

Correspondence

Postoperative analgesia following renal transplantation – current practice in the UK

We write to report the findings of a survey we performed of the current postoperative analgesia regimens that are utilised following renal transplantation in the UK. We carried out a telephone survey of all 27 NHS renal transplant units listed by the UK transplant directory [1] to establish current practice. We spoke to a member of the acute pain service (in 18 units), a consultant anaesthetist with a regular renal commitment (three units) or a renal unit ward sister (six units). The results are shown in Table 1.

Of the units that use patient-controlled analgesia (PCA) regimens, 18 use morphine (1-mg bolus) and five use fentanyl (20-µg bolus). Table 2 shows the variation in lockout period for

Table 1 Postoperative analgesic regimens.

Technique	No. of units (n = 27)
PCA	24
Epidural analgesia (selected cases)	2
Intermittent subcutaneous injection	1
Intravenous opiate infusion	1
Intermittent intramuscular injection	1

Table 2 PCA regimens.

Lockout time (min)	No. of units
(a) Fentanyl regimen (bolus 20 µg)	
3	1
5	3
6	1
(b) Morphine regimen (bolus 1 mg)	
3	1
5	14
6	1
15	2

morphine and fentanyl PCA. One unit uses morphine with a 0.5-mg bolus and a 10-min lockout period. Two units report experience with epidural analgesia but limit their use to selected recipients of grafts from living related donors. Neither unit reported significant complications, albeit with small numbers, and sited logistics as making this service difficult to provide.

This survey demonstrates that intravenous opioid administration provides the mainstay of analgesia following renal transplantation in the UK. The majority of centres favour the use of PCA morphine but a minority use PCA fentanyl. There is widespread acceptance of PCA as an effective postoperative analgesic technique. Although patient satisfaction is improved by PCA following major surgery, there is little effect on cardiovascular or pulmonary postoperative morbidity or

hospital stay [2]. There is no strong evidence to support the use of one opioid over another in patients with renal impairment. Despite the possibility of significant accumulation of potent metabolites, most UK NHS renal transplant units use morphine for analgesia without apparent problems. Potent active metabolites may act in a negative feedback loop to limit PCA use by the patient.

Solonyenko *et al.* reported epidural analgesia to be effective and without significant complications in a limited series [3]. It is used in selected patients in two units, but more extensive use appears limited by logistical problems and safety concerns. Only epidural local anaesthetic plus opioid mixtures have been shown to provide a reduction in postoperative pulmonary complications following major abdominal surgery [2]. Theoretical risks of the use of epidural catheters in renal transplant patients arise from the frequent tendency to disorders of coagulation (predominantly platelet dysfunction), cardiovascular dysfunction, peri-operative fluid shifts and resultant hypotension with reduced graft perfusion. Cadaveric kidneys with short-term dysfunction from acute tubular necrosis are commonly accepted for transplantation because of the good long-term outcome, but patients may need a period of supportive post-transplant dialysis requiring heparinisation.

It is difficult to quantify the contribution of good analgesia to a successful

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outcome from renal transplantation. However, good analgesia should not be achieved at the expense of graft function. The value of familiarity with a technique or drug should not be underestimated and may well outweigh other potential benefits. The use of regular paracetamol as an acute pain adjunct should not be forgotten. Finally, a functioning renal graft undoubtedly contributes to effective analgesia.

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Syringe labelling in critical care areas

It was with great interest and a combined sense of foreboding and *deja vu* that I read of the combined Royal College and Association of Anaesthetists' plans to implement a standardisation of syringe labelling in critical care areas [1].

Recent events in our department are illustrative of the risks associated with changing systems. Previously, our syringe labels had drug names on a white background. It was decided that we should adopt the international colour coding labelling system as proposed above and these were installed in all anaesthetic rooms. Despite notices of the change, within a fortnight we had two serious drug incidents in which trainees gave succinylcholine instead of fentanyl to conscious patients. Our trainees informed us that most departments in the South West region use a labelling system in which opioids are on orange backgrounds and muscle relaxants are blue and this was the source of confusion. We rapidly removed all the

internationally correct labels and replaced them with the 'local standard' ones. We had a large programme of alert notices, education and training to prevent labelling errors and have had no further problems. We now face returning to the recommended system. Evidence that orange for opioids and blue for muscle relaxants is common labelling practice is backed up by a recent survey, with 95% of UK units responding using these colours [2]. Thus most anaesthetic departments in the UK face the same change.

Whilst in principle I am fully in support of standardisation in this area, in order to prevent many mishaps occurring, changes must be brought in with a fanfare of publicity both nationally and locally and backed up with on-going education. Furthermore, there is a particularly unfortunate hazard in that the commonly used system in this country has the colours for opioids and muscle relaxants almost exactly in reverse to the international standard that is proposed.

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A reply

Dr Souter is correct in all his observations on syringe labelling. The important message, however, is that colour coding is a guide only to the type of drug being used. Reading the text on the label is by far the most important discipline for any practising anaesthetist. In the drug error described by Dr Souter, it seems that this did not happen. The last two major drug errors that I have been aware of have been when the wrong drug has been given in spite of correct labelling of the syringe! I agree, though, that extra vigilance is required during any changeover period. At the end of March and beginning of April, all Clinical Directors will be informed again of the change. This is

in addition to the preliminary notice in *Anaesthesia News* and the next *Royal College Bulletin*. The websites of both organisations will also carry details and the manufacturers will begin supply in May. Most anaesthetists in the UK believe in the need for a colour-coding standard for syringe labelling as Dr Christie's article discovered. The Medilabel® standard, although widely used, is not universal. To develop a European standard is likely to take time to implement. The standard used in Australasia and North America over the past 10–20 years has been selected. We know from discussion with colleagues in these countries that the standard works well. Who knows, it may eventually become a World standard.

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Failed gastric tube insertion with the ProSeal™ laryngeal mask airway (PLMA)

Failed gastric tube insertion via the drain tube of the PLMA can be caused by malposition, inadequate lubrication or selection of the wrong size. We report a case of failed gastric tube insertion due to cuff herniation that also illustrates the importance of appropriate sterilisation procedures and pre-use check tests.

A 33-year-old healthy female patient (height 160 cm, weight 62 kg) presented for anterior cruciate ligament repair. A size 4 PLMA was easily inserted using the digital technique and the cuff inflated to an intracuff pressure of 60 cmH₂O. Ventilation was adequate with tidal volumes of 800 ml without oropharyngeal, gastric or drain tube air leaks; however, a well-lubricated 14 Fr gastric tube could not be inserted due to resistance in the distal portion of the drain tube. Fiberoptic inspection revealed kinking within the bowl. The PLMA was removed and found to have a herniated cuff that was compressing the drain tube from behind (Fig. 1). Further investigation revealed that the

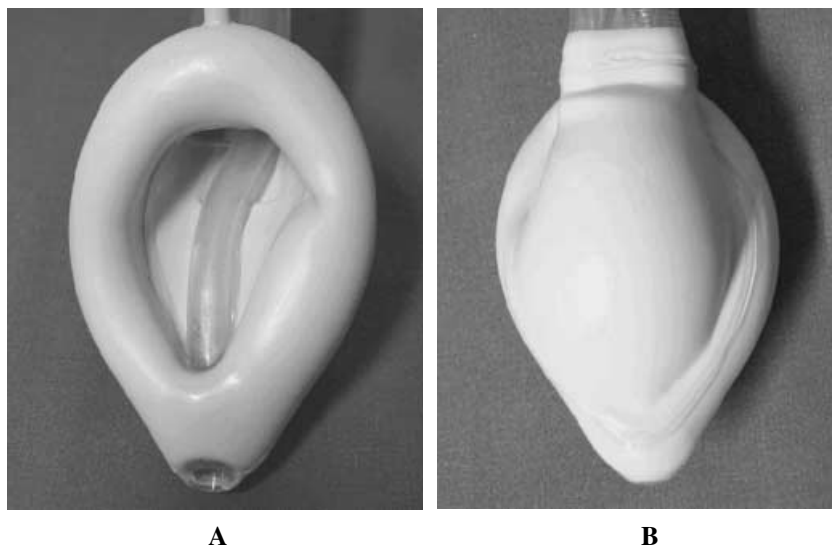


Figure 1 Ventral (A) and dorsal (B) views of the herniated cuff of the ProSeal™ laryngeal mask airway. The arrow indicates the location of the kinked drain tube.

cause of the herniation was incorrect sterilisation in ethylene oxide. The pre-use check tests had not been performed.

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Retropharyngeal abscess presenting with upper airway obstruction

Retropharyngeal abscess is a rare but distinctive cause of upper airway obstruction in children. With the availability of effective antibiotics in recent years, acute retropharyngeal abscesses have become increasingly rare [1]. Torticollis may result from an inflammatory process irritating the neck muscles in an acute retropharyngeal abscess [2].

A 13-month-old girl weighing 9 kg was admitted to the Paediatric Intensive Care Unit (PICU) from the Accident & Emergency Department of University Hospital, Lewisham. The girl had an upper respiratory tract infection 3 weeks

previously and now presented with inspiratory stridor, drooling of saliva and dysphasia. The child, although afebrile, had both a tachycardia (160 $\text{beat}\cdot\text{min}^{-1}$) and tachypnoea (32 $\text{breath}\cdot\text{min}^{-1}$). In addition, she was dehydrated as evidenced by poor skin turgor. The child was drowsy and in respiratory distress with obvious intercostal recession and inspiratory stridor. She was breathing with her mouth open. The tongue was pushed up and nearly touching the palate. The sternocleidomastoids were prominent bilaterally and there was a fixed flexion of the head on the neck.

Investigations revealed a reactive leucocytosis ($24.3 \times 10^9\cdot\text{l}^{-1}$). A lateral X-ray of the neck demonstrated widening of the retropharyngeal space. The ENT consultant decided to perform emergency incision and drainage of the abscess. The anaesthetist accompanied the patient during transfer from the intensive care unit to the anaesthetic room.

In the anaesthetic room, standard monitoring was started. Anaesthesia was induced with inhalation of incremental concentrations of sevoflurane (up to 8% in 100% O_2 using an Ayre's T piece) with the child sitting comfortably on her mother's lap. The child was then transferred to the operating table. As soon as she was positioned



Figure 2 Line diagram showing the view on opening the mouth in the supine position.

supine, the airway was lost. Simultaneously, intravenous access was established. While maintaining jaw thrust and continuous positive airway pressure (CPAP) of 10 cmH_2O , propofol 20 mg was administered to continue induction. This resulted in an unobstructed airway and the child breathing spontaneously. Maintaining sevoflurane at 6% in 100% O_2 , direct laryngoscopy showed the tip of the epiglottis (grade III larynx). The pharyngeal mass was seen to fill the airway (Fig. 2). The trachea was intubated with a 4-mm uncuffed RAE tube by passing it behind the epiglottis. Anaesthesia was then maintained with oxygen, nitrous oxide, sevoflurane and assisted ventilation. The child received a paracetamol suppository (20 $\text{mg}\cdot\text{kg}^{-1}$), dexamethasone (150 $\mu\text{g}\cdot\text{kg}^{-1}$) and Hartmann's solution 200 ml intravenously during the procedure.

An intra-oral mucosal incision was made and the posterior pharyngeal wall abscess drained 30 ml of pus. The trachea was extubated when the child was awake. Postoperatively she was monitored in the PICU for 18 h. She was discharged home with antibiotics 24 h later. *Staphylococcus aureus* was cultured from the abscess.

A pharyngeal abscess usually presents in children with fever, upper respiratory tract infections, irritability and poor oral intake. This progresses to torticollis, neck pain and finally signs of upper airway obstruction [3]. In our case, we feel that the diagnosis was delayed because of the absence of respiratory compromise until late in the disease.

This child presented us with a number of problems. She was 13-months-old and would not allow awake intubation. She was dehydrated, which made venous access difficult. She had a dynamic airway obstruction, which was worsening with time. The retropharyngeal mass bulging into oropharynx prevented the placement of a laryngeal mask and made tracheal intubation challenging. The possibility of rupturing the abscess and subsequent laryngospasm during laryngoscopy increased the airway problems.

We report this case as a reminder that old problems like retropharyngeal abscess can appear unexpectedly. Attention to detail does not guarantee a smooth course during anaesthesia. In our case, CPAP using the expiratory APL valve of a scavenging T-piece breathing system and intravenous induction without causing apnoea saved the situation. We feel that lateral thinking is useful when traditional methods are challenged.

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To refuse or not to refuse, that is the question?

I read with interest your Forum article on the changing use of syntocinon at Caesarean section following the 1997–99 Confidential Enquiry into Maternal

Deaths (Bolton *et al.* *Anaesthesia* 2003; **58**: 277–8). I too have reduced the amount of syntocinon I routinely give and have reduced the rapidity of injection. As yet, I have still not refused the almost routine request by some surgeons to provide a postoperative infusion of syntocinon (most commonly 40 units in 500 ml saline 0.9% over 4–5 h). I am fortunate in that I have recently finished providing first on-call cover for obstetrics after many years, but I wonder if one of your authors could express a learned opinion for those who must continue?

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A reply

Dr Grewal's observations mirror my own; it seems that a postoperative syntocinon infusion has become almost the norm these days – and despite the battling with the volumetric pump that invariably accompanies setting up such an infusion, it does seem a sensible and apparently harmless way of giving the drug. In our patients with cardiac disease, for example, we usually omit even a slow bolus of syntocinon altogether, and instead use an infusion as Dr Grewal describes (though sometimes double-strength if we are worried about fluid overload), with no noticeable side-effects. Another advantage of a postoperative infusion is that it would oppose the effects of any under treatment with syntocinon at the time of delivery, caused by our desire to avoid its adverse effects.

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Another central line complication: failure of inotrope delivery due to equipment incompatibility

A young male was admitted with severe hypovolaemic shock due to limb trauma. The nature of his injuries prevented

placement of wide-bore peripheral cannulae, and so an 8.5 F percutaneous introducer (Swan sheath) was inserted into his left subclavian vein to secure adequate intravenous access. He was transferred to the operating theatre to enable control of ongoing bleeding. He underwent surgery for approximately 8 h and received a considerable quantity of blood products. During the latter part of surgery, he appeared to develop a systemic inflammatory response, and required a norepinephrine infusion (3 ml.h^{-1} of norepinephrine $4 \mu\text{g}$ in 50 ml of dextrose 5%) to maintain an adequate blood pressure. The inotrope was administered through a 16-cm 7.5 F central venous line that had been inserted through the introducer sheath.

In the immediate postoperative period on intensive care, he developed a recurring pattern of haemodynamic instability during administration of fluids. At the start of fluid bolus delivery, he became hypotensive. His blood pressure slowly returned to its previous value as the infusion continued. He then became hypertensive following cessation of fluid administration. The cause of this was not immediately apparent. The fluids were administered as a 250-ml bolus at a rate of 999 ml.h^{-1} via a volumetric pump through the side arm of the percutaneous introducer sheath. The norepinephrine was administered via the medial lumen of the central line. The observed changes in blood pressure could be explained if the medial lumen of the central line remained within the introducer sheath. This would mean that administration of a fluid bolus would cause backpressure in the medial lumen of the central line, and reduce delivery of norepinephrine from the syringe pump. At the end of fluid bolus delivery, a bolus of norepinephrine was delivered due to the sudden drop in pressure within the central line.

In order to measure these presumed pressure changes, an invasive pressure transducer was attached to both the side arm of the introducer sheath and the medial lumen of the central line, and pressures measured before and during administration of a fluid bolus. These pressures are given in Table 3. Subse-

Table 3 Pressure (mmHg) measured in the medial lumen of the central line and in the side arm of the introducer sheath during infusion of inotrope only, and during inotrope and fluid bolus administration.

	Inotrope only	Inotrope + fluid bolus
Medial lumen of central line	20 mmHg	66 mmHg
Side arm of introducer sheath	10 mmHg	145 mmHg

quent fluids were given via a peripheral cannula. Following this, he no longer demonstrated these changes in blood pressure.

Syringe pumps deliver fluid by pushing a plunger along the syringe barrel, forcing fluid out of the end of the giving set. There is resistance to the force generated by the syringe pump as a result of factors such as resistance to flow due to the viscosity of the infused fluid and hydrostatic pressure due to any difference in height between the syringe driver and patient. Fluid will only flow when the driving mechanism has generated a driving pressure sufficient to overcome this downstream resistance. Changes in hydrostatic pressure while an infusion is in progress may cause a temporary change in infusion rate whilst the syringe driver adapts to the new downstream pressure [1].

The effect that the observed pressure changes may have had on the delivery of inotrope were investigated with an experiment designed using principles described elsewhere [1, 2]. A 50-ml syringe containing dextrose 5% was placed in a syringe driver mounted horizontally. A 200-cm fine-bore giving set was connected to the syringe. A 1-ml syringe with the plunger removed was inserted vertically into a three-way tap connected to the other end of the giving set. The height of the three-way tap was raised and lowered to simulate the changes in backpressure. The pressures in Table 3 were converted to a height of water using the principles:

pressure = height × density × gravity [3],

where the relative density of water is 13.6 times that of mercury.

The effect of a change in backpressure on the syringe driver was assessed by measuring the volume of dextrose delivered in a 15-min period having raised or lowered the three-way tap to simulate either application or removal of backpressure.

These measurements were repeated 10 times. When backpressure was applied, the increase in hydrostatic pressure prevented the syringe pump from delivering fluid for 84 s. Release of the pressure resulted in a bolus of 0.067 ml of fluid being administered. This would appear to confirm that the haemodynamic instability seen in this patient was caused by backpressure due to the central line being within the introducer sheath. Other studies have demonstrated similar no-flow times and bolus delivery with changes in hydrostatic pressure produced by moving the end of the giving set in relation to the syringe pump [2].

A straw poll amongst colleagues revealed that the practice of inserting central venous lines through percutaneous introducer sheaths is not uncommon. It may have been assumed that the central line would protrude from the end of the introducer sheath. However, as shown in Fig. 3, a 16-cm central line only just protrudes from the end of the percutaneous introducer sheath. The positions of the proximal and medial lumens are marked, and can be seen to remain within the sheath. The use of a longer 20-cm central line would have allowed all lumens to protrude from the end of the sheath (Fig. 4) and would have prevented such haemodynamic instability. Anaesthetists

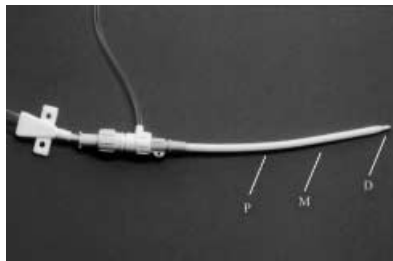


Figure 3 A 16-cm central line inserted through a percutaneous introducer sheath. The positions of the proximal (P), medial (M) and distal (D) lumens are marked.

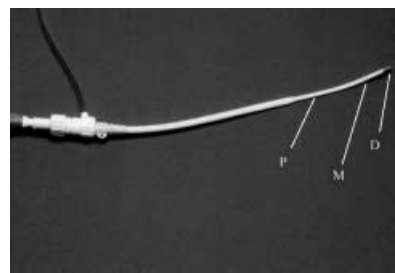


Figure 4 A 20-cm central line inserted through a percutaneous introducer sheath. The positions of the proximal (P), medial (M) and distal (D) lumens are marked.

should ensure that a central venous line of sufficient length is used if inserting it through a percutaneous introducer sheath.

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Fibreoptic equipment and variant Creutzfeldt–Jakob disease

Following recent correspondence in this journal (*Anaesthesia* 2003; **58**: 90–1, *Anaesthesia* 2003; **58**: 482–3), we are delighted to report that flexible fibre-optic equipment for intubation is available now, on request, from the National Creutzfeldt–Jakob Disease (CJD) Surveillance Unit in Edinburgh [1], for use in patients with variant or sporadic CJD.

A process similar to the one whereby the Surveillance Unit retains a gastro-scope for use in CJD patients who require percutaneous endoscopic gastrostomy (PEG tube) insertion will be adopted for the intubating fiberoptic equipment. An Olympus Bronchoscope 1T 30 with light source CLE/3 was donated to the unit by a Hospital Trust, which wishes to remain anonymous, following use in a patient with known vCJD. In addition, Pentax UK Ltd, distributors of fiberoptic equipment, are in the process of identifying a suitable endoscope that they will donate to the CJD Surveillance Unit.

Clearly, there must be a strict protocol for the use of this equipment because it is potentially contaminated and there is a risk of onward transmission of infection. When a patient with known or suspected vCJD is scheduled for surgery, an experienced anaesthetist should determine the potential need for fiberoptic intubation. The CJD Surveillance Unit should be contacted as soon as the availability of the fibrescope is considered necessary. Telephone: 0131 5372658 or 0131 5373073; e-mail: C.A.Mckenzie@ed.ac.uk.

Patients in whom the fibrescope is to be used should fulfil the current clinical criteria for a diagnosis of definite or probable CJD [2, 3]. Fully informed consent must be obtained from the patient and/or relatives. The current diagnostic criteria and a suitably worded consent form are available from the CJD Surveillance Unit.

The CJD Surveillance Unit will coordinate delivery of the instrument using designated couriers. The couriers are licensed to carry Class 6.2 items [4] and have experience of transporting the gastro-scope to and from hospitals. After use, the fibrescope should be cleaned locally, taking suitable protective precautions, using the protocol for manual cleaning of endoscopes [5]. It is not necessary to include the disinfecting step with glutaraldehyde. The Medical Devices Agency and the CJD Policy Team have been informed of this arrangement. In case of breakage or malfunction, the instrument should not be sent to a manufacturer but should be returned to the CJD Surveillance Unit.

Information regarding this equipment will be available on the Difficult Airway Society's website (<http://www.das.uk.com>) or via the chairman of the society (chairman@das.uk.com).

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Transient lingual ischaemia during anaesthesia

We would like to describe a case in which a 72-year-old man unexpectedly developed transient lingual ischaemia. He had been taking several antihypertensive drugs for 20 years. He underwent a total cystectomy under general anaesthesia with propofol, fentanyl and sevoflurane. An infusion of prostaglandin E1 (PGE1) was required for the excessive blood pressure responses. At the end of surgery, we were surprised to note that the whole body of

the tongue looked severely cyanotic. However, the cyanosis gradually recovered following tracheal extubation. Postoperatively, the patient underwent an ultrasonographic study of the extracranial carotid artery. It showed the presence of atherosclerotic plaques with a 40–60% reduction in the intravascular area at the carotid bifurcation bilaterally and a moderate stenosis of 45% in the right external carotid artery.

It is likely that compression by the tracheal tube at the base of the tongue is a possible cause of lingual ischaemia. As the ultrasonographic findings showed, our patient was considered to have a potential for lingual ischaemia. If relatively stenotic and inelastic arteries supply the patient's tongue, it might be more vulnerable to ischaemia by minor compression. Additionally, the use of PGE1 presumably further reduced the residual blood flow distal to the obstruction [1].

This case suggests that the arteriosclerotic vascular change affecting the external carotid artery [2, 3] may be a risk factor for lingual ischaemia. Ultrasonography is a non-invasive and useful tool to evaluate such pathological vascular changes. However, it is unclear how closely they relate to the incidence of lingual ischaemia in the anaesthesia setting.

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Consenting for direct laryngoscopy

A very interesting debate about whether the laryngeal mask airway has caused a decline in the intubation skills of anaesthetists took place during the Royal College Annual Meeting in March 2003. The result was an honourable draw; but is there a way we can still use the laryngeal mask, which both debating parties thought to be an important tool in anaesthesia, without jeopardising intubation skills? I think there is.

The reduction in intubation skills is usually a problem during the difficult intubation situation. So, we need to identify the difficult cases before they appear for an anaesthetic that requires intubation. Because most current operations are undertaken using a laryngeal mask and the difficulty in intubation is largely due to laryngoscopic problems, we can at least record a laryngoscopy result during laryngeal-mask-based anaesthesia. Then the patient will benefit from knowing whether he/she is a potentially difficult intubation. This is far more accurate than anatomical-based assessments and the trainee will benefit from practising the most important part of intubation. The anaesthetic time will not substantially increase by waiting to deepen the anaesthetic level enough to allow direct laryngoscopy.

Finding a difficulty and then not intubating the trachea may not be the best training opportunity but it is certainly a good one. This information will make the trainee prepare for any mishaps that may occur during the current procedure and allow a plan if in the future he or she is faced with this case or a similar one.

The ethics of this manoeuvre might be controversial on the basis of being unnecessary for the patient. The following argument, however, could be put. If the patient does not have a documented previous anaesthetic in which intubation was required, laryngoscopy will be of benefit for future anaesthetics and perhaps for the current one. If the patient has had a documented anaesthetic with an easy intubation and nothing currently suggests any expected difficulty, there is no need to perform laryngoscopy.

If all of this is not convincing, why not take consent from the patient for direct laryngoscopy?

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Bronchospasm or blocked tracheal tube?*

A 54-year-old man with severe rheumatoid arthritis was scheduled for cervical spine decompression and fixation (C2–T2). The trachea was intubated nasally using a 6.5-mm ID (internal diameter) Mallinckrodt™ reinforced tracheal tube and an awake fibreoptic technique; the cuff of the tracheal tube was inflated with air. The patient was anaesthetised with propofol and sevoflurane, and paralysed with vecuronium 10 mg. Pulmonary compliance appeared normal on manual ventilation and mechanical ventilation was continued using a Blease 6200 ventilator connected to a Bain circuit.

Whilst gaining further intravenous access, the capnograph demonstrated a dramatic change in re-breathing with an inspiratory carbon dioxide partial pressure greater than 2 kPa. Peak inflation pressures of 45 cmH₂O were noted. On auscultation of the lungs, there was bilateral air entry with polyphonic expiratory wheeze. The patient was cardiovascularly stable, and there were no other signs of an adverse drug reaction. Tracheal tube patency was confirmed by the easy passage of a suction catheter. Fibreoptic examination of the lower airway through the tracheal tube showed no sign of cuff herniation; however, the tube orifice was partially occluded by the posterior tracheal wall. Deflation of the cuff immediately revealed an unobstructed view of the carina and following this manoeuvre there was a dramatic reduction in inflation pressure and the expiratory wheeze disappeared. Re-inflation of the cuff immediately resulted in high

inflation pressures again. It was decided to proceed with the operation with the cuff deflated. A throat pack was positioned to reduce the leak around the tube. Anaesthesia and surgery progressed uneventfully. At the end of the procedure, the trachea was extubated and the patient made a good recovery. The tube cuff was inspected afterwards and found to inflate symmetrically.

We think that this incident occurred as a result of cuff inflation causing the tracheal tube tip to deviate towards and to abut the unsupported posterior tracheal wall. In retrospect, rotation of the tube may have negated this effect by positioning the lumen away from the posterior tracheal wall. We suspect that the change in airway pressure that occurred shortly after the initiation of mechanical ventilation arose following a spontaneous rotation of the tracheal tube within the trachea, which caused the tube lumen to face posteriorly. We had used a 'drilling' movement to advance the tracheal tube over the endoscope and perhaps this had introduced some torque into the tracheal tube. Another possible explanation is that the bevel was positioned against the posterior tracheal wall from the beginning, and this acted as a one-way valve, allowing a number of 'normal' breaths before inflation pressures began to rise and the physical signs described earlier then became apparent.

This type of tracheal tube does not have a Murphy's eye and perhaps the presence of this would have minimised the airway pressure changes that we observed with tracheal tube cuff inflation.

There are previous case reports of tracheal tube lumen obstruction by impingement on the tracheal wall associated with tracheal tube movement [1] and uneven cuff inflation [2]. Our case serves as yet another reminder that mechanical causes for bronchospasm during anaesthesia should always be considered.

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Expiratory obstruction caused by inappropriate connection of the expiratory limb of a breathing circuit

An important safety feature of breathing systems is the use of non-interchangeable connectors. The British Standards Institute recommend a 22-mm taper for connections within breathing systems (BS EN 1281-1: 1997). The presence of any other part on the anaesthetic machine that can fit a 22-mm connector constitutes a potential hazard. We describe a critical incident caused by connection of the expiratory limb of a breathing circuit to a 22-mm-diameter circuit block pressure relief valve.

A 61-year-old patient presented for elective coronary artery bypass graft surgery. During induction of anaesthesia, the patient's lungs were ventilated manually using a facemask attached to a circle breathing system that included a Scandia absorber circuit block (Blease Medical Equipment Ltd). Immediately prior to tracheal intubation, the expiratory limb of the breathing circuit became disconnected and was hastily reconnected. Following intubation and onset of mechanical ventilation, the patient's capnograph trace declined to zero, the ventilator low- and then high-pressure alarms sounded and the patient's blood pressure rapidly fell to 40 mmHg systolic. Resuscitation measures included reintubating the patient's trachea, manual ventilation using a Mapleson-C circuit connected to the fresh gas flow port of the anaesthetic machine and vasoconstrictor administration. The capnograph trace returned and the patient's blood pressure returned to normal. Subsequent surgery and postoperative course were uneventful.

Inspection of the circle breathing system revealed that during reassembly, the expiratory limb of the breathing



Figure 5 Breathing system correctly connected to the Scandia absorber circuit block. The patient safety valve and the expiratory limb of the breathing system are highlighted.



Figure 6 Expiratory limb of the breathing system misconnected to the patient safety valve of the Scandia absorber circuit block resulting in expiratory obstruction.

system had been accidentally connected to the patient safety valve of the Scandia absorber circuit block (which fits a 22-mm connector) rather than the appropriate 22-mm expiratory port of the circuit block (Figs 5 and 6). This resulted in expiratory obstruction subjecting the patient to a sustained Valsalva manoeuvre at a pressure of

40 cmH₂O, the upper pressure limit set on the ventilator.

Notwithstanding the fact that all those involved in the care of anaesthetised patients should be familiar with the equipment that they are using, we believe the presence of an extraneous 22-mm connector on this piece of equipment represents an unnecessary

and avoidable hazard. We have reported this incident to the Medical Devices Agency and to the manufacturer.

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A reply

The incident described is very unfortunate. However, we feel we must point out the need for full training of clinical staff on the equipment they are using. They should also be conversant with the contents of the *User Instructions*. These precautions combined with a pre-use check would have averted the incident described in the article.

There is a good argument made in the correspondence that suggests that, wherever practical, the possibility that the absorber can be used incorrectly is designed out. Blease Medical endeavour to do this with all products and we have made a modification to the absorber to 'design out' this particular incident. The modification will make it impossible to fit the breathing tube to the PRV in the manner described.

We believe we have acted in a very responsible manner regarding this matter by designing out the issue after having seen your report on it. Objective assessment of the facts suggests to us that non-compliance with the *User Manual* and training have played a larger part in this matter than the possibility that the system can be connected incorrectly.

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Total intravenous anaesthesia for oculoplastic surgery in a patient with myasthenia gravis without high-dependency care

We read with interest the use of total intravenous anaesthesia (TIVA) without muscle relaxation for eye surgery in Kugelberg–Welander syndrome (Watts.

Anaesthesia 2003; 58: 96). We wish to report our experience in a patient with myasthenia gravis who underwent major oculoplastic surgery using TIVA without muscle relaxation.

Myasthenia gravis is an autoimmune disorder caused by production of auto-antibodies against acetylcholine receptors of the muscle endplate. In myasthenia, the number of activated receptors may be insufficient to cause a normal response and with repeated stimulation there is fatigue. Problems for the anaesthetist include: (1) respiratory muscle weakness with the risk of respiratory failure; (2) bulbar muscle weakness with the risk of pulmonary aspiration; (3) interaction with relaxant drugs, resistance to depolarising agents, sensitivity to non-depolarising relaxants [1].

Following surgery, although planned extubation is usually possible, postoperative care in a high-dependency area has been recommended where rapid respiratory support can be instituted if necessary [2]. Several authors have reported the use of TIVA in patients with myasthenia followed by HDU care [3, 4].

Our patient was a 45-year-old, 68-kg male with a 10-year history of myasthenia gravis graded as Osserman classification II [5]. His treatment consisted of prednisolone, azathioprine, neostigmine and pyridostigmine; in addition, he received immunoglobulin therapy every 5 weeks. He presented to the Oculoplastic Unit with bilateral facial weakness and consequent severe eyelid closure problems. He was listed for mid-face lifts and posterior lamellar hard palate grafts on both sides. It was estimated that each side would take 2–3 h, so plans were made to undertake one side at a time. As our eye unit is geographically separate from the remainder of the hospital, arrangements were made to relocate the operating theatre list to the general unit for ease of access to the HDU. It took some months to organise operating theatre time owing to the very busy schedule in the general unit. Anaesthesia consisted of propofol via a Target Controlled Infusion (TCI) Diprifusor system and remifentanyl by infusion with oxygen in air; no muscle relaxation was used for tracheal intubation. Local anaesthesia

with vasoconstrictor was infiltrated at the operative site. The patient was extubated 5 min after the infusions were discontinued at the end of surgery. He was transferred to HDU where he was fully awake and able to take his medication within 1 h.

For the second operation, no special arrangements were made. In particular, as the surgery was identical, no arrangements were made for high-dependency care. Anaesthesia again consisted of TIVA without muscle relaxants. Following awake extubation, the patient returned to the eye hospital ward and was mobilising within 1 h.

Non-relaxant techniques are a recognised method of anaesthesia in myasthenia [6]. Remifentanyl is ideal in this situation because of its potent analgesic effects, the ability to provide apnoea and hence use controlled ventilation with minimal effects on neuromuscular transmission and a rapid offset. Its use in myasthenia gravis has previously been described [4].

This patient had repeated prolonged peripheral surgery that did not require relaxation or potent postoperative analgesia. The use of a short-acting intravenous anaesthetic technique virtually eliminated the risks of respiratory failure or aspiration despite surgery lasting 2–3 h. In addition, the avoidance of volatile agents reduced the risk of postoperative nausea and consequent intolerance of necessary oral maintenance medication for myasthenia gravis. Having demonstrated on the first occasion the safety of the technique, we were able to avoid the delays associated with the need for HDU facilities.

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Anterior commissure laryngoscope

We describe the use of a laryngoscope commonly used by ENT surgeons in two cases of unexpected difficult intubation where the presence of blood in the airway would have made fiberoptic laryngoscopy difficult.

A 79-year-old man was scheduled for panendoscopy for investigation of sore throat, earache, blood-stained sputum and weight loss. He had no history of stridor or breathing difficulties. The results of his flexible laryngoscopy in the ENT clinic were not available in the case notes and the surgeons failed to communicate the presence of a supraglottic tumour noted at this clinic visit. He was Mallampati grade 1 with good mouth opening and a thyromental distance of more than 6.5 cm. There were no symptoms or signs of upper airway obstruction. Anaesthesia was induced with propofol 120 mg, alfentanil 750 µg and atracurium 25 mg. Initial laryngoscopy with a Macintosh size 4 blade demonstrated a florid, friable tumour at the base of the tongue and a Cormack and Lehane grade 3 view. Repeat laryngoscopy with a McCoy blade made no improvement and several attempts to pass a gum elastic bougie blindly into the trachea failed, resulting in bleeding of the tumour. Mask ventilation was easy. Fiberoptic laryngoscopy would have been difficult due to the presence from blood and therefore the ENT surgeon used an anterior commissure

laryngoscope with ease to achieve a good view of the vocal cords. A gum elastic bougie was passed through the barrel of the anterior commissure laryngoscope. This laryngoscope was then removed and a size 5.0 mm microlaryngoscopy tube railroaded over the bougie. Tracheal intubation was confirmed with capnography. Panendoscopy demonstrated a large tumour involving the base of the tongue with distortion and narrowing of the pharynx.

In the second case, a 41-year-old man was scheduled for a left-sided superficial parotidectomy. Pre-operative assessment of the airway demonstrated good mouth opening, a Mallampati grade I pharyngeal view and a thyromental distance of 6 cm. Anaesthesia was induced with fentanyl 100 µg and propofol 200 mg. After confirming that mask ventilation was possible, muscle relaxation was achieved with cisatracurium 12 mg. Laryngoscopy with a Macintosh size 4 blade produced a grade 3 Cormack and Lehane view that could not be improved with a McCoy size 4 blade. The larynx was noted to be very anterior. Several attempts at blind intubation with a tracheal tube over a gum elastic bougie led to oesophageal intubation, which was confirmed by capnography and auscultation. Bleeding caused by the multiple laryngoscopies and blind insertions of a gum elastic bougie would potentially have made fiberoptic laryngoscopy difficult. With our previous experience we asked the ENT surgeon to use an anterior commissure laryngoscope. This was performed without difficulty and achieved a good view of the larynx. A gum elastic bougie was passed through the barrel of this laryngoscope and the anterior commissure laryngoscope was removed. A 7.0-mm armoured tracheal tube was railroaded into the trachea without difficulty. Anaesthesia and surgery proceeded uneventfully. The patient was reviewed the next day. He had no recollection or awareness of the events but complained of a sore throat.

ENT surgeons using an anterior commissure laryngoscope rarely fail to visualise the larynx. This laryngoscope (Fig. 7) has a straight enclosed barrel, which prevents the tongue from obscuring the field of view and allows



Figure 7

easy suctioning. The distal end is flared anteriorly with a recessed light permitting a good view of the larynx. The technique of straight-blade laryngoscopy is different to the Macintosh technique [1]. In this paraglossal or retromolar technique, the anterior commissure laryngoscope is passed from the right corner of the mouth, displacing the tongue to the left and advanced towards the larynx between the tongue and the tonsil. The neck is flexed and the head extended to bring the axes of the mouth, pharynx and larynx into alignment. Displacement of the tongue to the left removes the tongue from the line of view. This prevents the tongue from limiting anterior movement of the blade, which may be the cause of difficult intubation in some cases when a Macintosh blade is used. This technique is not new and has been described before [2–4] but is forgotten amongst most anaesthetists.

In cases of unexpected difficult intubation, particularly where the presence of blood in the airway would result in difficult fiberoptic laryngoscopy, an anterior commissure laryngoscope with a gum elastic bougie can be used for tracheal intubation. However, this may be better achieved in skilled hands even if it requires the assistance of an experienced ENT surgeon. Training of anaesthetists in straight-blade laryngoscopy is essential if they are to become competent in tracheal intubation with this method when faced with an unexpected difficult airway.

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The oesophageal tracheal combitube: a pilot study on airflow resistance and ventilatory pressures in 26 anaesthetised patients

Airway pressure levels during controlled ventilation are greater when using the combitube 37 F (CT 37 F; Tyco Healthcare, Mansfield, MA, USA) device instead of a tracheal tube (TT) [1–4]. The higher airflow resistance exhibited by the CT with respect to the laryngeal mask or TT depends on its reduced cross-section and turbulent flow induced by eight perforations at the pharyngeal level. The goal of our

study was to determine the real pharyngeal pressure in patients ventilated by a CT 37 F.

After approval from the Ethics Committee and informed consent, we recruited 26 consecutive patients scheduled for routine elective surgery. These comprised 11 women and 15 men, ASA status I and II, aged from 34 to 79 years (mean 55 years), body weight from 48 to 85 kg (mean 65 kg) and height from 157 to 179 cm (mean 171 cm).

The measurement of pressures during controlled ventilation was carried out with the aid of an experimental unit specifically adapted for the purpose. The airflow resistance of various size TTs and CT 37 F was evaluated by *in vitro* laboratory tests within the ventilation flow range used in clinical tests (Fig. 8).

The laboratory studies showed that the pharyngeal lumen of the CT 37 F has an airflow resistance higher than that of a 7-mm TT and lower than that of a 6-mm TT. Consequently, clinical evaluation of the effective airflow resistance of the same devices when inserted into the patient's airway (*in vivo* tests) was performed. From the laboratory evaluation, a 7-mm TT was chosen for the *in vivo* study because the reduced cross-section of a 6-mm TT produced a higher pressure than the CT 37 F (Fig. 8). A Servo 300 ventilator (Siemens, elema A.B., Sweden, Stockholm) was used to apply volume-controlled ventilation. A tidal volume of 8 ml.kg⁻¹ and a respiratory rate of 12 breath.min⁻¹ were used.

The peak pressure during ventilation was measured at three points: inside the pharyngeal lumen of the CT 37 F, at the proximal end (P1) and distally in front of the eight perforations at the pharyngeal level (P2); in the pharynx, external to the CT 37 F (P3).

In patients intubated with a TT, the peak ventilation pressure was measured at two points: inside the TT lumen, at the proximal end (P1) and in the trachea; outside the TT (P3).

The anatomical features of six of the 26 patients allowed us to use a 7.0-mm TT: in these patients the peak pressures were measured and compared using both the CT 37 F and the TT (PIP, P1, P3).

The results confirm that the airflow resistance *in vivo* exhibited by the CT 37 F and the 7-mm TT are comparable without significant difference (Table 4). Thus, in neuromuscular blocked patients, the tracheal pressure with a 7-mm TT was comparable to the pharyngeal pressure with a CT 37 F.

In order to detect the critical points that determine a higher drop in pressure along the profile measured (PIP, P1, P2, P3) when the CT 37 F was inserted in the oesophageal position, the pressure profile occurring in the 20 other patients with a weight higher than the first six patients was investigated. The ventilation mode and parameters used (tidal volume 8 ml.kg⁻¹; respiratory rate 12 breath.min⁻¹) remained unchanged. The results (mean value) indicate an increase of PIP, P1 and P2 values, while the P3 value remained constant (Table 5). This is due to an increased drop in pressure between the inside and outside of the device; P2 (19.0 cmH₂O)/P3 (7 cmH₂O) occurred because of turbulent air flow resistance produced by the angular

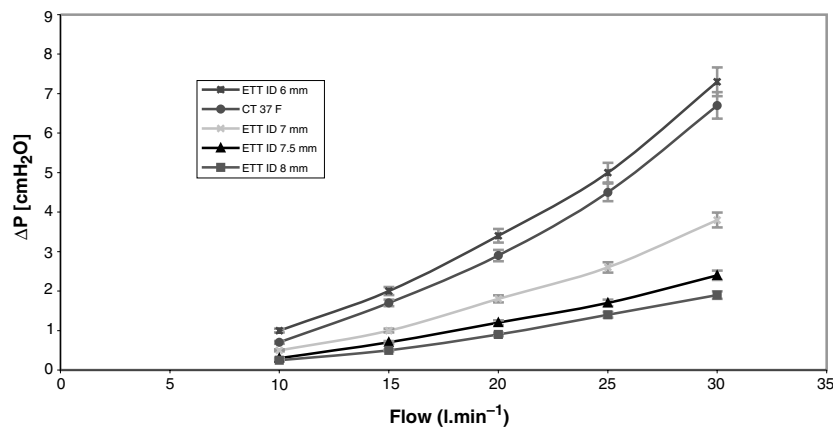


Figure 8 Air flow resistance of various size TT and a CT 37 F evaluated in laboratory tests.

Table 4 Mean value (cmH₂O) of peak pressure measurement at different levels along the airway profile (PIP, P1, P3) in six patients ventilated with CT 37 F and 7 mm TT.

	CT 37 F	7-mm TT
PIP	19.6	19.3
P1	17.8	17.1
P3	6.7	6.1

Table 5 Mean value (cmH₂O) of peak pressure measurement at different levels along the airway profile (PIP, P1, P2, P3) in 20 patients.

	CT 37 F
PIP	21.0
P1	19.9
P2	19.0
P3	7.0

deviation (about 90°) of flow crossing the eight perforations of the device.

Preliminary data show that PIP of 21 cmH₂O corresponds to a pharyngeal pressure of one-third this value, about 7 cmH₂O.

In conclusion, the results indicate that the CT 37 F does not incur a higher risk of barotrauma than the TT.

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Giant axonal neuropathy and anaesthesia

Giant axonal neuropathy (GAN) is an autosomal recessive gigaxonin gene disorder, leading to sensory and motor disturbances and mental retardation [1]. The disorder is characterised by large

axonal swellings consisting of accumulated neurofilaments because of cytoskeleton changes [2]. Clinical signs begin in early childhood and progress to late adolescence death [3]. Initially, delay in clinical motor milestones, proceeding to clumsiness of gait and progressive weakness starting at the distal lower limbs, become apparent. Sensation is impaired and tendon reflexes are absent. Subsequently, dysarthria, nystagmus, facial weakness and mental retardation become apparent. Patients usually become wheelchair bound in the first or second decade of life and have kyphoscoliosis and tightly-curved hair.

More than 200 patients are described in the literature [4, 5]. However, only one reference exists on anaesthesia in GAN patients [6]. The potential anaesthetic hazards are autonomic disturbances, restrictive pulmonary disease (kyphoscoliosis), prolonged muscle weakness in response to drugs, hyperkalaemia after succinylcholine, and existing aspiration pneumonia.

We recently anaesthetised a patient with GAN for acute torsion of the testes. This wheelchair-bound 13-year-old boy had morphologically confirmed GAN, which was later genetically proven. Besides the usual morphological characteristics, pulmonary function was impaired: vital capacity 1.59 l (normal 2.35 l) and forced expiratory volume at 1 s of 1.30 l (normal 1.92 l). Both maximum inspiratory airway pressure (–23 cmH₂O) and maximum expiratory airway pressure (+ 31 cmH₂O) were low. Arterial blood gases were normal. Pre-operatively we found a Mallampati grade 1 with normal neck mobility.

No premedication was given. Basic monitoring was established (ECG, pulse oximetry, NIBP). After establishing venous access, anaesthesia was induced with propofol 2 mg.kg⁻¹ remifentanyl 0.14 µg.kg⁻¹, and maintained with continuous infusions of the same drugs. A laryngeal mask was inserted. The lungs were ventilated using 50% oxygen at low inflation pressures. Surgery and recovery were uneventful. For postoperative analgesia, oral paracetamol 1000 mg was provided if needed. The patient was discharged on the first postoperative day.

This patient illustrates some anaesthetic considerations. Although in GAN patients regional anaesthesia may be considered, we chose general anaesthesia because of the severe thoracolumbar kyphoscoliosis present. We avoided tracheal intubation because of existing weakness of respiratory muscles and disturbed pulmonary function. This was justified because of no history of gastric aspiration and the presence of normal facial and neck muscle strength. A laryngeal mask was introduced. In these patients, the effect of non-depolarising neuromuscular blockers has not been evaluated and succinylcholine must be avoided.

Use of a nerve stimulator to assess neuromuscular block was considered of limited value because of the patchy loss of nerve function in such patients. Intraoperative respiratory support prevented hypoventilation, which may occur during anaesthesia in severe kyphoscoliosis.

Although in GAN, the autonomic nervous system is involved, our patient did not have urinary incontinence, or cardiovascular or gastrointestinal dysfunctions.

We believe total intravenous anaesthesia using propofol and remifentanyl and avoiding premedicants influencing muscle strength is a safe technique in GAN patients.

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Dystonic reactions to cyclizine

We read with interest the recent case report of dytonia after a single dose of cyclizine (King *et al.* *Anaesthesia* 2003; **58**: 257–60), having experienced a similar patient reaction. Our patient was a 27-year-old female day case, presenting for a diagnostic laparoscopy for menorrhagia and intermenstrual bleeding. She had undergone a previous uneventful general anaesthetic and had no significant past medical history. The operative procedure was uneventful and, following extubation, the patient was transferred to the recovery area. Her postoperative pain required a total of 15 mg of morphine over the next hour, plus two cocodamol tablets. As she was still in pain, a further 2 mg of morphine was given together with intravenous cyclizine 50 mg, following which she became unresponsive. Cardiorespiratory examination was unremarkable except for sinus tachycardia. Neurological examination revealed spontaneous eye opening with no verbalisation or motor response to pain. She had non-conjugate oculogyric eye movements but normal corneal and gag reflexes. Muscular tone was initially mildly increased but later became flaccid with normal reflexes and she had a normal blood glucose level.

Two doses of procyclidine 5 mg had no obvious effect but, after 15 min, she became more responsive and was able to obey simple commands such as hand squeezing. She still appeared unable to speak clearly and had marked conjugate gaze disturbance. Within 4 h, her symptoms had entirely resolved and she was discharged the following

morning with a provisional diagnosis of a 'dystonic' reaction to cyclizine. Interestingly, she had no recollection of any of the events in the recovery area.

In contrast to the case report published, this would appear to be a case where cyclizine appears to have precipitated a dystonic reaction in an individual with no previous neurological history. As King *et al.* noted, procyclidine had minimal effect, and it is more likely that supportive care and withdrawal of the drug led to recovery.

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Problems performing a sciatic nerve block in an amputee

We would like to suggest a possible solution to the problem identified by your correspondents (Lok & Kirk. *Anaesthesia* 2003; **58**: 289–90), namely performing a sciatic nerve block in an amputee. In this setting we would propose doing a parasacral sciatic nerve (actually a sacral plexus) block as described by Mansour [1]. With this proximal approach, it would still be possible to elicit hamstring muscle contractions as an end point with nerve stimulation in an anaesthetised patient. Depending on the extent of the ischaemia, it may not have been possible to elicit foot contractions, even if the patient was awake. There are other advantages [2] using the parasacral approach. It has a high success rate because the bony landmarks are easily identified, is an easy block to perform, and the obturator nerve is also blocked in a high percentage of cases, which may be desirable for an above knee amputation. Lastly, a catheter can also be threaded and tunnelled for continuous infusions and pain relief.

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Difficult central venous access – another role for the portable Doppler device (Dopplex®)

I would like to bring to your attention a difficult venous access in a 43-year-old short obese woman with Down's syndrome. She presented at the accident and emergency room with a history of cough, shortness of breath and feeling unwell for 2 weeks. On examination, she was 147 cm tall with a body mass index of 35 kg.m⁻². She was tachycardic with heart rate of 160 beat.min⁻¹ and hypotensive. Her ECG showed supraventricular tachycardia. Following initial assessment and resuscitation by the medical team, adenosine up to a total of 24 mg was administered to control her heart rate. Her only venous access was an 18-gauge cannula in her left antecubital fossa, which had an infusion of amiodarone running. Venous access was difficult, and attempts at central venous cannulation (for fluids and other medications) using anatomical landmarks failed. Owing to increasing patient discomfort and increased risks of potential chest complications, further attempts at cannulation of central veins in the neck and chest were abandoned for a femoral access. Palpation of the femoral arteries proved difficult. A SiteRite ultrasound device, which would have been ideal, was unavailable but a mini Dopplex (Huntleigh Healthcare Model D900), commonly used in surgical wards to assess blood flow in limbs, was used to locate and delineate the left femoral artery. The left femoral vein, which is medial to the artery, was subsequently cannulated and a quadruple central venous catheter inserted without complications.

Difficult venous access is not an uncommon problem and anaesthetists

are often called to assist in gaining venous access. In most situations, the problem is easily resolved but there are times when it is tricky and central venous cannulation may be necessary. Recently, the Tricky Vein Society (TVS) was inaugurated to raise awareness of this problem [1]. Our patient had not only tricky peripheral veins but also difficult central access. In a recent review, ultrasound-guided central venous catheterisation was recommended to minimise the risk of complications [2]. Unfortunately, not all intensive and coronary care units have this device and most patients requiring central venous catheters have them inserted blindly. Obviously, there are patients for whom a blind approach may be associated with higher complication rates, such as difficult surface landmarks (obese patients, local swelling), previous difficulties, previous complications (arterial puncture, pneumothorax, nerve injury), limited sites, uncorrected coagulopathy, inability to tolerate supine position and vascular abnormality. Although Doppler devices are not ideal, they can be used to enhance the surface-landmark-based techniques, especially for cannulation of femoral veins. It may also lend itself to use in the cannulation of internal jugular veins. These devices are portable, relatively inexpensive and readily available in most clinical areas.

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Anaesthetised for evacuation of retained products of conception; delivered a baby instead

A 16-year-old girl presented to our A & E department complaining of abdom-

inal pain and vaginal bleeding for about 24 h. The pain was a constant dull ache, and was associated with nausea and vomiting. She gave a history of 3 months amenorrhoea. The Gynaecology senior house officer (SHO) examined her, confirmed a 12-week parturient uterus, bleeding per vagina and diagnosed a miscarriage. She was booked on the emergency operating theatre list for evacuation of retained products of conception (ERPC).

During pre-operative assessment, the anaesthetic SHO was informed by the patient about the 3-month pregnancy and her presenting symptoms. There was no other significant past medical history. On examination, she was fully conscious, and appeared mildly dehydrated. Haemodynamically, she was stable with heart rate of 92 beat.min⁻¹, and blood pressure of 128/84 mmHg. Her chest was clear and airway assessment was normal.

Her investigations revealed haemoglobin of 11.1 g.dl⁻¹, white cell count of 9.7 and platelets 136 000. She was sickle cell negative. Blood was grouped and saved.

To correct the dehydration, she was started on Hartmann's solution 1 litre to be transfused, intravenously, over 4 h. Because of her nausea and vomiting, the decision was made to intubate the trachea and hence a rapid sequence induction was discussed with the patient.

Two hours later, anaesthesia was induced with fentanyl, propofol and succinylcholine, and her airway was secured with a cuffed orotracheal tube. When the abdomen was exposed, the actual fundal height seemed to be full term. The gynaecology SHO therefore called her registrar for assistance, who, after examining the patient, confirmed that she was term. Fetal well-being was assessed with Doppler, which confirmed maturity and normal heart sounds. At this stage, the patient was fully anaesthetised on controlled ventilation with oxygen, nitrous oxide and isoflurane. We could not proceed with a Caesarean section because of lack of consent. The patient's mother could not be traced for the consent either. The decision was taken to wake her up and proceed with labour and possible delivery in labour ward.

A nasogastric tube was inserted and about 100 ml of acidic fluid was aspirated. Neuromuscular blockade was reversed and the patient was extubated on her side when awake. The gynaecology team left for the labour ward to organise the delivery. The labour ward was 15 min trolley drive away. The gynaecology registrar was informed and he hurried back to the recovery room with the necessary delivery kit. However, on examination, she was not fully dilated. So, she was wheeled down to labour ward with an anaesthetist, paediatrician, an obstetrician and nurses in tow.

She delivered within 5 min of arrival in the deliver suite. A 1706-g live female baby was born with an estimated age of 30–32 weeks and Apgar score of 8. The baby was taken to the special care unit though she did not require any intervention and is making a good progress till date.

We feel the following issues need to be highlighted.

1 The diagnosis of 12-week pregnancy was made by a junior gynaecology SHO and the patient scheduled for ERPC prior to examination by a senior gynaecologist. As anaesthetists, we would not be expected to confirm the fundal height but it would be appropriate to check if an experienced gynaecologist has examined the patient and to confirm the exact duration of pregnancy. Although an ultrasound scan confirming fetal age would be helpful, most gynaecologists do not insist on one prior to an ERPC. Burnhill & Armstead [1] reported on their experience with over 7000 first trimester abortions and concluded that routine use of ultrasound 'when gestational age is unclear' is an essential measure in reducing the incidence of abortion complications. Also, on the basis of one mortality in the triennium 1994–96, the Confidential Enquiry to Maternal Death (CEMD) Report recommends: 'Ideally, all women should undergo ultrasound examination before termination of pregnancy to establish gestational age, viability and site.' [2].

2 It was fortuitous that the patient gave a history of nausea and vomiting and so a decision to perform a rapid sequence

induction and intubation were made. The consequence could have been disastrous if this had not been done and the patient had regurgitated and aspirated.

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Pulse oximeter ear probe

We have recently had to deal with a malfunctioned pulse oximeter probe. This probe was placed on one of the patient's fingers, but his hands were aligned to his body and therefore not accessible during surgery. Unfortunately, there was no pulse oximeter ear probe available. We therefore took



Figure 9 Probe secured to earlobe by means of a clothes peg.

a Nellcor Oxiband pulse oximeter probe (Nellcor, Pleasanton, CA, USA), normally used with single-use adhesive band, and attached it to the patient's ear lobe with a clothes peg. The clothes peg was applied to the part connecting the light-emitting unit and the light sensor unit. No direct pressure was applied to the ear lobe but there was enough indirect pressure to hold the probe on

the patient's earlobe. The probe produced an excellent signal for the rest of the surgery and at the end of surgery no signs of pressure were detected on the patient's ear lobe (Fig. 9).

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