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Correspondence

Herbal medicines in the United Kingdom

I read with interest the review of perioperative implications of herbal medicines (Hodges & Kam, *Anaesthesia* 2002: **57**: 889–99) and wish to add some information pertinent to the UK.

The herbal medicines selected by Hodges and Kam for detailed review may be those most commonly used in Australia, but are *not* the most popular in the UK. The popularity of herbal medicines will vary in different parts of the world. Hence the authors of two other recent reviews selected somewhat different lists of herbs for discussion [1, 2]. Also in any area, the order of popularity can be expected to change with time.

There are four legal categories of herbal products in the UK [3].

1 'Medicinal products' with a product licence under the Medicines Act 1968. These may be prescription only, pharmacy only or on the general sale list.

2 'Medicinal products' exempt from licensing under section 12 of the Medicines Act 1968. Intended for herbalists, initially these were only plants (dried, comminuted or crushed), sold under botanical name with no written instructions as to their use. However, this category came to include manufactured products (under a specials manufacturing licence).

3 Herbal products marketed as food supplements.

4 Herbal products marketed as cosmetics.

Licensed products receive a Medicines Control Agency (MCA) number. It is not possible to determine the top selling unlicensed products, since the sector is largely unregulated. However, a survey has shown that the majority of British consumers buy the licensed products [4]. I have obtained lists of the top selling licensed herbal medicines from the five most major U.K. manufacturers (Potter's, Chefaro, GR Lane's, Herbal Concepts and Bioforce). Based on this data the most commonly sold licensed herbs in the UK are:

usually sole agents – echinacea, panax ginseng, ginkgo, St John's wort;

usually in combination with other herbs – valerian, garlic, passiflora, elder, white horehound, senna, uva-ursi.

To the best of my knowledge, there have been no reported interactions between the last five herbs (not mentioned in the review by Hodges and Kam) and anaesthetic drugs. However, some are possible if these herbs are taken in excessive doses [5]. The following herbs (used in combinations) are also common in UK manufactured products: aloes, buchu, cascara, clivers, dandelion, elecampane, hops, kelp, lobelia, saw palmetto, wild lettuce, yarrow. For information on these I commend the herbal medicines guide commissioned by the Royal Pharmaceutical Society [5]. Ephedra, kava and ginger are not commonly used as herbal remedies in the UK. In 2002 the MCA, on advice from the Committee on Safety of Medicines (CSM), revoked licences for preparations containing kava because of the risk of liver toxicity [6]. However, kava capsules (unlicensed) are readily available, including on the Internet! Further regulation of herbal medicine products (HMP) is currently developing in Europe under the auspices of the European Agency for the Evaluation of Medicinal Products (EMEA) – the aim is to establish a new, simplified system of licensing for traditional HMPs by 2005 [7].

Every herbal medicine is a complex mixture of usually at least 50 chemical constituents, the active principles being mostly unknown. It is considered that the constituents may all contribute, perhaps synergistically, to the therapeutic action. In combination HMPs there are even more chemical constituents for interaction – additive, synergistic or negative? Also, brands containing the same or similar ingredients produced by different manufacturers can never be considered identical. There are concerns about use of the correct plant species, presence of contaminants such as insecticides, fungicides or microorganisms, and deliberate adulteration [7]. This sort of information is beginning to be included in new undergraduate medical curricula, but most medical graduates know very little about HMPs.

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One wonders about the level of risk that herbal medicines might add to anaesthesia *per se*, and I suggest that this is small. As of September 2002, the MCA had not received *any* adverse drug reaction reports involving interactions between anaesthetic drugs and herbal remedies (personal communication – MCA/CSM). This does not of course mean that nil adverse interactions have occurred, but it is perhaps not surprising as skilled anaesthetists titrate anaesthetic drugs and adjuncts according to patient response. Of course, I support the eliciting of herbal medicine usage by anaesthetists at pre-operative visits. However, this information may be more relevant to surgeons and physicians with regard to the longer term well-being of the patient.

The example of the patient in whom rejection of a liver transplant was caused by the ingestion of St John's wort as an antidepressant after discharge from hospital, comes to mind [8].

Discontinuing herbal medication for 2 weeks prior to surgery may be sensible advice in most cases, but this remains debatable for some herbal medicines. For example, abruptly stopping valerian may precipitate symptoms mimicking an acute benzodiazepine withdrawal syndrome [9]. The editorial on herbal medicines (Sehgal & Hall. *Anaesthesia* 2002; **57**: 947–8) mentioned valerian and alluded to alternative therapy. Substituting a benzodiazepine is an option, but I suggest that in many cases it would be perfectly reasonable to simply continue the valerian in the peri-operative period. Furthermore, in emergency cases, 2 weeks discontinuation is impossible, and the anaesthetist must proceed with due attention.

The UK herbal medicine industry is currently booming. Anaesthetists can be sure of encountering many patients taking herbal remedies and some knowledge of the subject is clearly required. A survey of current anaesthetic practice is called for.

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Evaluation of an expired fraction carbon dioxide monitor – incorrect statistical analysis?

I read with interest the recent article concerning the evaluation of an expired fraction carbon dioxide monitor (Ratnasabapathy *et al. Anaesthesia* 2002; **57**: 900–4). Figures 5 and 6 both illustrate the difference between two variables (arterial P_aCO_2 – portable F'_ECO_2) plotted against the one of the same variables (arterial P_aCO_2). If the arterial P_aCO_2 and portable $F'_ECO_2$are considered to be two different methods of estimating a single clinical measurement (legitimised by plotting the difference between these two variables on the y-axis of Figs 5 and 6) then the correct method would be to plot the arithmetic mean of the arterial P_aCO_2 and portable F'_ECO_2 on the x-axis. This error may arise with the use of correlation to study the relationship between an initial measurement and a change in that measurement over time, and for any two quantities X and Y, X will be correlated with X–Y; indeed, even if X and Y are samples of random numbers then one would expect the correlation between X and X–Y to be 0.7 [1].

The same problem appears to occur when measuring the agreement between two methods of clinical measurement: Ratnasabapathy *et al.* cite a reference by Bland and Altman which reports a statistical method for assessing agreement between two methods of clinical measurement [2]. Bland and Altman point out that it is a mistake to plot the difference between two variables against either value separately because the difference will be directly related to each; this statistical artefact is further illustrated in an earlier article by Gill *et al.* [3]. The *correct* method, as indicated above, is to plot the difference between two variables against the arithmetic mean of the two variables. Figure 3, from the article by Ratnasabapathy *et al.* appears to use the *correct* method in illustrating the relationship between fixed and portable F'_ECO_2. The general effect of using the *incorrect* method appears to be to increase the value of the correlation coefficient [3].

I would be pleased to hear if Ratnasabapathy *et al.* agree with my interpretation of the above methods. I cannot comment upon the effect of any new analysis of the data by Ratnasabapathy *et al.*

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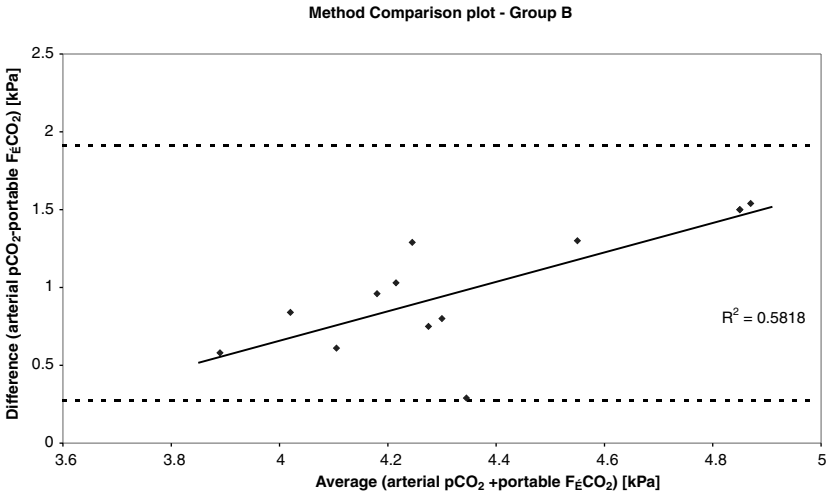

Figure 1

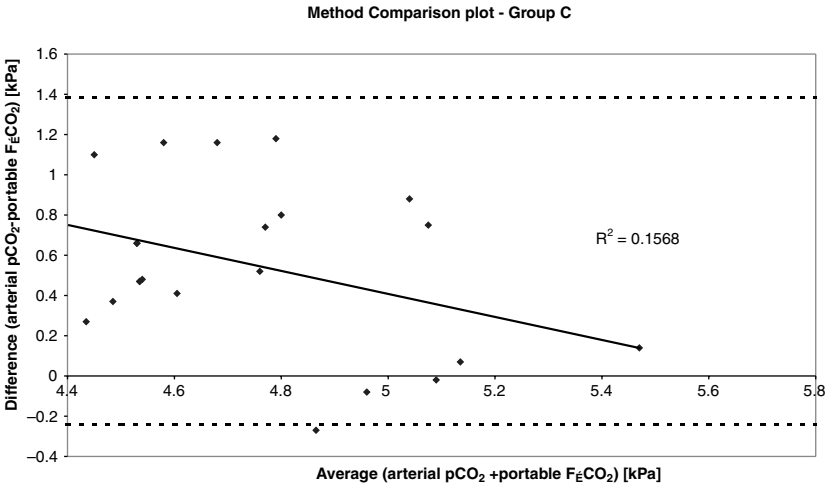

Figure 2

A reply

We are grateful firstly to Dr Lewis for his interest in our paper, and for cogently illustrating the statistical argument, and secondly for the opportunity to reply to his points. He is absolutely correct in terms of statistical accuracy – there are errors inherent in charting the difference between a standard and test measurement against only the standard, in terms of the correlation produced.

For interest's sake, we include the revised graphs of our data using the 'statistically correct' methodology (Figs 1 and 2). They indeed show a change in the slope of correlation in Fig. 2, whilst Fig. 1 is largely unchanged. There remains, however, a measurement difference of 0.4 kPa at an average of 5 kPa pCO_2 in Fig. 6.

Herein lies part of the problem facing any researcher in depicting this type of data. What does charting the average of pCO_2 and $F_{É}CO_2$ tell the clinical user? We would argue that this was a study of clinical accuracy, and the graph was structured to depict the deviation between what we accept as our clinically important variable (i.e. pCO_2) and the tested variable ($F_{É}CO_2$). Our error here lay in including the correlation lines – we should have refrained from including these in the graphs.

While Dr Lewis is entirely correct to say that the correlation between the measurements is more accurately related to the average of the measurements, the charting methodology does not change the difference between the two measurement techniques, which we believe to be significant.

We also believe the reader needs to appreciate the difference over the clinical range of pCO_2, so that they may then relate that to the clinical changes that appear with increasing pCO_2.

An average of the test and standard do not tell the reader that at a pCO_2 of 5.6 kPa, the portable capnometer under-read by 1.5 kPa. The figure of 5.6 has more immediate clinical relevance, in terms of cerebral blood flow and intracranial pressure, than the average of 4.85. This is perhaps an argument between the practically useful graphical depiction of data and the accurate statistical interpretation of graphed data.

Our essential point remains, however, that this type of monitor needs to be tested against the clinically relevant variable, i.e. pCO_2, and in a variety of circumstances, so as to gain insight into its performance in the various environments it may be used.

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Nasotracheal intubation

In their excellent article on nasotracheal intubation for head and neck surgery, the authors (Hall & Shutt. *Anaesthesia*, 2003, **58**: 249) discuss the techniques of intubation under direct vision and blindly. However, they do not mention the highly successful technique which is a combination of these two [1]. Here, the laryngoscope is used merely to place the tip of the tube optimally (i.e. in the midline and posterior to the epiglottis) under direct vision, a limited procedure producing far less stimulation than formal laryngoscopy. The upward lift of

the laryngoscope is then relaxed, allowing the soft tissues to fall back into their normal anatomical positions before the tube is advanced blindly into the larynx. Even in inexperienced hands this technique is almost always successful, and it deserves to be more widely known. It is suitable for routine use and is often the quickest and easiest solution to the more difficult case.

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Laryngeal mask cuff damage during interscalene block

Inadvertent iatrogenic injury to airway management aids resulting in a loss of airway security is a possibility of which every anaesthetist should be aware. Damage to a tracheal tube during an operative procedure in and around the neck is a possibility and has been reported [1]. We would like to report a recent case of accidental puncture of a laryngeal mask airway cuff while performing an 'interscalene' brachial plexus block.

A 10-year-old boy (25 kg) had presented to the orthopaedic surgeons following a fall from height. A diagnosis of a supracondylar fracture of the left humerus was reached and closed reduction was planned. After performing the routine pre-anaesthetic check-up and confirming the fasting status, a general anaesthetic technique was planned. Fentanyl 40 μg iv was given and anaesthesia was induced with propofol 60 mg iv. A size 2.5 laryngeal mask airway was inserted and the cuff filled with about 12 ml of air. Anaesthesia was maintained with O_2, N_2O and halothane (1%). Closed reduction was attempted under fluoroscopic guidance but stability could not be achieved. The operative plan was changed to open reduction and internal fixation. At this point we decided to administer an 'interscalene' brachial plexus block, to achieve better intra-operative and postoperative analgesia. A 24 gauge (1-inch-long) intradermal needle was taken and the landmarks for the block identified according to the standard interscalene technique as described by Winnie [2]. On introducing the needle, a 'give way' was felt and it was assumed to be the prevertebral fascia covering the scalene muscles forming the 'interscalene' space. After negative aspiration, 15 ml of 0.375% bupivacaine was injected. No resistance to injection was experienced. As the surgeons prepared and draped the operative site, gurgling sounds with each breath were heard. Another 4 ml of air was injected into the cuff. Over the next minute, the gurgling sounds reappeared. In an attempt to inflate the cuff correctly with the prescribed volume, we decided to deflate the cuff and re-inflate it. On aspirating, clear, transparent fluid (ca. 5 ml in volume) appeared. Attempts at re-inflating the cuff failed and it was decided to intubate the trachea. The laryngeal mask was removed, thorough oropharyngeal suctioning was done, vecuronium bromide 3 mg iv given and the trachea intubated with an appropriate sized cuffed tracheal tube. Tracheal suctioning was done and no significant aspirate was found. The rest of the procedure was uneventful and the patient's recovery was adequate.

On careful examination of the laryngeal mask, we found fluid in the inflating lumen and a small puncture on the left dorso-lateral aspect of the cuff. Our needle had accidentally entered the cuff of the laryngeal mask and its silicone material had given a false positive 'loss of resistance' feel. As the drug had been injected into the cuff, some of it leaked into the oropharynx from the puncture site while some was aspirated from the cuff inflation port.

This report highlights the risk that vital anaesthetic equipment is exposed to when interventions are done around them. The laryngeal mask airway has revolutionised anaesthesia because of the ease, simplicity and safety it offers over the conventional tracheal tube. It has gained widespread use in procedures like percutaneous tracheostomies, central venous line insertion, cervical spinal nerve blocks (stellate ganglion blocks, cervical plexus blocks, etc.), endoscopies and surgery on and around the neck [3–5]. Most of these procedures involve the use of needles or sharp instruments, which, if not used carefully, especially in patients not having well defined anatomical landmarks, may endanger our anaesthetic equipment and, more importantly, our patients. A high degree of caution and vigil is recommended during procedures being performed around the trachea with an airway device *in situ*.

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Hard to swallow

We wish to report a retrothyroid haematoma complicating apparently straightforward tracheal intubation.

An 83-year-old 67-kg man, ASA class II and Malampati I, was scheduled for a laparoscopic cholecystectomy under general anaesthesia. Pre-operative anaesthetic assessment identified chin recession and a slightly decreased range

of neck movement secondary to osteoarthritis. There was no history of neck trauma, thyroid pathology, dysphagia, cerebrovascular disease or bleeding disorders. Laryngoscopy (grade 2) was performed with Macintosh size 4 blade, and an 8.0-mm cuffed tracheal tube inserted easily by an experienced consultant anaesthetist. An 18F nasogastric tube was passed, and neck veins were not cannulated. Surgery was straightforward and extubation uncomplicated.

On the first postoperative day, the patient complained of being unable to swallow and neck pain, without respiratory difficulty. Examination elicited tenderness around the left thyroid lobe but was otherwise unremarkable; there was no neurological deficit. A speech and language therapy assessment reported dysphagia and aspiration, but flexible nasolaryngoscopy was normal.

A barium swallow (Fig. 3) showed minor deviation of the cervical oesophagus to the right, with aspiration of a small amount of contrast. Ultrasound (Fig. 4) and computed tomography scans (Fig. 5) of the neck showed an eccentric $4 \times 2.2 \times 1.7$ cm fluid collection, displacing the left thyroid lobe and isthmus, consistent with a retrothyroid haematoma.

The patient reported a progressive decrease in discomfort over a fortnight. However, persistent dysphagia necessitated placement of a fine bore feeding nasogastric tube, later replaced by a percutaneous endoscopic gastrostomy tube for nutrition. Complete resolution of symptoms took 8 months.

A sore throat often follows tracheal intubation, is generally short-lived, and is reportedly reduced by light wand intubation [1], preferential use of the laryngeal mask [2], and spontaneous as opposed to intermittent partial pressure ventilation [3]. In this case, symptoms were due to a retrothyroid haematoma, probably caused by bleeding from small neck veins. Despite an apparently straightforward anaesthetic, thyroid movement with the trachea during intubation or coughing on extubation may have initiated bleeding. Similarly, nasogastric intubation may have caused unsuspected trauma.

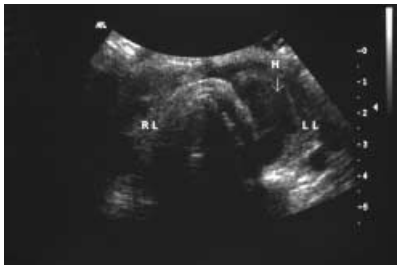

Figure 3

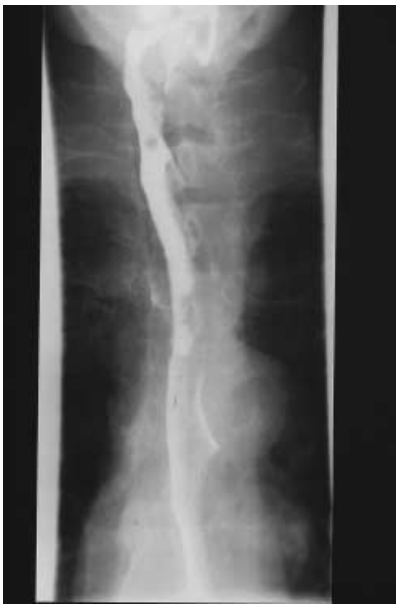

Figure 4

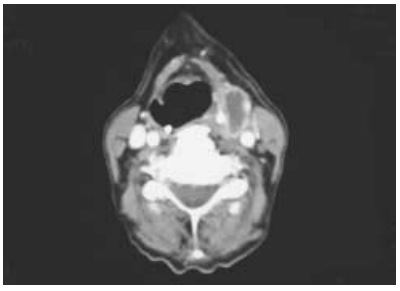

Figure 5

This case presents an unpredictable, serious and potentially life-threatening complication of tracheal intubation. The retrothyroid haematoma displaced the oesophagus causing dysphagia; fortunately, airway compression and respiratory compromise did not occur. However, this raises particular concern for day case laparoscopic cholecystectomy. Similar symptoms to those described by this patient are commonly reported following tracheal intubation, and their implications, were he in the community, may have been underestimated. Prior to embarking upon day surgery, it is crucial that robust systems are in place in the community to provide a safety net for similar potentially serious if isolated cases.

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An unusual case of postextubation stridor

A 63-year-old lady presented to the day surgery unit for nasal polypectomy. Her past medical history included allergic rhinitis, nasal polyps and mild asthma. Her current medications were salbutamol and becotide inhalers. She smoked 15 cigarettes per day and had recently finished a course of antibiotics for a chest infection. Apart from her normal smoker's cough, she denied any symptoms of cardiorespiratory compromise. Previous anaesthetic history was unremarkable having had an uneventful abdominal hysterectomy 20 years previously. Examination revealed a highly

anxious patient with normal cardiovascular and respiratory systems. Her weight was 59 kg and her calculated BMI was 29 $kg.m^{-2}$.

Anaesthesia was induced with midazolam 1 mg, fentanyl 100 μg, propofol 120 mg and vecuronium 4 mg. Laryngoscopy revealed a grade II view and the patient's trachea was intubated with a 7.0-mm cuffed RAE tube. A throat pack was inserted and anaesthesia maintained with a mixture of oxygen, air and isoflurane. At the end of the procedure, 40 min later, the throat pack was removed and the patient's postnasal space, pharynx and larynx suctioned under direct vision. The patient was reversed with neostigmine 2.5 mg and glycopyrronium 500 μg and placed in a left lateral position. Following the demonstration of adequate tidal volumes and a good cough reflex, the patient's trachea was extubated. Immediately following extubation the patient demanded to sit up. Over the ensuing couple of minutes the patient became increasingly distressed and complained of difficulty breathing. At the same time, it appeared that she had an inadequate cough and that secretions were accumulating in her upper and lower airways. She demonstrated good muscle strength and a train-of-four stimulation showed no fade. Over the next 5 min, she developed increasing inspiratory stridor, respiratory distress and her oxygen saturations fell to 85% despite high-flow oxygen and manual CPAP by facemask. Following propofol 80 mg and succinylcholine 75 mg, she was re-oxygenated and direct laryngoscopy surprisingly revealed no blood or secretions in the larynx. The patient's trachea was intubated with a size 7.0 mm cuffed tracheal tube. Fibreoptic bronchoscopy demonstrated no blood or secretions in the lower airways but blood could be visualised tracking down the outside of the tracheal tube. An ENT surgeon was summoned, as laryngeal trauma could not be excluded. The tracheal tube was removed and direct laryngoscopy performed with ventilation of the lungs via a Sanders injector. Anaesthesia was maintained using a target-controlled infusion (TCI) of propofol (range 4–6 $\mu g.ml^{-1}$). Direct laryngoscopy revealed the presence of an oedematous polyp on the left vocal cord, which moved to partially occlude the laryngeal glottis with jetting of the Sanders injector. Although the partial occlusion did not appear significant enough to account for the patient's respiratory compromise, the decision was made to excise the polyp. The rest of the laryngeal structures appeared normal. Following neuromuscular reversal and demonstration of the return of airway reflexes, the patient was extubated. As before, the patient initially appeared fine, but then became distressed with concomitant inspiratory stridor and hypoxia. The hypoxia responded to high flow oxygen; however, the stridor and agitation remained. The possibility of a vocal cord palsy was raised and it was decided to perform nasendoscopy. The patient was sedated with a propofol infusion (TCI 1.5 $\mu g.ml^{-1}$) and the nasal mucosa anaesthetised with lidocaine 1%. As the propofol sedation approached the target level of 1.5 $\mu g.ml^{-1}$, it was noted that the patient's stridor had resolved and she was able to cough effectively and phonate normally. Nasendoscopy was performed and demonstrated normal symmetrical movement of the vocal cords. With constant reassurance to the patient, the propofol sedation was weaned over the next 20 min with no further problems. Once fully recovered, discussion with both family and patient revealed that the patient was prone to panic attacks. During two of these attacks, it had been noted by family members that the patient's breathing had become 'noisy' and had necessitated review by the emergency on-call doctor.

Although our patient had a polyp on her vocal cord, we do not feel that this was the primary cause of her postextubation laryngospasm. The most likely cause was an acute panic attack precipitated by the stress of extubation and initial post anaesthetic disorientation. The evidence to support this comes from the patient's previous history and the fact that it settled spontaneously with conscious propofol sedation. Functional paroxysmal laryngospasm (laryngeal dyskinesia) can be associated with psychological disorders including panic attacks [1, 2]. Propofol at a dose of 0.25 $mg.kg^{-1}$ has previously been used to control post extubation and functional laryngospasm [3, 4]. Studies have demonstrated that peri-operative respiratory events including laryngospasm are more common in smokers [5, 6]. The ENT surgeon who performed the nasal polypectomy felt that the procedure might need to be repeated in the future. Our plan would be to anaesthetise the patient using a laryngeal mask airway and propofol TCI.

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Training method of applying pressure on the neck for laryngoscopy: use of a videolaryngoscope

Application of pressure on the neck, such as cricoid pressure [1] or backward, upward and rightward pressure (BURP) on the thyroid cartilage [2], may improve

the view of the glottis at laryngoscopy. However, inappropriately applied pressure may worsen the view [1]. Therefore, training of the correct application of pressure is mandatory. One major difficulty in teaching is that it is difficult to show trainees or assistants the change in the view of the glottis by pressure on the neck. We suggest that the use of a device that enables the view of the glottis to be displayed on a video screen is useful for teaching purposes.

One simple method is to place a small camera or tip of a fibrescope (with a video screen) near the mouth during laryngoscopy to show the image, but three people may be required to insert a laryngoscope, to hold a camera system, and to apply pressure on the neck. A better way is to attach a fibrescope to a laryngoscope. Henthorn and colleagues were the first to report a system of attaching a fibrescope to a Miller laryngoscope [3]. Several Macintosh blades that incorporate fibreoptic systems are now commercially available, and we have been using such a device (X-lite Video Laryngoscope, Rüsch, Germany). We have found that an anaesthetist inserting the laryngoscope can show an effective way of applying pressure on the neck because both the anaesthetist and the assistant applying pressure on the neck can see the glottic area on a video screen and see the effect of the applied pressure on the view of the glottis. We believe that, although availability of a video-monitored laryngoscope is currently limited, this should become more widely available.

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Clopidogrel and platelet transfusion in patients undergoing coronary artery bypass graft surgery*

We read with interest the timely review of clopidogrel (Kam & Nethery. *Anaesthesia* 2003; **58:** 28–35). Current recommendations for withholding clopidogrel for 6–7 days before elective cardiac artery bypass graft (CABG) surgery has recently been echoed in the American College of Cardiology/American Heart Association practice guidelines for management of patients with unstable angina [1]. Nonetheless, there has been a relative absence of data examining risks of bleeding and blood product transfusion in CABG patients receiving pre-operative clopidogrel. We wish to share our data after evaluation was prompted by a perceived increase in bleeding and blood product use in such patients.

After institutional approval, a retrospective study was conducted of all first-time CABG surgeries at a university-based teaching hospital for the first 9 months of 1999. Patients with abnormal baseline coagulation parameters, undergoing combined procedures or with an intra-aortic balloon pump were excluded. Intra- and postoperative blood loss and BP administration were recorded for four groups: patients receiving aspirin (A) alone ($n = 80$), clopidogrel (C) alone ($n = 16$), A + C ($n = 47$) or no antiplatelet therapy ($n = 41$). Transfusion data were not normally distributed and analysed with Kruskal–Wallis tests: significant differences were evaluated by Wilcoxon rank-sum tests between groups with Bonferroni correction.

There were no differences in patient demographics, baseline coagulation parameters, duration of cardiopulmonary bypass (CPB), lowest temperature on CPB, estimated blood loss, postoperative haematocrit or red cell, fresh frozen plasma or cryoprecipitate use between groups. However, significantly more platelets were administered to patients in the clopidogrel (mean = 6 units) and aspirin + clopidogrel (mean 3.4 units) groups compared to the no antiplatelet therapy group (mean 3.3 units) ($p = 0.005$). Patients in the aspirin only group were minimally exposed (mean 1.4 units, $p > 0.2$).

We believe that this data showing a 2–5 donor exposure increase in CABG patients receiving clopidogrel alone or with aspirin is significant for anaesthetists. Because of the retrospective investigation, patient group sizes were unequal and there was no prospectively defined transfusion algorithm. However, there was a stable cohort of cardiac anaesthetists and surgeons over this period, and blood product transfusion was guided, in part, by the Thrombelastograph®. Similar rates of other transfused blood products and postoperative haematocrits between groups suggest that platelet transfusions were a timely intervention to correct the defect in the clopidogrel groups without significant observer bias. These data were presented to our cardiology colleagues, who will be the most likely physicians able to identify appropriate patients for clopidogrel withdrawal, before anaesthetic or surgical evaluation, which is often within the 7-day window.

Until publication of further prospective data of the peri-operative implications of clopidogrel, we agree with Kam and others [2] that clopidogrel be withheld for 5–7 days before elective CABG surgery.

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Monitoring of platelet function

We read with interest the recent review about thienopyridine antiplatelet drugs (Kam & Nethery. *Anaesthesia* 2003; **58**: 28–35). A major problem faced by anaesthetists is the monitoring of the effects of antiplatelet medication. Although the platelet function analyser (PFA-100, Dade Behring, Dearfield, IL) appears to be the answer to this problem, it is a global test of primary haemostasis and is not specific for antiplatelet drugs [1]. The PFA-100 has been used to monitor the antiplatelet effect of aspirin [2]; however, the results may be affected by the citrate concentration in the sampling tube [3]. There is less experience in use of the PFA-100 to assess the effect of clopidogrel, and it is uncertain whether the citrate concentration is also important in this situation. Results from the PFA-100 may also be affected by smoking, haematocrit and gender [1].

The Thrombelastograph® (TEG®) Haemostasis Analyser (Haemoscope Corp., Niles, IL, USA) is a near-patient method of monitoring whole blood coagulation. Standard use of the TEG® system cannot detect the effect of aspirin [4]. However, a new TEG® assay has been developed specifically for this purpose (Personal communication: Haemoscope Corp., Niles, IL, USA).

The use of antiplatelet medication has implications for the safety of regional anaesthesia and propensity to bleed during surgery. Further studies are needed to provide a sufficiently robust method of platelet function assessment to guide anaesthetic management.

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Combined carotid endarterectomy and oesophagectomy

The benefits of combined operations of carotid surgery and coronary artery bypass graft (CABG) are well recognised in decreasing the incidence of stroke [1]; however, combined carotid surgery and major non-vascular surgery have not been described. We have recently been involved in managing a patient who had a combined endarterectomy and oesophagectomy, and we would like to highlight several issues.

A 58-year-old man was diagnosed with an operable adenocarcinoma of the gastro-oesophageal junction. He was referred for neoadjuvant chemotherapy prior to oesophagectomy. He described symptoms of dizziness, tinnitus and intermittent temporary visual field loss. An urgent carotid Doppler showed bilateral severe stenosis, more on the left side. A left carotid endarterectomy was performed 2 months later under general anaesthesia with an uneventful recovery. He was then scheduled for a combined oesophagectomy and right carotid endarterectomy.

A thoracic epidural and left radial arterial line was inserted prior to induction of general anaesthesia, the trachea was intubated with a RAE tube and anaesthesia was maintained with O_2, N_2O, and isoflurane. Laparoscopy was first performed to exclude distal disease. A right carotid endarterectomy was performed and heparin given prior to insertion of a Javid shunt. Heparin was reversed with protamine sulphate upon completion of the endarterectomy. The tracheal tube was changed to a left double lumen tube and right subclavian vein cannulation was performed. An Ivor Lewis oesophagectomy was performed which involved a laparotomy to mobilise the stomach and form a gastric tube, mobilisation of the oesophagus through a right thoracotomy and an end-to-end anastomosis. Abdominal and chest drains were inserted. The double lumen tube was exchanged for a cuffed tracheal tube and the patient transferred to the intensive care unit. He was extubated after 4 h, moved all limbs and answered questions appropriately. Recovery was uneventful.

Carotid surgery reduces the late incidence of stroke when compared to medical treatment and overall mortality ranges between 1 and 2% [2, 3]. Oesophageal resection represents a major surgical and physiological insult and carries major morbidity and mortality. Early recognition and aggressive management can minimise subsequent mortality [4]. Ivor Lewis oesophagectomy is associated with fewer anastomotic complications than the transhiatal approach [5]. A combined operation increases the risks; however, carotid surgery combined with CABG has been described as a safe technique and can decrease the incidence of stroke and long-term morbidity [6, 7]. It is speculated that to have a patent carotid artery is a beneficial protective factor before major surgery. Our patient responded well to the neoadjuvant therapy [8] and delaying the oesophagectomy would increase the possibility of metastatic spread. It is important to take all patient specific factors into consideration [9].

In this case, the patient benefited from reduced risks of neurological events occurring during major surgery, while avoiding delaying the oesophagectomy, which might have reduced his chances of a curative procedure. Such combined procedures can be safely undertaken with appropriate resources.

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Spontaneous reversal of the 'wall effect': another possible cause for inaccurate continuous intra-arterial blood gas monitoring data

The 'wall effect' is known to be one possible cause of inaccurate continuous intra-arterial blood gas (CIABG) monitoring data, where PaO_2 values are underestimated due to the intra-arterial sensor touching the arterial wall [1]. We recently encountered a spontaneous reversal of the 'wall effect', which caused an overestimation of PaO_2.

A 53-year-old, 162 cm, 52 kg, male patient, was scheduled to undergo oesophagectomy under thoracoscopy. After an epidural tube was inserted, anaesthesia was induced with propofol 160 mg and was maintained with sevoflurane. Tracheal intubation was facilitated by the addition of vecuronium 10 mg. A 37 Fr, left-sided, double-lumen tracheal tube (Broncho-Cath®, Mallinckrodt Inc., Argyle, NY) was placed and the accuracy of the position confirmed by auscultation and by fibreoptic bronchoscopy. The patient's lungs were ventilated with an inspiratory oxygen fraction of 0.5, a tidal volume of 510 ml, and a respiratory rate of 8–10 during two-lung ventilation. An intra-arterial sensor of the CIABG monitor (Paratrend 7 + TM, Diametrics Medical Limited, High Wycombe, UK) was inserted through a 20-gauge catheter placed in the left radial artery and advanced to a length of 15 cm. No resistance was felt during the insertion of the sensor. The arterial pressure wave was not damped after sensor insertion.

As is usually done in our institution, 2 ml of arterial blood was drawn for sensor calibration after tic marks on the display confirmed that the CIABG data were ready for calibration. The arterial blood gas data displayed on the CIABG monitor (PaO_2 165 mmHg, $PaCO_2$ 38.2 mmHg, pH 7.45) were calibrated to the arterial blood gas data analysed by ABL 720TM (Radiometer, Copenhagen, Denmark) (PaO_2 252 mmHg, $PaCO_2$ 33.5 mmHg, pH 7.469). We immediately noticed the large difference between the CIABG monitor PaO_2 and the ABL analyser PaO_2 values. Though we assumed that the tip of the sensor might be touching the wall of the artery, we did not change the position of the sensor because the reconstruction surgery involving the stomach had already started and the left arm of the patient was not easily accessible. While the reconstruction involving the stomach was being performed, we found that the PaO_2 values on the CIABG monitor increased linearly for approximately 20 min, even though no ventilation setting had been changed. The Paratrend 7 + CIABG monitor can document either the arterial blood gas data that are calibrated to ABL or data that are not calibrated to ABL. Therefore, both calibrated and uncalibrated arterial blood gas data were displayed and recorded on the CIABG monitor every time arterial blood gas samples were drawn for analysis. When the rise in PaO_2 was equilibrated, the PaO_2 as measured by ABL was much lower (219 mmHg) than the calibrated CIABG value (337 mmHg) but was closer to the uncalibrated CIABG data (210 mmHg), which was not affected by calibration to the ABL gas analyser. The PaO_2 data on the ABL analyser were closer in value to the uncalibrated CIABG data, except when the PaO_2 values were higher during one-lung ventilation. Thereafter, the ABL analyser PaO_2 values were lower than the calibrated CIABG data (Table 1).

Table 1 Sequential changes in PaO2 (rnmHg) data measured by conventional blood gas analyser (ABL 720) and Paratrend 7± with and without calibration.

	ABL 720	Paratrend 7± with calibration	Paratrend 7± without calibration
10:12 (T)	252		165
Calibration			
11:41 (T)	219	337	210
12:51 (O)	92	146	88
13:45 (O)	208	285	181
15:17 (O)	241	345	224
16:33 (O)	388	496	297
18:05 (O)	250	304	207
20:10 (T)	155	234	148
20:45 (T)	129	192	120

(T) = two-lung ventilation.
(O) = one-lung ventilation.

The 'wall effect' is usually manifested by PaO_2 values displayed on the CIABG monitor that gradually decrease with time but which are not accompanied by changes in either $PaCO_2$ or pH values [1]. Although the phenomenon was rarely seen in our total clinical experience of over 200 cases, the 'wall effect' is considered to be one of the major causes of inaccurate CIABG data. In our previous research, specifically involving a total of 28 oesophagectomy patients [2–4] and 50 patients who underwent thoracotomy [5], the 'wall effect' was never observed.

Since the $PaCO_2$ and the pH data were unaffected in this case, we assumed that the 'wall effect' occurred when the first arterial blood sample was drawn for calibration. The subsequent linear increase in PaO_2 on the CIABG monitor might be explained by the reversal of the 'wall effect', although it would be very difficult to prove this hypothesis. As far as we know, a spontaneous reversal of the 'wall effect' has not been previously reported. This case suggests that a 'wall effect' reversal can occur spontaneously and that it may cause the CIABG monitor to overestimate PaO_2 values, especially when the CIABG data has been calibrated to an arterial blood gas analyser.

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Airway obstruction following potassium permanganate ingestion

Potassium permanganate ($KMnO_4$) is a highly corrosive, water-soluble oxidizing antiseptic for cleansing and deodorizing suppurative eczematous reactions and wounds, used in baths and wet bandages. It is presented as a 0.1% solution, diluted 10 times to yield a 0.01% solution, or crystals that are dissolved, and applied until the skin becomes dry [1]. Both the crystals and concentrated solutions are caustic to skin, eyes, upper respiratory tract and mucous membranes, resulting in coagulation necrosis.

An 81-year-old woman was admitted to Accident and Emergency following deliberate ingestion of an unknown amount of $KMnO_4$ crystals, which were being regularly applied to suppurative chronic leg ulcers. She had a history of heart failure, chronic renal failure and atrial fibrillation, for which she was receiving warfarin. She had refused oral fluid replacement on admission and intravenous replacement was started. She remained cardiovascularly stable without respiratory compromise and her SpO_2 remaining above 92% on air. She was also commenced on intravenous antibiotics for presumed incidental cutaneous infection.

Twelve hours after admission, I was asked to review the patient on the ward, because she had a reduced level of consciousness and was snoring. The nursing staff also noticed that her tongue had swollen in the preceding hour. On initial assessment, she was rousable with a Glasgow coma scale (GCS) of 11. She was afebrile, pale, with cool peripheries. There was no skin rash evident. She had obvious macroglossia, with bloodstained secretions on her pillow, and she was stridulous. On air, her SpO_2 remained above 92%, with a pulse rate of 70 beat.min^{-1}, and BP 108/55 mmHg. There was no bronchospasm on auscultation, and no evidence of heart failure. In view of her airway compromise and fluctuating GCS, the patient was transferred to the ICU, where an anaesthetic machine was available for inhalational induction of anaesthesia.

Once on the ICU, further intravenous access was secured, together with invasive arterial BP monitoring. During this period, nebulised epinephrine (5 ml, 1 : 1000) was administered, with immediate improvement in her airway. The degree of macroglossia improved enough to allow speech. Humidified O_2 was continued, and hydrocortisone and antihistamines, both type 1 (H1) and type 2 (H2), given. She was not intubated, and remained stable over the next 8 h with adequate arterial oxygen saturations, and arterial blood gas analysis showing normal acid-base balance with good gas exchange. She was discharged to a medical ward with no further compromise. Her macroglossia gradually settled.

Potassium permanganate is regularly used in the management of suppurative eczema, and ulcers, and has previously been used as an abortifacient. Ingestion of dilute solutions can cause brown staining of the mouth and throat, sore throat, abdominal pains, vomiting and dysphagia. Concentrated solutions, or dry crystals as in this case, can cause swelling and bleeding of lips and tongue, pharyngeal oedema and swelling of the larynx, as well as gastrointestinal burns. Systemic effects do not usually manifest, due to poor absorbance, but can include tachycardia, hypotension, hallucinations, methaemaglobinaemia and cyanosis, metabolic acidosis, haemolysis, pancreatitis and coma [2, 3]. Some effects can be delayed up to 36 h postingestion including disseminated intravascular coagulation (DIC), cardiac failure and hepato-renal failure. In 1996, Young *et al.* postulated that the damage from ingestion of $KMnO_4$ crystals was due to

oxidative injury from free radicals generated by the absorbed permanganate ion. They found that the clinical course following $KMnO_4$ poisoning closely resembled severe paracetamol overdose, and recommended early administration of N-acetylcysteine [4].

The fatal dose for an adult is 10 g, and death usually results from pharyngeal oedema and cardiovascular collapse. Current treatment guidelines include avoidance of gut decontamination, and liberal oral fluids unless perforation is suspected. Indeed, there has been a documented case of fatal poisoning when charcoal was mistaken for $KMnO_4$ [5]. Supportive treatment is the mainstay of treatment thereafter, and if stable after 2 h following ingestion of dilute solutions, further treatment is unnecessary [6]. N-acetylcysteine should, however, be given within the first few hours, in the same dose as used in paracetamol poisoning.

This patient was reviewed 12 h after initial presentation and had not received N-acetylcysteine, but had been stable until 1 h before referral. Stridor is characteristic of upper airway obstruction, and suggests at least 50% reduction in airway diameter. Its presence, together with an inability to swallow secretions, should be a warning of severe airway compromise, and further management is dependent on patient factors and the skill of the anaesthetist. In an expected difficult intubation, an awake fibreoptic intubation may be indicated but the presence of secretions, including blood, may make this technically difficult. Inhalational induction with sevoflurane in oxygen allows intubation in a spontaneously breathing patient, and in the event of a failed intubation allows emergency tracheostomy in the anaesthetised patient. In this case, the patient was transferred to the ICU, where there is always a pre-checked cylinder-driven anaesthetic machine with sevoflurane available. Epinephrine was started as a temporary measure but resulted in a significant improvement and, with stable SpO_2, satisfactory blood gas measurements and a talking patient, it was felt that intubation was not required. She had also received a dose of intravenous antibiotics 3 h prior to referral, so an allergic reaction could not be excluded, although, aside from an enlarged tongue, there were no other manifestations of allergy. Secondary therapy with steroids and antihistamines was started but, as $KMnO_4$ can cause gastro- intestinal burns and inflammation, histamine 2 receptor blockers and steroids also have a role following its ingestion. The patient was discharged from ICU after close monitoring for 8 h with no sequelae.

Potassium permanganate is a commonly used antiseptic with astringent properties, although many anaesthetists have probably not encountered it since growing crystals in chemistry classes at school. It can cause severe caustic damage to the airway when ingested. As anaesthetists, relief of airway obstruction is one of our primary roles, both in theatre and on the intensive care unit (ICU), whether due to upper airway, mid or lower tracheal, or bronchial obstruction. Occasionally, the anaesthetist can encounter an unusual agent as the cause of obstruction, but airway management should not be influenced by this unfamiliarity.

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Progressive external ophthalmoplegia and ambulatory remifentanil-propofol based anaesthesia

With the advent of modern technology, new diseases are described. As patients with these 'new' conditions may require surgical procedures, the anaesthetic implications need to be addressed [1]. Mitochondrial myopathies are disorders of fatty acid utilisation or mitochondrial function that may affect skeletal muscle, or defective aerobic metabolism that effect several organ systems. The CNS is often affected. Other organs affected include the heart, liver, kidney, endocrine glands and gastro-intestinal tract [2].

We present a 61-year-old woman, weight 69 kg, height 160 cm, scheduled for breast biopsy. Her family history included maternal myasthenia gravis. Her medical history included hypothyroidism of unknown aetiology treated with thyroxine 50 mg daily, and deafness that needed a tympanoplasty. She was diagnosed as having progressive external ophthalmoplegia (PEO) 2 months before surgery because of recent and progressive bilateral ptosis. Muscular biopsy showed ragged red fibres and subsarcoleme lipofucsine granules. Lactic acid levels were high after exercise and 55 min after cessation. No therapeutic recommendations were made. Pre-operative haematology, biochemistry and ECG were normal.

We planned an anaesthetic technique avoiding triggers for malignant hyperthermia (MH), potential respiratory complications and with the initial plan of keeping the patient in an ambulatory surgery setting. Monitoring was: ECG, non-invasive blood pressure (NIBP), pulse oximetry, capnography and oesophageal temperature. Dantrolene was immediately available. The ventilator was previously purged with high flow oxygen. An intravenous saline infusion was started. Pre-anaesthetic medication

consisted of i.v. midazolam 1.5 mg, followed by a remifentanil infusion rate of 0.25 μg.kg $^{-1}$.min^{-1} Induction was performed with propofol 140 mg and a laryngeal mask size 3 was inserted. Anaesthesia was maintained with a mixture of oxygen and nitrous oxide with a remifentanil infusion and assisted manual ventilation until spontaneous ventilation started after 5 min. Duration of surgery was 40 min and the remifentanil infusion and nitrous oxide were discontinued at the completion of skin suturing. The patient awoke within 5 min. The total dose of remifentanil was 0.39 mg. At the end of surgery the patient was transferred to the recovery unit for 1 h and discharged home 6 h later.

Mitochondrial myopathies (MM) are classified according to their histological appearance, inmunochemical and biochemical profile or genetic analysis. The histological hallmark of MM is the ragged red fibre. PEO is associated with sporadic deletions of DNA. A negative family history indicates that deletion occurs in embryogenesis. Age of presentation ranged from 47 to 82 years in a nine-case series [3], with asymmetrical ptosis as the commonest sign, as in our patient. In this series, CPK was normal in eight cases and myopathic changes were found in three cases. The clinical course is usually progressive and diabetes mellitus, hypothyroidism, hyperparathyroidism and short stature are often associated.

The anaesthetic implication in MM and PEO is an increased susceptibility for MH, which has been described in at least one case. This requires a non-triggering anaesthetic technique. This can be addressed with propofol and remifentanil infusions [4, 5]. Depressed ventilatory response to hypercarbia or hypoxia, independent of muscle weakness or sleep apnoea, has been described in patients with various MMs including PEO [2]. Marked sensitivity to intravenous induction agents, opiates or sedative-hypnotics may be present and weaning difficulty has been described.

A few cases of pregnancy and other surgical procedures successfully managed with regional anaesthesia have been described in patients with MM [6].

Ambulatory care surgery is possible in PEO patients, as possible for many other conditions whose MH susceptibility is low and a non-triggering technique is used. Total intravenous anaesthesia (TIVA) based on propofol and alfentanil has been used successfully for eye surgery [7], or ketamine with fentanyl for cardiac surgery [8].

The use of ambulatory care surgery continues to grow and increases the likelihood of encountering MH-susceptible patients or of experiencing a malignant hyperthermia crisis. The management of this population creates new challenges A comprehensive plan of patient care under these circumstances includes the ability to identify the high-risk patient and to plan their care accordingly, early recognition of the signs and symptoms of MH, and being prepared to promptly and efficiently treat a malignant hyperthermic event [9].

MM is an heterogeneous group of rare conditions that will need to be anaesthetised more often in future and we believe that new anaesthetic drug combinations offer the patient safe anaesthesia with low MH risk and allow ambulatory surgery to be possible.

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Postoperative respiratory arrest in a post poliomyelitis patient (1)

I read with interest this report of a patient who had had poliomyelitis and developed postoperative respiratory failure (Magi *et al*. Anaesthesia 2003; **58**: 98–9). This should always be considered a potential complication when poliomyelitis has affected the respiratory muscles [1]. It is important to ascertain in the pre-operative assessment whether or not these were involved in the acute illness, and in particular whether respiratory support, usually in the form of an iron lung, was required.

The subject of this report had recurrent headaches, probably due to nocturnal hypoventilation. This arises particularly during rapid eye movement (REM) sleep, which occurs mainly at the end of the night. The headaches are characteristically bifrontal, occur early in the morning and clear within 20–30 min of waking. The authors comment on other 'vague' symptoms such as somnolence but this is a characteristic feature of nocturnal hypoventilation. The repetitive respiratory related arousals fragment sleep significantly to lead to the sensation of waking feeling unrefreshed and of excessive daytime sleepiness.

While measurement of respiratory muscle strength may be of value, a simpler and much more widely available screening test for chest wall muscle involvement is the vital capacity. This should be carried out in the supine as well as the sitting position in order to detect bilateral diaphragmatic weakness, which may only be apparent from the fall in vital capacity of up to 50% while lying flat [2]. If the clinical features and the vital capacity of less than 1.0–1.5 litres suggest significant respiratory involvement, arterial blood gases should be measured pre-operatively and non-invasive ventilation, usually in the form of nasal or oro-nasal mask ventilation, should be available postoperatively. The risk of hypoventilation can be reduced by avoiding muscle neuromuscular blocking agents during surgery and only discontinuing mechanical ventilatory support when the effect of the anaesthetic agents has completely worn off.

This report does highlight the risks of those who have had poliomyelitis when they undergo surgery, but accurate assessment pre-operatively can usually prevent postoperative complications

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Postoperative respiratory arrest in a post poliomyelitis patient (2)

We were distressed to read the letter describing a patient who suffered severe hypoxic brain damage following an operation on her foot (Magi *et al. Anaesthesia* 2003; **58**: 98–9).

We consider a number of factors contributed to this unfortunate outcome:

1 The pre-operative assessment failed in its prime purpose, to identify risk factors for anaesthesia; it was not lack of knowledge of polio but of respiratory function that was the problem. Even minor physical disability may conceal exertional dyspnoea, yet we suspect the indicators were there. The patient suffered from headaches that were sufficiently serious to be noted pre-operatively, yet their cause was not considered. They were simply regarded as a contra-indication to spinal anaesthesia, though the logic of this escapes us. (Would you avoid antibiotics in the treatment of cholera because they themselves sometimes cause diarrhoea?) We assume that they were mainly morning headaches, provoked by nocturnal hypoventilation. Respiratory insufficiency would also have been detectable in a quiet voice and tachypnoea during speech. The authors made the correct diagnosis in retrospect, but there would have been no need for blood gas analysis, a simple vital capacity measurement is all that is needed to identify restrictive respiratory disease, particularly in association with headache.

2 As those concerned with anaesthesia in this case overlooked respiratory insufficiency, it is perhaps to be expected that the danger of a respiratory depressant drug in these circumstances was also overlooked. The administration of three different opioids during brief surgery can only have compounded the problem. Remifentanil may be ultra-short acting, but then why also give fentanyl – 0.1 mg no less? The pharmacokinetic characteristics of fentanyl have long been known to pose a particular danger in the early postoperative period after short operations [1, 2] when moving the patient off the table or onto her bed causes a surge of drug back into the circulation. This increases the danger of respiratory depression, particularly if coinciding, as is often the case, with the onset of effect from a slower acting opioid such as morphine, given in this case subcutaneously 'during the procedure'.

3 We are not clear why she was also given atracurium 'for intubation and maintenance of relaxation'. We assume that as her operation was elective she was adequately starved, and she was not having an abdominal or thoracic procedure. We would not regard either intubation or relaxation as indicated, even for a fit patient in this case. Again, for a brief procedure a competitive neuromuscular blocking drug introduces yet another postoperative hazard. Whatever happened to the KISS principle in modern anaesthesia? (*Keep it simple, stupid!*)

4 Postoperatively she was left 'in her room' unmonitored for 50 min. Can this now ever be regarded as safe practice after general anaesthesia?

In 1990 we described in this journal the guiding principles that led to successful conduct of anaesthesia for 142 operations of all grades in 83 patients with restrictive respiratory disease, 44 of whom had had poliomyelitis [3]. *Nostra culpa* that neither the key words nor the abstract mentioned poliomyelitis.

- Pre-operative care included careful assessment, with training, if needed, in the use of non-invasive methods of respiratory support, which may be necessary during recovery from anaesthesia even among those not hitherto dependent on it.
- Competitive depolarizing muscle relaxants were not used (though it was often necessary to assist the patient's respiratory effort by hand), not because of any expectation of abnormal sensitivity, but because they were unnecessary and would increase the postoperative risk.
- Opioids were kept to a minimum and used in small doses for postoperative analgesia only.

- The recognised dangers of the post-operative period mandated special attention to respiratory function.

Every one of the simple measures that we recommended for patients with restrictive respiratory disease was inadvertently transgressed in this case.

The condition of patients who have once had polio may deteriorate in later life, not only because of any so-called post-polio syndrome, but for a variety of reasons largely connected with ageing [4].

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A reply

We are grateful for the opportunity to reply to the comments by Reynolds and Spencer on our recent letter.

The patient we described was a 51-year-old woman who had poliomyelitis since 2 years of age, without chest deformities, major motor deficits or respiratory symptoms; her status was classified ASA 2. We cannot add more details because although she was admitted to our intensive care unit, we were not involved in the pre-operative assessment or anaesthesia. We therefore cannot comment on whether her respiratory problems could have been better assessed. Headache is a very common complaint and it is very improbable that it could be considered as a possible symptom of respiratory failure during a routine pre-operative assessment of an apparently healthy patient. Patients who suffer recurrent headaches are reluctant to accept a technique such as subarachnoid anaesthesia when told that headaches are a complication.

The anaesthetic risk and the respiratory problems in patients with previous poliomyelitis described in the literature and in the article cited by Reynolds and Spencer [3] are related to overt respiratory failure and major disability. In our patient and in the case described by Gaul [5], the respiratory failure was occult; our conclusion is that every patient with previous poliomyelitis has to be evaluated in this regard.

In cases of suspected occult respiratory failure, we also have to consider primary hypoventilation and sleep apnoea syndrome as possible causes, so we cannot limit our investigations to the vital capacity, and at least blood gas analysis is required.

We totally agree with the idea that the anaesthetic technique has to be simple and that we have to minimise the use of opioids and neuromuscular blocking agents. Cases like this remind us that in our practice we may encounter patients with occult disease that can expose them to abnormal responses to standard doses of anaesthetic drugs.

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Convulsions following axillary brachial plexus blockade with levobupivacaine (1)

We read with interest the case report of convulsions following axillary brachial plexus blockade with levobupivacaine in an anaesthetised patient (Pirotta & Sprigge. A*naesthesia* 2002; **57**: 1187–9). The authors postulate that the convulsions were probably due to an inadvertent intravascular injection of levobupivacaine and with that we concur completely. The use of levobupivacaine with a better cardiovascular safety profile than traditional agents may have prevented more serious consequences, but the practice of a safe technique remains paramount.

We use a different technique to block the axillary plexus than that described and believe that our technique confers some significant safety advantages. The multi injection technique described by Coventry *et al.* [1] uses a peripheral nerve stimulator and a starting current of 2 mA – the reported starting current of 5 mA seems rather high and will cause muscle twitching when the needle is some distance from the nerve. The authors then began injecting levobupivacaine when the twitch disappeared at 1 mA; again, this still may be some distance from the nerve and it is our current practice to locate the nerve and maintain a twitch at 0.5 mA. Patience is a virtue with this technique but Coventry *et al.* report a success rate of 97% with the multi injection technique as opposed to 53% with a single injection technique.

It appears from the article that the authors began injecting when there was no twitch present. This is contrary to our practice, as we would only inject when the twitch was present; if immediate abolition of the twitch did not occur, we would stop injecting, as this is a sign of possible inadvertent intravascular injection. Negative aspiration does not guarantee the absence of intravascular needle placement.

Using the multi injection technique, the total volume of local anaesthetic is, of course, split between different sites, potentially decreasing the dose injected intravascularly at any particular site and

allowing a short time lag between each dose for questioning the patient for any symptoms of intravenous injection.

Finally, the authors used a technique described by Brockway and Wildsmith [2]. Professor Wildsmith is vocal in his support of performing blocks in awake patients [3]. Again, this would have conferred a safety benefit in this case. There may have been warning signs such as peri-oral tingling or tinnitus, which would have alerted the authors and prevented them from injecting the full dose.

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Convulsions following axillary brachial plexus blockade with levobupivacaine (2)

We read with interest the case report involving levobupivacaine-induced convulsions while performing axillary block (Pirotta & Sprigge. *Anaesthesia* 2002; **57**: 1187–9) and would like to make a few comments regarding the conduct of the regional block technique.

Firstly, the main reason for performing the axillary blockade seems to be the avoidance of postoperative nausea and vomiting (PONV). If that were the case, it would have been prudent to avoid a general anaesthetic altogether or at least to avoid nitrous oxide and consider the use of a total intravenous anaesthetic technique instead of using an inhalational anaesthetic agent up to 2 MAC.

Even though the patient had been told the previous episode of PONV was entirely due to opioid analgesia, it is common sense to consider the cause of PONV in a woman undergoing gynaecological surgery under general anaesthesia to be multifactorial.

Secondly, there does not seem to be a good reason for not performing the block awake.

Regarding the conduct of the blockade, commonly the initial current used is between 1 and 2 mA, facilitating the general location of the nerve, and then reducing to 0.3–0.5 mA to ensure close proximity to the nerve [1, 2]. The time when one would expect the twitches to disappear is after the injection of the initial few millilitres of the local anaesthetic. We do not see the logic in injecting local anaesthetic when the twitches disappear at 1 mA and the needle-tip is probably positioned anywhere except in the vicinity of the nerve to be blocked! Further evidence as to the distance between the intended nerve and the position of the local anaesthetic depot is suggested by the fact that the block had completely worn off after only 4 h, a block that, when performed optimally, should last longer.

It is also helpful to disconnect the syringe each time the needle is moved. This ensures that there is a free flow of blood down the cannula (assuming that the immobile needle technique is used) should one of the larger blood vessels be punctured accidentally (commonly axillary artery and vein) and helps remove the problem of negative pressure apposition of smaller vessel walls.

We would also like to point out the miscalculation of the total dose of local anaesthetic as 125 mg instead of 112.5 mg. However, this does not make a big difference in the interpretation of toxicity (1.61 $mg.kg^{-1}$ instead of 1.78 $mg.kg^{-1}$).

Finally, we are interested to know whether the authors were successful in achieving the aim of the blockade, namely avoidance of PONV!

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A reply

Thank you for giving us the opportunity to reply to the comments of Drs Cumming and Barker.

We acknowledge that other techniques to block the brachial plexus by the axillary route may offer advantages. The use of a starting current of 5 mA may be rather high, as they suggest. This is quickly reduced in our practice once a twitch is obtained. However, there is an argument for starting with a lower current. We do not feel that the amplitude of the starting current affects the likelihood of intravascular injection. Ideally, twitch loss should occur with a lower current of around 0.5 mA as they suggest. This is not always possible to achieve, as was the case with our patient. In any case, referral to the anatomy of the brachial plexus shows that the axillary vein and its tributaries lie very close to the branches of the brachial plexus [3], so that it is not inconceivable that the needle might produce a twitch at 0.5 mA and still be in a vein.

It appears we gave the incorrect impression that injection started in the absence of twitches. This is in fact not the case. Once the twitch is lost, the current is increased slightly until muscle activity is recaptured. Then loss of the twitch is witnessed secondary to injection of local anaesthetic as they recommend.

We agree that blocks should be performed in the awake patient whenever possible and we highlight this in our discussion. This is not always possible. In our case, we were concerned that stimulation of muscles crossing the fracture site might have increased patient discomfort. However, we acknowledge that this in itself is not necessarily a contra-indication for performing blocks in the awake patient. Nevertheless, thousands of blocks are still performed in anaesthetised patients in the UK each year.

We are grateful to Drs Dhileepan and Davis for their comments about our anaesthetic technique, which plainly was not perfect. If it had been, our patient would not have convulsed. As they note, the previous episode of postoper-

ative nausea and vomiting (PONV) was probably multifactorial and due to the combination of the factors they highlight. The avoidance of a general anaesthetic (GA) altogether would indeed have been the surest way to avoid PONV. However, this is not always possible. In order to avoid a GA, one would have to be confident that the block was sufficiently dense to allow surgery to take place. This is by no means certain even in the best hands. Furthermore, avoidance of a GA depends on our surgical colleagues being happy to perform the surgery under such conditions. Our orthopaedic surgeons do not routinely perform this procedure under regional anaesthesia alone.

They suggest that the patient's susceptibility to PONV might have been better served if total intravenous anaesthesia had been used. This may be so [4]. However, our patient did not experience PONV. We acknowledge our error in calculating the total dose of local anaesthesia.

Drs Dhileepan and Davis also comment that the detection of a twitch at 1 mA, but not less, is evidence that the needle tip must have been in the wrong place. We would argue that the needle must have been quite close to the brachial plexus, or there would not have been a twitch. Of course we agree that the needle was not in the right place. If it had been, the incident would not have occurred. The fact that the block had worn off completely by 4 h may be due to a significant proportion of the local anaesthetic having been deposited intravascularly rather than perineurally.

Our case report is anecdotal evidence that when up to 125 mg levobupivacaine was injected intravascularly, the effect was a transient convulsion without any cardiovascular effect. It is unlikely that there would have been such a benign response with bupivacaine.

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Lower limb blocks following general anaesthesia

We read with interest the letter concerning lower extremity blocks in the amputee (Lok & Kirk. *Anaesthesia* 2003; **58**: 289–90). The letter raises a number of valid points: first, the choice of peripheral nerve block technique in amputees, and second, the safety issues surrounding regional techniques following the induction of general anaesthesia. We have been faced with similar clinical situations on previous occasions. In the co-operative amputee, we have sought paraesthesia in the phantom limb using either a needle or a peripheral nerve stimulator with a high stimulating current (> 1 mA). In the unco-operative patient, a response to paraesthesia may be difficult to elicit. However, a motor response may be sought within the remaining proximal limb. In this case, we would suggest either a classic Labat or a parasacral approach to sciatic nerve block. During the classic sciatic nerve block, the operator may stimulate the hamstring muscles as a result of either tibial nerve innervation or direct hamstring stimulation with the needle. The latter may fail to establish a satisfactory block if not recognised during block placement. If a piriformis contraction is elicited prior to the onset of hamstring contraction, this is suggestive of correct needle placement.

A parascaral approach [1] avoids direct hamstring contraction. If a hamstring contraction is obtained, it is due to stimulation of the sciatic nerve as it emerges through the greater sciatic notch.

An alternative to peripheral nerve stimulation of the femoral nerve is the fascia iliaca block, which only requires identification of two fascial 'clicks'. This has been shown to provide reliable and effective analgesia within the femoral nerve distribution [2]. In particular, it excludes the possibility of inadvertent intraneural injection.

In the reported clinical situation, the patient presented with toxaemia and single shot peripheral nerve blocks were entirely appropriate. In the absence of sepsis, consideration should be given to the use of peripheral nerve catheters, which would provide excellent postoperative analgesia and reduce the exposure of opioids that may exacerbate acute confusion.

In addition, ultrasound has been used to aid nerve block placement for femoral nerve [3] and posterior lumbar plexus [4] blocks. Whilst this technique may be useful, its use for sciatic nerve blocks has not yet been described.

The ability of patients to report directly the symptoms of intravascular injection or intraneural needle placement is reduced in the confused patient. Alternative methods may aid the detection of these potential complications. Intravascular injection may be identified by the alteration of heart rate and/or blood pressure following injection of an epinephrine-containing solution [5]. Intraneural injection may be identified by a continued motor response with a low stimulating current (< 0.25 mA) or by marked resistance to injection.

In conclusion, peripheral nerve blocks can be used successfully in this patient population.

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Burning issue: a danger of epidurals

We would like to highlight a recent problem encountered while using epidural anaesthesia. A triple arthrodesis of the hind foot was performed on a 60-year-old man with ankylosing spondylitis, using combined spinal-epidural anaesthesia. Due to underlying disease, placement of the epidural catheter was technically difficult though ultimately successful. Surgery was uneventful. With an epidural infusion (bupivacaine 0.4%), the patient was comfortable during the first postoperative night, although the following morning he complained of severe foot pain and was given two boluses (8 ml each of bupivacaine 0.4%) as prescribed by an infusion protocol. Fifteen minutes later he complained of tingling in his mouth and difficulty coughing. Hoarseness was noted and the epidural infusion was discontinued. The patient initially was unable to feel ice at the base of his neck (C3 dermatome), although motor function was unaltered in the upper limbs. His ice levels had fallen to the nipple line (T3 dermatome) when he asked for a cup of coffee. Ten minutes later, he noticed a strange feeling in his fingers, which subsequently became very painful and blistered. He had sustained second degree burns to his thumb, index and ring fingers.

Thermal injury from a hot drink while under spinal or epidural analgesia is an unusual mechanism of iatrogenic injury. It is possible that the level of sensory blockade may have been incorrectly evaluated. This could have been due to the double innervation from the 4th and 5th cervical nerves along with the upper thoracic nerves above the nipple line. As the motor blockade characteristically lags behind sensory blockade by two segments [1, 2], power for lifting the cup to mouth may have been unaffected. It is more likely, however, that the epidural catheter had been placed in, or migrated to, the subdural space. As this is a potential space, small amounts of local anaesthetic can produce very rapid sensory blockade [3]. This classically occurs with minimal motor block and cardiovascular disturbance, which is in complete contrast to an extended spinal or epidural block. Recognition is the most important element of treatment, it is not always necessary to remove the catheter, although the infusion doses need to be adjusted appropriately.

This case illustrates that a simple error in evaluating the level of sensory blockade can lead to serious thermal injuries from something as innocuous as a cup of coffee. It is important to be aware of the possibility of a spinal or epidural block becoming subdural. Monitoring, including pain intensity and severity, level of motor and sensory blockade on chest wall and upper limb, and cardiorespiratory observations must be done to ensure appropriate patient care.

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Transient hearing loss with labour epidural block

We report a case of transient bilateral hearing loss with each top-up for an otherwise uneventful epidural block for pain relief in labour. Cranial nerve palsies associated with epidural blockade have been described in the literature, especially of the trigeminal, abducens and facial nerves [1]. Transient hearing loss has been described in association with accidental dural puncture, which was successfully treated with autologous epidural blood patch (AEBP) [2]. Deafness following spinal anaesthesia has been cured with AEBP [3, 4].

A 30-year-old primagravida with a body mass index (BMI) 54 $kg.m^{-2}$ was admitted to hospital for induction of labour. She was brought to the delivery suite for artificial rupture of membranes (ARM) and augmentation of labour. In anticipation of difficulties during labour associated with morbid obesity, an epidural was sited immediately after the ARM. The epidural was sited using a standard 16G Tuohy needle at the L3/4 interspace under aseptic conditions using a loss of resistance to saline technique. The procedure was uneventful and the catheter was passed at the first attempt. A test dose of 3 ml lidocaine 2% was given with no effect. She was given a first top-up of 10 ml plain bupivacaine 0.25%. The level of the block was checked after 15 min with ethyl chloride and loss of sensation to cold was detected up to the T10 dermatome bilaterally. With the first dose and every subsequent top-up (15 ml plain bupivacaine 0.1% with fentanyl 2 $\mu g.ml^{-1}$) she complained of bilateral hearing loss, which disappeared spontaneously after 30–60 s. After 10 h on delivery suite, the patient underwent lower segment Caesarean section for failure to progress in labour. Anaesthesia was achieved with 20 ml plain bupivacaine 0.5% with fentanyl 5 $\mu g.ml^{-1}$ given as two 10-ml boluses. Transient

deafness occurred with each top-up similar to that described previously.

The immediate postoperative period was unremarkable. She was followed up regularly for 3 days and was discharged home on the fourth postoperative day.

We have not been able to find similar cases described in the literature. Low frequency hearing loss associated with spinal anaesthesia has been described in the literature [5], presumed to be as a result of cerebrospinal fluid (CSF) leak. There was no suspicion of dural puncture in this case. It is possible that pressure changes in the CSF associated with injection of an epidural bolus could have contributed to the hearing loss. We would be interested to hear from other parties as to their opinion of the cause of this patient's symptoms.

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Oxygen delivery from a distance

When no portable oxygen bottles were available for patient transfer in a four-theatre suite, the option of oxygen delivery via tubing from wall mounted

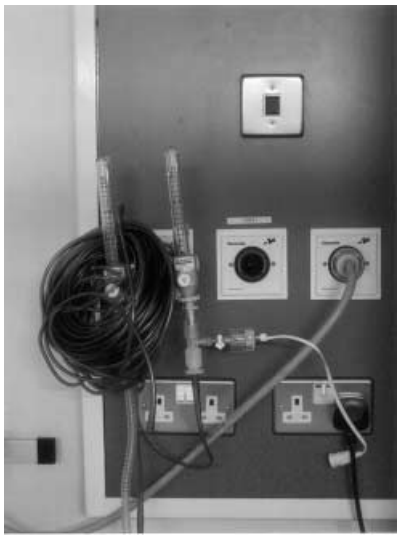

Figure 6

Table 2

Oxygen flow; l..min^{-1}	Oxygen tubing pressure; mmHg	Suction tubing pressure; mmHg
2	98	14
3	125	31
4	132	48
5	250	77
6	338	90
10	> 350	94
15	> 350	173

recovery oxygen flowmeters to operating room was considered; the tubing to follow the patient's trolley. Thirty metres of both green oxygen 'bubble' tubing and electrically conductive suction tubing (Pennine Healthcare) were studied to measure resistance to oxygen flow.

A wall mounted East Healthcare flow meter delivered oxygen through a breathing circuit carbon dioxide analyser connector to which was attached a Becton Dickinson Gabarith pressure transducer linked to a Siemens SC7000 monitor (Fig. 6). The 30 m length of tubing was laid out as straight as possible and pressure measurements at different flow rates were recorded (Table 2). At 10 and 15 l.min^{-1} the oxygen tubing was blown off the bottom of the flowmeter.

Delivery of oxygen at a distance is possible. Suction tubing should be used if high flow rates are required.

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Gum elastic bougies

I read with interest the background to the introduction of the gum elastic bougie as an aid to intubation. When I started anaesthesia training in Cardiff in 1976, gum elastic bougies (with straight tips) were widely available, and I learned to use them on a regular basis. On transfer to a region in the North of England in 1980, there were no gum elastic bougies anywhere to be seen. I asked if they could be obtained but this seemed to be resisted. I therefore ruthlessly applied the following strategy.

Every time I anticipated the need for a gum elastic bougie, or even when I needed one unexpectedly, I asked for the whole oesophageal dilator set, which comprised of a range of about 30 rubber and gum elastic bougies of varying sizes, all with straight tips (although the larger ones were tapered at the ends). I would have to open the whole pack and select the appropriate size (for a child or adult), after which, the whole set had to be re-sterilised. After about eight such uses, the message got home, and dedicated gum elastic intubating bougies were duly purchased.

Of course, we do not have to apply such juvenile tactics these days to obtain essential equipment!

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A better method of Laryngeal Tube insertion

The Laryngeal Tube (LT) [1] is a relatively new supraglottic airway device introduced as an alternative to the laryngeal mask airway. Like the laryngeal mask, it is inserted blindly, simply by opening the mouth and sliding the distal tip along the patient's hard palate until the central incisor teeth are at a prede-

termined level (indicated by a broad black line) marked on the LT shaft. The patient's position during insertion maybe neutral or in the sniffing position [2].

The difference in the method of insertion used for the LT compared to the laryngeal mask is that the index finger is not used. The index finger placed on the laryngeal mask cuff in a pen holding manner helps the advancing tip negotiate the soft tissue resistance encountered. However, in the case of the LT, this resistance is negotiated solely by the advancing tip.

We have been using the LT for elective surgery for the last 8 months and have consistently recorded poorer insertion rates for the LT as compared to the laryngeal mask. We believe that these poor results can be attributed to the difference in the method of insertion of these two devices.

To test our hypothesis we conducted a study in which 13 ASA I and II patients undergoing elective orthopaedic surgery and who had two failed attempts at LT insertion using the method of insertion advocated by the manufacturer [2] were chosen. We then recorded the success rate of LT insertion aided by anterior mandibular displacement (jaw thrust) in these patients.

We found that, aided with the jaw thrust, the LT could be inserted in six out of 13 patients on the first attempt and in another two patients on the second attempt; in the rest (five patients), the LT could not be inserted even with jaw thrust.

It is also interesting to note that (quite unintentionally) in all 13 patients selected for this study, ventilation using the face mask was difficult and was only possible when aided by a Guedel airway. Moreover, nine out of 13 patients complained of snoring while asleep.

The application of anterior mandibular displacement helps the advancing LT tip negotiate the soft tissue resistance, mainly by tensing the genioglossus muscle, which in turn lifts the tongue off the posterior pharyngeal wall. We have now started inserting the LT with the aid of anterior mandibular displacement and have found a significant improvement in the rate of successful insertions.

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Magnetic resonance compatible equipment: apply common sense

I read with interest the incident and subsequent measures taken by your correspondents (Farling *et al*. *Anaesthesia* 2003; **58**: 86–7) of painting a red line on the floor when a piece of apparently 'magnet-safe' monitoring equipment was drawn into their magnetic resonance (MR) scanner. The offending device weighed 25 kg and as such is rather heavy to be carried around on health and safety grounds. Presumably, therefore, it was on a stand with castors. Most MR monitors of recent manufacture involve a magnet-safe front end, often on a mobile stand with castors, to which all the patient monitoring leads and cables are attached, and a separate telemetry receiver display unit, which can be moved round but is intended to be used in the control room and is not necessarily magnet-safe. To reduce the need to take the display unit into the magnet room, the front end usually has a small display unit incorporated into it.

Had a patient been in the bore of the magnet when this incident occurred, it sounds as if serious injury could have occurred. I would not be prepared to work in a scanner where the equipment or the ways in which it is used could result in such an accident, possibly injuring me as well as the patient. Relying on a painted red line on the floor indicating the safe area is as useless as a painted white line down the middle of the road, which is singularly ineffective in keeping oncoming cars from crossing to the wrong side. Either their equipment should be returned to the manufacturer, on the grounds that it is not fit for purpose, or procedures in their scanner altered so that the offending piece of equipment never goes into the magnet room. Their hospital's clinical indemnity manager might also want a say in this, as well as the Medical Devices Agency.

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Estimating oxygen uptake

Your correspondents (Leonard *et al*. *Anaesthesia* 2002; **57:** 654–8) have evaluated elaborate techniques for the estimation of oxygen uptake, but it seems rather perverse to use the reverse Fick method, measuring cardiac output invasively, to do so. All they need is an anaesthetic machine, such as the Ohmeda CD, which has a 'bellows in bottle' ventilator. This can then be used to find the flow of oxygen required to keep the volume constant in a completely closed circle. Provided the carbon dioxide absorber is working satisfactorily, and nitrous oxide is either avoided or has been used for 30 min or more, it follows that the volume of oxygen added to the circuit is equal to that used by the patient.

Or doesn't it?

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Updating Wee's oesophageal detector

Wee's oesophageal detector, which aspirates gas from a tracheal tube but not a misplaced oesophageal tube, used a black rubber corrugated catheter mount, which is no longer available [1]. While trying to fit a 50-ml bladder syringe to a clear plastic 15 mm/22 mm angle piece, the operating department practitioner

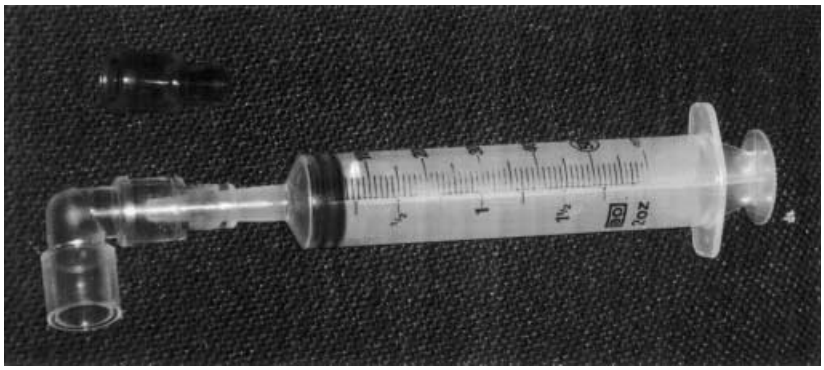

Figure 7

(S.P.H.) pointed out that the Mapleson C disposable breathing system (DAR/Mallinckrodt (286/7814) contained a green and clear soft plastic adaptor for wall mounted oxygen delivery cone to 15 mm female connection, and the clear adaptor fits the bladder syringe nozzle beautifully (Fig. 7).

Wee suggested using his device when a capnograph was not available, for example in an Accident and Emergency Department. The capnograph will not show a carbon dioxide trace in a patient with severe bronchospasm [2] or profound hypotension, both of which can occur in anaphylaxis. 'Hold up' of an Eschmann gum elastic bougie when passed through a tracheal tube will confirm tracheal placement and is recommended in these cases at the University of Washington Hospitals, Seattle (personal communication, Fred W. Cheney). Listening to the chest after every tracheal intubation will give an aural memory, which should alert when things don't sound right. If you haven't seen the tube pass through the vocal cords, then these are all 'useful tools in the fight against inadvertent oesophageal intubation' [3].

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Measuring non-invasive blood pressure in obese patients

Measuring blood pressure (BP) non-invasively in obese patients can be difficult when using a cuff at the upper arm, especially when it is conical in shape. Recently, I was able to overcome this problem by using a child's cuff at the wrist of an obese patient. However, it may not be suitable for very frequent measurements and for long cases because of several nerves that can get compressed at the wrist. By using a child's cuff at the wrist, the need for invasive BP due to difficulty in measuring may be avoided for short cases. The basic principles of non-invasive blood pressure and the relationship between the cuff width and the circumference of the limb are clearly explained in the reference given below [1].

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Reference

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Figure 8

Inspired advice for the FRCA vivas

The vivas for the most recent sitting of the Primary FRCA were held in rooms at the Bloomsbury Central Baptist Church. Outside the church was this sign (Fig. 8), presumably intended for potential worshippers but which also seemed to be fine advice for the exam.

I am sure that many candidates over the years have looked for divine inspiration but can we now assume that this has become a College recommendation?

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