Anaesthesia, 2000, **55**, pages 1127–1143

Correspondence

A case for co-ordinated investigation and reporting of hypersensitivity-type drug reactions in the UK

Despite the introduction of new drugs, improved monitoring and increased awareness, the incidence of life-threatening anaphylactoid reactions to drugs used in anaesthesia and intensive therapy is probably little changed from the observations of Macintosh in 1949 [1]. Prior to the virtual demise of the NHS Supraregional Assay Services (SAS) almost a decade ago, the majority of UK reactions were referred to the Supraregional Protein Reference Unit (PRU) based in Sheffield. Its anaesthetic service, initiated in 1975 and known as NAARAS, was funded by a group of leading pharmaceutical companies and provided a free referral system for anaesthetists and others faced with such immediate reactions. Following the demise of SAS, the anaesthetic service still continued but was then funded by assay fees. It was then known as NARCOS, in which form it continues to date. Similar centralised services were and are available in France, co-ordinated by Laxenaire and Moneret-Vautrin, and in Australia, co-ordinated by Fisher and Baldo. Active dialogue existed between these centres, some research laboratories (e.g. Lorenz, Marburg, Salo, Turku) and the pharmaceutical industries.

The availability of a commercial assay, plasma tryptase (Pharmacia), to provide simple accurate measures of mast cell degranulation based on the work of Schwartz *et al.* [2], transformed the scene, allowing rapid decision making as to the nature of the reaction mechanism (anaphylactic, anaphylactoid, other). This assay, first used as a large-scale screening technique in Sheffield, is now used worldwide. Other assays such as that for the histamine metabolite, methyl histamine, further 'fine-tuned' the investigation [3].

In the UK, the ready availability of such assays, coupled to the indication of the Association of Anaesthetists Working Party Reports [4] of 1990 and 1995 that any large-scale hospital laboratory should be able to investigate anaphylactoid reactions, has slowly devalued NARCOS to a simple assay service. The Unit still gathers the bulk of UK reactions, but the majority without any indication of the patient's problem, reaction details or management. Much valuable and 'cheaply obtained' research material is now being lost. Findings are relegated to the local requirement, and the wider perspective of combined data overlooked.

It is still not possible to identify the patient at risk except in vague generalities of previous family history and allergy. The necessary use of multiple drugs for anaesthesia and surgery still provides the bulk of anaphylactoid response, and isolating these non-specific reactions from those of error and of specific drug response (anaphylaxis) is essential for the future care of the patient involved and a defence in the increasing tide of litigation.

The current broad distribution of these mechanisms in France (as reported by Laxenaire [5]) from 1648 patients (1994–1996) is virtually identical to those in 53 consecutive UK reactions reported by NARCOS [6] in 1994 (Table 1).

A recent enquiry caused us to look at the time lapse between anaesthetic induction and the onset of clinical manifestations. General reading suggests that these are immediate, i.e. within a few seconds of drug(s) administration, although studies of the plasma histamine release curves in clinical trials [7] show histamine release peaking between 2 and 5 min. We wondered if time variation provided clues to mechanism: we were aware that dextran manifestations usually occur 10 min into infusion following 20–50 ml infused fluid and that such reactions are IgG antibody mediated. We selected two drugs for study – succinylcholine, where reaction is IgE mediated anaphylaxis, and propofol, infrequently immune with cardiovascular manifestations usually secondary to bronchospasm. The data extracted from 1990 reports are tabulated; missing data in more than 100 reports reduced the numbers to 15 (Table 2).

Although preliminary, this survey indicated that immediate bronchospasm (biased to propofol) is unlikely to be missed by the anaesthetist, but that the classic IgE-mediated manifestations of hypotension may be delayed or possibly unobserved for at least 5 min. Five minutes on from induction the patient may be in the anaesthetic room being ventilated and observation of the reaction may be delayed, with a disastrous outcome. Oddly, the distribution for succinylcholine here is mirrored by

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Table 1

Mechanism	France	UK
Anaphylactic (Immune)	42%	32%
Anaphylactoid (Non-immune)	37%	45%
Other	21%	23%

studies of Lorenz [7] infusing low concentrations of histamine into volunteers. Our reactor profiles may thus reflect both genetic variation in histamine release and variations in receptor uptake.

There is clearly much to be learned from the careful study of well-documented cases. Sadly, our own records deteriorate badly from 1993 for the reasons outlined. We are aware of the valuable work carried out by the Committee on the Safety of Medicines (CSM). It has been stated that NARCOS could dilute their input records. This was never the case; the argument against direct input of clinical observation by the anaesthetist is that cause and effect is not obvious without laboratory analysis, as compared with reports for antibiotics, antidepressants and other long-term 'single' medications. The original Sheffield PRU model is similar to that used in France and Australia in terms of sample analysis and interpretation. However, France relies on co-ordinated analysis from a chain of centres across the country. This is probably the way forward in the UK, with a co-ordinating centre identified to collate data from all units providing analysis of samples from adverse drug reactors.

Table 2

Time of onset (min)	Suxamethonium $n = 8$	Propofol $n = 7$
Immediate (< 30 s)	3	5
1	1	1
1–2	1	0
2–3	2	0
5	1	1

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New house officers' knowledge of resuscitation, fluid balance and analgesia

In August 1999, I conducted a postal survey of 242 new house officers in the first four weeks of their first house job. Using clinical scenarios, I tested their knowledge of resuscitation, fluid balance and analgesia. I received 63 replies (response rate 26%), of which 53 were useable. The results are summarised in Table 3.

The majority of respondents (32/53) had spent just one week on formal attachment to anaesthesia at medical school; 9/53 said they had spent no time in the specialty. For resuscitation, only 15/53 (28%) said that anaesthetists had played a major role in teaching. For fluid balance, the figure was 12/53 (23%) and for analgesia, 21/53 (40%).

The brevity of a letter barely does justice to the seriously deficient understanding shown by most respondents regarding these basic skills of peri-operative care. However, it demonstrates many of the common avoidable errors and omissions that vex those of us occasionally called to 'rescue' patients on the ward.

The profile of the anaesthetist is changing, as is the public's expectation of doctors' competence; it is time we pressed for greater involvement of departments of anaesthesia in providing structured undergraduate training programmes in peri-operative care.

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Pre-anaesthesia assessment clinics. Beauty in the eye of the beholder?

It is with interest that I read the correspondence [1–3] relating to my recent letter about pre-anaesthetic assessment clinics or PAACs [4]. It has obviously stimulated a great deal of thought. Of particular interest were the views of Dr J. R. Davies and Dr D. B. Baines, which I feel warrant further discussion.

Table 3 Summary of results from answers to questionnaire. (n = number of respondents able to attempt scenario; f = frequency of correct individual criteria.)

Scenario (style of answer in parentheses)	Summary of criteria assessed	*n*	Individual criteria assessed	*f*
1. Treatment of ventricular fibrillation (free text)	Adherence to major points of current Advanced Life Support guidelines	52	Assess rhythm	24
			Check pulse	17
			Defibrillate; 200 J x 2, thereafter 360 J31	31
			Cardiopulmonary resuscitation; 1 min or 10 cycles	28
			Adrenaline; 1 mg i.v. every 3 min	10
			All five of the above	2
2. Description of use of self-ventilating bag and mask device during cardiopulmonary resuscitation (free text)	Inclusion of major elements of procedure	51	Appropriate head position	12
			Manoeuvre to open airway	19
			Use of airway adjunct	6
			Connect oxygen	24
			Two-person manoeuvre	32
			1:5 breath:compression ratio	24
			Check for evidence of ventilation	9
3. Prescription of first 24 h maintenance fluid following hemicolectomy in otherwise healthy 60-year old male (blank fluid prescription chart)	Fluid volume and electrolytes prescribed	52	Volume, range (median): 500–4500 (3000) ml.24 h^{-1}	–
			Sodium, range (median): 0 – 450 (150) mmol.24 h^{-1}	–
			Potassium, range (median): 0 – 120 (40) mmol.24 h^{-1}	–
4. Simple postoperative oliguria following open cholecystectomy in otherwise healthy 30-year old female. Scenario strongly suggestive of hypovolaemia (free text)	Identification of: a. diagnosis	51	Correct	49
			Incorrect	
	b. correct supportive signs and symptoms	51	Tachycardia	24
			Hypotension	32
			Low JVP	7
			Dry mucous membranes	23
			Decreased skin turgor	33
			Decreased peripheral perfusion	16
			Thirst	17
			Sunken eyes	8
	c. correct treatment themes within answer	51	Appropriate fluid challenge	19
			Assess response	15
			Repeat and reassess	2
			Alert help if no improvement	2
			Consider/exclude surgical cause	2
5. Prescription of postoperative analgesia in fit 18-year old male, following internal fixation of ankle fracture (blank drug prescription chart)	Prescription containing regular non-steroidal anti-inflammatory drug and/or paracetamol with opiate bolus available for breakthrough pain.	43	Prescriptions matching criteria	3
6. Are you happy to administer intravenous morphine in a ward setting? (tick boxes)	–	53	Yes	18
			No	35
			Reasons indicated for answering 'no' (n=35):	
			Risk of respiratory depression	13
			Risk of nausea and vomiting	8
			Don't know dose	12
			No experience	28
			Never been taught	19
			Shouldn't be administered on ward	2
			Job of more senior doctor	1
			Unable to monitor patient on ward	5

Dr Davies states that while PAACs may be a valuable adjunct to anaesthetic practice in the UK, the cost of such a system may not comply with the cost-effective nature of the NHS. (A sentiment shared by many of us in relation to a variety of treatments and practices within the NHS!) In his argument he compares the pay structure of doctors in Australia and the UK, citing increased payments for 'out of hours work' as one reason for the possible use of daytime clinics in place of evening 'premeds'. This is not entirely true as it makes the assumption that all hospital doctors in the Australian system are paid in such a fashion. Trainees (SHO and Specialist Registrar equivalents) and self-employed Consultants or VMOs (Visiting Medical Officers) are paid higher rates for 'out of hours' work; however, Staff Specialists (an approximate equivalent of full/maximum part-time NHS Consultants) and Clinical Fellows receive fixed salaries. The PAAC of which I have experience was staffed by Clinical Fellows and Staff Specialists, a situation similar to that which may arise in the NHS if such clinics became commonplace. In isolation it therefore may not be cost-effective in terms of salaries, although Kerridge *et al.* [5] have demonstrated the potential for overall cost-effectiveness in the peri-operative process in terms of reduced length of stay, decreased cancellations and reduced non-attendance. Managers may yet see the funding of medical staff for PAACs within the NHS as a worthwhile cost-effective and quality of care exercise!

Other points of interest with respect to PAACs were made by Dr Baines 'giving balance' to my previous correspondence [4]. He/she states that whilst PAACs are cost-effective, their overall benefit remains unproven, particularly with respect to them 'enhancing our role' and 'improving training'. I certainly accept that my views on Australian PAACs are based entirely on my own experience as a foreign visitor and have not been subject to any controls of a 'double-blinded randomised nature'; however, I find it regrettable that he/she feels that they have been 'foisted on us by the minority'. The system I encountered (albeit in a different Regional Centre to that in which Dr Baines works) involved a major input from Consultants who were far from 'semi retired' and, in complete contrast to Dr Baines' experience, it allowed me to give more in-depth explanations of premedication, induction techniques and potential postoperative analgesic options/requirements. Indeed, compared with previous experiences, it reduced the 'sausage factory' concept of getting patients through the system. As an anaesthetist now re-ensconced in the UK, I therefore feel I can draw upon my experience of the Australian peri-operative system in a positive fashion.

Thus, given our polarised experiences of the PAAC, is it perhaps time for further appraisal of the effectiveness and problems of 'the peri-operative system' rather than relying solely on anecdote from people such as Dr Baines and myself?

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Gone but not forgotten!

Your correspondents (Brett & Wraith. *Anaesthesia* 2000; **55:** 593) have little to fear; the contribution of Dentistry to the development of Anaesthesia has not been forgotten. As it happens, some two years ago we began to explore the influence of the Edinburgh Dental Hospital and School on the advancement of Academic Anaesthesia. Our first paper covered the period from the introduction of chloroform to the outbreak of the First World War and was presented at the History of Anaesthesia (HAS) meeting in Cambridge in October 1999 [1]. It is perhaps unfortunate that we do not seem to have a dental surgeon among the members of the HAS.

The story is fascinating. Within days of James Y. Simpson's description of the anaesthetic properties of chloroform, his friend and neighbour, Francis Imlach, a distinguished dental surgeon, gave chloroform to one of his apprentices for the removal of a tooth. Imlach incidentally was the first dental surgeon to be elected President of the Royal College of Surgeons of Edinburgh. Subsequently, he and his contemporary John Smith became accomplished anaesthetists, as well as distinguished dental surgeons.

In 1860, John Smith, with the assistance of Francis Imlach and other colleagues, set up the Edinburgh Dental Dispensary, which with the passing of the Dentists Act, 1878 [2], became the Edinburgh Incorporated Dental Hospital and School. From its inception, the discipline of anaesthesia was encouraged. During the first decade, two chloroformists were appointed and a system of training was introduced which gradually became more formalised, first with demonstrations and tutorials, followed later by a more academic lecture programme. Thereafter, various anaesthetic techniques were introduced and developed, new apparatus was designed to exploit physical principles, and pharmacological and physiological responses began to be studied. Thus the Dental Hospital developed as a centre of anaesthetic teaching and research before the University teaching hospital. The Royal Infirmary had even appointed an anaesthetist. Accordingly, when William Guy was appointed Dean in 1899, he had little difficulty in persuading his colleagues that, in future, anaesthetics should only be administered in the hospital under the direction or in the presence of a qualified medical practitioner. It should be added that virtually all the

staff were doubly qualified, in medicine as well as in dentistry; in addition, a substantial number held higher qualifications in surgery.

It is surely significant that 50 years later at the end of the forties when we ourselves were in anaesthetic training posts in Edinburgh, three of the consultant anaesthetists responsible for our training were also qualified in dentistry, two of whom held simultaneous consultant appointments as dental surgeons.

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Ventilation through a metal tracheostomy tube

A 29-year-old female was scheduled to have a laryngotracheoplasty for severe tracheal stenosis thought to be secondary to prolonged tracheal intubation several years previously for severe asthma. Her past medical history was otherwise unremarkable. She had a silver Negus metal tracheostomy tube (28 French gauge) *in situ*. It was felt that any instrumentation of her airway could potentially precipitate coughing and bronchospasm. Appropriate monitoring consisting of ECG, non-invasive blood pressure and oxygen saturation was instituted. The patient was pre-oxygenated by placing the bevel of a size 3 laryngeal mask airway (LMA) firmly over the tracheostomy site with the silver Negus tube still in position (Figs 1 and 2). This simple manoeuvre produced an excellent airway seal and anaesthesia was induced with 100 μg of fentanyl and 150 mg of propofol. Prior to the administration of muscle relaxants it was confirmed that the patient could easily be ventilated through the LMA used in this fashion. Muscle relaxants were administered, the trachea sprayed with 120 mg of lidocaine and the tracheostomy tube easily replaced with a size 6.0 armoured cuffed tracheal tube without the patient coughing. Surgery proceeded uneventfully.

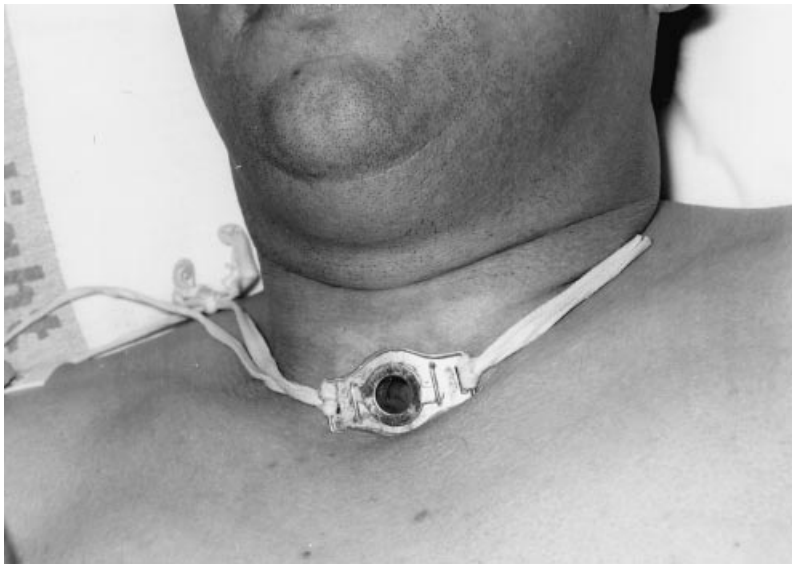

Figure 1

A silver Negus tracheostomy tube cannot be connected to an anaesthetic breathing circuit via the standard 15-mm connector. As the presence of the tracheal stenosis would have precluded any airway instrumentation above it, the options for ventilating this patient on induction of anaesthesia would have been the replacement of the Negus tube

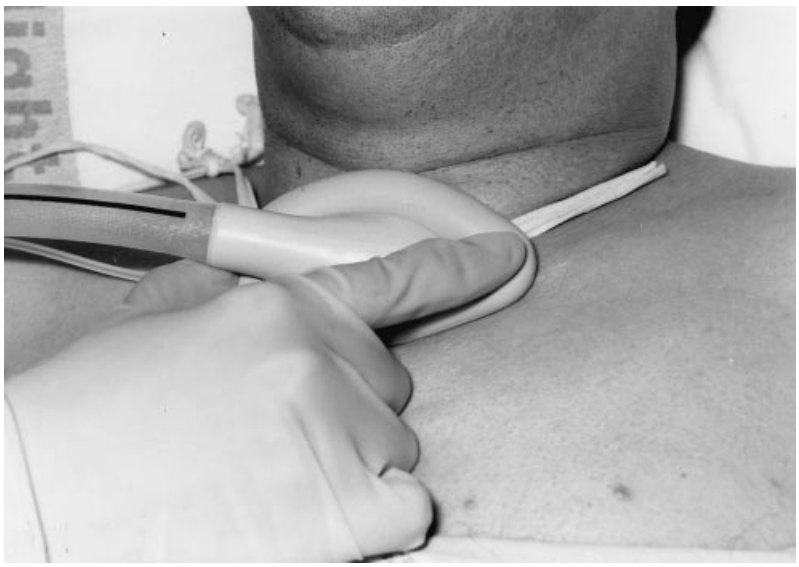

Figure 2

either with a Portex tracheostomy tube or a tracheal tube prior to the induction of anaesthesia. However, we felt that any difficulties encountered in inserting a different airway whilst the patient was awake would have led to unnecessary instrumentation and trauma to the airway, coughing and undue distress to the patient. Induction of anaesthesia without prior knowledge that ventilation could be accomplished in an atraumatic fashion was deemed unsafe. Another option could have been the use of a paediatric facemask employed in a similar fashion to the LMA [1]. However, it is often difficult to establish a tight seal over the stoma with this technique, and as an excellent seal was accomplished with the LMA this option was not explored further. We are aware of one other report in the literature describing the use of the laryngeal mask airway to ventilate a patient through a tracheostomy orifice [2].

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Translaryngeal tracheostomy in the high-risk patient

We read with interest the article (Byhahn *et al. Anaesthesia* 2000; **55:** 676–82) describing the use of the Fantoni tracheostomy technique in high-risk patients. We have used this technique in similar situations and fully endorse their view that this is a suitable technique for the high-risk patient. We would, however, wish to make a few comments.

1 The authors rightly point out that this technique requires an experienced team. This is essential and ideally should consist of three anaesthetists, one of whom is concerned solely with maintaining the airway and the patient's oxygenation.
2 The partial occlusion of the tracheal lumen by the bronchoscope during insertion of the guide-wire may lead to high inflation pressures and we find that switching to manual ventilation at this point results in more effective oxygenation.
3 The thin-walled tracheal tube allows ventilation of the lungs while the tracheostomy is being performed, but it is essential that this tube is correctly positioned and not allowed to move as unrecognised endobronchial intubation is potentially life threatening in the high-risk patient.
4 Our practice is not to remove the ventilating tube until the tracheostomy tube has been rotated and secured. While we agree that damage to the trachea is theoretically possible, we have not encountered any problems, and having observed the rapid development of tracheal oedema following removal of the tracheal tube, have our doubts about the advisability of relying on emergency oral re-intubation in unstable hypoxic patients should difficulties arise. We concede, however, that the majority of our patients are undergoing tracheostomy at a later stage than those described in the article (10–14 vs. 5–6 days) and that this may present us with more difficult operating conditions.

In conclusion, we feel that translaryngeal tracheostomy is a valuable technique but that the potential difficulties should not be underestimated.

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A reply

We would like to thank Drs Milligan and McCollum for their response to our article about translaryngeal tracheostomy (TLT) in high-risk patients [1] and their valuable comments. Unlike the authors, we perform TLT with a team of two anaesthesiologists, one of whom performs the tracheostomy, and the second colleague performs bronchoscopy and insertion of the small-bore ventilation tube. An ICU nurse trained in TLT is solely responsible for stabilising the ventilation tube to minimise the risk of airway loss by accidental extubation.

We use volume-controlled ventilation during TLT to ensure a given tidal volume regardless of pressure. A high inflation pressure is generated by the increased resistance of the tube's small diameter, but has been demonstrated not to affect the lungs [A. Pesenti, personal communication]. Correct position of the small-bore ventilation tube is confirmed by auscultation of the lungs. Once endobronchial intubation has been ruled out, the tube's position is maintained manually as described above.

The concept of maintaining the ventilation tube in place during cannula placement and rotation has also been advocated by Fantoni himself [2], but in the majority of reports on TLT the tube has been removed before cannula rotation was attempted. On the other hand, there are no reports of tracheal injury during cannula rotation with the tube in place. Based on the recent literature and personal communication, both techniques seem to be equally safe.

We have not yet encountered any problems with oral re-intubation should placement of the TLT cannula fail. This also held true in patients who had been intubated with a tracheal tube for an extended period of time before tracheostomy. Nonetheless, we feel that early percutaneous tracheostomy should be considered whenever possible, because several studies have shown that the critical period for the development of laryngotracheal lesions caused by the tracheal tube is between the 7th and 11th day of intubation and possibly earlier [3–5].

In conclusion, we agree with Drs Milligan and McCollum that TLT requires an experienced team especially in patients at high risk of adverse sequelae during tracheostomy.

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Difficult intubation

It was interesting to read the large study of failed intubation (Barnardo & Jenkins. *Anaesthesia* 2000; **55:** 690–4). However, I note that in the discussion they criticised the use of a second dose of succinylcholine and hand ventilation using relaxants. Although their opinions are widespread, they are totally contrary to those of Hewett and Livingstone [1].

In an age of 'Evidence Based Medicine' it is worrying that so many aspects of the bail out drills represent only personal conviction [2]. These include the wisdom of head down tilt; the use of the left lateral position; when and when not to continue cricoid pressure; the use of a repeat dose of succinylcholine, permitting a second (or third) attempt at intubation; the safety of continuing volatile anaesthesia with either spontaneous respiration intermittent positive pressure or ventilation on a facemask; the appropriate time to use a Laryngeal Mask Airway [3]; and so on.

Given the rarity of failed intubation in obstetrics (0.3%) [4], it would seem important that all the information on every failed intubation across the country should be obtained. Only when a large database has been obtained, including the techniques used and the problems that result, do we have any chance of discovering the relative risks of the different approaches. Only then could we have evidence-based guidelines. Might this be a role for the Obstetric Anaesthetists Association?

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Complete power failure

A 79-year-old man was scheduled for elective carotid end-arterectomy during a routine afternoon vascular list. Anaesthesia and surgery proceeded without incident until just as the surgeon was about to incise the carotid, there was a complete power failure. Initially, it was thought the emergency generator would take over after a brief pause but this did not happen. The operating theatre was completely dark apart from the operating light, which had a back-up battery. The patient was still being ventilated by the bag-in-bottle compressed-air-driven ventilator. The Datex monitor no longer functioned without power. The surgeon briefly became the only patient monitor available by virtue of his direct visualisation of the carotid artery, all other parts of the patient being hidden under drapes.

Within minutes a portable Pro-Pak monitor was obtained which had ECG, pulse oximetry and invasive pressure monitoring. Capnography and agent monitoring were still unavailable, as were proper suction and surgical diathermy. The power cut was affecting the whole hospital and there was speculation about its cause and how long it would last. The battery-operated monitor and lighting had an unknown lifespan as had the supply of compressed air driving the ventilator. A period of about 20 min had now passed and it was decided the only option available was to abandon the procedure and wake the patient up. The patient was recovered in the Intensive Care Unit, which having natural lighting was far preferable to the Recovery Unit, which had no light at all. The power was eventually restored after about 30 min.

The patient returned to the operating theatre the following morning where anaesthesia and surgery were carried out uneventfully.

The power failure was due to the following sequence of events: subcontractors working on the construction of a new building drove a steel pile through the hospital's main 11-kV incoming power cables. Fortunately, there were no casualties but a black out of the entire hospital site and the immediately surrounding area followed. Both the standby generators failed to run, resulting in a failure of the essential power supply for a period of time.

Initial investigation into the generator failure revealed that one generator did not start at all, and the other started, but quickly tripped out on overload. Further investigation revealed a project team design co-ordination issue, whereby an automatic generator changeover was enabled without the installation of any of the planned load shedding. Hence, the failure of one generator would result in overloading the other generator. The controls were reconfigured to prevent the automatic changeover of generators as an interim measure until phase 2

of the energy centre redevelopment and the associated load shedding was commissioned.

Repeated testing of the generators 'off line' failed to reproduce the original failure. 'Black start' testing (where by the main 11-kV supply is turned off, simulating a 'real' power failure) finally allowed the failure to be traced to a faulty component in the new generator control panel. This was replaced, and a third black start was performed satisfactorily.

This event highlights the fact that despite recent extensive planning and protocols to protect against the year 2000 bug, we cannot always predict when disaster may strike but have to deal with it as best we can. In the suite of 11 operating theatres, many were between cases or using regional techniques, which minimised disruption and, thankfully, this rare and potentially disastrous mishap did not result in harm to any patient.

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Continuous flow vs. draw-over apparatus

I welcome the recent contributions by Drs Fenton and Lunn suggesting that their experience in third world and military anaesthesia has useful lessons for the rest of us (Fenton. *Anaesthesia* 1999; **54:** 1111; Lunn. *Anaesthesia* 2000; **55:** 402). In particular, they advocate the use of draw-over anaesthesia for the growing number of anaesthetists who regard nitrous oxide as obsolete, and imply that perhaps the increasingly complex Boyle's machine is starting to look over-specified. My own view is that we should try to distinguish between two separate but related concepts, namely the use of room air as opposed to pressurised air, and the use of draw-over as opposed to continuous flow of this air. While it is difficult to question the logic of using room air enriched with oxygen, I would suggest that continuous flow, independent of back-pressure, has genuine advantages. The ability to use an accurate, high-resistance, vaporiser cannot be dismissed. Even if we were to commit ourselves to total intravenous anaesthesia, a Mapleson type rebreathing system with collapsible reservoir bag to give a feel for the state of the patient's lungs would probably be worth preserving. Continuous flow may fail to justify itself in the third world setting, and I have every respect for those who cope in its absence, but I cannot see anaesthetists in affluent countries electing to use self-inflating bags and draw-over vaporisers.

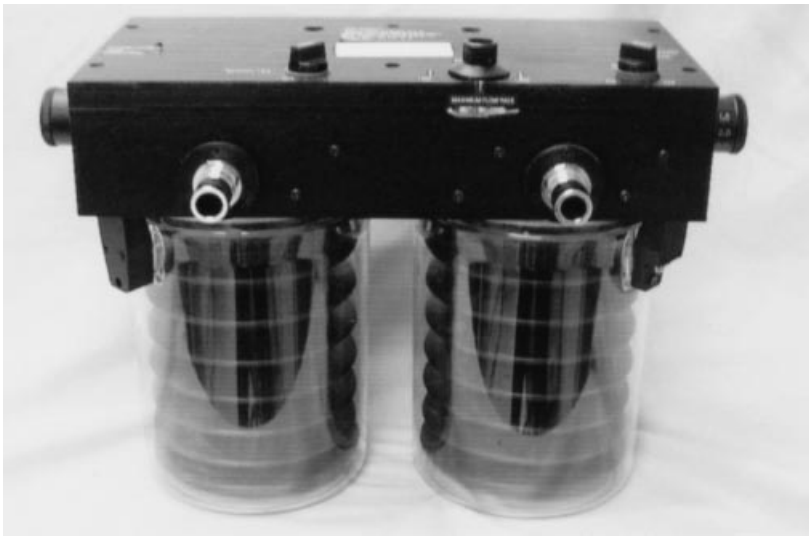

Figure 3

It is hardly surprising that the use of room air be considered synonymous with draw-over anaesthesia, as no apparatus has ever been available which can deliver oxygen from 21% to 100% at a continuous flow-rate that is independent of backpressure. However, I designed a device to address this issue. It was built by Penlon Ltd, Abingdon, UK, and further developed in the Medical Physics Department of the Royal Perth Hospital, Western Australia, and uses a double bellows arrangement to entrain room air and deliver it to the patient via a Mapleson type breathing circuit (see Fig. 3). The two bellows alternate in their action during spontaneous respiration in order to generate continuous flow. Perhaps it represents the answers to Dr Fenton's original questions.

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An unusual case of 'gastro-oesophageal reflux'

We wish to report a case of rupture of a nasogastric feeding tube *in vivo*. The tube in question was a 8 gauge, 110-cm-length Flocare® fine-bore polyurethane nasogastric tube (Nutricia Ltd).

Briefly, a 71-year-old woman had been on intensive care for several weeks with complications of atypical Guillain Barré Syndrome. She had a surgical tracheostomv performed on day 8 with no problems. During her admission, nasogastric feed had been noticed regurgitating around her tracheostomy site and several manoeuvres had been tried to stop this including motility agents, reducing the feed rate, and finally replacing the nasogastric tube. At that time the tube was not specifically examined, but no obvious defect was reported.

On day 24, further nasogastric feed was noted around the tracheostomy site. The ITU SHO passed a new fine-bore nasogastric tube on day 26 and its

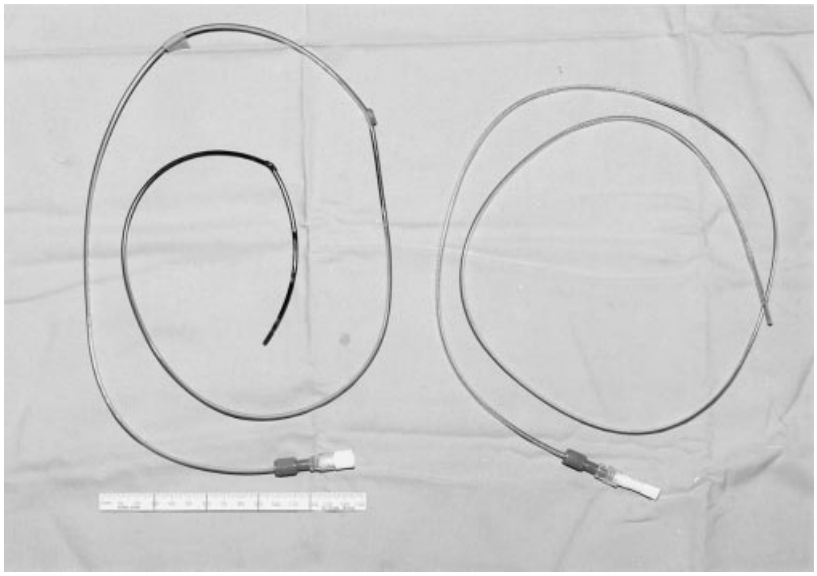

Figure 4

position was satisfactory on chest X-ray. Feeding recommenced without problems until day 38. At this time, feed was again pooling in the pharynx but the patient tolerated oral fluids without regurgitation.

The nasogastric tube was removed and found to be ruptured at two sites. (Figs 4–6). Our assumption is that this was due to flushing of the tube after blockage by nasogastric drugs on two separate occasions. The drugs given were omeprazole suspension, liquid cisapride and temazepam, and soluble paracetamol. None of these is specifically known to cause nasogastric tube blockage or erosion. A 2-ml syringe with soda water was used to unblock the tube on day 37 (manufacturer recommend a syringe no smaller than 30 ml). However, testing a new tube with both water and neat Coca-Cola using a variety of syringes from 1 to 10 ml and maximal force exerted by an ITU SpR could not rupture the tube.

We could find no reports of this in published literature. The manufacturer has heard of one possible case, but has no specific details. Since the manufacturer states that tubes can be safely used for up to 6 weeks, we assume that either the tube (or tubes, which may have come from the same batch) was defective, or that one of the drugs passed nasogastrically allowed a blockage to occur as well as corroding the polyurethane. Following this, the 2-ml syringe used caused the tube to rupture.

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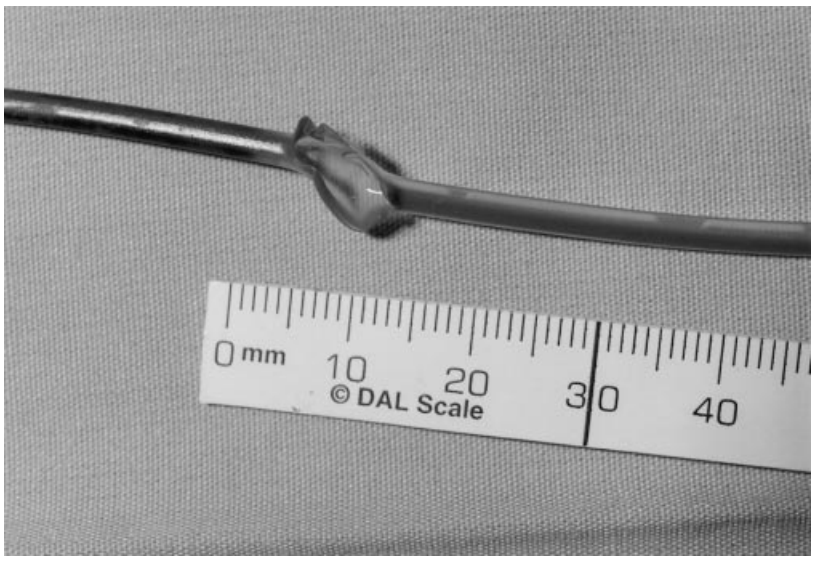

Figure 5

A reply

Having read with same concern Dr Carradice's case report, I should like to take the opportunity to comment.

The cause of regurgitation around the tracheostomy tube at the time the first nasogastric tube was placed could have been caused by a number of factors: tube malposition, patient position, delayed gastric emptying or severe gastro-oesphageal reflux. It would not have been caused by the Flocare feeding tube, which as Dr Carradice noted, had no signs of defect.

Tube occlusion in all fine-bore nasogastric tubes is known to be a recognised problem, occurring in 6–10% of cases [1]. The common causes of these occlusions include failure routinely to flush the tube [1], the effect of medication and gastric juices on enteral feed causing protein precipitation [2, 3] crushed medication particles [1, 4], liquid medication with a pH of 5 or less (causing changes in feed consistency), viscosity and particle size of the formulation [5–7].

Care must be taken whilst attempting to remove tube occlusions to prevent tube rupture. Tube rupture may occur when a pressure of more than 40 psi is applied by an infusion pump or syringe

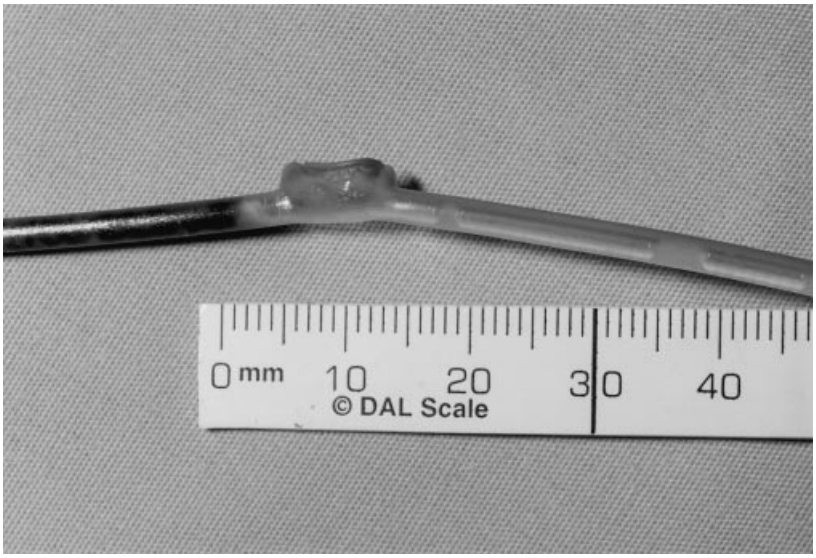

Figure 6

[8]. It is for this reason that Nutricia as the manufacturer of Flocare tubes does not recommend the use of syringes which are smaller than 30 ml. Using a 2-ml syringe to unblock a tube could cause the rupture of the tube. Tests performed by Nutricia Medical Devices on Flocare Nasogastric tubes, submerged in artificial stomach fluid at 37 °C for several weeks and then pressurised using a small 10-ml syringe, showed the same type of rupture. Unfortunately, there were no batch details available from the hospital for this particular tube, but as there have not been any similar reports for several years, it is unlikely that a tube or batch defect was to blame for this incident.

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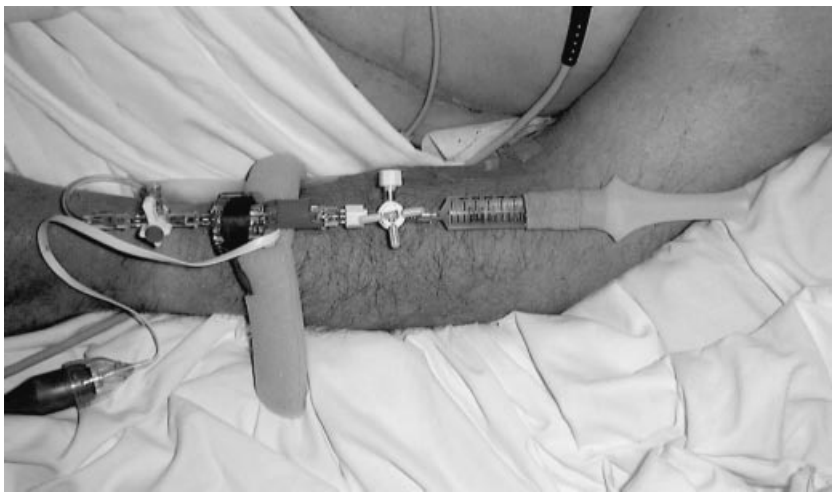

Figure 7

Pressure for success

The movement of critically ill patients, both within and between hospitals, is a fairly common event in British intensive care [1], and is fraught with potential hazards. The risk of misadventure during transit is proportional to the number of tubes connected to the patient (intravenous lines, chest tubes, drains, urinary catheter, etc.), which is related to the severity of the patient's condition. These tubes are there for a good reason, and most cannot be removed or disconnected prior to transfer; but those that can, should be. Some of the most annoying 'tubes' are those that connect the pressurised bags of heparinised saline to the arterial and venous pressure transducers. The pressure bags are bulky, frequently lose their pressure in transit, and have a tendency to entrain air from the drip chamber when they are laid flat, which is almost inevitable at some point in the process. This is particularly true during long-distance aeromedical transfers, when there are the additional problems of not having anywhere to

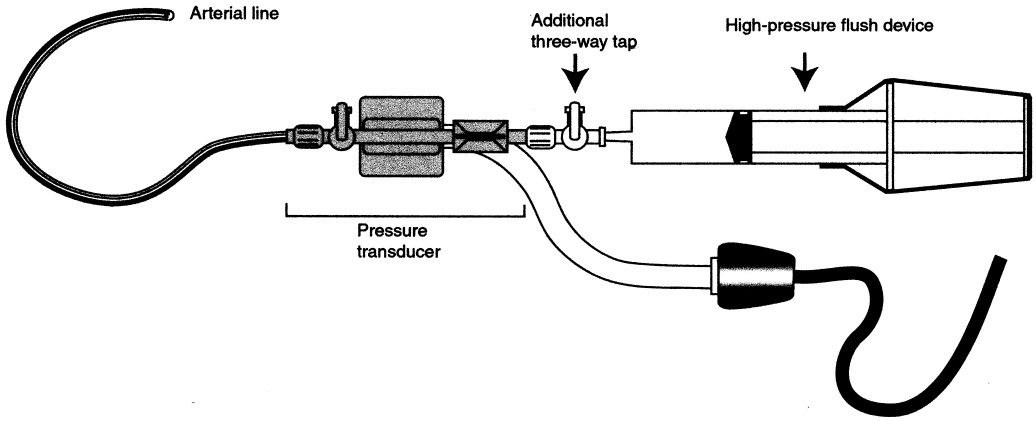

Figure 8

hang them in the confined space of a small commercial jet aircraft and the effect of changes in cabin pressure on the air-filled pressurisation devices. These problems can be avoided by replacing the bag of heparinised saline, pressure bag and giving set with a three-way tap and the device shown in Figs 7 and 8. The flush device is a 10-ml syringe which has had the plunger covered with two fingers cut from a size 8 surgical glove and taped to the barrel of the syringe. The first finger should be approximately 4 cm long, the second 5 or 6 cm long. Whilst empty, the syringe is fitted firmly to the three-way tap sited where the giving set would have joined the transducer unit, and is then loaded with 10 ml of heparinised saline from a second syringe.

Four of these devices were constructed and connected to a commonly used pressure transducer system (Medex Medical Inc.), and the pressure generated measured (Hewlett Packard). All four generated more than 350 mmHg for between 45 and 75 min (above the upper limit of measurement for the monitors used), and then generated the pressure/time profile shown in Fig. 9 (error bars indicate one standard deviation). At the end of 3 h of measurement, each of the syringes had delivered between 5 and 7 ml of fluid. In clinical use, the volume loss from the system (and therefore the pressure decay) is likely to be less when connected to an arterial line because of the backpressure effect of the patient's blood pressure. In transducers connected to the pulmonary artery or central veins, the pressure loss is similar to that shown. In either case, the system will generate at least 180 mmHg for at least 3 h, and can be easily topped up with more heparinised saline via the three-way tap. If this system had been in use by Scott and colleagues [2], they would have had the additional benefit of completely preventing the problem they described.

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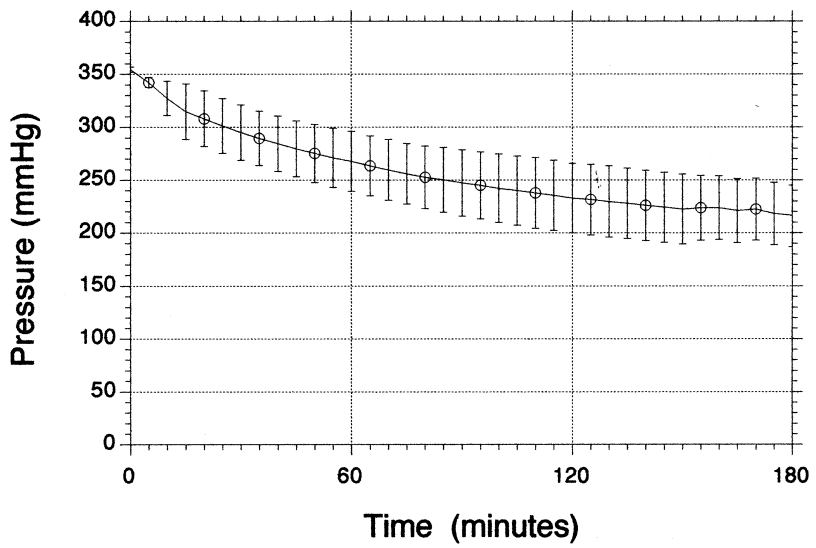

Figure 9

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Prediction of degree of hypomagnesaemia during general anaesthesia

Peri-operative infusion of sodium lactate solution can reduce serum

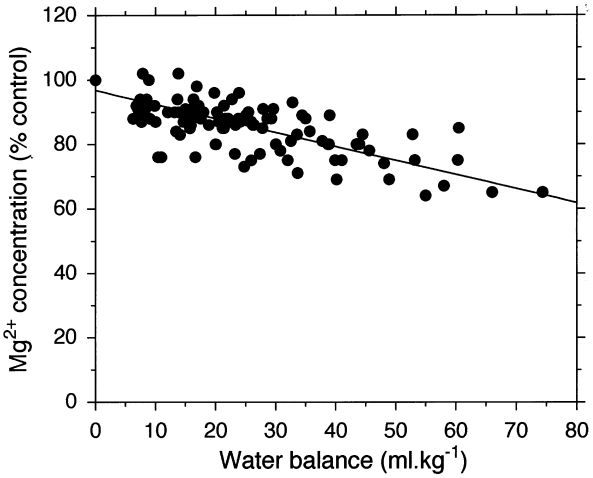

Figure 10 Serum magnesium ion concentration Mg (% of control) and the water balance per body weight. The straight line was satisfactorily fitted by the equation: $y = -0.44x + 96.8$, $r = 0.79$ ($n = 126$). The data points were obtained from 20 patients.

magnesium ion concentration (Mg) [1]. Hypomagnesaemia could induce cardiac arrhythmia [2] and lower the threshold for nociceptive reaction via modulation of the N-methyl-D-asparatate (NMDA) receptor [3]. In order to predict the degree of hypomagnesaemia during clinical anaesthesia, we have investigated the relations between amount of fluid infusion and magnesium.

After obtaining approval from our research ethical committee and informed consent, we studied 20 patients (American Society of Anesthesiologists physical status I or II, 16–84 years old) undergoing elective surgery under general anaesthesia (1.0–1.5% isoflurane and 60% nitrous oxide in oxygen, and 100–300 μg of fentanyl) at the Toyama Medical and Pharmaceutical University Hospital. Patients with renal impairment or receiving diuretics or steroid treatment were not studied. All patients received intravenous infusion of magnesium-free acetated Ringer solution at 15 ml.kg^{-1}.h^{-1} (0–60 min), 10 ml.kg^{-1}.h^{-1} (61–120 min) and 5 ml.kg^{-1}.h^{-1} (after 121 min). Arterial blood (3 ml) was sampled from a radial artery cannula using a blood gas sampling kit containing 7 iu of heparin lithium (QuickLite™; Marquest Medical, Englewood, CO, USA) for every 500 ml of fluid administration. Serum magnesium determinations were performed by an electrolyte analyser (CR-8; NOVA biomedical, Waltham, MA, USA). Results were expressed as mean (SD). The analysis of variance (ANOVA) was used for statistical analysis and $p < 0.05$ was considered significant.

The intravenous administration of magnesium-free acetated Ringer solution reduced Mg. Before fluid infusion Mg was 0.51 (0.03) mmol.l^{-1} ($n = 20$), and 1500 and 3000 ml of fluid administration significantly reduced Mg to 0.45 (0.02) ($n = 20$) and 0.41 (0.03) mmol.l^{-1} ($n = 5$), respectively. Figure 10 shows a good correlation between Mg (% of control) and the water balance per body weight. The water balance (ml.kg^{-1}) was calculated by the equation: (infusion volume) − (blood loss) − (urine volume)/(body weight). The correlation predicts that the water balance of 50 ml.kg^{-1} can reduce Mg to 75% of control. The results suggested that the magnesium supplementation should be considered when a large amount of magnesium-free fluid is infused during general anaesthesia.

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Combined emergency Caesarean section and intracerebral aneurysm clipping

Management of cerebral aneurysm clipping with acute subarachnoid haemorrhage during pregnancy involves medical issues and poses an interesting challenge to anaesthesiologists, obstetricians and neurosurgeons [1–3]. We present a case of acute subarachnoid bleeding in a pregnant patient in whom emergency Caesarean section was performed followed by craniotomy and clip obliteration of the aneurysm.

A 38-year-old patient at 36 weeks gestation experienced severe headache with loss of consciousness induced by Hunt and Hess Grade IV subarachnoid haemorrhage. Following emergency intubation with methohexitone and succinylcholine, anaesthesia was maintained with fentanyl, midazolam and atracurium. The fetal heart rate was monitored by Doppler sonography in addition to monitoring of maternal heart rate, invasive arterial blood pressure, end-expiratory CO_2 and urine output. Computed tomography and intra-arterial digital subtraction angiography revealed a large ruptured aneurysm of the right internal carotid artery (Figs 11 and 12). Superselective angiography was performed under moderate

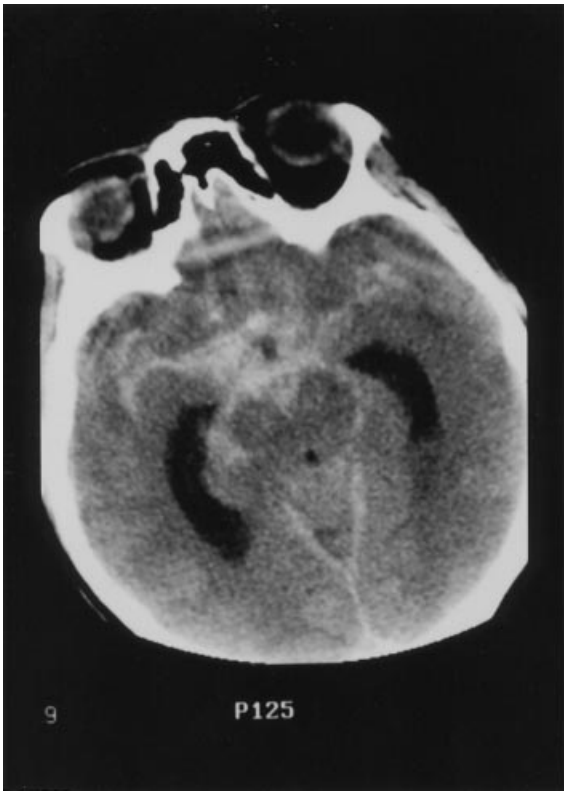

Figure 11 Axial CT scan without contrast showing a severe aneurysmal subarachnoid haemorrhage that fills the basal cisterns and produces a hydrocephalus (note the marked brain swelling).

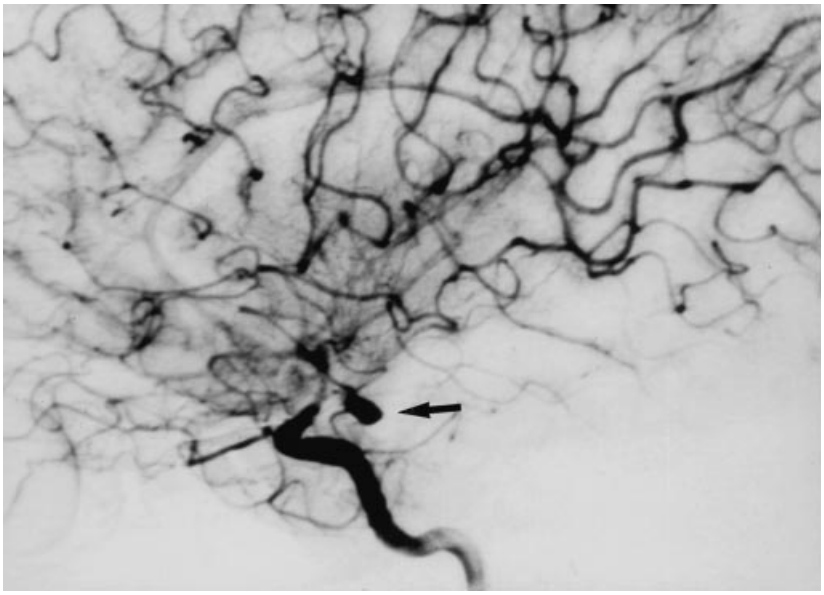

Figure 12 Cerebral angiography showing an aneurysm of the right internal carotid artery (arrow).

hypotension (mean arterial pressure ± 75 mmHg) and hyperventilation (arterial carbon dioxide pressure ± 36 mmHg). Owing to fetal bradycardia, the indication for scheduled craniotomy was revised and emergency Caesarean section was performed. The neonate was delivered 70 min after induction of anaesthesia; the Apgar scores were 2 and 7 at 1 and 5 min, respectively. Following intubation and ventilation, the neonate emerged from anaesthesia 12 h later. After Caesarean section, an external ventricular drainage system was installed and craniotomy and clip obliteration of the aneurysm were carried out consecutively. Due to the intracranial oedema, the patient was sedated with midazolam and fentanyl and hyperventilated for the next 5 days. Postoperative course was uneventful. Only minor neurological deficits were recognised which completely recovered within the first 2 months postoperatively.

Aneurysmal subarachnoid haemorrhage during pregnancy is an infrequent complication, presenting a hazard to both the mother and the fetus. Over 80% of aneurysmal subarachnoid haemorrhages during pregnancy occur during the second and third trimester [4]. Successful treatment requires thorough diagnostics and close monitoring in a flexible teamwork to address both the varying maternal and fetal needs. The fundamental aims of anaesthesia are to maintain oxygenation and stable systemic, cerebral and placental haemodynamics and to avoid increased intracranial pressure. In the situation of acute fetal distress, as occurred in the present case, one must, at any time, be prepared for immediate Caesarean section. Under these circumstances, it has proven helpful to have a special neonatal care team and unit to care for the 'co-anaesthetised' baby delivered at an unexpected time during the procedure. We strongly encourage the development of working relationships for guidelines for the management of aneurysmal subarachnoid haemorrhage and similar cerebrovascular diseases during pregnancy.

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Peripartum general anaesthesia without tracheal intubation

Ezri *et al.* recently reported ketamine-induced anaesthesia without tracheal intubation during obstetric procedures in the peripartum period. The observed risk of aspiration was comparable to that occurring in the general surgical population (Ezri *et al.* Anaesthesia 2000; **55:** 421–6). Nowadays, in many European countries, ketamine is administered, if at all, under emergency conditions in trauma patients. In areas with a lack of specialist staff and limited equipment, as is common in the developing world, ketamine may still serve as a sole anaesthetic during various surgical procedures [1, 2]. It produces a state of deep analgesia and dissociative anaesthesia in non-ventilated patients with preserved brainstem reflexes [3]. I wish to report my experience with ketamine-based primary anaesthesia at Rushere Community Hospital, Mbarara District, Uganda.

From November 1992 to October 1994, a total of 65 operations and 347 minor surgical interventions were performed; 34.9% of them were obstetric procedures. A single shot of ketamine was sufficient for short manoeuvres, e.g. incision and drainage, removal of foreign bodies or painful redressing in burn patients. Repeated doses of ketamine were given in lower-segment Caesarean section (LSCS), laparotomy, emergency trepanation, and dilatation and curettage. The drug was generally available, cheap and easy to store. In most patients, a dose of 0.5 $mg.kg^{-1}$ was sufficient to induce general anaesthesia. Additional doses for maintenance were half the initial dose at 10–15 min intervals. During anaesthesia, patients breathed ambient air. It was difficult to assess the accurate stage of ketamine-induced anaesthesia. Monitoring during anaesthesia was restricted to intermittent measurement of blood pressure, pulse rate and respiratory rate. Slight increase in muscle rigidity, blood pressure, heart rate and respiratory rate were frequently detected. Excessive salivation required atropine. Eye opening, nystagmus and spontaneous movements were common. Frightening hallucinations were infrequent and, when present, not completely controlled by benzodiazepines. Generally, in LSCS diazepam was not administered before the umbilical chord was clamped [4]. Consequently, neonates delivered by LSCS did not show drug-induced depression of Apgar score as compared with spontaneously delivered babies. Despite the absence of monitoring with ECG and pulse oximetry, there were no life-threatening complications, e.g. laryngospasm or aspiration associated with ketamine-induced anaesthesia [5].

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Pressure ulcers during labour: the effect of epidural analgesia

We were interested to read the recent letter regarding the occurrence of pressure ulcers during labour (Offori & Popham. *Anaesthesia* 2000; **55:** 194). We would like to reassure them that they are not alone in this problem. We would also like to share our experience of this problem, particularly because it is not confined to the labouring parturient as can be seen from another letter in the July issue of this journal [1].

The phenomenon of pressure ulceration occurring in patients with 'low-dose' epidurals is on the increase within maternity units across the country [2–6] and will probably be seen on other hospital wards too. However, this is not new and was reported as long ago as 1985 [7, 8].

In 1997, five cases of pressure ulcers, which appeared during labour, were investigated in the Royal Cornwall Hospital. In all cases, the women had normal vaginal deliveries with epidural analgesia for pain relief. In all cases the epidural was low-dose bupivacaine with opioid given intermittently rather than by infusion. Since then, anecdotal evidence has been gathered from 13 hospitals around the country, which

had also discovered similar problems. Our main conclusion was that direct pressure alone is not the major cause of intrapartum pressure damage.

For many hospitals, pressure area care within maternity units is not considered as important as other aspects of care; it is not expected that healthy young women in childbirth will develop pressure ulcers. Specific incidence monitoring often excludes maternity wards for this very reason. Friction and shear forces can destroy the microcirculation within the epidermis especially in sodden skin tissue, which is highly mobile, such as the buttocks of women in labour. This combination may cause damage within a matter of a few minutes in some cases [9]. The continued use of plastic draw sheets and poor quality incontinence pads further increases friction and shearing by adhesion to the skin and trapping moisture. We recommend that their use be discontinued.

In our experience, the quality of the delivery mattress further increases the risk of pressure ulcer formation. Some mattresses are too hard or too soft and some have a poor shape. Both these factors increase either direct pressure or shearing forces or both. Combine this with lack of knowledge of the problem, and we have a recipe for the continuing occurrence of pressure ulceration. All these risk factors are worsened in the presence of reduced sensory perception that exists during epidural analgesia: tissue pressure, friction and shear all remain unrelieved in the absence of movement induced by pain.

In our unit, we have stopped using certain delivery mattresses and incontinence sheets and, more importantly, developed a training awareness programme specifically to target problem areas within the hospital. In the labour ward, an assessment of risk using simple trigger factors has proved most effective. Education and training for midwives should be available and relevant to the women's needs. There is very little evidence to suggest that epidural analgesia in labour, even using 'low-dose' techniques, may be a factor in the formation of pressure ulcers. There are many variables that affect tissue tolerance and it is vital that each woman is assessed individually and a care plan devised accordingly. A dynamic and flexible approach to the care of the woman in labour, especially one with an epidural for pain relief, is essential. We have seen no further problems since the five cases identified in 1997.

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A novel method for epidural catheter fixation

Epidural catheter migration is a common problem that can inconvenience the anaesthetist and have potentially devastating consequences for the patient. Following insertion into the epidural space, the catheter can be simply taped with an adhesive dressing or tunnelled subcutaneously to reduce the incidence of migration [1]. Numerous fixation devices have been designed to tether the catheter to the skin, including simple adhesive devices [2] and more complex catheter clamps that adhere to the skin (Lockit®, Portex, UK). Devices such as a balloon-tipped catheter (Patent No. US4973305) and a self-retaining catheter with wings that can be expanded outwards at the tip (Patent No. EP0931559) have not reached the market. It appears that the weak link in adhesive catheter fixation techniques is in the tethering of the adhesive to the skin. Perspiration and shear forces combine to reduce adhesion over time. Directly suturing the catheter to the skin can cause occlusion and fracture of the catheter wall and so is not routinely used [2]. We describe a simple, cheap and highly effective fixation technique that attaches at the point of insertion with a single suture.

A 2-cm length of robust adhesive dressing (Veni-gard, ConMed, USA) is wrapped around the catheter at the point of insertion. Following infiltration with local anaesthesia, a single 2.0 silk stitch is passed through the dressing and sutured to the skin adjacent to the catheter entry site (Fig. 13).

We tested this technique using an *in-vitro* porcine skin model. A weight of one pound (0.45 kg) could be reliably held suspended from the catheter (Fig. 14). Further force applied to the assembly caused the catheter to stretch then ultimately slip from the adhesive tethering. This far exceeds the forces one would expect to be produced in clinical circumstances.

Securing the catheter at its point of entry into the skin does not, however,

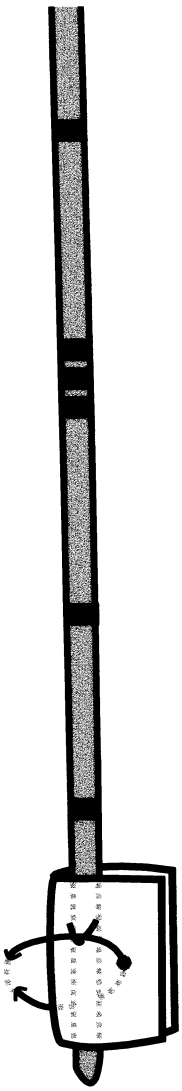

Figure 13

prevent movement of the skin relative to the epidural space. An epidural placed in the sitting position in an obese patient will retract into the subcutaneous tissues on returning the patient to the lateral recumbent due to an increase in the distance between the point of skin puncture and the point of entry into the epidural space [3, 4]. This positional migration is of 1.0–2.5 cm and could itself result in outward catheter migration unless the catheter is stitched in the deflexed recumbent position. Beilin *et al.* showed that a catheter inserted 5 cm into the epidural space of labouring

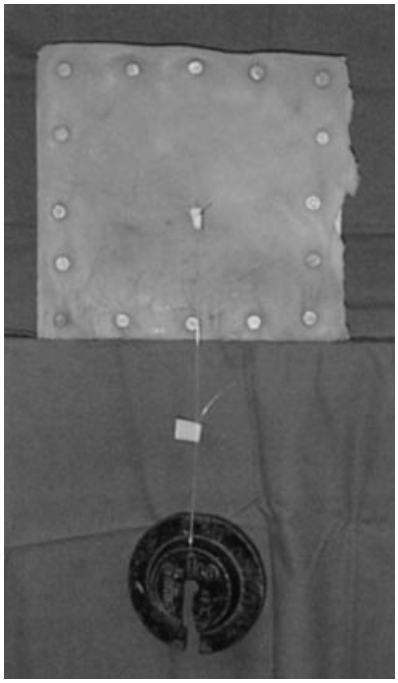

Figure 14

women was associated with the highest incidence of satisfactory analgesia [5]. This length of catheter insertion in a sitting patient with a deflexed back would allow for the possibility of a degree of initial migration of the tethered catheter out of the epidural space. Our clinical experience using the technique in 15 cases has indicated that this simple epidural catheter fixation technique is successful in preventing migration.

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Tunnelled epidurals

Your editorial on tunnelled epidurals (Kumar & Chambers. *Anaesthesia* 2000; **55:** 625–6) was most interesting, although the methods used to achieve this end were rather complex. I have always found the easiest way is to use a tunnelling rod such as the one supplied by B Braun Medical, France.

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Sleepers for body piercing

Metal body jewellery represents a potential hazard to the patient presenting for surgery, either through displacement from sites in or near the oro-nasal cavity [1], or by presenting a potential earth for diathermy. Although plastic sleepers are available, few patients have these available when they present for surgery [2]. Current best practice would suggest that metal body jewellery should be removed prior to the induction of anaesthesia, or where this is not possible, covered with adhesive tape; one or other of these solutions is possible in the vast majority of cases. Occasionally, the piercing is situated in the surgical field or is too recent for the subcutaneous track to have fully epithelialised, and on these (rare) occasions I would suggest that the intravenous portion of a cannula does the trick nicely (Fig. 15). Care should be taken when removing the metal ring or stud in a recent piercing, as it

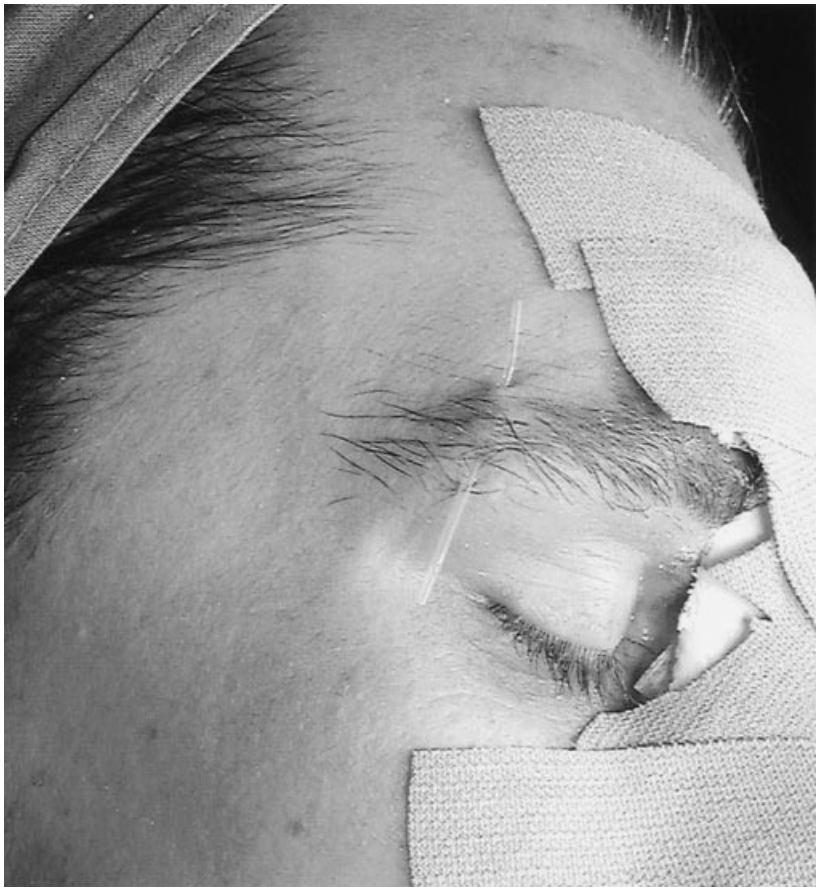

Figure 15

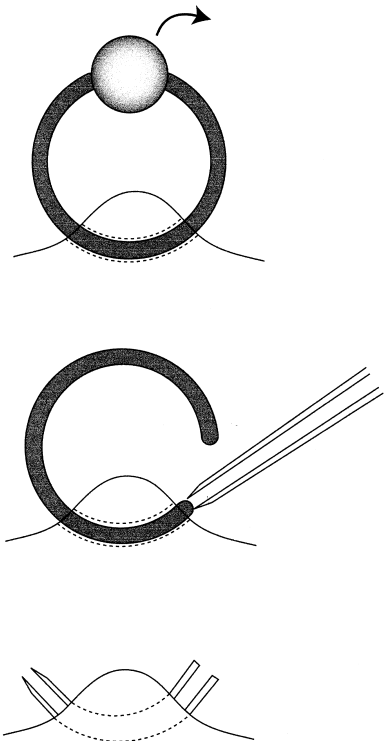

Figure 16

can be quite impossible to recannulate the subcutaneous track. In these cases the plastic cannula can be guided into place by choosing a cannula diameter that is the same size as the ring or stud, or only slightly smaller, and then use the ring or stud to guide the cannula into place (Fig. 16). Once in place the hub of the cannula can simply be cut off.

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Erratum

In the correspondence 'Pressure sore following low-dose epidural infusion' (*Anaesthesia* 2000; **55**: 709–10), the rate of epidural infusion of bupivacaine 0.1% and fentanyl 2 $\mu g.ml^{-1}$ should read 10 $ml.h^{-1}$ and not 10 $ml.min^{-1}$ as published.