs to be given prior to induction of anaesthesia and the commencement of surgery. Premedication is frequently the part of the anaesthetic procedure that people will experience and remember as they wait on the ward for their operation; therefore, it seems likely that they will have some opinions about its use. This brief survey indicates that this is the case and that there is an expectation on the part of the public and hence patients for premedication to be given. We should therefore include a full discussion about premedication with all patients, whether it is our intention to prescribe it or not. Our survey showed that most patients knew that anxiolysis and sedation are key reasons for prescribing premedication but far fewer were aware of the other, often more frequent reasons for giving premedication. It is important that patients are made aware of the nature of the premedication that has been prescribed for them so that they are not disappointed by the lack of sedation experienced following premed of, for example, ranitidine and metoclopramide. Whether to prescribe or not to prescribe a premed depends on the individual patient, anaesthetist, medical and surgical factors; however, it is worth remembering that patients have their own expectations and views on the subject.

Table 2 The public's view of the reasons for premedication.

Reason	Number (%)
To make you sleepy	59 (64%)
To stop you feeling anxious	62 (67%)
To settle your stomach	16 (17%)
To give you pain relief	18 (20%)
To stop you feeling sick	20 (22%)
To reduce the swelling caused by the operation	6 (7%)
To control your blood pressure	18 (20%)
To control the amount of saliva you make	26 (28%)
None of the above	0 (0%)
Don't know	7 (8%)

K. C. Hames
John Radcliffe Hospital,
Oxford, OX3 9DU, UK

Midwifes putting the pressure on…?

We read with interest the article relating to application of cricoid pressure using a part task trainer (Owen *et al. Anaesthesia* 2002; **57**: 1098–101). We recently undertook a similar audit in our department. The original idea was born after a

midwife was noted to have said to one of the specialist registrars (SpRs), in a rather indignant fashion, 'we can do cricoid too, you know!' whilst the said SpR was awaiting the arrival of the operating department practitioner (ODP) for an emergency Caesarean section. This lead us to wonder whether there was any difference in the accuracy of cricoid pressure applied by frequent and non-frequent users.

We surveyed 52 members of the theatre staff including all members of the anaesthetic medical staff, all ODPs, recovery staff and some intensive care staff. The survey consisted of a questionnaire relating to length of time since training, any training specific to cricoid pressure, frequency of application of cricoid, knowledge of correct forces and any complications. We then assessed accuracy of cricoid pressure using the 50 ml air-filled syringe technique mentioned by Ashurst *et al.* [1] and Flucker *et al.* [2]. After an initial attempt, the operators were shown the distance of the syringe that they had to move the plunger in order to apply a 30-N force.

Our results were similar to Owen's group in as much as the pretraining range of results was wide, with a range 8–60 N, median 30 N, mean 29 N, and mode 40 N. After training, there was a vast improvement, with the range being 20–40 N, median 30 N, mean 30 N and mode 30 N. We then compared those who performed cricoid regularly, i.e. more frequently than once a month, to those who performed it seldom or never. Training in cricoid pressure had been received in 54% of our group, mainly theatre ODPs, who were the group who performed cricoid frequently. The original training had been in the form of verbal instruction in 33%, a mixture of verbal instruction and hands on patient experience in 47%, whereas only one person had received any training on a mannequin or mechanical device. All training had been given during original ODP training, which varied from 1 year to 18 years previously. The results between the 2 groups showed that there was no difference in the accuracy of cricoid pressure applied. Frequent group range 8–60 N, median, mean and mode all 40 N. Infrequent group range 12–40 N, median 30 N, mean 29 N and mode 40 N. We conclude that there is no difference in the effectiveness of cricoid applied by frequent or non-frequent users, especially when the original training techniques were perhaps inadequate. So what should our answer have been to the midwife in question, when maybe she could have performed as accurate cricoid pressure as some of our trained staff? Another thought, perhaps, is that midwives could be trained to apply cricoid pressure, which would leave the ODP with hands free to assist with any potential airway problem (that idea ought to put the pressure on!…).

C. Mearns
J. Elliott
East Surrey Hospital,
Redhill RH1 5RH, UK

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Why so slow?

We would like to report an interesting case of bradycardia.

A 52-year-old-gentleman presented for bilateral excision of hidradenitis suppuritiva. His previous admission had been for the treatment of a 'black' toe secondary to an embolus. He was not on warfarin at the time as his down and out lifestyle was deemed not conducive to anticoagulation.

He initially declined to have a transthoracic echocardiogram but was convinced to undergo the procedure last year. The result hinted at a mild increase in the intraventricular septum but good left and right ventricular function. Although the left atrial appendage was not visible, there was no signs of any vegetations or mural thrombi.

Pre-operatively he appeared to be in total heart block [Figs 12 and 13] but had no symptoms of syncope. His exercise tolerance had decreased to walking approximately 20 yards. He was a poor historian and was generally argumentative and disinterested in our explanations.

We initially postponed his surgery to await a cardiology opinion, requesting a pre-operative pacemaker (preferably permanent). He was diagnosed as being in atrial fibrillation and having a left bundle branch block and left axis deviation. He was asymptomatic and assessed as being low risk, and therefore not needing a pacing wire; however, after further debate, the cardiologists placed a temporary pacing wire for the operation. It was hoped to remove this postoperatively.

We anaesthetised him and paced his heart at a rate of 70 beat.min-1 and he remained cardiovascularly stable. Monitoring included invasive blood pressure monitoring to assess beat-to-beat arterial blood pressure.

He was paralysed, intubated and ventilated and the operation seemed to be proceeding without a problem; we even sent for the next patient. We demonstrated four twitches on the nerve stimulator and added reversal of neostigmine 2.5 mg and glycopyronium 500 μg for good measure, and awaited the usual spontaneous respirations to return. His tidal volumes appeared to be adequate and he was extubated. The patient responded more slowly than normal and appeared to be finding it increasingly difficult to breath. He was resedated and we assisted his ventilation.

On reapplying the nerve stimulator, we found four strong and equal twitches with no fade and a sustained muscle contraction on tetanic stimulus.

The penny then dropped as we reflected on his characteristic frontal baldness, the ptosis and the decreased mental fortitude that is often present with myotonic dystrophy (synonym: Steinert muscular dystrophy).

Although the prevalence is about 3–5 in 100 000, it never the less is with us. It is inherited as an autosomal

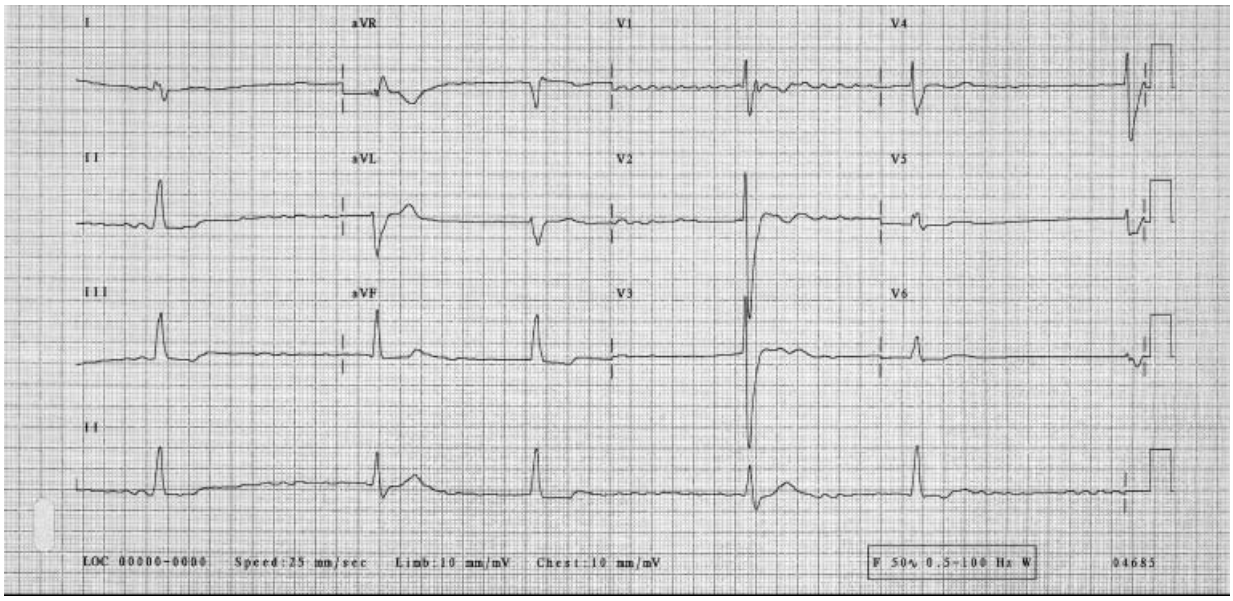

Figure 12

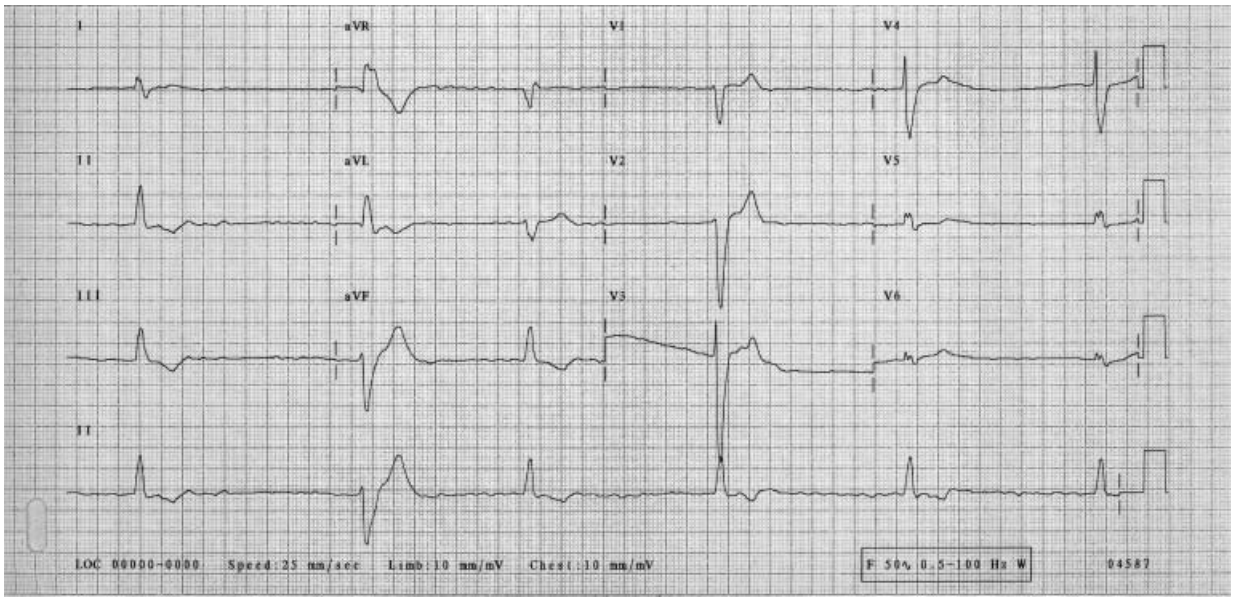

Figure 13

dominant trait with the gene responsible residing on the long arm of chromosome 19 [4]. Unlike other myotonic syndromes, it is a multisystemic disorder with most patients presenting between the ages of 15 and 35 years old. Cardiac abnormalities are usual and most commonly there is a progressive deterioration of the conducting system resulting in first degree heart block, bundle branch block and widening of the QRS complex with sudden death associated with third degree heart block [4]. Myotonia is demonstrable on EMG, but myotonic discharges are less abundant than in non-dystrophic myotonias. They are best demonstrated by examining the facial or distal limb muscles [4].

Aldridge in Aberdeen [3] looked at the characteristics in 16 patients (Table 3), and also found a 52% complication rate in previously diagnosed cases and a 35% complication rate in undiagnosed cases. We are not advising that we investigate all the these

Table 3

Symptoms and associated findings	Male %	Female %
Family history elicited	89	75
Ptosis	78	75
Facial weakness	89	62
Neck weakness	66	100
Dysarthria	55	75
Distal muscle weakness wasting	100	87
Abnormal gait/Foot drop	55	25
Decrease or absent reflexes	66	37
Symptomatic myotonia	78	75
Balding	78	12
Testicular atrophy	55	
Female infertility/endometriosis		25
Low IQ	44	12
Cataract	44	50
Associated diabetes	11	0
Family history of diabetes	11	25
Thyroid abnormality	0	12
ECG abnormality	33	37

symptoms as we are convinced that after checking for testicular atrophy, the patient will be wondering about the holistic role of the anaesthetist as a perioperative physician!

The intra-operative diagnosis is not unique and occurs internationally [1,2]. We should always attempt to think laterally and look for family history and attempt to explain why a sign is present. The questions to be asked are why the ECG was so slow, and more importantly why was our diagnosis so slow? The retrospectoscope is a powerful instrument and when we were debating the merits of a pacemaker pre-operatively, we should have been looking for the cause.

Our patient is no longer suffering from his previous malodorous condition and is enjoying life with his new pacemaker.

G. Morton
J. Bowes
Morriston Hospital,
Swansea, UK

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