

Correspondence

Research output in anaesthesia

A recent letter studied different countries' positions in the field of anaesthesia in the period 1991 to year 2000 (Rahman *et al.* *Anaesthesia* 2002; 57: 1213–14). Among other things, the analysis elicited the top ranking 20 countries for research in the field of anaesthesia. Also, they revealed countries where research shares went down and up, respectively. The results were interesting, but one sentence puzzled me. The authors write: 'Journals not available in the Medline database for the entire 1991–2000 period were excluded (Acta Anaesthesiologica Scandinavica and Clinical Journal of Pain)'. To my knowledge, which is substantiated by a recent search in Medline, both journals have been available in the Medline database for the entire period (Table 1).

I would be pleased if the authors could explain this difference.

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A reply

I apologise for our error and am grateful to Professor Viby-Mogenson for pointing out the inaccuracy in our letter. Our library's Medline was having difficulty and we failed to find Acta Anaesthesi-

Table 1 Published and registered articles according to Medline database in Acta Anaesthesiologica Scandinavica and Clinical Journal of Pain in the years 1989–2002.

Year	Acta Anaesth	Clin J. Pain
1989	143	80
1990	139	53
1991	161	65
1992	166	57
1993	156	47
1994	173	47
1995	216	49
1996	219	48
1997	257	50
1998	227	54
1999	207	48
2000	219	76
2001	234	70
2002	240	55
Total	2751	799

ologica Scandinavica and Clinical Journal of Pain online.

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Pandora's Box contains no easy solution

The recent editorial (Reade & Young *Anaesthesia* 2003; 58: 1–3) highlights the problems associated with critical care research. Although it concentrates on observational studies, the issues are equally applicable to therapeutic studies.

Both the legal and ethical issues arise because of the need to obtain the subjects' consent prior to being recruited to any research study. The dilemma facing researchers is that most critically ill patients lack the capacity to give (or refuse) consent.

The four possible solutions mentioned all have their limitations:

1. Retrospective consent. To be meaningful, consent must be given in advance. Once an act has been performed, it cannot be undone and retrospective consent cannot respect the person's autonomy. Any harm, no matter how small in the eyes of the researcher, has been done.

2. Prospective consent. Identifying an 'at risk' group and prospectively consenting them for a study may be possible within a defined population (e.g. those undergoing coronary bypass surgery) but is impractical and unethical in most situations. A blanket 'consent everyone and hope enough get ill' approach is unlikely to be successful. How will you identify those who have, or have not, consented? Are people going to make a truly informed decision about participating in a study if the chances of them actually participating are slight?

3. Proxy consent. As Reade and Young say, this is legally unacceptable in England, Wales or Northern Ireland, but since July 2002 proxy consent for research has been permitted in Scotland under the Adults with

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Incapacity (Scotland) Act 2000 [1]. There are strict criteria for permitting research on those unable to consent for themselves. These include that there is no alternative group of patients, minimal additional risk or discomfort and that consent has been obtained from a guardian or next of kin. The Multicentre Research Ethics Committee for Scotland must review all applications for research studies involving incapacitated patients. Whilst this may not be the perfect answer it is happening within the UK and may guide future developments elsewhere.

4. Removal of need for consent. This has been introduced in the US to facilitate research in the emergency situation where the patient is unable to give consent and there is no time to approach the proxy. It is specifically intended for emergencies such as the treatment of cardiac arrest or head injury research. Each study requires a specific application to the Food and Drug Administration (FDA) for the omission of consent to be approved. It would not be applicable to most critical care research being discussed by Reade and Young. Their suggestion that individuals should have to accept loss of autonomy as to whether or not to participate in research as the price for state-provided health care is unlikely to be acceptable to the public.

We agree that research on critically ill patients is essential and that most issues of consent need discussion. The use of proxy consent does seem the most promising route for most cases and we would encourage our colleagues to see how the situation develops in Scotland. Removal of the need for consent is a potential remedy for true emergency research but not for what most of us do.

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- 1 Adults with Incapacity (Scotland) Act 2000: http://www.scotland.gov.uk/health/cmo/incapacity_act_toc.asp.

Benefits of off-pump coronary artery surgery?

As rightly suggested by Heames *et al.* in the recent review of off-pump coronary artery bypass surgery (*Anaesthesia* 2002; 57: 676–85), further work is required to clarify certain aspects of the efficacy of off-pump surgery. In particular, the issue of postoperative cognitive function remains unresolved (Singh. *Anaesthesia* 2002; 57: 1219). However, since the review was published there have been several studies published comparing on- and off-pump technique and we feel these should be brought to your readers' attention.

In April 2002, the findings of two major studies were presented. Firstly, Angelini and colleagues published a pooled analysis of the Beating Heart Against Cardioplegic Arrest Studies (BHACAS) 1 and 2 [1]. These two studies were prospective randomised trials with a total of 200 patients undergoing off-pump and 201 on-pump coronary artery surgery between March 1997 and November 1999. Notably, they excluded patients with a history of stroke or transient ischaemic attack (TIA). They demonstrated significant reductions in the incidence of atrial fibrillation, chest infection, inotropic support and red cell transfusion, and the number of patients requiring more than 1 day on the intensive care unit and more than a total of 7 days in hospital in the off-pump group. Only gross neurological events such as TIA or stroke were recorded and there was no difference in the incidence of these between the 2 groups. Second, in a less well-designed, non-randomised study of on- vs. off-pump surgery in 300 patients (150 in each group), there were significant reductions in the time to extubation, intensive care and hospital lengths of stay in those patients undergoing off-pump surgery [2]. However, the initial off-pump patient population had only 1 or 2 grafts performed due to lack of surgical experience with the technique. Again, there was no difference in the incidence of postoperative stroke, but this end-point required radiological confirmation by way of CT scanning. Unfortunately, neither

study made any attempt to measure the incidence of cognitive dysfunction.

The study of Van Dijk quoted by Dr Singh is probably the best-designed study to date addressing the issue of cognitive dysfunction following coronary artery surgery [3]. A total of 281 patients with a mean age of 61 years were randomised to either on-pump ($n = 142$) or off-pump ($n = 139$) surgery. There was a small, but non-significant reduction in the incidence of cognitive dysfunction 3 months after surgery in the off-pump group (21% vs. 29%). This was not evident at 12 months. Again, many patients had only single or double-vessel disease and their anaesthetic management was not standardised.

Most interestingly, Zamvar and colleagues recently demonstrated a dramatic reduction in cognitive dysfunction after off-pump surgery [4]. In their randomised comparison of 60 patients undergoing either on- or off-pump surgery, the incidence of cognitive dysfunction was only 27% and 10% in the off-pump group vs. 63% and 40% in the on-pump group at 1 week and 10 weeks, respectively. Anaesthesia was standardised and all patients had triple vessel disease with a similar number of grafts in each group. This study, though well matched for patient characteristics, comprises a small low-risk group for cognitive dysfunction with a mean age of 62 years. Nevertheless, the findings are significant and emphasise the need for a larger trial, which should be designed with a more precisely defined cardiopulmonary bypass management protocol and intra-operative haemodynamic management in the off-pump group. Whilst these findings are encouraging for the advocates of off-pump surgery, it must be remembered that there is a significant incidence of cognitive dysfunction after major non-cardiac surgery.

The International Study of Postoperative Cognitive Dysfunction reported an incidence of 25.8% at 1 week after surgery and 9.9% at 3 months after surgery [5]. Clearly, a large and well-designed trial is still needed to see whether off-pump surgery, perhaps by total arterial revascularisation avoiding

all aortic manipulation as advocated by Taggart [6], can reduce the incidence of cognitive dysfunction comparable to that seen after non-cardiac surgery.

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Anaesthetic equipment – familiarity is comforting, but check it

Whilst preparing to anaesthetise an ASA grade-II woman, weighing 67 kg, for a hysteroscopy, I requested a standard size-4 laryngeal mask airway, appropriate for her weight and build. Due to the lack of standard laryngeal masks, my

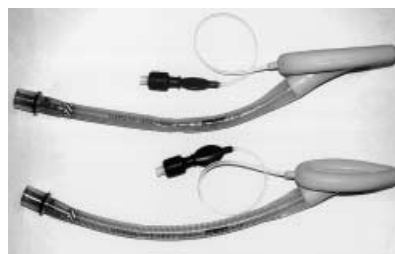


Figure 1

anaesthetic assistant returned with a reinforced size-4 laryngeal mask and prepared it for use. Monitoring was attached, intravenous access secured, and pre-oxygenation was started. It was during this stage of the induction I noticed that the laryngeal mask looked 'small', contrary to its labelling and packaging. No intravenous drugs or inhalation agents had yet been given. A new reinforced laryngeal mask was prepared, and induction of anaesthesia continued. The subsequent anaesthetic and surgery was uneventful. At the end of the list, the size 4 reinforced laryngeal mask was examined, and found to be size 3, which was confirmed by comparison. Figure 1 shows the mislabelled laryngeal mask, together with an actual size 4 one above. Both are labelled 'size 4r'. It has subsequently been withdrawn from circulation.

The laryngeal mask is one of the most familiar pieces of equipment to anaesthetists, in its various forms and sizes, and is used in elective and emergency situations. Frequently, equipment familiarity can reveal problems/errors even when it appears that there are none, as this incident demonstrates. My normal practice is to use size 4 laryngeal masks for adult females, and size 5 laryngeal masks for adult males, assuming no contraindications. I could have continued with the size 3 laryngeal mask without incident. However, this case highlights that, although familiarity is comforting, it should not allow us to forgo our standard checks. It is still important to check all equipment prior to induction of anaesthesia.

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Fibreoptic equipment and prion disease

We read with interest the recent letter (Dombrowski *et al.* *Anaesthesia* 2003; **58**: 90–1) relating to the incineration of fibreoptic equipment following its use in a patient who was considered to be at risk of transmitting new-variant Creutzfeldt-Jacob disease (vCJD). We would like to report a similar experience and suggest a possible solution to this problem.

One of us was asked to provide anaesthesia for an 18-year-old male patient, with a definite diagnosis of vCJD, who required insertion of a CSF reservoir. Pre-anaesthetic assessment of the airway indicated poor mouth opening and problematic dentition. Previous procedures included tonsil biopsy and insertion of a percutaneous endoscopic gastrostomy (PEG) tube. It was clearly understood that all equipment coming into contact with the patient would be incinerated at the end of the procedure. A 6-year-old fibreoptic laryngoscope, coming to the end of its usefulness, was available. Anaesthesia was induced using an infusion of propofol and use of the bag and mask easily achieved manual ventilation. Vecuronium was given in an attempt to relieve the muscle spasm but mouth opening did not improve and it was impossible to insert a laryngoscope or a laryngeal mask. Tracheal intubation was achieved with a 6.5-mm reinforced tube passed nasally over the fibreoptic laryngoscope and the operation proceeded uneventfully. We assume that the highly restricted mouth opening was secondary to disuse (no speech and PEG feeding) possibly exacerbated by muscle spasm. Myoclonus is a feature of vCJD.

We considered retaining the fibre-scope for use if this patient required further operative intervention. However, the Infection Control department were adamant that if the scope was retained, it would have to be cleaned. This would contaminate the disinfecting machine worth approximately £15 000. The old fibrescope was therefore incinerated.

We understand that the CJD Surveillance Unit based at the Western

Infirmiry, Edinburgh retains a gastro-scope that may be used for PEG tube insertion in patients with probable CJD. Clearly laid down criteria must be met before such equipment is made available and there are mechanisms in place for transport and cleaning of the endoscope. Since this precedent already exists, should a similar arrangement be instigated for airway equipment? Obviously, this would only be possible when the operative procedure was elective and difficulty with the intubation was anticipated. Given our experience, perhaps difficult intubation should be expected in patients with advanced vCJD. The Difficult Airway Society could liaise with the CJD Surveillance Unit to co-ordinate such a scheme.

It is likely that there will be an increased involvement by anaesthetists with patients who have a definite or potential diagnosis of CJD. In addition to published work on this topic, we would recommend <http://www.priordata.org> as a useful source of information.

Name and address supplied

Inadequate ventilation with the LMA ProSeal™

The most recent development in the evolution of the laryngeal mask airway is the introduction of the LMA-ProSeal™ laryngeal mask airway (PLMA). The PLMA includes a gastric access tube that allows for easy passage of a gastric tube and also serves as an escape conduit for regurgitant fluid, thus preventing aspiration. Additionally, some changes to the mask and cuff design allow for pressure control ventilation with higher airway pressures than the classic laryngeal mask. Trials conducted in Europe indicate that the PLMA has strong potential for clinical use, as it offers advantages over the laryngeal mask airway in most technical aspects of airway management in paralysed patients undergoing pressure control ventilation [1].

We wish to report a case where we had difficulty ventilating a paralysed patient with the PLMA. A 48-year-old, fit, ASA 1 male of oriental origin

presented for left ureteroscopy and basket lithoclast of a ureteric stone. Pre-operative airway assessment was unremarkable. Anaesthesia was induced with fentanyl 100 µg, propofol 200 mg, muscle paralysis achieved with rocuronium 40 mg, and anaesthesia maintained with isoflurane in 35% oxygen and nitrous oxide, morphine (10 mg) was used for further analgesia. A fully deflated size-4 PLMA was inserted easily with the aid of the introducing device and the cuff inflated with 30 ml of air as recommended. On instituting ventilation with the reservoir bag using a Bain circuit, the chest did not seem to inflate and a leak was apparent. Further inflation of the PLMA cuff with 10 ml of air did not rectify the problem. The PLMA was withdrawn and repositioned with the same problem recurring. On closer inspection, we could feel and hear air venting out from the gastric port of the PLMA. The leak led to the failure of chest inflation with ventilation, which was confirmed with the absence of capnography (Fig. 2). We rectified the problem by occluding the gastric outlet with a bung (Fig. 3). Inflation of the chest was then achieved with a normal capnographic trace (Fig. 4). We also checked for gastric distension, which was absent, and the surgery proceeded without any untoward effect. On extubation, there was no evidence of gastric secretions in the oral cavity.

There have been reports of oesophageal insufflation or aspiration of air through the drain tube [2,3] and of oesophageal breathing and upper airway obstruction [4]. Although we may have had oesophageal breathing, our case did



Figure 2



Figure 3



Figure 4

not have airway obstruction as evidenced by the ability to ventilate after occluding the drain tube. A similar case has not been reported and it would be interesting to hear if anyone else has had a similar problem.

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Natural airway before artificial airway

The letters responding to the editorial (Yentis. *Anaesthesia* 2002; 57: 105–9) on predicting difficult intubation made depressing reading. In a follow-up reply (Yentis. *Anaesthesia* 2002; 57: 613–14), the author in summarizing the work of Langeron *et al.* [1], did not point out that their conduct of mask ventilation omitted mention of head/neck positioning, oral airway and jaw prognathism use, face mask selection, anaesthetic circuit, anaesthetic, and rationale for 10 l.min⁻¹ gas flow, 4 min ventilation, and the many unparalysed patients. What is learned from such work? Similar criticism [2–4] has been levelled at references cited in the editorial and as a result, I have never used any of the tests or scoring systems. Laryngoscopy involves: the sniffing position, good relaxation, correct technique (holding handle, pull direction, tongue control, and blade position in mouth), Macintosh 4 blade (or Miller or other blades) if Macintosh 3 blades inadequate, blade put posterior to the epiglottis if anterior not sufficient, firm backward thyroid cartilage pressure, and a trial of increased neck flexion [5], etc. Apart from El-Ganzouri *et al.* [6] who seem to have employed all except the last, other workers mention only a few of them or nothing at all. Based on such work, have examinations been failed or sanctions applied? The lesson is that it pays to read the methods section of articles very carefully and critically.

In their respective letters (Calder. *Anaesthesia* 2002; 57: 612, Das & Pearce. *Anaesthesia* 2002; 57: 824, Cheater & Jameson. *Anaesthesia* 2002; 57: 615), Calder uses the word 'airway' nine times, Das & Pearce six times, and Cheater and Jameson five times, but was their meaning 'laryngoscopy' and/or 'intubation'? Yentis, in his editorial,

concludes that attempting to predict difficult intubation forces the anaesthetist to think of the airway. In general, it seems these, and other terms, are used synonymously, interchangeably, and inappropriately [7]. This follows from the way the Practice Guidelines for management of the difficult airway are defined [8,9]. More careful, specific, and unambiguous word use might produce more understanding and less confusion.

We breathe through our natural airway. If we snore when asleep it is our natural airway that obstructs. During mask anaesthesia, as Safar, Escarraga & Chang showed [10], certain measures are necessary to retain a clear (patent) natural airway, i.e. head extension and/or artificial oropharyngeal airway and/or forward displacement of the mandible. However, some patients may stay partially obstructed or prove very hard work to prevent it. In a rare patient, it may not be possible to acquire an open airway. What should these be named but (I) 'Difficult (natural) Airway'? On the other hand occasionally after induction, we find it hard to visualise part or any of the glottis, i.e. (II) '(Primary) Difficult Laryngoscopy'. I and II presumably arise from different expressions of anatomical variation of the natural airway because, fortuitously, they rarely present in association. If the measures to maintain a patent natural airway are restricted or prevented we have (III) 'Compromised Control of the (natural) Airway'. Finally, when the lumen is reduced by a cause outside, inside, or in the wall of the natural airway, we are facing (IV) 'Compromised (natural) Airway'. These discrete groups [7] can occur in combination. Each has different causes and implications. In I, II, and III, the natural airway is essentially normal. For III and IV, with progressive depression of consciousness, the potential for obstruction of the natural airway increases and the control available to prevent it is reduced. Also secondary difficult laryngoscopy can coexist. Usually III and IV are known about beforehand with the opportunity to plan, prepare, and approach appropriately, whereas I and II are invariably discovered after induction

has taken place. Unfortunately, adverse events reviews have not given such information or the detail to evaluate the cases oneself.

'Difficult Intubation' is not synonymous with (II) 'Difficult Laryngoscopy', nor 'Difficult Mask Ventilation' with (I) Difficult (natural) Airway. They are harder to define because they incorporate additional factors remote from the natural airway and a wide range of difficulty. Difficult Intubation can apply to laryngoscopic, blind nasal, lighted stylet, fibrescopic, retrograde guided, etc. and should be so qualified. Difficulty is potential before the event, and only true, or not, after. Difficult laryngoscopic intubation can arise from poor illumination, gap between upper teeth, type of tube, or incongruence between the axis of the tube and the axis of the larynx, etc. Difficult Mask Ventilation can be caused by facial hair, facial shape, obesity, rigid chest, tense abdomen, etc., of the patient, and factors related to apparatus and anaesthetist.

The tracheal tube airway (oro/nasopharyngeal airway, laryngeal mask airway, combitube airway, etc.) is an internal artificial airway device inserted into the natural airway for the purpose of resuscitation/ventilation or anaesthesia/surgery. Delay may be impossible for the first but not with the second. In either case, the natural airway may be normal or impaired. Always the anaesthetist has the dichotomy of two distinct tasks, and thus assessments: (a) preserve the natural airway until the artificial device is correctly positioned, and (b) decide the device, and technique, for insertion. Therefore (a) not (b) decides the approach to anaesthesia [11]. The natural airway can be kept by awake spontaneous breathing, asleep spontaneous breathing, asleep assisted mask ventilation, or asleep full mask ventilation (in which rarely difficult mask ventilation occurs). Very rarely internal artificial airways do not suffice and resort is made to external devices (cricothyroidotomy or transtracheal ventilation).

Long ago, Donlon [12] categorised management of the compromised airway (IV) according to severity and a

similar approach can be used for (III) compromised control group. For every case, the anaesthetist has to consider intravenous induction, or inhalational induction, or awake intubation (or other device), or awake surgical airway. The choices will be assessed on the history, physical examination, indirect laryngoscopy, fiberoptic nasopharyngoscopy, and X-rays (plain, tomography, CAT, MRI). The anaesthetist can also opt for diagnostic awake direct or fiberoptic laryngoscopy.

In the absence of apparent abnormal features, an intravenous (and relaxant) induction is invariably given. Failing to intubate may delay the surgery temporarily, but failing to oxygenate can delay it forever. Benumof and Schellen [13] stated an approximate 'cannot intubate/cannot ventilate' incidence in their hospital during six years, as 1 per 10 000 anaesthetics. Their definition of 'cannot mask ventilate' was a fully trained and/or reasonably experienced anaesthetist couldn't cause a life-sustaining amount of gas exchange to occur. They also quote an incidence of 0.01–2.0 per 10 000 from other sources. El-Ganzouri *et al.* [6] reported just 8 cases of difficult mask ventilation (inability to obtain chest excursion sufficient to maintain acceptable capnogram waveforms) in 10 507 consecutive patients. Surely, this is why 'trainees know that if they use a routine intravenous induction everything will usually be fine' (Yentis. *Anaesthesia* 2002; 57: 613–4)? Also it is why Kristensen's [14] suggestion 'in 10 000 patients, 1616 patients are intubated awake' is illogical, unnecessary, and horrifying. Mouth opening, head/neck movement, ability to prognath, thyromental distance, bodyweight, etc., are as significant to maintaining an unobstructed natural airway as to laryngoscopy, intubation, and other transoral devices.

It pays to study the internal anatomy of the natural airway of every patient when performing laryngoscopy and intubation. Not only is the tracheal tube more likely to finish in the trachea, and any pathology be noticed, but the variations in the size of the pharynx, and the length, width, shape, and position (from behind or touching the

uvula to a long distance from it) of the epiglottis will be learned. How do they correlate with external features? In rare subjects, after induction, the epiglottis may possibly obstruct the distal pharynx and glottis making artificial devices and mask ventilation fail. The ability to predict such cases is unlikely.

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Radiographic guided epidural placement

The experience of facet joint biopsy during attempted thoracic epidural catheter placement in an asleep patient (Picard. *Anaesthesia* 2002; **57**: 1233) will not be unique. The idea of a large gauge needle being 'wielded' blind into the anatomically abnormal spine of an unconscious patient is truly terrifying.

Patients who present difficulties in establishing effective epidural analgesia include those with anatomical variation, e.g. morbid obesity or scoliosis, degenerative change, e.g. spondylosis or osteoporotic collapse, where there is severe distress or anxiety and where there has been previous failure to catheterise the epidural space. Often they have the most to gain.

We have continued to develop the technique of radiographic-guided placement into routine practice since our previous report [1]. Our series of 38 challenging patients includes only two failures in which the interspinous and interlaminar spaces were obliterated: one patient with advanced ankylosing spondylitis (another with the same condition had an uneventful placement) and one patient with advanced osteoporosis and spondylosis.

We use the paramedian approach with the patient asleep in the lateral flexed position on a standard operating table with a horizontal X-ray beam. We can be certain of the exact location of the needle tip relative to the bony landmarks at the appropriate spinal level and can check any loss of resistance with injection of radiopaque contrast medium. The course of a radiopaque catheter can be followed before finally

performing an epidurogram to confirm appropriate spread of contrast solution. We feel that this visualisation much improves efficacy and safety whether the patient is awake or asleep.

One of us, a non-pain specialist anaesthetist, quickly learnt the technique and we are called on to assist with cases on other anaesthetists' lists. With planning, access to image intensifier equipment is usually not a problem and overall there is considerable time and distress saved.

Thoracic epidural analgesia is likely to improve outcome, particularly in high-risk patients; however, safe, accurate placement is essential. We recommend that anaesthetists consider using X-ray guidance where difficulty can be anticipated.

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Complication of the combined spinal epidural technique 1

We would like to report a problem with the needle through needle combined spinal-epidural technique using the RapID Extra-length Pencil Point Spinal Needle, 26G (Portex).

A 22-year-old primigravida presented for elective lower-segment Caesarean section. We decided to perform a combined spinal-epidural anaesthetic. She was fit and well with no past medical or surgical history.

The epidural space was located easily in the midline position at 5 cm from the skin with a 16G Tuohy needle using a loss of resistance to saline technique. The RapID Extra-length spinal needle was introduced easily through the Tuohy needle until a 'click' was felt. After several attempts it was impossible to remove the stillette, and consequently

we removed the needle as a whole. For reasons that were not clear the needle had bent acutely.

A second pencil point spinal needle was inserted and a successful block was established. The epidural was tested postoperatively in the recovery area and found to be working well. The patient did not complain of symptoms or demonstrate any signs of dural puncture headache, or any other complications during the follow-up period and was discharged home, fit and well.

While common sense dictates that some needle deformity is inevitable during insertion, our acute bending of the needle happened during an apparently atraumatic insertion. Our main concern was shearing of the needle tip on withdrawal, with the resulting foreign body within or near the intrathecal space. A significant dural tear whilst removing the bent needle could have occurred.

The problem has been reported to Portex who have yet to reply to our concerns. We wish to warn others of this particular problem in relation to difficulty in removing the stillette.

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Complication of the combined spinal epidural technique 2

We would like to highlight a potential problem we encountered using the needle-through needle Sims/Portex spinal epidural set (16G 80 mm Tuohy 26G 105 mm Whitacre –[100/396/816]).

A 68-year-old female wished to remain awake for her right total hip replacement. A combined spinal epidural technique was chosen as being suitable for the procedure and for providing postoperative analgesia. Following intravenous access and establishment of full monitoring, an epidural was performed in the sitting position under aseptic conditions. At the L3–4 interspace, loss of resistance to saline was encountered at 60 mm, the spinal

needle was inserted and at 100 mm, resistance to further advancement was felt. No pain or paraesthesia was noted. The stylet was withdrawn with some difficulty but no flashback was seen. The spinal needle was pulled back 5 mm but still no cerebrospinal fluid was seen. Further withdrawal then became impossible. The procedure was abandoned and both needles were withdrawn together. Figures 5 and 6 show that the spinal needle had kinked and on closer inspection, the tip was distorted and appeared to be at risk of shearing off.

The spinal epidural was repeated, this time uneventfully using separate spaces (epidural 16G Tuohy at L2–3 and spinal 24G Whitacre at L3–4). Surgery and recovery proceeded uneventfully.

Whilst we are aware of the low likelihood of metallic fragments being deposited from friction between the two needles [1], we are not aware of this particular complication being described previously. Fortunately, in this episode we were able to recover the spinal needle intact on the first attempt. We acknowledge that the kinking can occur with all techniques where a fine gauge

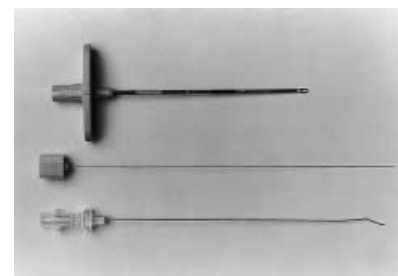


Figure 5



Figure 6

spinal needle is used, but the combined spinal epidural needle through needle technique means that there is a real possibility that the spinal tip could be sheared off especially if there are repeated attempts at removal, unrecognised distortion of the spinal needle, if the Tuohy needle has the spinal exit portal at the curved Huber tip and as the spinal needle gets finer.

We therefore recommend that if there is resistance to withdrawal of the stylet the procedure should be abandoned and both needles taken out together and carefully inspect the kit. Consider using two separate needles either in separate spaces or a single space, locate the epidural space first then perform the spinal anaesthetic and only then thread the epidural catheter to avoid potential puncturing of the catheter [2]. Alternatively, we could use kits where the epidural needle has a backeye or is double barreled (e.g. EspocanTM by Braun).

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A reply

Thank you for the opportunity to reply to these two letters. As Dr's Loc, Kirk, Sturgess and Brown point out in their letters damage or deformity to finer gauge spinal needles is generally accepted as a potential hazard of the combined spinal epidural technique.

The most likely explanation for these unfortunate events is the tip of the spinal needle contacting bone during insertion, indicating that the epidural needle was not correctly located. A review

of the clinical literature supporting this subject suggests the needle-through needle technique remains the most popular CSE technique [1]. Many thousands of procedures have been performed using this technique which is reported as being safe and efficacious [2–4]. If performed correctly, the epidural needle will reach the epidural space in the mid line without encountering obstruction.

The importance of communicating the danger of spinal needle deformation has increased over the last decade, as clinicians have demanded finer gauge needles from medical device manufacturers. Portex continues to provide clear warnings in the products' instructions for use, alerting users to this potential danger. In response to Drs Loc, Kirk, Sturgess and Brown's letters, we are reviewing the clarity of all our product instructions for use and safety recommendations supplied with Spinal needles and CSE kits.

All Portex RapID spinal needles supplied in CSE kits are manufactured to ISO 9626 : 1991 and Portex continues to invest in spinal needle research and development to achieve a product specification that delivers optimum performance without compromise to safety. Since Portex launched the 27G CSE kit in January 1999, we have sold many thousands of units and received only one complaint relating to the needle bending during use, none relating to the shearing off of metal fragments.

I would like to thank Drs Loc, Kirk, Sturgess and Brown for their comments, and for raising the awareness of this potential CSE complication. Portex always welcomes feedback from clinicians as this helps us to continually improve the products and instructions for use we provide.

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Loss of consciousness from epidural sufentanil for labour analgesia

We would like to report an unusual reaction to a systemic absorption of epidural sufentanil for labour pain mimicking hypoglycaemia and eclampsia in an Asian parturient. She had a history of insulin-dependent diabetes mellitus, chronic hypertension and hypothyroidism, controlled with nifedipine XL 30 mg a day, synthroid 0.15 mg a day and regular insulin 70 units and 40 units NPH AC breakfast, 60 units regular insulin AC lunch and 180 units regular insulin AC dinner. The patient was admitted to the hospital for induction of labour and delivery with intravenous oxytocin.

Epidural administration for labour pain was unremarkable. A combination of 20 ml ropivacaine 0.1%, sufentanil 1 µg/ml and epinephrine 2 µg/ml was administered in four divided doses every 2 min. The epidural catheter was connected to a pump with an infusion rate of 4 ml.h⁻¹, PCA dose of 4 ml and a lockout time of 10 min.

One and a half hours later, the patient complained of pain, received 10 ml epidural bolus dose and the infusion rate was increased to 6 ml.h⁻¹. Four and a half hours later, the patient began experiencing itching, somnolence, aphasia, tremors, jerky movements of hands

and an inability to speak. Then the patient became unresponsive and had BP 171/95 mmHg, heart rate 150 beat.min⁻¹, and SpO₂ 100% with a normal ECG. The insulin, pitocin, and epidural PCA infusions were discontinued. Ten minutes later, the patient was able to nod her head, the blood pressure at that time was 216/88 mm.Hg with a heart rate of 155 beat.min⁻¹ and her blood glucose was 6.9 mm.l⁻¹.

Eclampsia was suspected and a magnesium sulphate intravenous infusion was started immediately. The patient was examined by a neurologist and an endocrinologist to rule out hyperthyroidism, hypoglycaemia, pre-eclampsia/eclampsia or any neurologic event. The patient had no hyper reflexia and the Babinski test was negative. There were no focal symptoms or postictal symptoms. Her recent thyroid stimulating hormone (TSH) level was 6.20 mUI⁻¹ compatible with uncontrolled hypothyroidism. Blood sugar ranged between 6.5 and 7.2 mm.l⁻¹ after a careful insulin infusion since the start of labour.

Fifteen minutes later, the patient recovered, was orientated, co-operative and complained of pain. She stated that she was awake during this episode, could not talk, was scared, and explained that her jerky hand movements were intentional to alert the staff of her condition. For pain treatment, the patient received two boluses of 5 ml of the epidural solution 2 min apart. The same symptoms returned and were reversed by a total of 0.2 mg of naloxone. A test dose of 3 ml lidocaine 1.5% with epinephrine 5 mg.m⁻¹ was negative. Tachycardia, tinnitus, numbness around the lips, and metallic taste in the mouth were not present. Epidural analgesia for labour pain was preceded with ropivacaine 0.2% 5 ml boluses. Her blood pressure came down slowly but effectively with ropivacaine epidural analgesia. Her vital signs were: BP 167/71 mmHg, heart rate 129 beat.min⁻¹ and SpO₂ 100%. The magnesium sulphate infusion was discontinued at this time.

The patient was later scheduled for Caesarean section for failure to progress. Four doses of 5 ml epidural 2% lidocaine and epinephrine 5 mg.m⁻¹ provided a satisfactory epidural anaesthesia at

T4–S5. Uneventful postoperative analgesia was provided by epidural-PCA ropivacaine 0.1% with epinephrine 1 mg.l⁻¹ at 10 ml.h⁻¹, PCA dose 4 ml, and a lock out time of 10 min.

We reported a similar reaction [1] when post-Caesarean analgesia was provided by epidural-PCA sufentanil 0.8 mg.m⁻¹ bupivacaine 0.01%, and epinephrine 1 mg.m⁻¹ at 15 ml.h⁻¹. When the epidural-PCA was discontinued 48 h from the start of the infusion, the sufentanil plasma level surged. The patients became somnolent, were unable to speak, had jerky movements of their upper extremities and had nausea and vomiting for about 30 min. Several patients stated that they felt as if they were having a stroke. Gradual reduction of the epidural infusion rates over time eliminated this problem. Hamilton *et al.* reported a high sensory block in six patients during intrathecal sufentanil injection [2]. Swallowing difficulties, facial numbness, and agitation were the clinical findings for extreme cephalic sensory changes that showed up around 10–25 min after uncomplicated combined spinal-epidural techniques. However, these symptoms were different from the symptoms we described before and with this case report following the discontinuation of epidural-PCA sufentanil. We suspect a rostral CSF spread of sufentanil for Hamilton's patients, and excessive systemic absorption of sufentanil in our patients. We suspect that in this case, untreated hypothyroidism also played a role in this unusual reaction.

In summary, we report a case of unusual reactions to systemic absorptions of epidural sufentanil and ropivacaine for labour pain mimicking hypoglycaemia and eclampsia in a parturient with a history of diabetes, hypertension and hypothyroidism. This is the first time that we witnessed excessive somnolence and aphasia following the administration of epidural opioids and local anaesthetics for labour pain. This possible reaction should be considered when epidural sufentanil is administered.

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Acute hypoglycaemia in a healthy parturient following induction of a combined spinal-epidural analgesia for labour

The literature concerning acute hypoglycaemia following induction of a combined spinal-epidural (CSE) technique for labour analgesia is limited to one case of a parturient with pre-existing type-2 diabetes mellitus [1]. I herein report a case of a healthy (non-diabetic) parturient that developed acute onset hypoglycaemia following subarachnoid injection of 5 µg of fentanyl in combination with 2.5 mg of levobupivacaine as part of a CSE technique for pain relief in labour.

A 29-year-old, 163 cm, 85 kg, gravida 5, para 3, healthy female at 40 weeks gestation was in active phase of labour (with painful uterine contractions) and requested analgesia. She had no known drug allergies and no prior anaesthesia. Her admission blood pressure was 114/66 mmHg, heart rate was 68 beat.min⁻¹ and respiratory rate was 18 breath.min⁻¹. Fetal heart rate was 140 beat.min⁻¹ and reactive. The course of her pregnancy had been uneventful. Her serum glucose levels had been normal throughout pregnancy. She had nothing by mouth for approximately 2 h.

No contraindications to regional anaesthesia were identified and the patient consented to CSE analgesia. Pre-hydration was accomplished with

intravenous infusion of 1000 ml of Lactated Ringer's solution. The epidural catheter insertion technique consisted of using the sitting position and a median approach at the L2–3 vertebral interspace. An 18-gauge Tuohy-Schliff epidural needle was introduced with the loss of resistance to saline technique and the epidural space was identified at the first attempt. After an uneventful epidural needle placement, a 27-gauge Pencan needle was inserted inside the epidural needle. Following the appearance of cerebrospinal fluid (CSF) at the hub of the spinal needle, 5 µg of fentanyl combined with 2.5 mg of levobupivacaine (1 ml of 0.25% solution) was injected into the subarachnoid space. A 20-gauge multi-orifice epidural catheter was inserted 6 cm into the epidural space. Aspiration from the epidural catheter was negative for blood and CSF. The patient reported complete pain relief 3–4 min after the subarachnoid injection.

Approximately 12–15 min after the subarachnoid injection when the anaesthetist was securing the epidural catheter with tape (with the patient still in sitting position), the patient reported sudden onset of dizziness, headache, and clouding of vision. She became slightly confused, pale and diaphoretic. Her arterial blood pressure fell to 72/40 mmHg, and her heart rate increased to 94 beat.min⁻¹. Her blood haemoglobin oxygen saturation on room air, as documented by pulse oximetry was 99%. Fetal heart rate was 140 beat.min⁻¹ and reactive. The uterus remained normotensive with a normal pattern of contractile activity. Oxygen was promptly administered via facemask, and fluid bolus (500 ml of Lactated Ringer's solution) was rapidly infused. A total of 25 mg of ephedrine was administered intravenously in divided doses over 3 min. Blood pressure returned to baseline (112/62 mmHg), however, the patient continued to report dizziness, clouding of vision and headache. The pinprick sensory level of analgesia was T10. No signs of motor blockade were documented. The chest was clear to auscultation and chest X-ray was normal. There was no clinical evidence of placental abnormalities (placental separation or placenta praevia)

and the transabdominal ultrasound examination was normal. Pelvic examination ruled out vaginal bleeding. Arterial blood gas analysis revealed serum glucose level of 3 mmol.l⁻¹, but was otherwise normal. Dextrose 5% was administered intravenously, resulting in the return of serum glucose to normal and prompt alleviation of the symptoms of hypoglycaemia. The patient did not require any further treatment.

The standard epidural test dose with lidocaine and epinephrine was administered and was found to be negative for intravascular and subarachnoid catheter placement. Labour epidural analgesia consisted of maintenance by continuous infusion of 0.0625% levobupivacaine with fentanyl 1.9 mg.m⁻¹ at the rate of 10 ml.h⁻¹, and provided good pain relief for the rest of her labour and the uneventful vaginal delivery of a female newborn weighing 3650 g, who had Apgar scores of 9 and 10, after 1 and 5 min, respectively.

It is well established that labouring parturients have increased serum concentrations of stress hormones (e.g. cortisol) and catecholamines (e.g. epinephrine), both of which are known to stimulate hyperglycaemia [2]. With the rapid onset of labour analgesia (as provided with CSE), a significant abrupt decrease in the concentrations of circulating catecholamines and cortisol has been shown to occur, leading to a decrease in serum glucose levels [3]. The time of onset of symptoms of hypoglycaemia in the patient described in this report seems consistent with a reaction to sudden decrease in the concentration of serum catecholamines and cortisol levels following induction of CSE labour analgesia. Although our differential diagnosis included other possible causes of dizziness, mental status changes and clouding of vision, including aortic caval compression (the patient was in the sitting position), and/or high sensory block-induced hypotension (persistence of dizziness even after the blood pressure had been restored), given the above clinical scenario, they seemed unlikely. Additionally, what may seem to confirm/reconfirm our unexpected diagnosis of hypoglycaemia is the fact that dizziness, clouding of vision and

headache all disappeared shortly after the rapid infusion of intravenous dextrose solution had been initiated. Crites and Ramanathan [1] reported a case of a parturient with pre-existing type-2 diabetes mellitus who developed similar acute onset hypoglycaemia after receiving subarachnoid injection of fentanyl and bupivacaine as part of a CSE technique for labour analgesia. However, this author is not aware of any reports documenting acute hypoglycaemia following induction of CSE with fentanyl and levobupivacaine in a healthy (non-diabetic) pregnant patient.

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Lateral cutaneous nerve of the thigh pain in association with inguinal hernia repair

We would like to draw attention to the problem that occurs when an anaesthetist performs a procedure in the area in which the surgeon is operating and a complication arises. We present two patients who suffered chronic pain in the distribution of the lateral cutaneous nerve of the thigh following open inguinal hernia repair. The pain was blamed on peripheral nerve blockade but was more likely to have been caused by surgery.

The two patients had inguinal hernias repaired under general anaesthesia. Both had an ilioinguinal nerve block performed medial to the anterior superior iliac spine with a standard 21G needle after induction of anaesthesia. The first patient complained of shooting pains in the lateral aspect of the right thigh the following morning. The pain persisted but changed to a burning sensation with anaesthesia over the lateral thigh. Nerve conduction studies confirmed complete transection of the lateral cutaneous nerve of the thigh. The second patient reported sharp pain in the left antero-lateral thigh the next day. There was no sensation to pin prick over the antero-lateral aspect of the thigh below the inguinal ligament. There was allodynia on the left side of the shaft of the penis and below the medial half of the inguinal ligament. Both patients suffered persistent, disabling pain.

The syndrome of a dull ache, sharp pain, burning sensation, tingling or numbness in the distribution of the lateral cutaneous nerve of the thigh is known as meralgia paraesthetica. Entrapment by staples of the lateral cutaneous nerve of the thigh and femoral branch of the genitofemoral nerve is a documented complication of

laparoscopic inguinal hernia repair [1]. Surgical procedures on the lower abdominal or inguinal region have resulted in damage to nerves including the ilioinguinal, iliohypogastric and genitofemoral nerves [2]. On the other hand, local anaesthetic blockade of the lateral cutaneous nerve of the thigh has been used for harvesting split skin grafts from the thigh without complications [3,4]. We could find no cases reported of permanent damage to the nerve following open inguinal hernia repair.

The lateral cutaneous nerve of the thigh is entirely sensory and originates from the anterior divisions of the second and third lumbar nerves. It emerges from the lateral border of psoas major and courses obliquely across the surface of the iliac muscle towards the anterior superior iliac spine. Before it reaches the inguinal ligament, a number of anatomical variations have been described (Fig. 7) [5,6]. The commonest route for the nerve is downward and under the inguinal ligament, crossing anterior to the sartorius muscle and dividing into an anterior and a posterior branch. A recent study of cadavers [6] described five anatomical positions of the nerves that varied from superficial to posterior and from lateral to medial. This study

found that, in 13.5% of cases, the nerve divided into two branches before passing beneath the inguinal ligament with the medial branch forming the femoral branch of the genitofemoral nerve. Another anatomical study [7] found that the nerve contributed to the genitofemoral nerve 30% of the time.

The first patient had nerve damage in the usual distribution of the lateral cutaneous nerve of the thigh but the second patient had damage in an area involving both the genitofemoral nerve and the lateral cutaneous nerve of the thigh. In both cases, the peripheral nerve blocks were blamed for the nerve damage resulting in chronic pain because they had been performed close to the operative site. However, local anaesthetic injection is unlikely to cause complete nerve damage, in contrast to surgery which is more likely to cause neurotmesis.

While damage to the lateral cutaneous nerve of the thigh is presumed to be rare, as judged by the paucity of reports, it is probably underreported. These patients serve as a reminder that regional anaesthesia can be blamed for surgical complications and highlight the importance of explanation and proper patient consent.

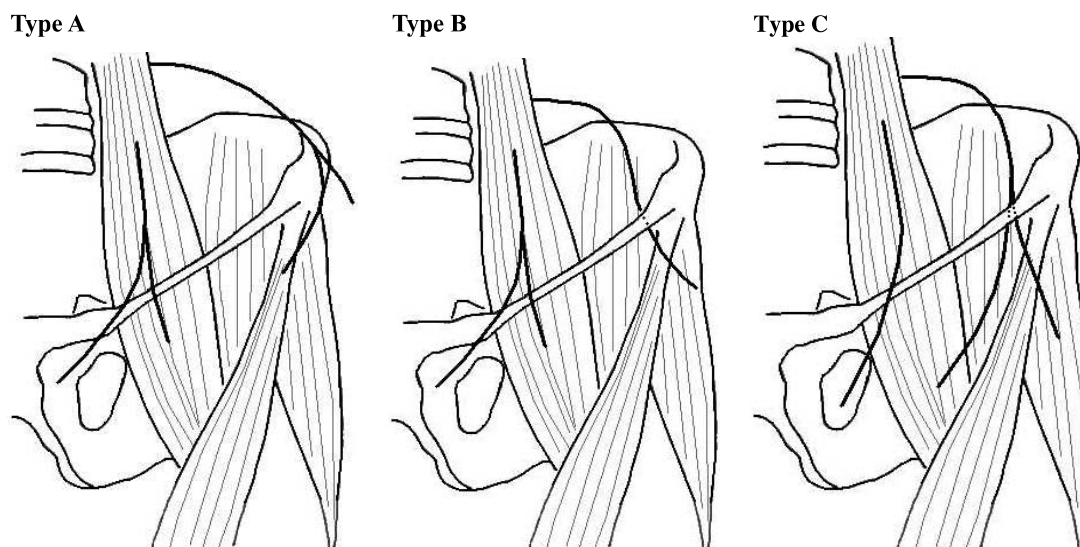


Figure 7 Variations in the position of the lateral cutaneous nerve of the thigh as it exits the abdomen. Type A – nerve courses across the iliac crest posterior to the anterior superior iliac spine. Type B – nerve is medial to the anterior superior iliac spine ensheathed in either the inguinal ligament, tendinous origin of sartorius muscle or found between sartorius and iliopsoas muscles. Type C – nerve is in most medial position superior to iliopsoas muscle, contributing the femoral branch to the genitofemoral nerve.

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Citalopram – a risk factor for postoperative hyponatraemia

Hyponatraemia is a common finding in postoperative patients, and while usually benign, its consequences can be severe and even life-threatening. The aetiology is often complex and multifactorial, and vigilance to newer pharmacotherapy is needed, as illustrated by the following case.

A fit 74-year-old lady (57 kg, m⁻² BMI 24) was admitted for an elective total knee replacement. She suffered

from osteoarthritis and mild asthma, and had a history of gastric ulceration secondary to non-steroidal anti-inflammatory drug-use. She also had a long history of anxiety and depression, but was established and remained well on zopiclone 3.75 mg *nocte* and citalopram 20 mg daily. Her other medications included omeprazole 20 mg daily, beclomethasone inhaler twice daily, and co-proxamol.

Her pre-operative assessment and investigations were unremarkable, including serum sodium of 136 mmol.l⁻¹, creatinine of 79 µmol.l⁻¹ and haemoglobin of 13.2 g.dl⁻¹. Surgery was carried out uneventfully under subarachnoid anaesthesia and midazolam sedation, and postoperative pain relief was provided by femoral nerve block, morphine delivered via patient controlled analgesia and simple oral analgesia.

She initially made a good recovery, but complained of postoperative nausea and dizziness on the first postoperative day. On the second day, she was less communicative and confused, and later became unresponsive, making uncoordinated clonic limb movements. Her Glasgow Coma Score was 5 and her pupils were dilated bilaterally and responded sluggishly to light. Clinically euvoelaemic, she remained well oxygenated and cardiovascularly stable throughout. She was intubated, her lungs ventilated and she was transferred to the Intensive Care Unit (ICU). A chest radiograph, 12-lead ECG and computerised tomography scan of her head were normal. However serum sodium was 116 mmol.l⁻¹.

The patient received a total input of 6 L of fluid intravenously and 1.48 l of water orally from the start of surgery to the time of fitting and ICU admission (50 h). The former were composed of 3 l sodium chloride 0.9%, 1 l Hartmann's solution, and 2 l of 4% dextrose/0.18% sodium chloride. The recorded output was 2185 ml of urine and 550 ml of blood into the drain. The fluid balance charts were, however, inaccurate because the patient was not initially catheterised, and there was at least one episode of urinary incontinence. The calculated fluid

balance when fitting occurred was 4745 ml positive.

Ventilation was continued for 48 h. She was transfused two units of blood but was otherwise fluid restricted and citalopram was discontinued. She was treated empirically with broad spectrum antibiotics, and made a full and uneventful recovery. Her serum sodium at discharge from ICU 4 days postoperatively was 130 mmol.l⁻¹ and from hospital 30 days postoperatively was 134 mmol.l⁻¹ despite normal fluid intake, but her citalopram had not been recommenced.

Hyponatraemia (generally defined as a serum sodium level of less than 135 mmol.l⁻¹), is the most common electrolyte disorder of all hospitalised patients, and is particularly prevalent amongst the elderly and those on antidepressants [1]. The commonest causes postoperatively include injudicious use of dextrose containing intravenous fluids combined with a stress response to surgery causing fluid retention. In addition, nausea and pain themselves are a potent stimulus for antidiuretic hormone release, which is independent of osmolar or baroreceptor regulation.

Citalopram is a selective serotonin reuptake inhibitor (SSRI), which is increasingly used to treat depression. It is well tolerated, relatively safe in overdose, and particularly efficacious in treating agitated depression in the elderly. The syndrome of inappropriate antidiuretic hormone secretion (SIADH) is a rare but potentially life threatening complication of this therapy, and is well described in the psychiatric literature [2]. It is usually transient, and resolves on discontinuation of the drug. Risk factors include increasing age, female gender, higher doses of citalopram, and concomitant use of other drugs known to cause hyponatraemia [3].

SIADH associated with citalopram has not been described in the anaesthetic literature and as far as we are aware, this is the first report of citalopram contributing to severe postoperative hyponatraemia. We hypothesise that a combination of risk factors including increased age, female gender, stress response to surgery and citalopram

therapy particularly predisposed this patient to hyponatraemia.

This raises a number of issues relating to peri-operative and postoperative care. There is often a discrepancy between the prescribing of intravenous fluids and what is eventually administered. Accurate monitoring of fluid balance is notoriously difficult, particularly on busy surgical wards. Patients' oral intake may also be forgotten when estimating intravenous fluid requirements. A standardised postoperative fluid regime needs to be tailored to each individual patient's weight and requirements, and reviewed and modified in response to current investigations. Particular caution is needed in the elderly, those having major surgery, and those on multiple and perhaps unfamiliar medications. Finally, pharmacovigilance remains paramount in anaesthetic practise, with heightened awareness of new therapies for common conditions being essential.

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Study of postoperative nausea and vomiting: recommending risk models for group comparisons

Having read with great interest the recent paper (Thomas *et al.* *Anaesthesia*

2002; **57**: 1119–28), as it questions the validity of predictive models [1–4] that we endorse, we would like to make the following observations.

The authors compared the incidence of postoperative nausea and vomiting (PONV) predicted by several risk models, based on the demographic characteristics of patients from their previous study [5]. The predicted risks did not correlate very well. However, to conclude that the use of risk scores for group comparisons is limited, ignores the fact that some scores may be considerably better than others [6,7].

Furthermore, if the authors wish to determine whether one of the risk models provides an acceptable prediction, the method of Bland and Altman is certainly not the appropriate choice [8]. This relatively simple but elegant method was originally developed 'for assessing agreement between two methods of clinical measurement' in the absence of a gold standard. However, in risk modelling the gold standard is clearly the actual outcome, i.e. the estimated incidence should be compared with the number of patients that actually suffered PONV or not. Unfortunately, such a comparison was not performed.

Therefore, neither the presented material nor the performed statistical analysis in the discussed paper [4] allows one to conclude that risk scores in general do not provide an appropriate estimate for patient risk (and therefore are not useful for group comparisons). In contrast, the simplified risk scores [3] did provide a realistic estimate of the patients risk for PONV in adults as recently shown in several other centres [6,7,9]. For this and other reasons, we would strongly recommend the use of a simplified risk score for group comparisons in anti-emetic trials in adults [10]. The only exception is paediatric anaesthesia due to the lack of a reliable predictive model for children. This is also the reason why a risk score for group comparison was not used in a recent study, since 593 out of 1180 patients were children [11].

If the authors are still not convinced, we would be pleased to support them

by performing a validation study of risk scores in their own centre.

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A reply

We thank Dr Apfel and his colleagues for their interest in our paper and would like to respond to the comments they have made.

Our study demonstrated that different scoring systems produce very different estimates of the incidence of postoperative nausea and vomiting (PONV). We found this both perplexing and surprising bearing in mind the similarity of the demographic details that make up the systems that we analysed. While we accept Apfel's statement that some scoring systems may be considerably better than others, without a placebo group, no researcher is going to be able to know which system will be the best or most accurate. We still maintain that the inclusion of a placebo group within an anti-emetic trial involving ambulatory surgery for gynaecological patients is unethical.

For the reason stated above, by necessity, we were working without a known 'gold standard'. Our decision to use the method of Bland and Altman was therefore quite correct. Furthermore, it would have been impossible to perform a comparison in the absence of such a gold standard. This limitation of our study was covered fully in our original discussion.

We quite understand the attractiveness of a simplified risk score to be able to either predict anti-emetic require-

ments or to use within the structure of a randomised control trial. What we believe we have demonstrated is that all the current instruments are too blunt, since moderate accuracy is probably the best one can hope for when trying to construct risk scores by current techniques of modelling.

We thank Professor Apfel for the offer of support in performing a validation of Apfel's risk score in our own centre and would welcome such co-operation. Indeed we are still confused as to how we should be scoring occasional smokers and patients with no previous anaesthetic history (do they really have the same risk of PONV as patients who have had a general anaesthetic and did not suffer from PONV?). Even gender has become contentious and the boundaries blurred. Is the risk of PONV genotypically, phenotypically or hormonally derived?

Moreover, we wish to draw on professor Apfel's teams expertise in testing the utility of his scoring system by challenging the null hypothesis: 'there is no difference in PONV in a population prescribed the currently available anti-emetics based on derived risk (using Apfel's scoring system) and a population where prescribing is left to chance'.

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Fire and frost!

We read with interest the recent letter regarding skin colour changes associated with the use of ethyl chloride spray when testing the height of an epidural block in a parturient with black skin (Kopka. *Anaesthesia* 2003; **58**: 102). The final paragraph asks the question 'Any lesson to be learnt?' In addition to his reply of 'know your basics well and always be prepared for the unexpected', we would like to add the following. Ethyl chloride is a hazardous substance. It is a highly flammable agent and can cause a spectrum of skin damage by rapid evaporation [1]. Therefore, for

health and safety reasons, alternative methods of testing the height of regional anaesthesia should be devised. We have used frozen sachets of sodium chloride 0.9% (25 ml Sterets Normasol, Seton Pebbles Ltd) for the last 5 years and have had no complications.

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Reference

- 1 Ethyl Chloride COSH. information sheet, Roche products Ltd, 2003.

Allergic reaction to hyaluronidase after a peribulbar injection

Hyaluronidase is a testicular protein, which hydrolyses hyaluronic acid, a structural component of the intercellular ground substance. It is often mixed in the local anaesthetic solutions used in ophthalmic regional anaesthetic techniques in order to improve the anaesthetic action in terms of speed of onset and spread of block. It is known to be a potential cause of allergic adverse events although there are few reports and little detail in the literature. Consequently, we are reporting a recent case at our institution.

A 41-year-old, 56 kg female presented for removal of a circumferential explant placed a year previously to treat a left sided retinal detachment. Her past medical history included breast carcinoma, treated 2 years ago with mastectomy, irradiation and chemotherapy. Her only regular medication was tamoxifen and she had no known allergies. She reported severe postoperative nausea and vomiting following general anaesthesia, so after discussion it was decided to use a local anaesthetic technique. Sub-Tenon's anaesthesia was impossible due to the explant positioning, so an infero-temporal peribulbar injection using a 25 gauge 25 mm needle was performed. A solution of 2 ml prilocaine 3% with felypressin 0.03 μmol^{-1} and 3 ml levobupivacaine 0.75%, containing 15 μmol^{-1}



Figure 8 Allergic reaction to the local anaesthetic.

hyaluronidase was injected. Within 5 min of the injection, she developed marked periorbital erythema and then oedema, which spread to the contralateral side (Fig. 8). Hemorrhage was considered briefly, but excluded as the appearances were more consistent with an allergic response. There was no systemic evidence of anaphylaxis. Intravenous chlorpheniramine 10 mg and dexamethasone 8 mg were administered. The swelling settled over the next 20 min, and it was possible to remove the explant uneventfully.

Her postoperative course was unremarkable and at last follow-up she had a flat retina with 6/6 visual acuity. In view of the implications for further surgery and anaesthesia, she was referred to an immunologist who performed skin prick testing to chloramphenicol, hyaluronidase, levobupivacaine and prilocaine with felypressin. This test showed a marked positive reaction to hyaluronidase consistent with Type 1 IgE mediated hypersensitivity. She had no known prior exposure to hyaluronidase.

Although allergy to hyaluronidase is a known side-effect, there have been only three case reports of adverse events during ocular anaesthesia [1–3]. Similar to our case, the reactions were limited locally, and terminated following treatment with antihistamines. However, in one, the rapid rise in orbital pressure led to vitreous loss during surgery [1]. Rapid peri-orbital swelling following an orbital injection suggests haemorrhage, but although rare, the possibility of hyaluronidase allergy should be considered and appropriate investigations performed even in individuals with no known prior exposure.

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Antihistamines and potentiation of opioid induced sedation and respiratory depression

In some areas of therapeutic practice we still follow traditions rather than logic. Treating opioid-induced pruritus with antihistamines was based on the assumption that pruritus caused by opioids was due to histamine release. However, scientific research does not rationalise the use of antihistamines in the treatment of opioid induced pruritus. Moreover, antihistamines are more likely to enhance the sedative effects of opioids [1]. One patient in our hospital had serious interaction of opioid with antihistamines, which raises a few questions.

A young lady received fentanyl and bupivacaine lumbar epidural analgesia and anaesthesia for labour pain, Caesarean section and then for postoperative pain relief. She received a bolus of 10 ml of 0.25% bupivacaine for labour pain, which was followed by an infusion (fentanyl 2 µg/ml with bupivacaine 0.125% at 10 ml.h⁻¹). After 3 h, she required delivery by a Caesarean section. For this she was topped up with 14 ml bupivacaine 0.5% and fentanyl 100 µg that produced a block from T4–S5. For postoperative pain relief, she received an epidural infusion (fentanyl 4 µg with bupivacaine 0.03%) at the rate of 7 ml.h⁻¹.

She remained pain free with stable vital signs, although slightly drowsy, for 4 h after surgery. She then complained of itching, for which she received intramuscular chlorpheniramine 10 mg. Thirty-five minutes after the injection, she was found somnolent with a respiratory rate of 4 breath.min⁻¹ and responded quickly to assisted ventilation and naloxone 0.4 mg.

Treatment of pain with opioids always carries the risk of side-effects. All opioids, regardless of route of administration cause respiratory depression [2]. The greatest risk of respiratory depression occurs 4–8 h after epidural or intrathecal administration of opioids [3]. The delayed respiratory depression is more likely to occur with less lipid soluble (such as morphine) than more lipid soluble (such as fentanyl) opioids. However, early respiratory depression can occur with continuous epidural infusions of highly lipophilic opioids as systemic uptake leads to significant plasma concentration of drug. In a national survey, the overall incidence of respiratory depression was 0.2% to 0.4% among non-obstetric patients who received opioids epidurally [4]. Respiratory depression in this survey was most commonly observed in elderly patients who received parenteral narcotics, hypnotics and neuroleptic drugs in the peri-operative period. Elderly patients are sensitive to opioids but young patients can also suffer from somnolence and respiratory depression if they receive excessive amounts of opioid. Profound respiratory depression with as little as 100 µg of epidural fentanyl has been reported [3]. The epidural catheter can migrate in the subarachnoid space [5]. This can lead to delivery of increased amounts of narcotic in the CSF causing central nervous system depression.

The pruritus associated with opioids is seen more commonly after epidural and subarachnoid administration than after systemic administration and is more common in pregnant than in non-pregnant patients [6]. For many decades, it has been traditional to treat narcotic-induced pruritus with chlorpheniramine, which is an antihistamine with sedative properties. Surprisingly, PubMed and Medline searches did not show

any case report or study that looked at the risk of serious interaction between epidural fentanyl and intramuscular chlorpheniramine. Some authorities still recommend antihistamine for opioid-induced pruritus [2,7] despite their belief that the main benefit of using antihistamine is that it induces sleep, so the patient no longer 'cares' about the itching [2]. However, there is no evidence that this pruritus is because of histamine, and the exact mechanism remains unclear. Moreover, fentanyl does not cause histamine release.

Our patient received fentanyl 300 µg epidurally over the period of 9 h. Somnolence and severe respiratory depression in our case might be either due to patient's increased sensitivity to opioids or migration of the epidural catheter into the subarachnoid space, resulting in deposition of relatively large dose of fentanyl in the CSF. But the rapidity with which her sedation increased after chlorpheniramine, causes little doubt in its role in potentiating fentanyl-induced side-effects.

This case has led us to review the rationale of using chlorpheniramine for treating opioid-induced pruritus. Opioid antagonists such as naloxone or partial agonists such as nalbuphine [8] or 5-HT₃ antagonists such as ondansetron [9,10] but not antihistamines should be used to treat narcotic-induced pruritus. This will not only reduce the risk of potentiating other serious narcotic side-effects, but also be more logical.

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Paediatric dental sedation

We were interested to read the article on paediatric dental sedation (Wilson *et al.* *Anaesthesia* 2002; **57**: 860–7). This study contains a relatively small number of patients (no power analysis is described in the paper) and we feel the conclusion that oral midazolam in a dose of 0.5 mg.kg⁻¹ in children aged 10–16 years is a safe technique compared with nitrous oxide in oxygen, may be premature. Our local tertiary paediatric hospital has an upper limit of 15 mg to the dose of midazolam given as a sedative premedication. The paper does not give the weights of the children so we do not know what doses

were given, but without an upper dose limit over-sedation is a possibility.

We were curious as to why an emotional status rating scale (Brietkopf and Buttner Classification of Emotional Status) was used to measure sedation, in which the highest level of sedation (grade 4) was 'very sleepy: drowsy, without reaction but rousable' (Reference 26 in the paper). Of the midazolam treated patients, 46% were in this group, which does not differentiate between rousable to verbal or physical stimulation, and the scale does not include a further level of sedation for the unresponsive.

The range of oxygen saturations in the group sedated with midazolam was 90–100% and the authors state this to be 'within acceptable and safe limits for conscious sedation'. We take issue with this lower level in children. The reference quoted to support the lower limit of 90% in fact states: 'Patients undergoing sedation should always have an oxygen saturation well above 90%' (Reference 14 from original paper). Children have a higher metabolic rate and lower oxygen reserves. The non-linear relationship between SpO₂ and PaO₂ at saturations less than 92% means that below 92%, severe hypoxia is imminent. The authors do not state the number of children in the midazolam group that had low oxygen saturations or for how long the desaturation was sustained. If, however, these children with low oxygen saturations were also in the sleepy/drowsy category, we suggest they should be classified as over-sedated. The majority of patients in the nitrous group were either very good or excellent on the Houpt Behaviour rating scale and this suggests that midazolam has little to offer over nitrous oxide in terms of efficacy while raising the possibility of the risk of over-sedation and hypoxia.

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A reply

We appreciate the comments made regarding our paper and wish to respond.

We would like to clarify that the conclusion of our paper does not state that midazolam in a dose of 0.5 mg.kg^{-1} is a safe technique compared to nitrous oxide in oxygen. Rather, our conclusion suggests that this form of sedation appears to be a safe and acceptable sedation agent in 10 to 16 year-old healthy children and may therefore offer a further method of paediatric dental sedation. We accept that further work is required in this field and certainly do not advocate the use of oral midazolam outside of a secondary care setting.

It is accepted that the doses of midazolam given may have been above the 15 mg ceiling used in Burns *et al.*'s department; however, in our study midazolam was being used as the sole agent to produce slightly higher levels of sedation than perhaps would be required from a premedication agent.

The comments regarding the use of the Brietkopf and Buttner Classification of Emotional Status are very valid. In searching the literature, many scales have been used to record levels of sedation and the scale used in the study was chosen for ease of recording. Verbal and physical stimulation are indeed very different and may be reflected differently with regard to levels of sedation. A level for unresponsive would, we agree, provide a more complete scoring system and this will be considered for future work.

The range for the lowest oxygen saturation recorded for the children receiving midazolam group was stated as 90–100%. It is important to note that the oxygen saturation was recorded every 5 min and the lowest reading noted for the series of readings throughout the appointment. Only one child recorded a lowest value of 90% and this was for one reading at 5 min into the appointment. For the remainder of the appointment, the oxygen saturation remained well above 92%. In future studies, this value may need to be recorded in a clearer fashion.

The fact that nitrous oxide oxygen sedation was generally well accepted by children in the study does indeed

suggest that it is a suitable form of sedation. However, as stated in the introduction to the paper, nitrous oxide does pose an occupational hazard to those using it on a regular basis. It is also apparent that not all children can tolerate inhalation sedation and there is therefore a need to seek other methods of sedation for paediatric dental patients. Our suggestion was not that oral midazolam should replace nitrous oxide and oxygen sedation, but rather that it might provide an alternative for some individual patients.

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About submental intubation

I read with great interest the paper of (Amin *et al.* *Anaesthesia* 2002; 57: 1195–212) in which the authors make a very detailed description of the

procedure of submental tracheal intubation. They do an adequate review of the surgical and anaesthetic aspects, detailing interesting items, so the reader has a very useful guide to developing this technique. They also note its indications, not only in cranio-maxillofacial trauma but in other fields such as orthognathic surgery or congenital malformations surgery. For our part, we would like to add the usefulness of submental intubation in skull base surgery, transfacial surgery and pediculated craniofacial surgery.

Nevertheless, we advocate investigation, as the authors have done, of the first description of the technique [1] with its drawings demonstrating the need for respect for the important structures of the floor of the mouth and submandibular region. Hence, we wish to emphasise the surgical performance of the submental incision (Figs 9 and 10). The incision should be made in the paramedial region, in the anterior

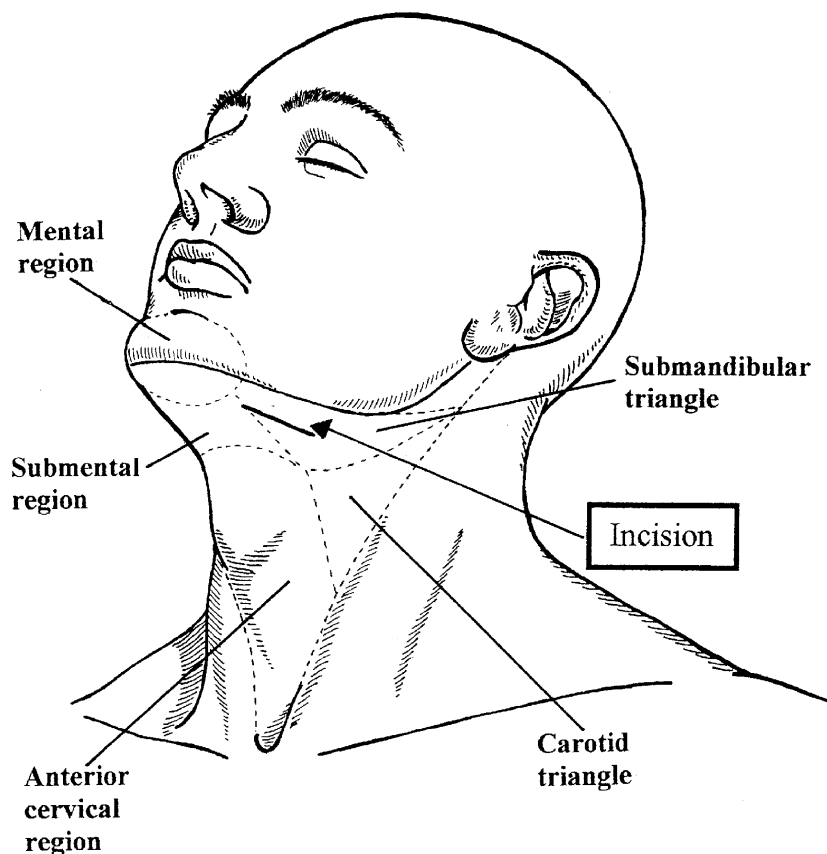


Figure 9

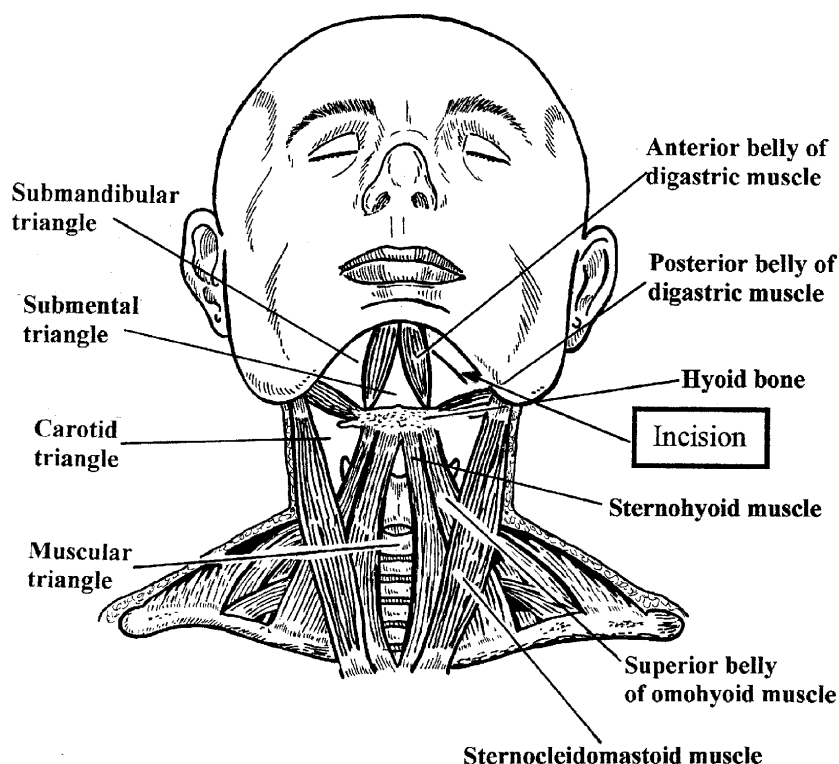


Figure 10

vertex of the submandibular triangle parallel with the mandibular lower border, which is an anatomically "clear" zone. The track of the tube must be made from oral to submental region in the area of the decollement of the inner mandibular mucosa at subperiosteal level in the parasymphysarial portion. The place of the incision may be modified only if wounds or previous scars exist. It is likely that a submandibular medial incision would cross more significant structures, such as the muscular insertion zones on the same level as the genial tubercles. Furthermore, medial incisions may damage important structures of the floor of the mouth. We want to clarify that the floor of the mouth has to be displaced, but also respected.

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Simultaneous coronary bypass surgery and same anaesthetic technique in identical twins

I have recently had the chance to compare the same anaesthetic technique for major surgery in 59-year-old identical twins who presented consecutively for coronary bypass surgery. Different anaesthetic modalities for outpatient cases of twin patients have been reported. Our patients both had ejection fractions of 63% and stenotic lesions in the left anterior descending (LAD), circumflex (Cx) second obtus and right coronary artery on coronary angiography. Coronary artery bypass grafting (CABG) was planned in both patients.

Both patients were premedicated with midazolam. Anaesthesia was induced

with etomidate (3 mg.kg^{-1}), vecuronium (1 mg.kg^{-1}), dehydrobenzperidol (DHP) 2.5 mg and fentanyl ($5 \text{ } \mu\text{g.kg}^{-1}$) and maintained with air, oxygen and isoflurane 0.5% and fentanyl ($3 \text{ } \mu\text{g.kg.h}^{-1}$). Standard cardiopulmonary bypass (CPB), anaesthetic and surgical techniques were used for both cases. During the bypass, left internal mammary artery–left anterior descending artery, aorta–obtus marginal, aorta–right coronary artery anastomoses were made. Before the end of cardiopulmonary bypass, dopamine $5 \text{ } \mu\text{g.kg}^{-1}.\text{min}^{-1}$ was administered to both patients for similar periods, as the myocardial performances were not satisfactory. During the operations, both patients had similar activated clotting times (ACT), total drug doses and haemodynamic parameters. ACT values of both patients were 97 s before heparin, 424 s and 434 s after heparin; 110 s and 112 s after protamine, respectively. Total fentanyl administered was 0.6 mg for the first case and 0.7 mg for the second; total vecuronium bromide was 16 mg for both. Operation durations were 210 and 205 min, CPB time was 73 and 74 min and cross-clamp time was 37 and 39 min for the first case and second case, respectively. For the first patient, systolic blood pressures were 120 mmHg (preinduction), 140 mmHg (postinduction), 120 mmHg (before CPB), 100 mmHg (end of CPB); and 130 mmHg , 130 mmHg , 120 mmHg , 100 mmHg , respectively, for the second patient.

At the end of the operations, the patients were admitted to the intensive care unit, sedated with propofol, and were extubated after 6 h. At 16 h post surgery, surgical drainage was 500 ml and 475 ml and urinary outputs were 4000 ml and 4940 ml for the first and second patient, respectively. The patients stayed at the ICU for 48 h, and were discharged from the ward on the eighth postoperative day.

In these cases, we compared the results of the same anaesthetic technique of two different anaesthetists, performed for CABG, at the same time intervals. We saw that their haemodynamic parameters, ACT and administered total drug doses were very close to each other. We believe that observing the

results of two different anaesthetic approaches in such a situation of major surgery to be quite interesting.

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Preventing loss of nasogastric tubes in the critically ill. The 'nasal lasso' improved

Nasogastric tubes (NGTs) are essential in some critically ill patients to allow feeding, administration of drugs and to empty the stomach. Unfortunately, the patient often removes them. Replacement of nasogastric tubes may cause distress to the patient, who may need sedation if the new tube has to be inserted under direct vision. The 'nasal lasso' is a technique described by Lawler [1] where two lengths of oxygen (green bubble) tubing are inserted through the nose and brought out of the mouth. The two tubes are then tied together and one pulled out of the nose. One tube now goes completely around the nasal septum to form an anchor. A NGT is then inserted through the nose into the stomach. It is then attached to the 'lasso'.

We have found this technique to be useful and prevent the need to stitch the nasogastric tube to the nose. However, we were concerned that the use of oxygen tubing was not optimal. Trauma to the nasopharynx caused by two oxygen tubes tied together and the difficulty of teaching and performing this was at times considerable. Furthermore, we were concerned that the relatively rigid plastic of the oxygen tubing, especially when kinked, may traumatise the nasal septum unnecessarily. We have therefore modified this technique to reduce these problems.

The new technique uses a length of silastic drain tubing (Silicone Tubing 3.2 mm bore, Cat N° TSRO200100P; Bibby Sterilin Ltd, Stone, Staffordshire, UK) instead of oxygen tubing to 'lasso' the septum. Silastic tubing is thinner, softer, more elastic, and does not kink when bent. The potential for damage to the nasal septum is thus reduced, in addition to reducing the waste of two

nasogastric tubes during the insertion process.

An artery forceps should be attached to the end of a 20-cm length of well-lubricated silastic rubber tubing. The free end is passed through one nostril and into the posterior oropharynx. This is then brought out of the mouth using Magill's forceps. The artery forceps prevents the whole length of tubing disappearing into the mouth and nasopharynx. Both ends of the silastic tubing are then secured using the artery forceps. A lubricated nasogastric tube is passed into the oropharynx via the other nostril, and the free end also brought out through the mouth using Magill's forceps. Pushing the nasogastric tube into the end of the silastic tubing then joins the oral ends of the silastic and nasogastric tubes (Fig. 11). Using gentle traction, the nasal end of the nasogastric tube is pulled out of the nose entirely so that the silastic tubing now 'lassos' the nasal septum (Fig. 12).

The two ends of silastic tubing are again secured using artery forceps, preventing the silastic tubing from becoming dislodged while the nasogastric tube is passed into the stomach in the normal way. The nasogastric tube and two ends

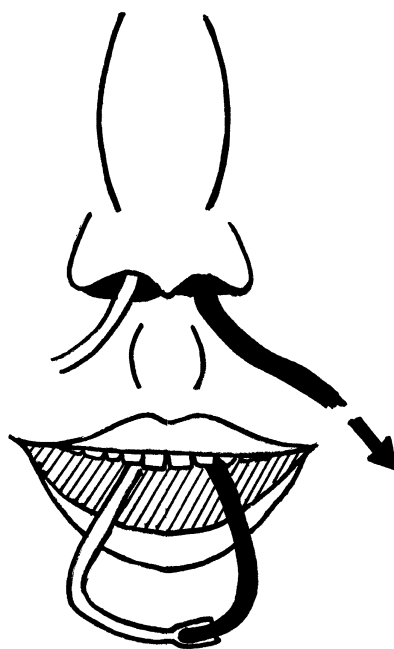


Figure 11

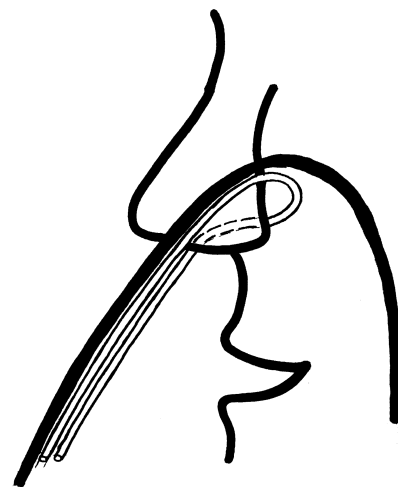


Figure 12

of silastic tubing are cleaned and secured together using tape, leaving a gap of at least 2 cm between the nose and tape.

Using this technique, we have found nasogastric tubes to last for over a month in patients who previously needed several each day. We have not seen any evidence of septal trauma, infection or other problems.

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James Matthews Duncan

Dr Wilkinson is to be congratulated for his eloquent description of further details surrounding 'the discovery of chloroform' (Wilkinson. *Anaesthesia*, 2003; 58: 36–41). The manuscript 'James Matthews Duncan: A sketch for his family', which he presumes never to have been published was privately printed in 1891 by Cornwall of Aberdeen [1]. The book has 181 pages and is approximately 25 cm in height; the

only copy I have been able to trace is held by the Wellcome Library in London (personal communication – Wellcome Library). The deposit of many of Matthews Duncan's personal belongings in the Medical College Library, St Bartholomew's Hospital, has been noted (1959) by John Thornton, who was honorary librarian and an authority on Duncan. Readers may be interested to know that, on Duncan's death, letters of condolence were sent to his widow from numerous distinguished contemporaries, including Queen Victoria and Lord Lister [2].

The letter written by Matthews Duncan to Sir Robert Christison in 1875 has been mentioned in the book 'Simpson and Syme of Edinburgh' by J.A. Shepherd (1969) [3]. Shepherd's references for this came from the honorary librarian, Thornton [4], and an Obituary of Duncan in St Bartholomew's Hospital Report [5].

In the 15th J.Y. Simpson Oration given at the Royal College of Obstetricians and Gynaecologists in 1975, Donald Fraser devoted some time to the life of Matthews Duncan, acknowledging (as sources of information) Simpson's grand-daughter, Mrs Jane McBarnet, and the honorary librarian, Mr Thornton. He mentioned that Simpson had sent a paper to Paris for Duncan to translate into French and also stated that 'Duncan had certainly tried it (chloroform) two nights before' the famous dinner party of 4th November 1847. Fraser pointed out that if James Young Simpson took all the credit for the discovery of chloroform, he also took all the risks – no one knew what adverse effects it might have in obstetrics [6]!

In all the above references, the role of Matthews Duncan in the discovery of chloroform has received little more than a glance. By quoting the most relevant passages from Isabella Newlands' manuscript and Duncan's letter to Christison, Dr Wilkinson has brought this blurred image into focus.

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- 3 Shepherd JA. *Simpson and Syme of Edinburgh*. Edinburgh, Livingstone, 1969, 94.
- 4 Thornton JL. The relationship between James Matthews Duncan and Sir James Simpson. *Medicine Illustrated* 1949; **3**: 261–5.
- 5 Turner W. Obituary of James Matthews Duncan. *St Bartholomew's Hosp. Report* 1890; **26**: 23.
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Early books and pamphlets on anaesthesia

There have been four articles entitled 'Early textbooks on anaesthesia' published in *Anaesthesia* [1–4]. Each of these articles provided information extending the list of publications on anaesthesia in the year 1847. It became apparent that some of these publications were pamphlets rather than textbooks, and some were reprints of journal articles. I am now in a position to add 11 further titles to the list and to further categorise some of the works already presented.

The major source of my information is the outstanding bibliography by Rosemary Faraday of the Nuffield Department of Anaesthesia, University of Oxford – a thesis submitted in 1966 for Fellowship of the Library Association [5]. Faraday divided her bibliography into two sections:

- (1) books and pamphlets;
- (2) dissertations.

She pointed out that 'a wholly disproportionate idea of research in anaesthesia would have been given if doctoral dissertations had been included in the main sequence', because French and German universities had a policy of printing and distributing their doctoral theses, whereas English-speaking universities did not. She found over 2000 dissertations on anaesthetic subjects

printed between 1846 and 1950, the majority being French and German.

Six of the 25 'books' listed in the first article by Secher were in fact *dissertations*. These are as follows (pagination from Faraday):

Bromberger T. *De aetheris sulphurici vaporibus...* Berolini, typis fratrum Schlesinger. [1847] 26 [4] p. 20 cm.

Chambert H. Des effets physiologiques produits par l'inhalation des différents éthers. Paris 1847, 4°.

Diesterweg J. *De aetheris sulphurici in organismus per vias respiratorias inducti efficacitate*. Berolini [1847] 8°.

Dupuy J-M. *De l'étherisation*. Paris, 1847. 4°.

Martin A. *Zur Physiologie und Pharmakodynamik des Aetherismus*. München, 1847. 8°.

Merck WC. *Der Schwefelaether in seiner Bereitung, Wirkung und Anwendung*. München, 1847. 8°.

Interestingly, in Secher's next list of 13 titles the 'book' by Jablonski was declared to be a *dissertation*. Dinnick provided a further 16 definite additional titles. He also suggested another 6 possible titles (obtained from the book by Lach), but did not put these in the additional list, because of the lack of full descriptions. Three of these 6 appear in Faraday's bibliography, and are marked with an asterisk in the list appended.

For readers' information, Faraday found a further 2 printed *dissertations* besides the 6 already stated. All the above information refers to the year 1847. What about 1846? Faraday found two *dissertations* printed in 1846. She found one book:

Esdaile J. *Mesmerism in India, and its practical applications in surgery and medicine*. London, Longman, Brown, Green & Longman, 1846. xxxi, 287p.

Regarding pamphlets, William Morton sent out (to physicians) his first printed document on anaesthesia on 20th November 1846. This circular also appeared on the back of the *Boston Medical and Surgical Journal* of 25th November and 2nd December 1846. From 26th November 1846, Morton distributed a series of circulars entitled *Morton's Letheon*. The text of the first was probably identical to the advertisement on the back cover of the *Boston*

Medical and Surgical Journal of 9th December 1846.

Morton's *Letheon* 2nd edition (4 pages) and 3rd edition (14 pages) were issued in December 1846 [6]. Franco, Cortés, Vidal and Alvarez included in their list of six additional 1847 publications the 4th edition (42 pages) and 5th edition (88 pages) of Morton's *Letheon* issued, respectively, in January and May 1847.

It may be of interest to historians of anaesthesia to know that I have compiled a database (Microsoft Access) of books and pamphlets on anaesthesia 1847–1900. This lists nearly 600 publications arranged in chronological order with the authors of each year in approximate alphabetical order. The holdings of five libraries are given plus an 'other library' column. Journal articles are *not* included unless these were re-issued as reprints; furthermore dissertations are *not* included. The Access system enables a wide range of reports to be derived from the Table. This database is available in CD-ROM and the Association of Anaesthetists of Great Britain & Ireland has a copy.

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- Nuland SB. *The Origins of Anesthesia*. Birmingham (Alabama), The Classics of Medicine Library, 1983 Chap. IX, pp. 74–7.

Appendix

Additional 1847 books and pamphlets

Blandin PF. De l'usage des inhalations d'éther dans les opérations chirurgicales. Mémoire lu à l'Académie royale de médecine, 23 mars, 1847. Paris, Labè, 1847. 60p. 8° (repr. From J. de l'Union médicale, mars, 1847)

*Delabarre AFA. *Guide du praticien dans l'administration des vapeurs d'éthyle*. Paris, 1847

Hayward G. *Remarks on the comparative value of the different anaesthetic agents*. Boston, D. Clapp, 1847. 11p. 8°

Hering CE. *Die Schwefel-äther-Frage nach eigener Erfahrung und nach den neuesten Forschungen beleuchtet*. Leipzig, Friesse, 1847. ix, 90p. mit I lith. Taf. gr. 8

Minich A. *Sui pericoli dell'inspirazione dell'etere solforico*. Venezia, 1847. 24p

*Pajot C. Des effets de l'inhalation des vapeurs d'éther, de son action sur l'homme sain et dans les opérations chirurgicales comme moyen d'éviter la douleur, résumé de toutes les expériences faites à l'étranger et en France, par MM Roux, Velpeau, p. Dubois, Gerdy, Blandin, Malgaigne, Baudens, Jobert, etc. Paris, 1847. 8°

Simpson JY. *Anaesthetic and other therapeutic properties of Chloroform*, 1847. (Continuation of original report, Remarks on the superinduction of anaesthesia in natural and morbid parturition, Edinburgh, 1847). 'First edition. Smith P. *On the employment of ether by inhalation in obstetric practice*. [1847]

*Villeneuve. *De l'éthérisation dans les accouchements...* Marseille, imprimerie Bellande, 1847. 32p. 22½cm. No.3 in a volume of pamphlets lettered: Gestation. Hymen. Etherisation. Ms. puerperales. Matrice et ovaires. Cystocele vaginal. Polypes.

Waldie D. *Chloroform, the new agent for producing insensibility to pain by inhalation*. [Liverpool, 1847] 8°

Ranking WH (ed.). *Half-yearly abstracts of Medical Science: Vol.5 Ether; Vol.6 Chloroform*. London, Churchill, 1847.

Caution with your arterial lines

A 69-year-old lady was admitted with right lower lobe pneumonia. Despite physiotherapy, medical therapy and

non-invasive positive pressure ventilation, she tired and required invasive positive pressure ventilation. Lines were also sited for invasive monitoring and secured prior to transfer to another Intensive Care Unit within the region, as no beds were available in the admitting hospital. On arrival, it was noted that she had developed a pressure sore where her arterial line had been looped over her thumb and dressed with crepe bandage (see Fig. 13).

Previous literature has identified a number of risk factors for pressure sores, the most significant of which are norepinephrine infusion, APACHE II score greater than 13, faecal incontinence, anaemia, and length of stay [1]. In this example, norepinephrine was required, APACHE II was greater than 13, there was a prolonged period of stay/illness, and although of lesser significance in the study (but not in this individual case), both tissue hypoxia (secondary to her respiratory failure) and peripheral oedema had developed.

We therefore advocate the use of caution when securing any equipment/peripheral lines to the patient, especially the common practice of looping arterial and venous lines around



Figure 13

digits where there is little room to accommodate any developing peripheral oedema. With patients at increased risk, we also suggest the use of padding between skin and giving set, such as green gauze or 'Microfoam' surgical tape by 3M.

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Vegetarian anaesthesia

Last year, I had to anaesthetise a Hindu lady for a minor day care gynaecological procedure. During that month she was fasting for religious reasons. She insisted that she should not be given any drugs or medicines that come from animals including substances from eggs. I discussed this with the surgeon and theatre staff. We reassured the patient that we would not use anything 'non-vegetarian'. I anaesthetised her with an inhalational induction and maintenance of anaesthesia. The surgical procedure took only about 10 min. Her peri-operative period was uneventful and she went home the same evening.

I performed a brief search of the medical literature including Medline and the Internet, but could not find any relevant information. My list of 'non-vegetarian' drugs/substances that we might use peri-operatively include: propofol, gelatine based colloids, heparin, protamine, older insulins, blood and blood products.

It would be interesting to hear views from colleagues, if they have encountered a similar situation and if they can add to the list of 'non-vegetarian' substances.

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