Anaesthesia, 2001, **56**, pages 82–97

Correspondence

Anaesthesia without induction rooms

We read with interest the recent editorial: 'For the times they are a changing – or are they?' (Harmer. *Anaesthesia* 2000; **55**: 735–6), with regard to the continued use of anaesthetic rooms and we would like to make the following comments:

In 1993, following Meyer-Witting and Wilkinson's editorial [1], our clinical director asked us to review the use of anaesthetic rooms at the Ipswich Hospital and suggested a trial period of in-theatre inductions. We planned to establish a practice which would comply fully with the monitoring recommendations made by the Association of Anaesthetists in July 1988 [2], meet the concerns raised in the 3rd NCEPOD report of 1993 [3], and promote optimal patient safety during induction. The proposed trial was, as you predicted, met with widespread scepticism.

Surgical, anaesthetic and nursing staff had to be convinced that the benefits of in-theatre induction would outweigh the disadvantages. Several surgical colleagues anticipated an increase in turnover time between cases. Anaesthetists were more concerned with the loss of a quiet environment for anaesthetic induction and the potential for heightening patient anxiety. Theatre nursing staff were worried by the prospect of having to prepare instruments with a patient present in theatre. Paediatric nurses were concerned about parents not being able to come into theatre for induction.

It has frequently been suggested that time is saved by allowing junior anaesthetists to induce patients in the anaesthetic room during the conclusion of the previous case. We have always tried to teach our trainees, rather than using them as 'an extra pair of hands'. The change in practice was therefore unlikely to delay lists appreciably. Despite this, the general feeling was that the introduction of routine in-theatre induction would be detrimental to all. There were, however, a few enthusiasts prepared to undertake a timing and feasibility study in a limited number of theatres.

The results of this showed that there was an insignificant delay to the list with in-theatre induction, that patients found it perfectly acceptable, that the nursing staff were able to lay up instruments and that parents could be accommodated in theatre while their children underwent induction of anaesthesia. In view of the preliminary study's favourable findings, a working party, consisting of anaesthetists, a surgeon and senior nursing theatre staff, then carefully planned the transition to in-theatre induction, which was phased into all our theatre suites over a 9-month period. This gradual introduction enabled us to address the preconceptions of all the staff concerned, as outlined above.

What of the advantages? In 1993, anaesthesia was induced in our anaesthetic rooms with minimal monitoring – an oscillotonometer and a pulse oximeter. Our options were to purchase 15 sets of monitors duplicating the theatre monitors, devise a mobile monitoring trolley, or move into theatres for inductions. We opted for the third. As a direct result of not having to purchase monitors for the anaesthetic room, we could afford to improve our theatre monitoring equipment (the present trend towards anaesthetic workstations with integrated monitors would also make equipping anaesthetic rooms prohibitively expensive). Not only are patients monitored *ab initio*, but also we avoid the movement of an unmonitored, unventilated, disconnected anaesthetised patient between the anaesthetic room and theatre, described as 'clumsy and ill-conceived' by D. Brahams, Barrister-at Law, in his commentary on an accidental anaesthetic death [4].

We consider in-theatre induction with uninterrupted monitoring to be better for training, and to comply with the guidelines for basic specialist training published by the RCA [5]. It has been our standard practice for the last six years and has passed the test of time.

Would we get rid of the anaesthetic room? No, we still need a room for preparation prior to theatre. BP cuffs and ECG monitoring electrodes are applied, intravenous cannulation performed and some regional anaesthetic techniques undertaken. We still have a mobile machine with full monitoring available in each theatre suite for those cases where induction in the anaesthetic room may be more appropriate, but this is rarely used.

The continuing minor problem we encounter is ensuring a quiet environment at the time of induction. Teaching new nursing and surgical staff is therefore required and the re-education of existing staff is also important. The practice of in-theatre induction for high-risk patients is widespread, because

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it is the safest option. Why not extend this to all patients? Our sceptics have been convinced and would not go back to the former use of the anaesthetic room. Try it – you might like it, or perhaps you feel that, as in the Guinness advert, 'I've never tried it because I don't like it'.

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Paediatric intensive care transfers

We write in reply to the recent correspondence (Griffiths and Smith. *Anaesthesia* 2000; **55**: 610) that raised concerns about the safe conduct of paediatric intensive care retrievals. They highlighted the case of an 18-month-old child with epiglottitis and the difficulties surrounding his re-intubation while under the care of the Paediatric Intensive Care Team despatched to pick him up. Whilst this case graphically illustrates the value of a well trained and experienced senior anaesthetist in securing a compromised airway, the authors draw other conclusions, which we feel are unwarranted.

The 1997 Troop report stated quite clearly that children with single system failure requiring intubation do not necessarily need to be transferred to a Paediatric Intensive Care Unit (PICU) if the admitting hospital and their ICU meet certain predefined standards [1]. In these circumstances, common sense should prevail; children who are expected to make a rapid recovery, say within 24 h, need not be transferred, especially if the transfer distance is large. For such children, ongoing intensive care may be undertaken by the DGH clinicians, who should liaise with their lead centre PICU. However, on a cautious note, it must be remembered that although many senior clinicians in referring hospitals may feel confident of their ability to deal with the ongoing management of children in primarily adult ICUs, this confidence is not always shared by other staff who may feel exposed in terms of training, experience and professional liabilities.

If the standards laid down in the Troop report are not met, or if the child continues to deteriorate, then, once stabilised, the child should be transferred to a PICU. It has never been suggested that transfers take place 'when physiology is most compromised'; indeed, this goes against the whole philosophy of transferring children safely. Full resuscitation and maximal stabilisation should be achieved by the referring hospital and subsequently by the retrieval team prior to any child being moved. Well-documented studies have shown that children may be moved safely without physiological deterioration [2, 3]. Moreover, the suggestion that clinicians in DGHs be encouraged to undertake the first 24–48 h of intensive care, only to call a PICU when they get into difficulty, has been generally felt to be both inappropriate and unsafe [4, 5].

The issue of transport personnel and their ability to intubate children with upper airway obstruction is more difficult. All consultant paediatric intensivists should have training in the management of acute paediatric airway obstruction, as is stipulated in the requirements of the Intercollegiate Committee for Training in Paediatric Intensive Care Medicine. However, even for paediatric anaesthetists, who perform numerous inhalational inductions each day, the inhalational induction of a child with upper airway obstruction is a relatively rare and challenging event. In our opinion it seems totally inappropriate that the paediatricians amongst us, even those with some previous anaesthetic experience, should attempt this technique, either in our institution or while out with the retrieval team.

Thus, when organising the retrieval of such children, one must consider carefully who is going to perform the intubation. To a large extent this will depend upon the skills available at the referral hospital, the degree of urgency involved and the transfer distance. For the most part, in our region, this means that the intubation will be performed by anaesthetists at the referral hospital. With reference to the notion of 'deskilling', children will continue to require all the skills of the DGH clinicians with regards to resuscitation, stabilisation and initiation of specific management. The retrieval service must be seen as complementary to those skills.

When the complexity of the case demands, we send a consultant intensivist with the retrieval team and, when the referral involves a difficult airway, this intensivist is usually a paediatric anaesthetist. All lead-centre PICUs should eventually be able to achieve this standard, so long as they maintain a good balance between senior paediatric and anaesthetic staff.

In summary, the recommendations of the Troop report have been formulated in the best interests of critically ill children. In the final analysis, the safe management of a child with upper airway obstruction, or any critically ill child for that matter, depends on teamwork, close co-operation between the referring clinicians and the PICU team, and a good deal of common sense.

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A reply

Thank you for the letter by Murphy *et al.* regarding Paediatric Intensive Care Transfers. We agree with all the points that have been made but would like to add some further discussion.

The information regarding single organ system failure has not been disseminated widely and the reaction over the last 4 or 5 years has been to refer to a specialist unit as soon as a child is unwell. We suggest that a national audit of sick children, their pathology and more importantly the geography needs to be carried out urgently to target resources more efficiently. We agree with Murphy *et al.* that there should be an anaesthetist present at all transfers involving a difficult airway; this must be mandatory.

Finally, there must also be a rethink on the training requirements for DGH consultants and also their need to be updated regularly on paediatric emergencies. Those in the front line are often not paediatric specialists but generalists, and resources must be made available to ensure that any hospital with a significant emergency workload can cope with all paediatric emergencies.

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I want to be an anaesthetist

I was glad to see that the conclusion reached by Webster was that we as anaesthetists should not become peri-operative physicians (Webster. *Anaesthesia* 2000; **55**: 839–40). The point that I would like to make is that I do not want to become a peri-operative physician, I want to be an anaesthetist. I trained in South Africa where surgeons are excellent at looking after their patients and they fulfil their role as peri-operative physicians in an exemplary manner. Before the patients are transferred to theatre, their postoperative fluids and analgesia are already prescribed by the junior surgical trainees. The reason for their ability to look after their patients is that they have been taught by the senior surgical staff who see this as an integral part of being a surgeon. Anaesthetists are said to be better at fluid management in this country and therefore often called for simple advice. There is no reason why surgical trainees should not be taught fluid therapy as part of their training. In fact I think that it should be a part of undergraduate education. As anaesthetists, we see only a small proportion of surgical patients. I wonder how many more are incorrectly managed while the surgical seniors make themselves unavailable.

Fluids are just one aspect. I feel that if we do become more involved in non-anaesthetic peri-operative care, those who should be doing it will become worse at their jobs. Surgeons will become further deskilled and in the end patient care will be compromised. Six months of medicine should perhaps be included in surgical rotations. The need for high-dependency units may decrease if undergraduate training were to be improved and surgical trainees and their consultants made to realise that being a surgeon involves more than just operating. I want to be an anaesthetist, not somebody who does other people's work because they do not want to.

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Pre-anaesthesia assessment clinics

Dr Davies suggests that the cost effectiveness of the pre-anaesthetic clinic (PAAC) system in Australia depends on avoiding overtime payments to anaesthetists at penalty rates (Davies. *Anaesthesia* 2000; **55**: 812–13). This is an oversimplification which I feel should be corrected. The financial imperative, at least in our hospital, came from a wish to save money by increasing the proportion of patients admitted on the day of surgery. Pre-admission clinics were a cost-generating solution for the anaesthetic department to a cost-saving strategy by the surgical division of the hospital. Furthermore, overtime payments were not avoided as many anaesthetists now start work earlier in order to make their own assessment of their patients.

The Perioperative system as originally described by Kerridge [1] and used in a modified form at our institution is a co-ordinated approach to the management of elective surgery. The financial benefits accrue to the whole institution and are largely the result of efficient bed utilisation rather than the possible reduction in anaesthetists' overtime payments.

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Reference

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Anaesthesia clinics: the Canadian experience

We would like to comment on the recent editorials and correspondence regarding anaesthesia consultation clinics [1–6]. As British trained anaesthetists working in Canada we would like to submit our experiences of working in an anaesthesia clinic.

We agree with Professor Webster [2] that advantages are gained in enhanced training opportunities and perception of the physician anaesthetist's role but also feel that significant benefits come from reduction in late cancellations with a subsequent reduction in patient inconvenience and wasted operating time. At our institution, no patients admitted on the day of surgery were cancelled due to inadequate pre-operative preparation in the latest 3-month period. During this time, 98% of elective surgical patients (total 3161 cases) arrived in hospital on the day of surgery and 77% were discharged the same day. We do not agree with Dr Davies' assertion that it will be difficult to show clinics to be cost effective [5]. At Toronto Western Hospital, the cost of an hour lost in the operating room is $CDN200 (£93) excluding the salaries of medical staff, and an overnight stay costs $CDN3000 (£1400). By combining 'same day-admit' for inpatient elective surgery with anaesthesia clinics, savings are made not only in reduced hotel costs but also from a reduction in wasted operating time.

The practice of admitting patients on the day of surgery offers little opportunity to ensure that patients with complex comorbidity are adequately investigated or counselled. Our experience, in line with the literature [7], is that the anaesthesia clinic plays a major role in reducing cancellations and gives more time for patient assessment. A consultant-led clinic can therefore lead to an improvement in quality as well as cost savings. We would share Dr Baines' concerns [6] if clinics were delegated to unsupervised trainees. At Toronto Western, all consultations are undertaken by, or with direct supervision from, fully trained anaesthetists.

An argument against clinics is that individual anaesthetists may feel that pre-operative investigation or counselling differs from that which they themselves would recommend. In our experience, open dialogue with colleagues leads to an improved awareness of other clinical viewpoints and can be educational. We find that the presence of the anaesthesia clinic encourages teamwork in this department. Having seen a patient where a complex anaesthesia plan is required, a note is made and stored in a file of difficult patients. This file is of use both to the anaesthetist in charge of the case and to trainees, both to direct them to challenging cases, and for case study teaching. Clearly the anaesthetist giving the anaesthetic will also wish to see the patient on the day of surgery. Such a visit is considerably facilitated with all information to hand.

The anaesthesia clinic at Toronto Western has been a valuable tool for improving operating room efficiency whilst enhancing our public profile and offering valuable educational opportunities for anaesthesia trainees and consultants alike. We arrived in Toronto with a sceptical view of the benefit of anaesthesia clinics, but now see that there are clear advantages to be gained from a well-organised, well-staffed clinic.

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Brainstem death and ventilator trigger settings

We read with great interest the recent case report (Willatts and Drummond. *Anaesthesia* 2000; **55**: 676–84) and wish to share a similar experience.

A 22-year-old male was admitted to our ICU after a motor vehicle accident. On arrival, Glasgow Coma Scale (GCS) was 3 and both pupils were fixed and dilated. CT scan of the brain showed marked cerebral oedema and almost complete effacement of the quadrigeminal cisterns suggestive of some degree of coning.

Twelve hours after admission, he fulfilled all the criteria for brainstem death testing and was certified brain-dead by two qualified physicians on two separate occasions. In our country, kidney donation is compulsory by law under the Human Organ Transplant Act. Victims of motor vehicle accidents between 18 and 60 years of age who are certified brain death are considered for compulsory organ donation unless they had opted out of the Act previously. The transplant coordinator was accordingly notified. The patient's father also consented to donation of his son's heart, liver and cornea – organs not covered under the Act.

Just before the patient was transferred to the operating theatre, parents and relatives who had gathered by the bedside saw the patient turn his head and take a single 'spontaneous' breath. The patient was artificially ventilated with a Siemens Servo 300 ventilator using the synchronous intermittent mandatory ventilation (SIMV) plus pressure support. The ventilation pattern was set at 12 breath.min^{-1} with a tidal volume of 700 ml and PEEP of 5 cmH_2O. Pressure support was set at 10 cmH_2O with a triggering sensitivity of -1 cmH_2O below PEEP.

The relatives were understandably distressed and demanded another inde-

pendent opinion to confirm brain death. Organ procurement was put on hold and the on-duty neurologist was called to give a further opinion. A separate set of brainstem-death certification testing was done and again the results confirmed brainstem-death. The relatives were finally convinced and organs procurement proceeded 3 h later than was initially scheduled.

There have been several case reports of complex reflex movements and respiratory-like patterns in patients diagnosed brain death [1, 2]. Respiratory-like patterns are usually reported during the apnoea test.

Although we did not lose the opportunity for organ donation, we agree that the ventilatory set-up for patients diagnosed as brain-dead should be done with more care to avoid unnecessary distress and the possible loss of organs for transplantation. In our patient, the sensitivity setting of -1 cmH_2O below PEEP was perhaps too sensitive and may have caused the ventilator to trigger spontaneously. Perhaps placing a patient on a controlled mode of ventilation with a lower triggering sensitivity after the certification of brainstem death would prevent the appearance of any 'spontaneous' breaths.

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The Do Not Resuscitate order

In light of recent media attention and further to recent correspondence in *Anaesthesia* regarding the issue of the 'Do Not Resuscitate' (DNR) order, I would like to suggest that there still needs to be some differentiation between 'resuscitation' and what is management in the event of a cardiac arrest. There are many circumstances in which the resuscitation of a patient in terms of oxygen therapy and fluid optimisation may be wholly appropriate; however, in the event of a cardiac arrest, further invasive therapy may not be the right course of action.

For example, a patient admitted with an acute exacerbation of end stage COPD is still suitable for resuscitation in terms of medical management, but it may not be in the best interest of the patient to instigate further intensive care therapy.

To doctors trained in the era of ALS/ATLS/APLS/PALS guidelines, the phrase 'to resuscitate' has meaning far broader than purely the actions taken in the event of a cardiac arrest. A joint statement from the British Medical Association, the Resuscitation Council and the Royal College of Nursing emphasises that a DNR order applies solely to cardiopulmonary resuscitation (CPR). It carries on by saying that 'it should be made clear that all other treatment and care appropriate for the patient are not precluded and should not be influenced by a DNR decision'.

Should we not avoid ambiguity surrounding this highly emotive topic by abandoning the use of the phrase 'Do Not Resuscitate' and replacing it with the expression 'not for resuscitation in the event of a cardiorespiratory arrest' as recommended by the joint statement?

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A cautionary tale for percutaneous tracheostomy

We would like to report an incident, which occurred during the performance of a percutaneous tracheostomy under bronchoscopic guidance.

Following skin incision and blunt dissection, a Cooks 'rhino-horn' single dilator technique was used. This does not come prepacked with a tracheostomy; therefore, a size 9.0 Portex tube had been checked and lubricated at the start of the procedure. However, the operator was unable to insert this into the trachea and requested instead a size 8.0 tube. The size 8.0 Portex tube was rapidly checked and lubricated and inserted with ease into the trachea and the cuff inflated. However, on connecting the breathing circuit to the tracheostomy it was not possible to ventilate the patient adequately because of a large leak. This leak was noticed by the bronchoscopist as air coming up the oral tracheal tube, which had not yet been pulled completely back through the vocal cords. The position of the tracheostomy was then confirmed by bronchoscopy through the tracheostomy and appeared to be satisfactory. The pilot-tube balloon was palpated and seemed inflated to a satisfactory degree. The cuff was inspected from above via the tracheal tube and appeared intact, but surprisingly the carina was visualised as the bronchoscope passed with ease behind the cuff. The size 8.0 tracheostomy was therefore quickly removed and the original 9.0 passed into the trachea with a little force. Following cuff inflation, the lungs could now be ventilated adequately without a leak.

The 8.0 tracheostomy tube was inspected and found to have a cuff that was not only smaller than expected but which was also smaller than another 8.0 Portex tube taken from a similar looking box (Fig. 1). There were no markings on the tube itself to explain this; however, on the packet from which it came were the words 'profile cuff' compared with 'standard cuff' on the packet of the second tracheostomy tube used for comparison. Although this particular tube was 'in date', many others in the storeroom on the intensive care unit were not, and as there is probably no clinical indication for using these low-volume, high-pressure cuffed tubes, they were all consigned to the bin.

We recommend that other units inspect their stores for similar tubes, and that clinicians performing tracheostomies check the tracheostomy tube carefully prior to use.

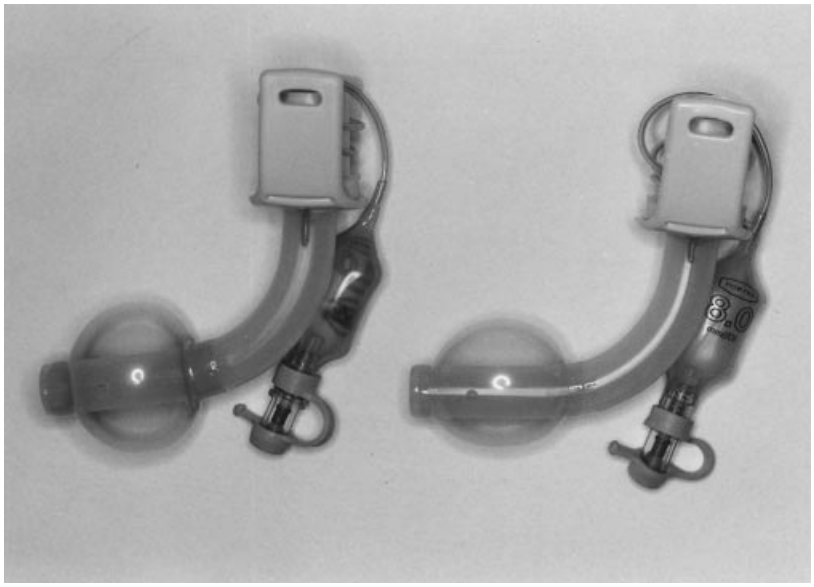

Fig. 1.

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Risk of infection from laryngeal mask airways

There is an important point that does not appear to have been raised by your correspondents, A. R. Wilkes and C. Deakin (Wilkes & Deakin. *Anaesthesia* 2000; **55**: 917) which I feel deserves to be made.

Irrespective of whether withdrawing the laryngeal mask airway inflated or deflated is associated with more or less bloody secretions (and is it not the traumatic insertion that causes the bleeding anyway?), surely the important issue should not be concern over cross-contamination from the anaesthetist's gloves but emphasising the point of changing gloves between patients and discarding blood-stained gloves at the first opportunity. In the days before universal precautions made the wearing of gloves mandatory, one washed one's hands between cases; woe betide any trainee anaesthetist at Great Ormond Street Hospital who transgressed this rule whilst under the tutelage of Dr Ed Battersby. The consequences were imprinted upon the memory for the rest of one's professional career.

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Sterilisation of laryngoscopes

Having recently reported a study of occult blood and microbial contamination of laryngoscopes following routine cleaning and disinfection [1], we were interested to read the letter by Drs Sacks and Carney regarding sterilisation of laryngoscopes (Sacks and Carney. *Anaesthesia* 2000; **55**: 700). Although they advocate the use of peroxyacetic acid and The Steris System™, they present no evidence for the validation of this procedure. We would refer them to a recent publication using this same technique to sterilise laryngoscope blades [2]. The blades were soaked in Haemosol®, mechanically washed and then sterilised using The Steris System™. The laryngoscope blades were then tested for occult blood using a three-stage phenolphthalein indicator test. Although none of the blades was visibly contaminated, 13/65 (20%) tested positive and the authors concluded that their procedures for cleaning, disinfection and sterilisation of laryngoscopes were not effective. We would agree with these conclusions and the results of this and our own study suggest that further work is required before a validated, effective method for decontaminating laryngoscopes can be recommended. The Medical Device Agency guidelines cited by Drs Sacks and Carney [3] are detailed and informative but make no specific mention of the sterilisation of anaesthetic laryngoscopes. Nor do they address the important issue of the storage of laryngoscopes, which are commonly left exposed to the risk of environmental contamination after cleaning. Numerous national associations (e.g. The British Society of Gastro-Enterology, The British Association of Urological Surgeons and the British Thoracic Society) have issued guidelines and protocols relevant to their specific practice. We think it timely for The Association of Anaesthetists to take a lead in formulating such policies for the decontamination, disinfection and sterilisation of laryngoscopes.

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Fig. 2.

Editorial comment
The Association of Anaesthetists of Great Britain and Ireland is in the process of setting up a working party to look at this issue.

Mechanical failure of the McCoy laryngoscope during a difficult intubation

I should like to report the mechanical failure of a McCoy laryngoscope blade [1] during the course of what was suspected to be a difficult intubation.

The patient, a 58-year-old man with long standing asthma controlled on salbutamol and beclomethasone inhalers, was scheduled for microlaryngoscopy and biopsy of the vocal cords, followed by a right mastoidectomy for cholesteatoma. An anaesthetic for a microlaryngoscopy 5 years previously had revealed a Cormack and Lehane Grade III laryngoscopy [2]. The patient was systemically well and examination of the airway revealed mouth opening of two and a half fingers with good jaw protrusion and neck mobility. He was judged to be a Mallampati II on inspection of the pharynx [3]. He was premedicated with temazepam 20 mg and prepared for theatre.

Following adequate pre-oxygenation, anaesthesia was induced using propofol. After establishing that manual ventilation using a mask and oral airway was easy, the patient was paralysed using atracurium 0.5 $mg.kg^{-1}$. A McCoy laryngoscope with size 4 blade was used. Laryngoscopy was found to be Cormack and Lehane Grade II once pressure had been exerted anteriorly on the neck. Operation of the lever arm of the laryngoscope only slightly improved this view. Tracheal intubation with a 7.0-mm microlaryngeal tube was achieved without incident. The microlaryngoscopy was performed in theatre, during which a small nodule was biopsied.

The proposed technique for the subsequent mastoidectomy was to exchange the microlaryngeal tube for a larger tracheal tube as a blind technique using a gum elastic bougie. The patient's lungs were ventilated with 100% oxygen, paralysis was confirmed using a nerve stimulator and the pharynx was suctioned. The bougie was introduced into the microlaryngeal tube, which was then removed. Difficulty was subsequently encountered when a 9.0-mm tracheal tube was passed over the bougie, the tube sticking at the level of the larynx. After two attempts, and prior to attempting with a smaller tracheal tube, I became unsure about the position of the bougie in the larynx. The bougie and tube were removed in order to ventilate the patient with a mask and airway, the plan being to re-intubate the trachea in the conventional way.

The same McCoy laryngoscope was again used. After introduction of the blade into the mouth the lever mechanism failed to operate. Despite this, laryngoscopy was again Grade II and intubation was achieved with the help of a gum elastic bougie. An 8.0-mm tracheal tube passed through the vocal cords without any difficulty. Closer inspection of the blade showed that

Fig. 3.

the braze that connects the proximal pivot of the lever mechanism to the blade itself had broken (Fig. 2) preventing leverage of the tip (Fig. 3). Failure of the distal hinge mechanism has been reported previously [4], but I believe that this is the first report of failure of the proximal end.

I believe the McCoy laryngoscope to be an important piece of equipment, particularly when a difficult laryngoscopy is anticipated. The flexible tip may convert a Grade III laryngoscopy to a Grade II, thereby allowing easy intubation under direct vision [5]. Fortunately, in this case this facility was not necessary. Despite the damage, the laryngoscope was able to function as a rigid-ended blade would have done, thereby allowing a reasonable view of the glottis.

The faulty laryngoscope, supplied by Penlon, was kept in a receiver alongside the patient between the first and second intubation attempts. It is stored between cases on the difficult intubation trolley inside the rigid box as supplied by the manufacturer. This box is lined by foam and has special cut-outs to hold and protect the laryngoscope and blade. To my knowledge, this laryngoscope had not previously been damaged or dropped and had been cleaned in the standard way as recommended by the manufacturer.

The lever mechanism of the McCoy laryngoscope is operated by the thumb whilst clenching of the fingers around the laryngoscope handle. This grasp allows the potential for considerable force to be exerted on the mechanism, particularly during those anxious few seconds before the larynx is seen. While I do not believe that undue force was exerted during this instance, I would be interested to hear if any anaesthetist has experienced a similar problem, and if any modification in the manufacture of the laryngoscope could prevent such a failure.

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A reply

Thank you for giving us the opportunity to comment on the above failure. This is the only complaint of this type that Penlon has recorded on its complaints database and therefore we would be interested to learn from any other anaesthetists who have experienced a similar problem.

Following our inspection of the blade, we believe that this is an isolated component fault. However, knowing that considerable force can be exerted on the lever mechanism, the design of the hinge pin has been improved to prevent a recurrence of this problem. The design of the blade is such that failure of the hinge pin results in the blade tip resting back against the tongue guard, enabling the blade to be used as a conventional Macintosh blade and thus minimising any risk of injury to the patient.

Penlon is committed to the quality of its products and has already replaced the faulty blade under the laryngoscope lifetime warranty policy.

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Capnography and errors with gas sampling

Drs Brownlow and Bell are correct in their explanation of the abnormal capnograph obtained with a cracked capnograph sample port in a 14-year-old patient undergoing fixation of a supracondylar fracture (Brownlow and Bell. *Anaesthesia* 2000; **55**: 832–3). When a leak exists at the juncture between the gas sampling line and the gas analyser, an artificially low exhaled gas alveolar plateau is due to entrainment of room air and the following peak is due to pressurising the sampling

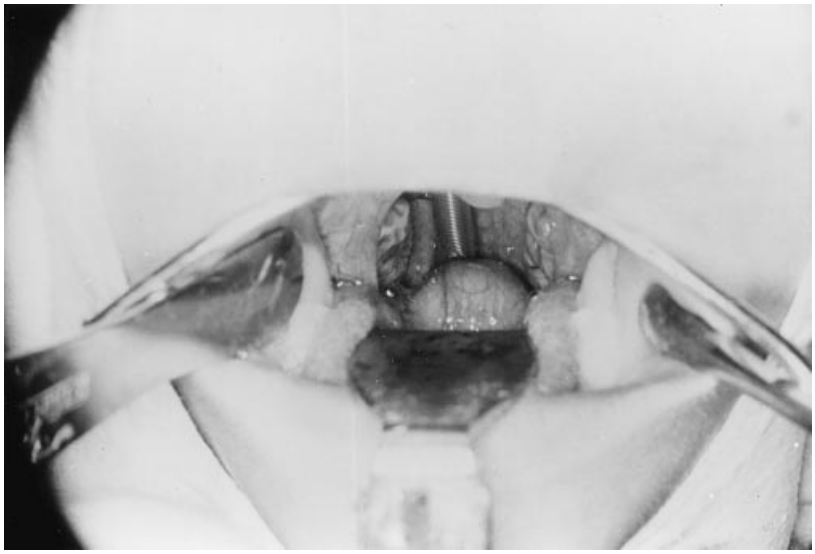

Fig. 4.

line with the next positive pressure ventilation so that the analyser receives true end-tidal gas.

The purpose of this communication is to point out that this phenomenon has been well studied in terms of the size of the leak and the next positive pressure ventilation tidal volume [1]. As might be intuitively obvious, the larger the leak, the lower the plateau and the larger the positive pressure tidal volume, the greater the following peak.

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Reference

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The two different-sized fibreoptic bronchoscope method in the management of a difficult paediatric airway

A 6-year-old boy weighing 20 kg with a vallecular cyst was scheduled for surgical excision. The CT scan revealed a 1.5-cm × 2.5-cm sized round, soft and cystic mass on the base of the tongue. The patient received an intramuscular injection of glycopyrronium 80 μg (4 $\mu g.kg^{-1}$) 30 min before surgery.

In the operating room, after obtaining cooperation, we evaluated the feasibility of conventional orotracheal intubation using direct laryngoscopy. However, we decided against this because we feared total airway obstruction by the cyst. We felt that complete airway obstruction during induction with an inhalational anaesthetic was also likely.

Following intravenous injection of ketamine 10 mg, 4% lidocaine was sprayed into the right nostril. As a 5-mm OD uncuffed wire-reinforced tracheal tube (Mallinckrodt, Ireland) could not be advanced through the nostrils, a well-lubricated 4-mm OD uncuffed tube was introduced through the right nostril and positioned in the nasopharynx. A 60-cm Olympus LF-P fibrescope with an external diameter of 2.2 mm was threaded inside the tube. The fibrescope was advanced, the vocal cords identified and their surrounding structures identified as intact. The tracheal tube and fibrescope were withdrawn. Ketamine 10 mg was injected intravenously again. As the 60-cm Olympus LF-2 fibrescope with an external diameter 3.8-mm could not be passed through the 4-mm wire-reinforced tracheal tube, it was directly advanced into the nasopharynx through the right nostril without tube placement. Topical anaesthesia of the larynx was achieved by spraying 1 ml lidocaine 2% through the biopsy channel of the fibrescope. After 30 s, it was passed into the trachea and again sprayed with 1 ml lidocaine 2%. The patient coughed lightly and the fibrescope was withdrawn. A well-lubricated 4-mm uncuffed wire-reinforced tracheal tube was passed again through the right nostril. The ultra thin Olympus LF-P fibrescope was then threaded inside the tracheal tube, which passed through the vocal cords to the mid-trachea. The tracheal tube was slipped gently over the fibrescope and there was no discomfort to the patient. During the procedure, there was no increased salivation. It took 15 min to excise the mass (Fig. 4) under general anaesthesia with enflurane. The patient was discharged uneventfully the next day.

When fibreoptic intubation is attempted in the awake patient, either a superior laryngeal nerve block and transtracheal block [1] or the spray-as-you-go technique with local anaesthetics [2] has been recommended to prevent laryngospasm and the cough reflex. As there is a difficulty performing these blocks in children, especially those less than 6 months old, the spray-as-you-go technique is best performed under sedation. However, there are no suction or biopsy channels in an ultra thin fibreoptic bronchoscope. As a solution, Kleeman *et al.* [3] used a different-sized bronchoscope method for difficult tracheal intubation in children under 30 months old. Following ketamine injection, topical anaesthesia of the larynx through the biopsy channel of the Olympus BF-3C4 fibrescope with an external diameter of 3.5 mm was obtained and followed by successful use of the over-the-scope method with the Olympus PF-27 L FOB (external diameter 2.7 mm) through a nostril. Although there was no problem in advancing the tube into the trachea in their case, severe coughing or laryngospasm may occur when

the tracheal tube passes through the vocal cords if the trachea is not anaesthetised. To spray the small amount of local anaesthetic into the trachea through the working channel of the FOB may provide greater inhibition of reflex.

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Cremasteric reflex is not a useful indicator of spinal anaesthesia in anaesthetised children

In a previous study, we reported that disappearance of the cremasteric reflex is a useful indicator of effective spinal anaesthesia in adults [1]. Subsequently, we studied if this test was useful in children. After obtaining approval from the institutional ethics committee and informed consent from patients' parents, we studied 60 children, aged 2–5 years, to examine the presence or absence of the cremasteric reflex, before and after induction of anaesthesia (using sevoflurane) and after caudal anaesthesia. The reflex was always present before induction of general anaesthesia, but it always disappeared after induction of general anaesthesia but *before* caudal anaesthesia. The reflex also disappeared after induction of anaesthesia using isoflurane ($n = 10$) or halothane ($n = 5$). Therefore, in anaesthetised children, the cremasteric reflex test is not useful in confirming effective spinal anaesthesia. Whether this is also the case in anaesthetised adults or in awake children has not been elucidated.

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Viagra®: are anaesthetists rising to the challenge?

September 15th 1998 was the dawn of yet another drug to the United Kingdom – the wonder drug, Viagra. While its obvious benefits are well known to all, the lesser-known effects may have gone unnoticed although these may have significant implications for the anaesthetist.

The addition of Viagra (sildenafil citrate) to the dispensing repertoire for men with erectile dysfunction was first discovered by accident during testing as an agent for treating heart disease. However, its appeal soon became universal. Since its launch in the United States in March 1998, Viagra has become the fastest ever selling drug [1]. Prescriptions for Viagra in the US peaked at around 260 000 a week and are currently around 160 000 a week. Its appeal is far reaching and, in addition to the estimated 140 million impotent men worldwide, it is fast becoming a popular recreational drug and being used increasingly by woman [2].

Sildenafil is a potent and selective inhibitor of phosphodiesterase type 5 (PDE5). Sildenafil is 4000–10 000 times more selective for PDE5 compared to the 1–4 and 7 isoforms and 10 times more selective compared to PDE6. The PDE5 isoform is responsible for breaking down cyclic guanosine monophosphate (cGMP) in the corpus cavernosum. In response to sexual arousal and stimulation, nitric oxide (NO) is produced in cavernosal tissues. NO stimulates the secretion of the secondary messenger cGMP, which relaxes the smooth muscle causing local vasodilatation and swelling of the corpora as it fills with blood. Inhibition of PDE5 by sildenafil causes accumulation of cGMP, resulting in a 35% increase in cGMP levels [3] with observed benefits.

A terminal half-life of 4–6 h safeguards sildenafil from influencing an anaesthetist's elective list, but it may have implications in the emergency setting. The pharmacological profile of sildenafil is summarised in Table 1. The cardiovascular and gastrointestinal effects are those most likely to affect anaesthetic practice and may generally be explained by its mechanism of action [4]. NO and cGMP are important mediators of smooth muscle relaxation throughout the body and PDE5 the major enzyme degrading cGMP in a variety of smooth muscles, including those of the vasculature and gastrointestinal tract.

Dilatation of systemic blood vessels causes a transient drop in blood pressure (8.4 mmHg systolic and 5.5 mmHg diastolic) in healthy volunteers following a 100-mg tablet [5]. Nitrates potentiate this transient hypotension and, following results from phase 1 studies, their concurrent use is contraindicated [5]. However, sildenafil is often used recreationally in combination with amyl nitrate ('poppers') and other illegal drugs without apparent adverse effects [2]. Furthermore, sildenafil has not been found to potentiate the hypotensive effects of alcohol in healthy volunteers. To date there are no studies of the effects of sildenafil on the cardiovasular system during general anaesthesia but caution should be observed with the concomitant use of hypotensive agents.

Table 1 Sildenafil citrate: a brief synopsis

Presentation	25/50/100 mg blue diamond-shaped tablets
Dose	50 mg recommended (Maximum 100 mg OD)
Route of administration	Oral
Therapeutic indications	Erectile dysfunction
Mechanism of action	cGMP specific PDE_5 inhibitor
Pharmacodynamics	Median onset time 25 min
	Effect duration 4–5 h
Pharmacokinetics	Peak plasma concentration 60 min
	Bio availability 41%
	Volume of distribution 105 l
	Protein bound 96%
	Terminal half-life 4 h
	Total body clearance 41 $l.h^{-1}$
	Excreted 80% faeces/13%urine
	Active N-desmethyl metabolite
Contraindications	Known hypersensitivity
	Nitrates
	Severe hepatic impairment
	Hypotension (< 90/50 mmHg)
	Recent stroke or myocardial infarction
	Degenerative retinal disorders
Drug interactions	Nitrates
	Cytochrome inhibitors (cimetidine, erythromycin)
Side-effects	Headaches, flushing, nasal congestion, dyspepsia
	Altered vision
	Transient hypotension, dizziness
Overdose	Standard supportive measures
	Not cleared by renal dialysis

No ECG changes have been reported following sildenafil and phase 2/3 placebo-controlled studies showed no increased risk of myocardial infarction [6]. While many agree that sexual activity is the main cardiac risk factor, there are reports of myocardial infarction prior to exertion while taking Viagra [7]. This may possibly occur due to a coronary steal phenomenon.

Dyspepsia is experienced in 7% of users and is assumed to occur because of relaxation of the lower oesophageal sphincter [6]. Precautions to prevent reflux and regurgitation in the peri-operative period should therefore be taken.

Metabolism is primarily mediated by the cytochrome P450 isoenzymes – 3A4 (major route) and 2C9 (minor route). Drug interactions with cytochrome P450 inhibitors, e.g. ketoconazole, erythromycin and cimetidine, can be expected and a reduced dose of Viagra is recommended during concomitant therapy.

Many studies have shown that sildenafil is efficacious in its licensed use [8–10] and has relatively few serious adverse effects. Pooled safety data from 21 studies, totalling over 3700 men aged 18–87 years (equivalent to 1631 years of exposure), show no evidence of serious adverse effects attributable to sildenafil [6]. The most common side-effects are minor and include headaches, flushing, dizziness, dyspepsia, nasal congestion and transient disturbance of blue–green colour discrimination. The latter effect is due to some activity of sildenafil on PDE6, which is important for phototransduction in the retina. Up to 30% of participants experienced a side-effect, but the authors described these as transient and only 2% discontinued treatment as a result [8]. The US Food and Drug Administration (FDA) reported details of 69 deaths in people taking sildenafil between March and July 1998 during which 3.6 million prescriptions were dispensed [11]. However, the FDA has not found any need to take regulatory action and none of these reported deaths has clearly been linked to sildenafil or its concomitant use with nitrates [12]. Notwithstanding this, it is important to have an active programme to monitor the cumulative effects of long-term use, particularly with regard to effects on the retina [4].

Controversy and publicity will continue to surround Viagra and the government bodies and medical boards that debate its use [13, 14]. Sildenafil is an effective treatment for a highly prevalent and distressing disorder and it is inevitable that it will be taken by large numbers of men. This number is likely to increase with time, particularly if the drug becomes deregulated and sold over the counter [15]. It is inevitable that we will be seeing more of it in our practice.

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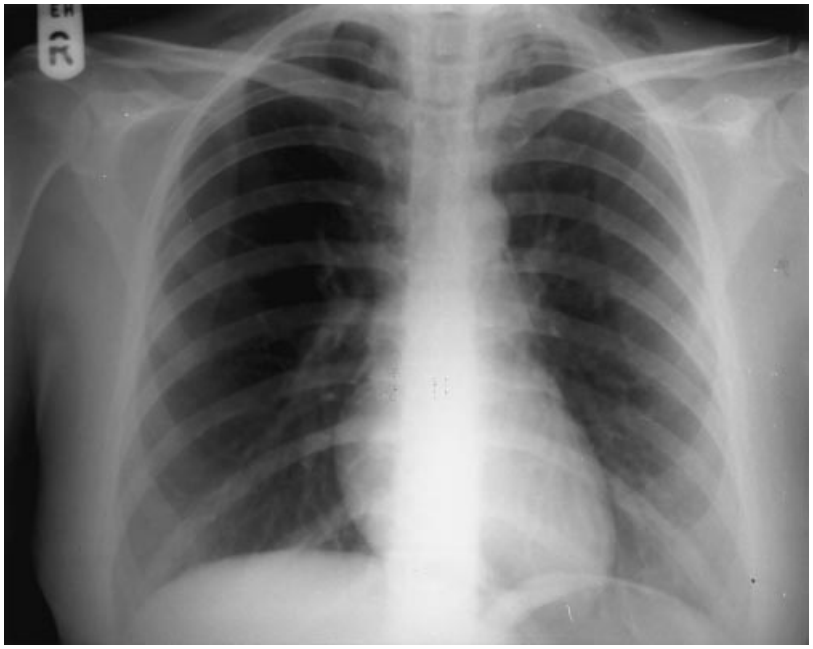

Figure 5 Antero-posterior chest X-ray showing subcutaneous cervical emphysema, pneumomediastinum and pneumopericardium.

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Spontaneous cervical and mediastinal emphysema following childbirth

A primiparous 36-year-old doctor was admitted in spontaneous labour at 37 weeks gestation. A non-smoker, she had a history of mild bronchial asthma. During her labour she used transcutaneous electrical nerve stimulation, and then Entonox, for pain relief. She vomited once during her labour. After a second stage of labour lasting 4 h, she had a spontaneous vaginal delivery of a 2.7-kg baby. A little more than an hour after delivery, she experienced some difficulty breathing with associated chest discomfort. She told her midwife that she thought she had cervical emphysema. Examination revealed crepitus over the neck, but was otherwise unremarkable. An anteroposterior chest X-ray (Fig. 5) showed a pneumopericardium, pneumomediastinum and subcutaneous emphysema, but no pneumothorax. A lateral chest X-ray showed air anteriorly in the mediastinum. A contrast swallow showed no evidence of oesophageal perforation. The patient was reassured and managed symptomatically with simple analgesics and oxygen. The subcutaneous emphysema had almost gone by the time she was discharged 48 h later. Repeat chest X-ray one month later showed complete resolution of the pneumopericardium, pneumomediastinum and subcutaneous emphysema.

Spontaneous subcutaneous cervical emphysema, with associated pneumomediastinum and pneumopericardium, following childbirth may occur as infrequently as one in 100 000 deliveries [1]. The emphysema arises from rupture of alveoli due to the high intra-alveolar pressures generated by Valsalva manoeuvres associated with bearing down and pushing during labour. It usually occurs during the second stage of labour and, as in this case, symptoms are often not noticed until after delivery. However, it may occur during the first stage of labour [2] or earlier in the pregnancy in association with hyperemesis gravidarum [3]. Whilst spontaneous pneumothorax during labour has been reported several times [4], pneumothorax has only rarely been found in association with subcutaneous cervical emphysema and pneumomediastinum [2]. Spontaneous cervical and mediastinal emphysema is commoner in primiparous mothers and was thought to be associated with prolonged labour and large babies, but Reeder reviewing 187 cases in the literature found the duration of labour and birth weight to be within normal limits [5]. If the cervical emphysema follows vomiting, oesophageal rupture needs to be excluded.

Spontaneous cervical and mediastinal emphysema is usually a self-limiting condition; therefore, observation, reassurance and symptomatic treatment with analgesia and oxygen are all that is needed in most cases. Only four maternal deaths have been reported, all before 1908 [5]. Whilst the majority of cases present after delivery, if the condition is diagnosed earlier in labour, delivery of the fetus using forceps or vacuum extraction to shorten the labour has been recommended [1, 2, 5]. The subcutaneous emphysema usually resolves within a few days and the X-ray changes within 2–3 weeks. Only two cases of re-occurrence of subcutaneous emphysema in a later pregnancy have been reported [5], both before 1900, and there is no reason for the management of subsequent pregnancies and labours to be altered.

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Unplanned administration of atropine, succinylcholine and lidocaine

Of late, there has been a trend to develop and implement a range of strategies to control rising drug expenditure [1]. This trend has occurred in all fields of medicine. Like all specialties, anaesthesia needs to examine carefully its clinical practice so that excessive costs and waste can be reduced without comprising patient care and safety [2].

We used to draw up succinylcholine, atropine and lidocaine before the start of each list in the 11 operating rooms of our hospital. These drugs were then readily available in an emergency. Unused drugs were discarded after 24 h. Usually all of these drugs were wasted each day at an approximate cost of 572 rupees per day or 143 000 rupees per year (approximately £1703 per year).

We conducted an audit to find out the incidence of unplanned administration of these drugs. Audit was for a period of 3 months. The indication for giving the drug, ASA status, age of patient and whether the drug was used in elective or emergency case were noted. During the 3-month period, 2231 operations were performed in most surgical subspecialties except cardiac surgery. At the end of this audit, we found two patients required succinylcholine for laryngospasm, nine patients received atropine for acute bradycardia and seven patients received lidocaine for premature ventricular contractions. This represented an incidence of 0.089% for succinylcholine, 0.40% for atropine and 0.31% for lidocaine. It was also noted that in none of these cases were the drugs given in a hurry. As a result of this audit, it was decided that these drugs did need not to be drawn up at the start of the operating list, the only exception being for paediatric surgery. It was also recommended that a box containing ampoules of succinylcholine, atropine and lidocaine together with syringes be kept on the anaesthetic machine so that these drugs are easily accessible in case of emergency. These drugs are checked at the start of each operating list as part of the anaesthetic machine check. This has led to substantial savings in anaesthesia expenditure.

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Reinforced epidural catheters

My attention was drawn to two recent letters (Scawn and Pennefather. *Anaesthesia* 2000; **55**: 304; Roddin and Dancey. *Anaesthesia* 2000; **55**: 831) recounting problems with kinking of epidural catheters. The former authors have described how securely fixing the epidural catheter to the skin seemed to result in kinking of the catheters in the subcutaneous tissue. The second letter also notes several instances of kinking of Portex epidural catheters at various sites between skin and epidural space.

I would like to draw attention to the Arrow FlexTip Plus – a reinforced epidural catheter with kink-resistant properties (Fig. 6). It is an open tip, single-orifice catheter constructed of a circumferential stainless steel coil impregnated in soft polyurethane. The coil-reinforced body imparts the firmness needed for insertion while providing resistance to kinking and collapsing. The soft distal tip and the flexibility of the catheter reduce the risk of paraesthesia and inadvertent penetration of the dura and epidural veins (probably due to reduced tip forces).

I had the opportunity to use this catheter in my previous department. Reviewing the literature (studies conducted mainly in parturients [1–3]) the incidence of paraesthesia is 2.16–2.7% compared with 35.5% with Portex, 15.16% with Kendall and 32.34% with Braun catheters. The reported incidence of epidural venous cannulation is 0–0.53% compared with 10% in Portex, 5.61% in Kendall and 4.68% Braun catheters. Banwell *et al*. in their study [1] described five instances when Portex catheters could not be threaded into the epidural space, while Arrow catheters were successfully passed in all five cases through the same needle in the same position.

In our experience, we found a higher incidence of venous cannulation (1 in 60, 1.6%) and difficulty in threading the catheter (4 in 60, 6.6%). However, this could be due to the small number of insertions and the initial inexperience with the new equipment.

Overall, this epidural catheter could be useful in obstetrics and long-term indwelling catheters where patency will be ensured. It may well be the solution to kinking of epidural catheters.

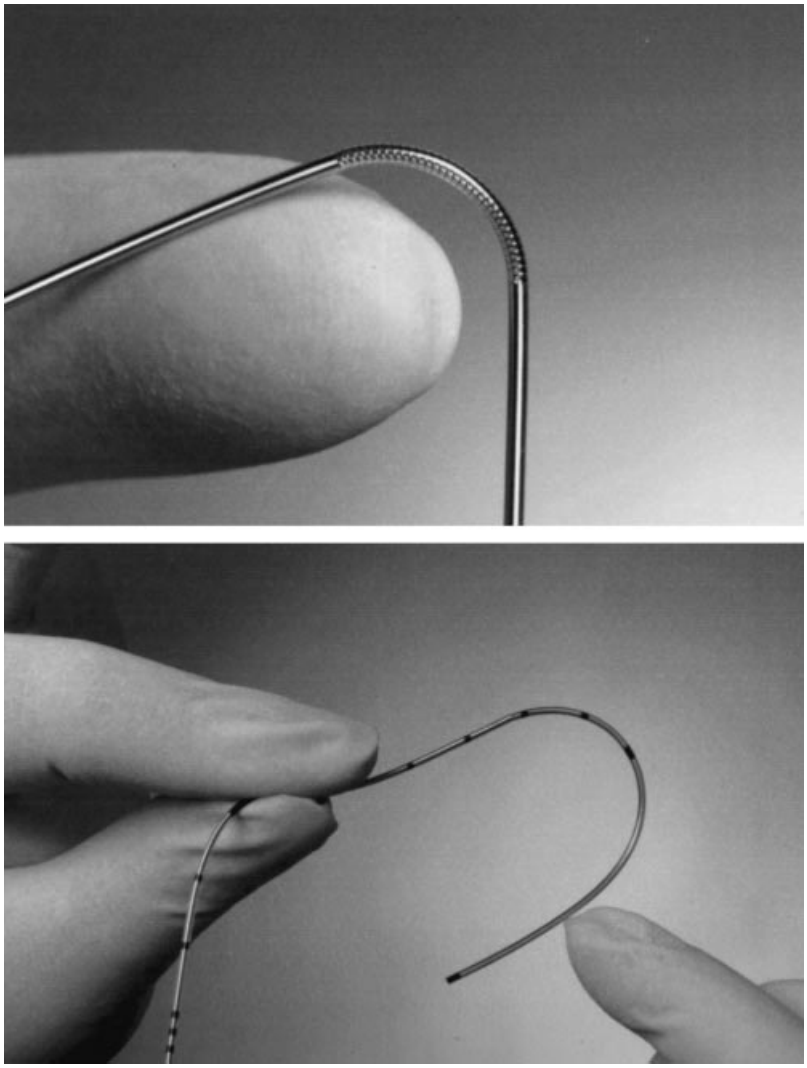

Fig. 6.

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Comparison of epidural catheter induced anaesthesia in parturients. *Regional Anaesthesia* 1995; **20** (Suppl.): 152.

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Spinal needle introducer hub detachment

Today, disposable regional block needles are made to a high standard. The incidence of needle breakage or hub/needle separation is exceedingly rare. I should like to report a needle/hub separation, which could have had serious consequences.

Spinal anaesthesia was required for a rather large (119 kg) parturient presenting for lower section Caesarean section (LSCS) for fetal distress. A 25G spinal needle (conical–eliliptic shaped) made by Polymedic was used. The patient was placed in the sitting position. Midline insertion of the introducer needle through the skin, supra spinal and intraspinal ligaments at $L_{3/4}$ was felt to be rather tight. Due to the patient's size, the introducer needle was introduced up to the hub. Subsequent insertion of the spinal needle and intrathecal injection were uncomplicated. On withdrawal of both needles, it was noted that the hub of the introducer had become detached from the introducer needle (see Fig. 7). This left approximately 8 mm of the introducer needle protruding from the skin, with the remaining 30 mm still embedded. Fortunately the needle could be removed by hand. The patient was then placed supine with 15° of left lateral tilt and, following testing of the block, the LSCS proceeded uneventfully.

The detachment of the plastic hub from the needle could have been due to several factors. Firstly, the spinal ligaments gripped the introducer needle tightly. Second, I removed both needles together with a short sharp tug. Third, the bond between hub and needle may have been defectively weak. Thus the shearing force generated by removal obviously overcame the hub/needle bond strength. As I do not normally test the hub/needle bond strength prior to insertion, it is impossible for me to comment on whether the bond strength was normal or defectively weak.

The consequences could have been worse. If the introducer needle had been inserted such that the hub was indenting the skin, as is the case sometimes when performing spinal anaesthesia for larger patients, then if detachment had occurred on withdrawal, the skin would have closed over the needle, making its removal impossible and the ability to place the patient supine for the LSCS questionable.

A *Medline* review 1967–2000 revealed only two other reported episodes of hub-detachment from spinal introducer needles. One was a plastic hub [1] and one a metal hub [2]. Several

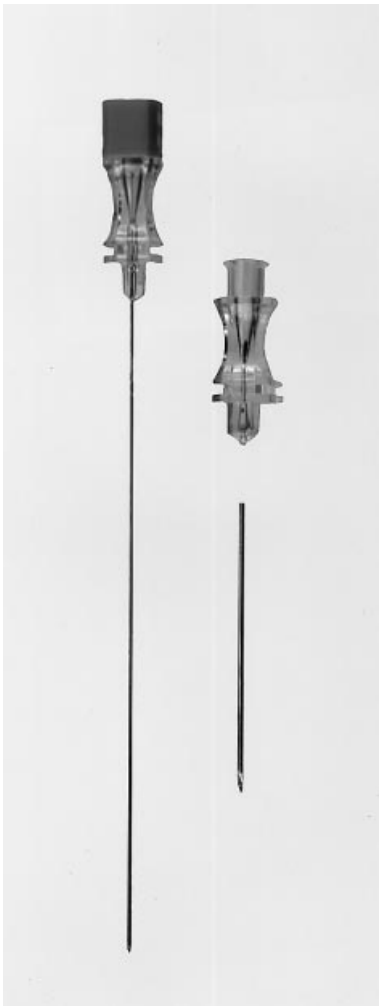

Fig. 7.

episodes of hub detachment from epidural needles have been reported [3–7], only two of these being plastic hubs [5, 6].

In the era of the reusable regional block needle, the metal hub was 'crimped' on to the needle. Breakage or detachment at the needle/hub junction was not uncommon due to the stresses imposed by bending and subsequent re-straightening. Reusable spinal or epidural needles used to have a security bead on their shafts near the hub. This was designed to limit the depth to which they could be inserted and if detachment did occur then the needle end would not disappear beneath the skin surface. Today the plastic hub is glued to the needle with epoxy. There is no longer a security bead, presumably as the incidence of detachment is so rare and the increased cost of manufacture.

Previous authors [1] have recommended that if the needle end should disappear beneath the skin then a second needle should be inserted along its track to aid subsequent retrieval. In this instance, the needle was instantly retrieved. Given the urgency of the LSCS, if the needle had disappeared it would have been necessary to prevent pressure over the mid lumbar region before placing the patient supine. This could have been facilitated by the use of bags of colloid/crystalloid placed cephaloid and caudal to $L_{3/4}$.

There are several recommendations I would make in the light of this incident.

1 The needle/hub bond strength should be tested prior to use by axial traction.

2 Avoid inserting the introducer needle such that it indents the skin. This may be avoided in larger patients by ensuring the availability of longer length spinal needles.

3 If the spinal ligaments grip the introducer needle tightly, then introducer withdrawal should be slow and controlled.

4 If the hub does become detached and the needle is no longer visible then mark the insertion site, thus making subsequent retrieval easier.

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A reply

Thank you for the opportunity to reply to Dr Smith's letter.

Unfortunately, Dr Smith has been unable to provide us with the needle batch number and we are unable to investigate the exact cause of the detachment. Detachment of the hub from the body of introducer needles is more common than is reported in the scientific literature. Usually, this does not cause the sort of problem highlighted by Dr Smith and therefore does not get reported. Epoxy glue, commonly used to fix hubs to needles, is gradually being replaced by monomere glues which are hardened by exposure to ultraviolet light and produce a stronger bond.

The thicker the needle, the greater the traction forces exerted on the hub–needle connection. Such forces are increased when the introducer needle is swivelled in order to change the direction of the spinal needle instead of withdrawing and redirecting the introducer needle. This is in contrast to the forces encountered by intravenous needles.

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False negative CVP with a normal waveform

Various causes of 'erroneous' central venous pressure (CVP) readings include malposition of cannula, blockage, zeroing error, concomitant infusion of fluids through the same line, undiagnosed pathological conditions, positive pressure ventilation and transducer faults [1, 2]. We report yet another well-known but less reported source of CVP error in the presence of an apparently good waveform.

A 65-year-old otherwise healthy

female was scheduled for excision of parietal meningioma. After induction of general anaesthesia, a triple-lumen central venous cannula was inserted through the right internal jugular vein; insertion was uneventful. Once the patient was positioned supine on the operating table, the distal lumen was connected to the transducer. Since the transducer was near the foot end of the patient, two extension tubings connected in series were used to complete the circuit. The initial CVP reading was 2 mmHg with a standard waveform. After about 5 min, the reading on the monitor was showing − 11 mmHg. The waveform was still present and appeared normal. There was no difficulty in flushing the line and fluid could be easily aspirated with a syringe from the circuit. The transducer was at the level of the midaxillary line. The junction of the two extension tubings was lying outside the drapes about 3 feet below the level of the heart with no apparent signs of leak. The heart rate and radial arterial blood pressure were within the normal range with no acute changes. Initially it was thought that the tip of the cannula might have gone into the subclavian vein but the presence of a waveform made it difficult to accept this theory. Infusions of 500 ml crystalloid and 500 ml Gelofusine were given which raised the CVP to − 8 mmHg. Intermittent flushing of the line did not change the waveform or the negative reading. No more fluid was given. About 30 min later, blood-tinged fluid was noted to drip slowly onto the floor from the junction of the two extension tubings. A closer inspection revealed that the female end of one extension tubing was slightly skewed leading to a very small amount of leak. Repositioning the connector corrected the CVP to a true 8-mmHg reading. Postoperative chest X-ray confirmed the correct position of the tip of the cannula.

The leak was not big enough to allow rapid leakage of the saline in the tubing. Since the junction was located about 3 feet below the zero level, a bigger leak would have given a more negative reading up to − 65 mmHg (90 cmH_2O). Most monitors display a negative reading up to − 20 mmHg and anything beyond is shown as a question mark suggesting error. The small amount of leak ensured that the waveform of the pressure changes was still displayed. It is likely that blood would have showed up earlier in the tubing but for the repeated flushing attempts by the anaesthesia team to ensure patency of the line. In retrospect, the waveform of the negative CVP may have been a bit damped but there was no leakage of fluid from the junction of the tubings during the flushing attempts.

This report demonstrates that very small leaks can lead to erroneous CVP readings whilst maintaining a waveform. Apart from inappropriate infusion of fluid, this could have caused persistent and potentially significant haemorrhage if the connection was located under the drapes. All connections should be rechecked, especially in the presence of unexplainable readings.

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Lip balm: a novel way to 'flavour' facemasks for inhalational induction

Inhalational induction of anaesthesia remains a useful technique in paediatric practice, but children are sometimes put off by the smell of the anaesthetic vapour. One manufacturer produces 'flavoured' facemasks in an attempt to disguise this smell, and to encourage patient cooperation. However, these masks are expensive as they are intended for single use only, and we have found the mask's smell to be quite subtle.

The addition of fruit extract or essential oils to the anaesthetic gases or facemask has been shown to improve the acceptability and odour of inhalational induction in children [1], but doubts have been raised as to the safety of this technique [2], and fruit extracts may be detected as anaesthetic vapour by infrared gas analysers [3].

We have been using Lip Balm to add a smell to our standard facemasks. Lip Balm is inexpensively available in stick form in a variety of 'flavours' from most chemists; we have found Lypsyl® to be the best brand for our purposes. A generous smear of balm on the inside of the facemask produces a strong smell, which can then be washed off with detergent and water after use. We have tested masks scented in this way, and Lypsyl® is not detected as agent by an infrared analyser.

We have found this technique to be particularly useful for the Haematology/Oncology list, where patients are returning regularly for multiple anaesthetics, and often request gas induction. At the pre-operative visit, much ceremony can be made of the selection of a flavour for today's induction, providing useful distraction and involvement of the reluctant child.

This technique has proved popular with children and parents, and we believe it is a valuable addition to the Paediatric Anaesthetist's armamentarium.

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