Anaesthesia, 2003, **58**, pages 1235–1252

Correspondence

Consent for anaesthesia

The special article on consent for anaesthesia (White & Baldwin. *Anaesthesia* 2000; **58**: 760–74) is timely, relevant and merits detailed attention.

In 1994 when I wrote a paper on 'Problems with regional anaesthesia: hazards or negligence?' [1], I advocated a moderate view regarding informed consent and obstetric regional anaesthesia. Indeed, on reflection, I 'sat on the fence'. The way in which medicine is practised continually evolves and has to respond to the ever-changing values and philosophy of the society/community that it serves: our patients are better informed; their knowledge of both their illnesses and rights is continually being reinforced by increasing use of the Internet, the media and various pressure groups.

Early during my tenure as Postgraduate Dean, it became apparent that it was inappropriate for preregistration house officers to take consent for complex surgery about which they had very little understanding. The educational contract was appropriately modified. They also had little knowledge of anaesthetic techniques and postoperative pain relief and so appropriate tutorials were included in their teaching sessions but we were left with the problem of consent for anaesthesia, particularly where increasingly complex techniques were being used e.g. combined general and regional anaesthesia with postoperative continuous spinal or epidural analgesia.

Although the Association has produced thoughtful guidelines, I would agree with White and Baldwin's three criticisms and am persuaded that separate consent for anaesthesia should be taken. Obviously, for more simple procedures, e.g. anaesthesia for most day surgery and investigative procedures, an information sheet carefully worded may suffice.

How should the actual process of taking consent be implemented? First, every department should discuss this paper at a departmental meeting attended by consultants and trainees i.e. if such occasions still occur given the abundance of shift working. Second, a consensus should be reached, and thirdly pilot consent taking will be necessary followed by audit and the cycle closed. Finally, a plan should be presented to the managers to implement since presumably that is one of their functions. Such a development must improve patient/doctor communication, a basic tenet of a modernised Health Service. However, this will take time, which means sessional commitment, otherwise patient throughput will be reduced. The College or the Association may wish to coordinate this process with a view to achieving national consensus.

R. Macdonald
Leeds LS16 9JF, UK
E-mail: rosemacuk@yahoo.co.uk

Reference

1 Macdonald R. Problems with regional anaesthesia: hazards or negligence? *Anaesthesia* 1994; **73**: 64–8.

Academic anaesthesia

Academic anaesthesia is clearly at a cross roads and alternative structures for the provision of academic support for our speciality need to be found. As Jackson suggests (Jackson *et al*. *Anaesthesia* 2003; **58**: 911–2), one possible solution is to take academic anaesthesia away from the medical school environment. Our experience in the South-west of England over the past few years is an example of this.

In 1997, we had established an academic department of anaesthesia as part of the Plymouth Postgraduate Medical School. However, with the arrival of the new undergraduate Peninsula Medical School in 2001, things were soon to change. The Peninsula Medical School, for all of the reasons that have been elucidated by Jackson, did not feel able to continue to support an academic department of anaesthesia. Despite numerous attempts by anaesthetists in the region and recurrent funding being secured from the NHS, an academic department of anaesthesia within the Peninsula Medical School was not established and our young academic department was lost. We therefore took

All correspondence should be addressed to Dr David Bogod, Editor of *Anaesthesia*, Anaesthesia, Ist Floor, Maternity Unit, Nottingham City Hospital, Hucknall Road, Nottingham NG5 1PB, UK.
Letters (two copies) must be typewritten on one side of the paper only and double spaced with wide margins. In addition, please include your letter as a Word for Windows or .rtf document on disk or alternatively submit as an e-mail attachment addressed to *anaesthesia@nottingham.ac.uk*. Alternatively, a response to a previously published article or letter can be submitted online at *www.anaesthesia correspondence.com*. Copy should be prepared in the usual style and format of the Correspondence section. Authors must follow the advice about references and other matters contained in the Notice to Contributors to *Anaesthesia* printed at the back of the January and July issues. Author guidelines can also be found on *www.blackwellpublishing.com/journals/ana/submiss.htm*. The degree and diploma of each author must be given in a covering letter personally signed by all the authors.
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the only route open to us and established an NHS based academic department of Anaesthesia, Critical Care & Pain Management under the auspices of the South-west School of Anaesthesia. This provides a focus for academic activity within the NHS and the School of Anaesthesia. The academic lead is by NHS clinicians with a background in research and rotates on a three yearly basis. This provides academic time for NHS clinicians to allow us to continue to provide an academic environment for trainees in the South-west School of Anaesthesia.

I believe that this is a constructive and pragmatic way forward in the presently hostile climate that anaesthesia finds itself in, both in established and, from our experience, also within the new medical schools.

J. A. Langton
Plymouth Hospitals NHS Trust,
Plymouth PL6 8DH, UK
E-mail: Jeremy.langton@phnt.swest.nhs.uk

Patient safety

I read with interest the Editorial about patient safety (Jorm. *Anaesthesia* 2003; **58**: 833–4). Dr Jorm mentioned the concept of 'six sigma', which has been achieved in the airline industry and has made commercial flying without doubt the safest form of transportation. Part of this greatly increased safety has been due to the introduction over the past 25 years of Crew Resource Management (CRM) taking into account that over 80% of aviation accidents were caused by human error. This is defined as 'better recognition and utilisation of all available resources, information, equipment and people to achieve safe and efficient flight operations! The components of CRM include:

Communication/interpersonal skills including polite assertiveness, active listening and feedback.

Situational awareness and the avoidance of 'tunnel vision' in a crisis; problem solving, decision making and judgement.

Leadership with the pilot in command being responsible for accessing and managing all resources that are available for the safe completion of the flight and include non-command members being assertive when necessary.

Stress management and ensuring that an understanding of personal and work related stresses would make colleagues alert for any decline in performance.

Critique with the ability to analyse a future, current or past plan of action.

In anaesthesia we already use many of the above components in our practise but in the airline industry there is formalised training in CRM and a requirement for pilots to adhere to it. I think we have much to learn from the airline use of CRM to improve patient safety. Maybe we should consider that such CRM type training relevant to anaesthesia should be included in the FRCA syllabus?

A. Levison
Wexham Park Hospital,
Slough SL2 4HL, UK
E-mail: andrewflyboy1@aol.com

Underpowered and overbiased? Potentially unfair to the single-use bougie

I hope the authors (Marfin *et al. Anaesthesia* 2003; **59**: 852–5) will not feel offended if I use their article to illustrate some recent problems I have encountered surrounding the question of power estimation and sample size calculation in clinical studies.

Whereas it is a welcome change to now see referenced evidence of efforts to calculate a sample size which provides a study with the recommended power, we non-statisticians should not be lulled into a false sense of security by the apparent science. The authors used a standard deviation (s.d.) of 20 s from their preliminary trials to calculate the sample size. If this is the best available evidence then fair enough, but what should be done when the s.d. or variability of the studied parameter turns out to be different in the actual study? Should the authors perform a *post hoc* power analysis to prove that the study still had adequate power to support their conclusion? Evans *et al.* [1] fell foul of this problem when the standard deviation of morphine consumption in their study was greater than in their pilot, such that they had a clinically significant difference that was not statistically significant. Although the intubation times in the study by Marfin *et al.* were not normally distributed, the total ranges are only 20 s (single-use) and 27 s (multiple-use). This suggests a much smaller variability than the s.d. of 20 s used in the initial power/sample size calculation, which in this case only serves to increase the power of the study. However, with median intubating times of 22 and 26 s for the two bougies, how on earth could the authors expect to find a 'clinically important difference between intubating times' of 20 s?

Furthermore, let us consider the one significant result in the study: the proportion of successful intubations. Remember that the sample size was calculated to detect a difference in intubating times and no mention was made of the number of successes. To detect a reduction in the number of successful intubations from 95% to 55% (percentages changed slightly for ease of calculation) with 80% certainty would require about 30 patients in each group. Or, to put it another way, the current study had a 50% chance of detecting this difference, if it really exists [2]. Were the authors just lucky or is this a type I or α error? (In fact, a reduction from 95% to 55% is an incredibly ambitious target to anticipate from the outset. A reduction from 95% to 75%, perhaps more reasonable to expect, would require about 60 patients in each group for the same power).

The authors mention the possibility of bias in their method of laryngoscopy, but fail to mention the bias that might be introduced by their past experiences and preconceived ideas, clearly stated in the introduction, that the single-use bougie has '…very low rates of success…' and '…is intrinsically difficult to use'. So, rather than showing a 'striking difference between multiple-use and single-use bougies' we have a barely significant result ($p = 0.03$) in an underpowered and potentially biased study. Don't get me wrong, I'm not an apologist for the single-use bougie, but if we are to condemn it to a landfill site let's do it with good science.

One other thing whilst I'm having a bit of a power rant. Why is it that we accept a 5% (1 in 20) chance of finding a true difference that doesn't exist, but are happy to risk a 20% (1 in 5) chance of missing an intervention or treatment that may be beneficial to our patients?

S. Morris
University Hospital of Wales,
Cardiff CF14 4XW, UK
E-mail: Stephen.morris@
cardiffandvale.wales.nhs.uk

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A reply

Dr Morris criticises our study as being underpowered. The points made are mathematically interesting and require us also to consider more generally the limitations of power analysis.

Our study: power and significance

One small error in the text might have led to some misunderstanding. Our power analysis was based on a reduction in *total* time (not *intubating* time as written) of 20 s as being clinically important. The *post hoc* power analysis thus shows a mean difference in total time of ~12 s with a standard deviation of ~15 s. This gives a required sample size of ~20 [1]. We used 16 subjects, so (*post hoc*) our study had ~65% power [2] to exclude a difference in total times for intubation between the two groups at the $p < 0.05$ level. This is indeed slightly lower than conventional levels. However, it is clear from our paper that we were intentionally very conservative in our handling of the data. The total times for intubation we quoted for the single-use bougie were only for those attempts which were *successful*. Most investigators would have included the times at which *failure* of insertion occurred (i.e. ~120 s). If we had done this, the actual difference between the two groups would be ~40 s, yielding a *post hoc* power of > 95% and a statistically significant difference of $p < 0.01$ [2]. However, we were careful to avoid such dramatic treatment of the statistics and favoured a more careful approach, which minimised the risk of bias. Therefore, Dr Morris' view of power is simplistic: careful handling of the data is more important than any aspect of power analysis.

Dr Morris suggests that sample size also influenced our statistically significant result, and in this assertion makes a common error. The purpose of a prior power analysis is to be reasonably confident that a *negative* (i.e. non-significant) result is meaningful. If, on the other hand, a *positive* (i.e. statistically significant) result is obtained, the prior power analysis is irrelevant [1,2]. One simple illustration might help. Let us suppose that we wish to design a study to test the hypothesis that 'all swans are white', and prior power analysis suggests a very large sample size of (e.g. 10 000 swans). However, I choose to look at only one swan (which is black). Thus, the hypothesis is disproved in its entirety on the basis of a sample size of one. Regarding our study [1], one comparison was 'significant' at the $p < 0.03$ level. This simply means that this result might have occurred by chance no more than 3% of the time. Dr Morris' description of this result as 'barely significant' is therefore imprecise: it was significant at the 3% level: whether this is described as 'barely' or otherwise, is purely subjective. Note that sample size is not relevant to this debate. Generally, if there is concern about a false-positive result (α or type 1 error), the level at which a result is accepted as 'significant' would need adjustment. We can see no compelling need for this in our study.

General limitations of power calculations

Just as experiments have their limitations, so do power calculations. Blind faith in power calculations, as shown by Dr Morris, is misplaced. Power calculations are only *crude estimates* (with *crude* emphasised in the title of some relevant papers [3]). Two important factors contribute to power [1,2]: (a) the clinically important difference between groups; (b) the expected standard deviation of the measure of interest.

The choice of (a) can be open to much debate: why did we choose 20 s as the clinically important difference in our study; why not 15 s; or why not 45 s? This (artificial) precision has a large impact upon the power, yet the choice is often (and sometimes necessarily) arbitrary. Where no prior data exists, both (a) and (b) can be 'best guesses': this is self-evidently a poor (but clearly acceptable) means of calculating power [4]. Alternatively (a) and (b) may be estimated from a set of 'pilot' observations. In this case, great caution must be exercised: the pilot observations are, by definition, less robust than the results of the planned study. Pilot studies are not themselves subjected to a power analysis (and are rarely, if ever, subject to rigorous randomisation and control) [5]. Thus, philosophically, it seems curious to lend equal or greater weight to a small pilot study than to the larger substantive study. Perhaps the power analysis itself should be expressed in terms of a confidence interval (e.g. 'our power analysis indicated that we might use between 20 and 60 subjects to be between 70 and 90% confident of excluding a true difference between the means of 10–50 s…') [6].

The crudeness of power analyses is reflected in the different sample sizes yielded by different methods. If we assume that in a study the clinically important difference is 1 (arbitrary units) and the standard deviation is 0.8, then various power calculations give the sample size in each group (80% power at $p < 0.05$) to be: 10 [3], 11 [7], 13 [8], 18 [1].

Finally, I can answer the question in Dr Morris' last paragraph (at least in part) by reference to Lilford and Johnson [9]. Studies often compare two 'treatments', one established and one new. Usually, the new treatment carries greater 'costs' (e.g. financial, clinical or organisational). Therefore, in general it is appropriate to design studies with the slight risk of rejecting new treatments which might work. However, where 'costs' of two treatments are thought to be equal, it is entirely reasonable to power the study to greater than 80% [10].

Summary
Bacchetti [10] has explicitly stated that criticisms of power calculations should only be made when *unavoidable*: that is, when there is little else in a study to criticise, or when a study states clearly unrealistic goals, or fails to appreciate its other experimental limitations. Five of 11 paragraphs in our discussion drew attention to important experimental problems and limitations of the study. These potential criticisms (which we were careful to discuss of our own study) far outweigh in importance the rather obscure statistical points made by Dr Morris regarding power calculations.

J. J. Pandit
John Radcliffe Hospital,
Oxford OX3 9 DU, UK
E-mail: jaideep.pandit@physiol.ox.ac.uk

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Sleep deprivation and performance

I read with interest the study on sleep deprivation and performance (Murray & Dodds. *Anaesthesia* 2003; **58**: 520–5). I have been following the research into sleep deprivation and performance since, when driving home post 'on call', I found myself on the wrong side of the road with a large truck bearing down on me. This was in the bad old days of 72 h weekends, so things must be better now. But are they?

While juniors are given the day off post call, and many work shifts, consultants are expected to continue with the rest of the days work. Murray and Dodds do not state when their subjects started working in the morning, but 07:00–08:00 would cover most anaesthetic consultants. If you have then been supervising juniors, covering the ICU and/or maternity while on call, it is not uncommon to be up until 02:00 the next day before you can contemplate sleep, which will then be disturbed again 4–5 h later to get up to go to work again, or possibly sooner. You may well have been up all night and are then expected to continue to work the routine list the following day, as our contracts do not recognise 'on call' as work.

One study has equated the level of performance decrement using hand eye co-ordination after sustained wakefulness, and alcohol consumption [1]. After 18 h of wakefulness, the performance was equivalent to 0.05% blood alcohol concentration, and after 24 h, to 0.1% blood alcohol concentration. If the Trust found you to have such levels of alcohol in your blood while working, you would expect suspension and probably erasure from the medical register, and possibly criminal charges. However, if you have been working all night, it is considered acceptable to have the same level of performance, *as if* you had been drinking, and you still have to drive home.

In subjects who were sleep deprived for 24 h, Positron Emission Tomographic scanning (PET) for the utilisation of glucose as a marker for activity, showed decreased activity in the areas of the brain involved with attention and higher order cognitive thinking [2].

Consider the case of Gary Hart, after the Selby Rail Disaster he was sentenced to 5 years in prison for causing the deaths of 10 people; in summing up Justice MacKay said: 'In moral terms I see little to choose between a driver who sets off on a journey affected by drink and a driver setting off in your condition.' [3]. Gary hart had had little or no sleep the previous night.

Therefore, continuing to work when you have the subjective feeling of tiredness (with the support of objective research evidence), could be considered a breech of General Medical Council (GMC) guidelines, on the grounds of putting the patient at risk, when you know it is unsafe to continue [4,5]. Should something happen, it is not impossible that you could be proven 'negligent' on the grounds of 'forseeability' [6]. Even if you are not physically working, just being 'on call' will affect your sleep pattern and quality of sleep [7], which may have similar effects on your performance the next day if you had been working, as pointed out in the study. Appropriate management of this problem will result in reduced day time activity, but failure to act will continue to create risk. The problem will become more acute in August 2004 as the European Working Time Directive bites for junior doctors hours.

Anaesthesia has led the way in reducing patient risk and making life bearable for its staff; surely the time has now come for both the Association and the College of Anaesthetists to get together and support further research on the topic of performance and sleep deprivation?

In the meantime, there is enough evidence to suggest that it is unsafe for patient care, and the individual anaesthetist, to continue working the day after a busy 'on call', regardless of the grade.

S. R. Price
Burnley General Hospital,
Burnley BB10 2PQ, UK.
E-mail: srprice@doctors.org.uk

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Patients ideas of surgical risk

The survey on information that patients request (Moores & Pace. *Anaesthesia* 2003; **58**: 703–6) was very timely, and made interesting reading. The authors state that the commonest question asked of them by patients was that of the likely duration of surgery. This puzzled me slightly until it crossed my mind that perhaps patients were using the length of surgery as a signal of the level of risk and severity of the procedure. This is an analogous situation to that of the first question asked of many patients after surgery by relatives and friends, i.e. 'How many stitches did you have'. Patients may therefore be using surgical duration as a self selected guide to their particular level of risk, erroneously basing their question on newspaper reports and television documentaries where length of surgery is frequently discussed. It is easy to dismiss their question as one based on ignorance, but strangely enough, they may well be right [1]. It is also of interest to note that the question of surgical duration is not one highlighted in the recently published information booklet for patients, 'You and Your Anaesthetic' [2], but is briefly mentioned, and linked to risk, in 'Anaesthesia Explained' [3]. However, I doubt whether this can have influenced the patients taking part in the survey.

J. Palmer
Hope Hospital
E-mail: james.palmer@srht.nhs.uk

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Faulty unidirectional expiratory valve as a cause of rebreathing

We would like to report an incident involving a faulty unidirectional expiratory valve of a circle system with carbon dioxide absorber (Series 5 A absorber, Ohmeda) that led to rebreathing of carbon dioxide.

The anaesthetic machine (Ohmeda Excel 210) and the integrity of the carbon dioxide absorber and circle breathing system was checked for leakage prior to use and was deemed intact. The gas and anaesthetic agent monitor that was in use was the Datex Ohmeda (5250 RGM). This has a side-stream sampling port and was calibrated before use. After induction of anaesthesia for the first case, we noticed that the baseline carbon dioxide detected on capnography was elevated at about 4–7 mmHg. A check on the carbon dioxide absorber showed that it was neither due to tracking nor exhaustion of soda lime. The expiratory valve on initial assessment appeared intact. However, a further investigation into the possible causes revealed that the knife-edge of the valve seat of the expiratory valve was cracked and chipped (Fig. 1). It was most probably that the hole in the valve seat, despite the disc sitting in a correct position, allowed positive pressure ventilation to occur through the expiratory limb of the circle breathing system as well. This would explain the raised baseline carbon dioxide concentration.

A quick check of other similar Ohmeda series 5 absorbers showed that irregularities such as minute cracks and deformed edges were common. We speculate that this may be due to wear

Figure 1

and tear, or may be damaged during refitting of the cage onto the valve seat after cleaning. This demonstrates the importance of proper maintenance of the expiratory unidirectional valve, which may be a more common cause of rebreathing of carbon dioxide than reported. Vigilance of the attending anaesthetist and adequacy of monitoring are equally important as well.

W. S. Yew
N. C. Hwang
Singapore General Hospital,
Singapore
E-mail: ganyws@sgh.co.sg

Six smiths and safety

All the recent correspondence regarding syringe labelling and avoidance of drug errors reminds me of an occasion whilst working as a urology house officer in Bournemouth. At the time there were six patients named Smith on the ward. To my dismay, the sister in charge moved all six Smiths into the same six-bedded bay. However, her management was of course brilliant. Any intervention involving a Smith now demanded that the patient details were checked and checked again! I am certainly not advocating that all drugs be packaged in an identical manner, but I would agree that familiarity with the packaging and labelling of drugs definitely breeds a degree of complacency, as suggested by your correspondents (Amoako & Lavies. *Anaesthesia* 2003; **58**: 816). I am sure I am not alone in taking extra precautions whenever using 0.9% saline, water for injections or 1% lidocaine, all manufactured by Braun and presented in identical Mini-Plasco® vials.

J. G. Hudsmith
Peterborough Hospitals NHS Trust,
Peterborough PE3 9GZ, UK
E-mail: jonhudsmith@doctors.org.uk

Capnography detects tracheal placement, but is it 'correct'?

The recent study (Mallick *et al. Anaesthesia* 2003; **58**: 864–8) comparing capnography and bronchoscopy to detect correct needle insertion before performing a percutaneous tracheostomy concludes with a misleading statement. The study demonstrates that needle placement within the trachea is equally likely with either method. I take exception to that being called 'correct needle insertion'. As they state in their discussion, the bronchoscope 'ensured midline needle insertion at an appropriate tracheal level'. The capnographic method is a blind technique which is unable to provide any information about the proximity to the midline, or the level of insertion. A cadeveric study [1] demonstrated marked tracheal stenosis in two patients following oblique insertion of the cannula into the trachea. This complication would go completely unnoticed in a blind technique placement of the needle and tracheostomy. The other concern when trying to fashion tracheal access with a tracheal tube *in situ* is that the tracheal tube will be punctured by the needle insertion as happened in one patient in the capnography group in this paper. Again, this is another example of the needle being in the right place (the trachea), but not being in the correct place (within the indwelling tracheal tube).

I think a more accurate conclusion would be to say that the capnography method detects placement of the needle in the trachea, and in the absence of a bronchoscope being available, this should be considered. However, tracheal placement alone does not mean *correct* placement.

S. W. Benham
John Radcliffe Hospital,
Oxford OX3 9 DU, UK
E-mail: maguerite.scott@orh.nhs.uk

Reference

1 Van Heurn LWE, Theunissen PHMH, Ramsay G, Brink PRG. Pathologic changes of the trachea after percutaneous dilatational tracheotomy. *Chest* 1996; **109**: 1466–9.

A reply

We agree that there is a difference between needle placement in the trachea and the actual positioning of the needle through the anterior tracheal wall, with the latter only able to be confirmed with bronchoscopy. In our study, capnography was used solely to detect 'correct tracheal needle insertion' implying that the tip of the needle was in the right place. However, we clearly state that needle insertion remains blind and we make no reference to capnography locating correct needle position.

A. Mallick
S. C. Elliot
Leeds General Infirmary, Leeds, UK
E-mail: abhiram.mallick@leedsth.nhs.uk

A hidden safety feature in the event of oxygen failure

We would like to report a recent incidental discovery. Whilst checking an anaesthetic machine (Blease Frontline Plus 560™), it was discovered that failure of the oxygen supply not only shuts off the nitrous oxide and sounds the oxygen failure alarm, but also supplies compressed air to the air flowmeter irrespective of the position of the air/nitrous oxide interlock switch.

No mention of this feature is made in the company's brochure advertising the machine, although it is mentioned in passing in the user manual. This is achieved by means of a gas relay that, in the event of an oxygen supply pressure below 225 KPa, supplies air directly to the air flowmeter bypassing the interlock switch.

We believe that this is an important added safety feature, which ensures a continued supply of air to the common gas outlet in the event of an oxygen failure whilst remedial action is taken. However, the feature is only effective if the air flowmeter valve is left open, if it is not then no advantage is gained. We feel therefore that it should be recommended by the manufacturer that the air flowmeter valve is left routinely open even when oxygen and nitrous oxide are the gases in use.

C. M. Hearn
K. Chandradeva
Queen Mary's Hospital,
Sidcup DA14 6LT, UK
E-mail: chandra.chandradeva@qms.nhs.uk

A reply

We thank Drs Hearn and Chandradeva for drawing readers' attention to this feature. It is nice to know these hidden benefits in our equipment are being appreciated by anaesthetists.

A. Ferguson
Blease Medical Equipment Ltd,
Chesham HP5 2WX, UK

Tracheal ring fracture – dislodgement after Blue Rhino percutaneous tracheostomy

Percutaneous dilatational tracheostomy has become a standard procedure on many ICUs and we have carried out over 150 such procedures in the last 12 months. In spite of the frequency with which the procedure is carried out, it still carries the risk of serious complications. We would like to report a case of tracheal ring fracture–dislodgement following tracheostomy with a Cook 'Blue Rhino' percutaneous tracheostomy kit.

A 36-year-old man had had a coiling of a ruptured anterior cerebral artery aneurysm and required a tracheostomy as part of his postoperative care. Under general anaesthesia and bronchoscopic control, the trachea was punctured with the introducing cannula. The first puncture was not in the midline and a second puncture was required. The tracheostomy was then carried out uneventfully and a size 8 Portex tracheostomy tube was placed using the 24 French gauge introducer. Unfortunately, during the immediate post procedure bronchoscopy, a foreign body was seen in the mid trachea. This was removed with the suction port of the bronchoscope and was found to be a piece of cartilage 1.8 cm long and 4 mm wide. The dimensions suggested that it formed part of a tracheal cartilage. The patient made a slow recovery from the subarachnoid haemorrhage and the trachea was eventually decannulated after 8 weeks. The patient had no symptoms suggestive of tracheal narrowing and no change in voice; a further bronchoscopy was therefore not performed.

We are aware of only one previous case report of fracture dislodgement of a tracheal ring after serial dilatational percutaneous tracheostomy [1] and we are not aware of this complication having previously been reported after the 'Blue Rhino' technique**.** The incidence of tracheal ring fracture is, however, probably higher with the 'Blue Rhino' technique than when serial dilators are used. We are aware of at least three previous studies that support this observation [2–4].

It would seem likely that, in the case we describe, the same tracheal ring could have been transected during both needle introductions. The fact that the cartilage was only identified in the trachea after the tracheostomy tube had been sited, suggests the cartilage could have been dislocated during the insertion of the tracheostomy tube.

The case illustrates the importance of bronchoscopic control during percutaneous tracheostomy and of inspection of the tracheal after the procedure has been completed.

A. Thomas
S. Subramani
S. Mitra
Salford Royal Hospital,
Salford UK
E-mail: tony.thomas@srt.nhs.uk

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Fatal cardiac perforation by central venous catheter dilators: does the length matter?

Recently, the Medical Devices Agency has written to all parties involved with the insertion of central catheters. This letter dated 18th July 2003 (reference MDA/2003/020) warned of the dangers of over insertion of dilators that come with various single and multilumen central catheters. The solution proposed was to increase education and awareness of the issue.

Fatal cardiac or vascular perforation, although fortunately not common, is certainly not rare. There are many case reports published and most anaesthetists and intensivists will be aware of many unreported cases. The dilator has been implicated in both major vessel [1] and cardiac penetration [2]. Sensibly, clinicians should be aware of, and trained in the avoidance of, all the various mechanical complications of central venous access.

In almost all Seldinger method kits, the dilator is substantially longer than the needle or cannula used to insert the wire. This is unnecessary and we have asked various manufacturers why the dilators need to be so long. The dilator in percutaneous tracheostomy sets is the same length as the introducer cannula and functions satisfactorily. We would propose that dilators in central line kits do not need to be as long as they are, and I suspect that the reason is convenience of manufacture and possibly also historical acceptance.

W. C. Russell
J. Greiff
Leicester Royal Infirmary,
Leicester LE1 5WW, UK
E-mail: William.russel@uhl-tr.nhs.uk

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The Negus slotted laryngoscope

We read with interest your recent letter describing the use of the anterior commissure laryngoscope and we agree that this style of laryngoscope has particular advantages that have been described very well by the authors (Davies & Balachandran. *Anaesthesia* 2003; **58**: 721–2). As they state, the closed barrel style of laryngoscope may be of particular use in the patient with a difficult airway and in whom fibreoptic laryngoscopy may not be possible, e.g. when there is already, or is likely to be, significant bleeding. However, we would like to make a suggestion that in fact there may be an even more useful piece of equipment amongst the ENT surgical instruments. In common with the anterior commissure laryngoscope, the Negus slotted laryngoscope also has an enclosed barrel, with all the advantages previously described (Fig. 2). The cords may be visualised using the paraglossal approach from the right side, and the bougie advanced between the cords. However, the posterior section of the barrel of the Negus slotted laryngoscope may then be removed completely by sliding it towards the operator (Fig. 3). The tracheal tube may then be passed over the bougie and the laryngoscope removed completely without having to pass the laryngoscope back over the tracheal tube, and the tube connector may remain attached throughout. This removable section of the slotted Negus laryngoscope was originally incorporated to allow the advancement of a rigid bronchoscope, but this also allows the passage of a

Figure 2 The Negus slotted laryngoscope

Figure 3 The Negus slotted laryngoscope with posterior part of blade retracted

tracheal tube. Thus the trachea may be intubated under direct visualisation, and further local damage caused by blind advancement of the tracheal tube may be avoided.

Of course, it could be argued that fibreoptic intubation should be the 'plan A' in all patients in whom a pharyngeal, laryngeal or lingual lesion is suspected, but even then the use of the Negus slotted laryngoscope would make a very reasonable 'plan B'.

A. Dering
R. Corbridge
A. Kitching
Royal Berkshire Hospital,
Reading RG1 5AN, UK
E-mail: andydering@onetel.net.uk

Something to chew over?

We would like to present an unusual case of airway obstruction in a 30-year-old-primigravida female with a 35-week twin pregnancy who opted for vaginal delivery. An epidural was sited without incident, and the first twin was successfully delivered vaginally. Unfortunately, the second twin suffered shoulder dystocia and fetal distress, so the mother was taken for emergency Caesarean section. For expedience, the epidural was not used and a general anaesthetic was administered with intubation of the airway. The operation was uneventful and the patient was extubated at the end. Shortly afterwards, a stertorous noise was heard, although the patient continued to breath adequately with good oxygen saturations. Pharyngeal suctioning was repeatedly performed and oxygen delivered with a face-mask, but the unusual noise continued. The patient was turned into the left lateral position and subsequently suffered a bout of coughing, expectorating a lump of white material. The noise from her airway promptly ceased. On closer inspection, the object proved to be a piece of chewing gum. This had not been seen at laryngoscopy. Either it had been adherent to the teeth or gums, or she had swallowed it and it had been regurgitated into the pharynx. The patient was eventually discharged home with no problems.

This is not the first case report describing chewing gum obstructing the airway [1–4], although previously, the gum has adhered to an airway device (tracheal tube, etc.). We were not so fortunate. Chewing gum can be a very real cause of airway obstruction with potentially life-threatening consequences. Where there is a real chance of operative intervention, the obstetric population being a prime example, should chewing gum not be forbidden? After our experience, we feel that this should be the case, or at the very least it should be something the anaesthetist should chew over.

A. Afzaal
J. Kirk-Bayley
D. R. Uncles
Worthing Hospital,
Worthing BN11 2DH, UK
E-mail: azedafzaal@hotmail.com

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The ProSeal laryngeal mask airway: an easier and safer approach to tracheal tube/ laryngeal mask exchange

Exchanging the tracheal tube (TT) for the classic™ laryngeal mask airway (classic LMA) has been used following

a variety of surgical procedures to facilitate a smooth emergence, and offers advantages over the face-mask in terms of respiratory complications [1]. Although the technique is effective and probably safe, there are two reports of aspiration during the exchange [2,3]. In principle, the risk of aspiration can be reduced by maintaining anaesthesia or muscle relaxation (to prevent airway protective reflex activation), passage of a gastric tube (to empty the stomach) and placement of the classic LMA before extubation (to form a barrier in the hypopharynx). We describe a technique involving the ProSeal™ laryngeal mask airway (ProSeal LMA) that we consider easier and safer than using the classic LMA.

The technique involves the following steps, which should only take place with the patient deeply anaesthetised (>1.3 MAC [4]), or paralysed:

(1) pharyngeal suction,

(2) placement of the ProSeal LMA using the laryngoscope-guided, gum elastic bougie-guided technique [5],

(3) partial inflation of the ProSeal LMA cuff,

(4) holding the ProSeal LMA at the mouth to maintain the position of the distal cuff in the hypopharynx,

(5) inserting a gastric tube via the drain tube and emptying the stomach,

(6) disconnecting the anaesthesia breathing system from the TT and deflating the TT cuff;

(7) tracheal extubation while maintaining the position of the ProSeal LMA;

(8) connecting the anaesthesia breathing system to the ProSeal LMA;

(9) fixing the ProSeal LMA in position with tape;

(10) reversal of muscle relaxation, if required, and discontinuation of anaesthesia.

The advantages of the ProSeal LMA over the classic LMA are easy access to the stomach and improved ventilatory capability; a disadvantage is that a smaller fibreoptic scope must be used if laryngoscopy is required. The advantage of the gum elastic bougie over the digital and introducer tool techniques is that the distal cuff will be guided into the hypopharynx, increasing the likelihood that it will form an effective barrier to regurgitation. We have used this technique in 18 patients, mostly after thyroid surgery or suspension laryngoscopy, without any problems.

J. Brimacombe
N. Brimacombe
Cairns Base Hospital,
Cairns, Australia
E-mail: jbrimaco@bigpond.net.au
C. Keller
Leopold-Franzens University,
Innsbruck, Austria

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Contraindications to cricoid pressure

A 29-year-old man presented to the A & E department of his local hospital complaining of the sudden onset of painful dysphagia. This had occurred immediately following the ingestion of a piece of lamb shank as part of a three-course meal. Past medical history was unremarkable apart from an episode of atrial fibrillation following accidental electrocution one year previously. This had been successfully treated with DC cardioversion. A lateral neck X-ray demonstrated a radio-opaque foreign body in the oesophagus at the level of C6 [Fig. 4]. This was thought most likely to be a piece of bone. The patient was listed for urgent rigid oesophagoscopy.

Examination revealed an obese, 123 kg gentleman in obvious discomfort. He was cardiovascularly stable with heart rate 100 beat.min^{-1}, BP 140/85 mmHg and oxygen saturations in air of 97%. Airway examination was classified as Mallampatti II. The patient was noted to be exquisitely tender over the anterior part of his neck at the level of C6. The anaesthetist, concerned about the use of cricoid pressure during rapid sequence induction and the risk of this causing oesophageal perforation, consulted his ENT colleague. The opinion of the ENT surgeon was that there was a small risk associated with the use of cricoid pressure but this was significantly less than the risk of perforation if the patient was left for 6 h to allow his stomach to empty. On this basis it was decided to proceed with the case. The patient was premedicated with intravenous ranitidine 50 mg and metoclopramide10 mg. Anaesthesia was induced in theatre with alfentanil 1 mg, propofol 230 mg and succinylcholine 150 mg. Cricoid pressure was applied by a trained assistant immediately following loss of consciousness. Laryngoscopy revealed a grade II view of the larynx and the patient's trachea intubated with an 8.0-mm armoured tracheal tube. Anaesthesia was maintained in an oxygen/air mixture with 2% sevoflurane. Rigid oesophagoscopy revealed a segment of bone in the oesophagus with surrounding tissue oedema. The bone was removed and further examination demonstrated no evidence of perforation. The patient was extubated uneventfully and a postoperative chest X-ray was normal.

In Sellick's original paper, only vomiting is mentioned as a contraindication to the use of cricoid pressure [1]. Since then, a number of clinical conditions have been reported as possible contraindications to its use. These include actual or suspected cervical spine injury,

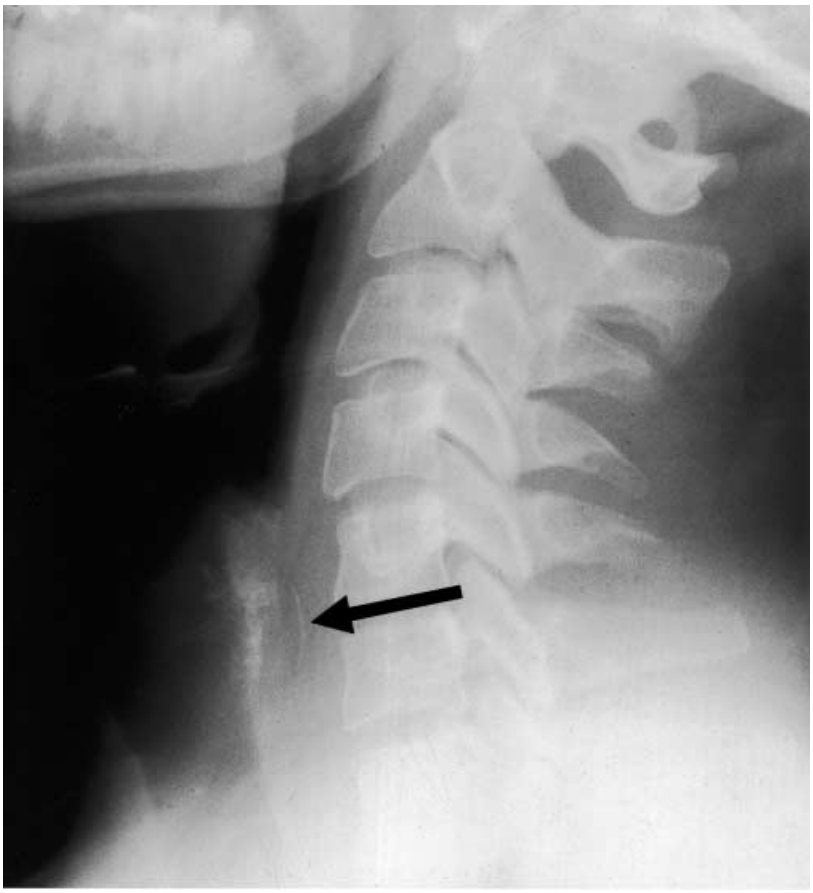

Figure 4

laryngeal trauma and the presence of a foreign body in the upper airway. Although intuitively it would seem reasonable to assume that the presence of a sharp foreign body (e.g. a bone) in the oesophagus would contraindicate cricoid pressure, a literature search revealed no case reports of oesophageal perforation in this context. A study by Canter *et al*. in cadavers [2] demonstrated no evidence of oesophageal trauma when sharp foreign bodies were placed in the cervical oesophagus and cricoid pressure applied. From this study and our literature search we would conclude that cricoid pressure can safely applied in this situation. We would welcome other readers' thoughts on the management of this case.

S. Lewis
P. Magee
Royal United Hospital,
Bath BA1 3NG, UK

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Incorrect blood results

Errors in blood transfusions have resulted in rigid protocols concerning the drawing of blood transfusion samples, their labelling, handling in the laboratory and subsequent checking of products for transfusion. However, a more casual approach to the handling of other blood samples remains. I would like to present a case where relying on incorrect blood results may have had as grave consequences as a mismatched transfusion.

Mr CC is a 73-year-old gentleman who presented to the Accident and Emergency Department with a month long history of left hip pain. He could not identify any traumatic precipitating event. His admission clerking states that his only past medical history was that of multiple myeloma diagnosed 6 months earlier for which he had received three courses of chemotherapy. X-rays revealed a fracture of the left neck of femur with evidence to suggest that the fracture was pathological. In the margin of the clerking, the blood results had been documented. Apart from a low white cell (WCC) count of $2.2 \times 10^9.l^{-1}$, the full blood count and clotting were normal, as were his electrolytes and renal function.

He was seen by an anaesthetist and both patient and anaesthetist agreed on spinal anaesthesia as the best course of action. His operation was delayed until the following day due to lack of theatre time and so he was seen by a second anaesthetist who questioned the presence of an isolated low white cell count in a patient who may well have poorly functioning bone marrow and other biochemical abnormalities. Indeed, upon checking the admission blood results on the computer they were found to be: WCC $2.2 \times 10^9.l^{-1}$, haemoglobin 11.5 $g.dl^{-1}$, platelets $62 \times 10^9.l^{-1}$, urea 7.8 $mmol.l^{-1}$, creatinine 151 $\mu mol.l^{-1}$.

The origin of the incorrect blood results remains unclear. Once the error had been identified, the advice of the haematologists was sought and the operation went ahead under general anaesthesia with broad spectrum antibiotic and platelet cover. The consequences of proceeding with the operation under spinal anaesthesia on the basis of the erroneous blood results may have been disastrous. Increased surgical bleeding may have been encountered and an epidural or spinal cord haematoma may have resulted from the choice of anaesthetic.

The question now is not who to blame for this incident but how to prevent a recurrence. The manual transcribing of results from computer to house officer notebook to patients' case

notes is obviously unacceptable, but laboratory generated results forms may take more than 24 h to return to the ward. My preference would be computer generated printouts on sticky labels complete with full patient details and date of test that could be stuck in to the patients' notes. This particular incident appears to have resulted from an error in transcription of results but perhaps it highlights the fact that all blood samples warrant the same degree of respect as blood transfusion specimens, with three forms of patient identification and signatures of the doctor, nurse or phlebotomist taking the sample on bottle and form. It also reminds me that all test results should be viewed in the context of the patient to whom they relate, and causes for any anomaly sought.

E. Combeer
East Surrey Hospital,
Redhill RH1 5RH, UK

Thrombosis related to emergency factor VIIa treatment

Factor VIIa is emerging as a treatment for a variety of coagulation disorders characterised by impaired thrombin generation and life-threatening bleeding. It initiates the coagulation cascade by binding to exposed tissue factor at the site of endothelial injury and causes the formation of thrombin to trigger coagulation [1]. Based on its mechanism of action, thrombosis is a potential risk, although this has only rarely been reported [2]. We report a case in which treatment with factor VIIa was life saving; however, the treatment was complicated by the clotting of an arteriovenous (AV) fistula.

A 58-year-old man with diabetes mellitus, end-stage renal failure and a right forearm AV fistula was scheduled for an emergency thoracotomy for a right-sided empyema. The pre-operative coagulation profile was normal. The surgery was uneventful and the patient was transferred to the Cardiac Intensive Care Unit (CICU) postoperatively. The patient underwent haemofiltration and the AV fistula was kept warm and examined regularly for a thrill.

Thirty-six hours post surgery, an increase in bleeding was noted and over the next 18 h this continued despite two re-exploration thoracotomies, neither of which revealed an obvious bleeding point. Non-operative treatment during this period included the administration of aprotinin 2000 000 units, desmopressin 16 μg, 17 units of packed cells, 2.5 L of fresh frozen plasma and 10 units of platelets.

The bleeding continued and 6 h after the second thoracotomy, he continued to lose an average of 500 $ml.h^{-1}$ into the chest drain. The haematologists were consulted and factor V11a (0.1 $mg.kg^{-1}$) was given. Within 30 min of receiving factor V11a the bleeding settled. Blood loss was only 250 ml in the subsequent 6 h after which, all bleeding ceased. He required only one further unit of packed cells.

The patient's AV fistula was documented to have had a thrill between the two re-exploratory thoracotomies. Eight hours later and 30 min after the factor VIIa was commenced, the renal team reviewed the patient. At this time, a thrill was initially absent although returned after the fistula was massaged. The next morning, 14 h after the factor VIIa, the fistula had no thrill and was presumed clotted. The patient recovered and was subsequently discharged to the renal unit.

Factor VIIa is a highly effective haemostatic agent that has been used in the prevention and treatment of bleeding in patients with coagulopathies and in the setting of massive haemorrhage. It's mechanism of action, dependent on the interaction with exposed tissue factor, localises its effect [3]. However, patients with other areas of abnormal endothelium may be at risk of thrombosis at these sites, for example those with coronary atheroma. Although we cannot be absolutely certain that the Factor VIIa initiated thrombosis of this patient's AV fistula, the timing is suggestive. High flows through the fistula may have led to vessel wall damage, predisposing it to thrombosis once the factor VIIa was given. This case suggests that thrombotic events in patients with abnormal vasculature are more than just a theoretical concern and that a cautious approach to the use of factor VIIa in such individuals is well founded.

J. L. Brown
S. Varma
N. J. Morgan-Hughes
G. Rocco
Northern General Hospital,
Sheffield S5 7AU, UK
E-mail: n.hughes@sheffield.ac.uk

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Malignant hyperthermia – patients don't always read the textbook

An individual anaesthetist may never see a patient with malignant hyperthermia during their career; they are even less likely to see an unusual presentation. The Association of Anaesthetists of Great Britain and Ireland has issued guidelines on the presentation and management of malignant hyperthermia (MH). While tachycardia and hypercapnea are invariably present and their onset often sudden, other features can be variable and the presentation insidious. A recent case illustrated this to us.

A 50-year-old man, who had not previously been anaesthetised, presented for re-implantation of his left forearm following traumatic amputation. He received an intravenous induction with fentanyl, propofol and rocuronium and anaesthesia was maintained with oxygen, air and isoflurane. He remained haemodynamically stable for 3 h but then became hypotensive requiring incremental metaraminol to a total dose

of 9 mg over 45 min Shortly after the onset of hypotension, his heart rate and end-tidal CO_2 started to increase slowly, reaching peaks of 150 beat.min^{-1} and 94 mmHg, respectively. Naso-pharyngeal temperature also increased slowly to a maximum of 39.5 °C by which time the diagnosis had been made and intravenous dantrolene started. After 100 mg of dantrolene had been infused, his heart rate, temperature and end-tidal CO_2 started to return to normal. The re-implantation was successfully completed and the patient made a full recovery. He has since undergone a muscle biopsy, and in-vitro contraction testing has confirmed his susceptibility to malignant hyperthermia.

This case illustrates two important points. Firstly, there can be a delay between exposure to trigger agents and the appearance of signs of MH. This may be particularly important in shorter cases where the onset may be in the recovery area where neither end-tidal CO_2, nor temperature is monitored. Second, despite a hypermetabolic state, the patient can be hypotensive rather than hypertensive. In addition, it is worth noting that susceptible patients will not always manifest the signs of MH on their first exposure to trigger agents.

S. Wimbush
J. Homewood
Frenchay Hospital,
Bristol BS16 1LE, UK
E-mail: wimbush@ukgateway.net

Ionised magnesium concentrations in non-neurosurgical patients undergoing spinal anaesthesia

It has been reported that changes in cerebrospinal fluid (CSF) ionised magnesium (iMg) concentration can modify analgesic effects *in vivo* [1] and alter the general anaesthetic actions *in vitro* [2]. Since it is important to know whether measurements of the plasma iMg concentration predict the alteration in CSF iMg concentration, we investigated the relationships between plasma and CSF iMg concentrations in non-neurosurgical patients with an intact blood–brain barrier (BBB).

After institutional approval and informed consent, 57 patients, aged 19–87 years, undergoing non-neurosurgical procedure under spinal anaesthesia were enrolled in this study. Two ml of arterial blood and 1 ml of CSF were obtained prior to the surgical procedure for determination of plasma and CSF iMg concentration with an electrolyte analyser CRT8^{TM} (NOVA, Waltham, MA, USA). The mean iMg concentrations in plasma (0.49 SD 0.05 mmol.l^{-1}, $n = 57$) were lower than those in CSF (0.77 SD 0.04 mmol.l^{-1}, $n = 57$). There was no correlation between plasma and CSF iMg concentrations (Regression equation: $y = 0.745 + 0.058x$, $r = 0.06$; $p = 0.65$, $n = 57$), suggesting that CSF iMg concentration tends to remain stable regardless of variation in plasma iMg concentration in patients with an intact BBB. However, Fuchs-Buder *et al*. [3] reported that an intravenous administration of magnesium sulphate led to a significant increase in CSF iMg concentration in neurosurgical patients and that it took 90 min to raise CSF iMg concentration following the injection. The present results cannot rule out the possibility that long-term loading or depletion of plasma iMg might affect the CSF iMg concentration.

R. Sasaki
K. Hirota
M. Yamazaki
Toyama Medical and Pharmaceutical University, Toyama,
930–0194, Japan
E-mail: koki@ms.toyama-mpu.ac.jp

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A novel role for magnesium?

We would like to highlight a recent example of the use of intravenous magnesium in a lady with severe asthma undergoing elective lower segment Caesarean section (LSCS) under combined spinal-epidural anaesthesia. Despite the increasing use of magnesium in a wide range of clinical settings, there have been no publications describing its use in this scenario.

A 30-year-old Caucasian lady was admitted for an elective LSCS for the delivery of her second child. She had been asthmatic since the age of 3 years, requiring numerous hospital admissions. On her previous admission for an acute exacerbation, she had responded well to intravenous magnesium administered in the emergency department. Her asthma was normally poorly controlled on a combination of long-term high-dose steroids (> 30 mg prednisolone daily), theophyllines, nebulised β agonists and nebulised antimuscarinics. Despite this therapy, she had an average peak expiratory flow rate (PEFR) of only 250 l.min^{-1} and required home oxygen, especially at night. She had a body mass index of 36 kg.m^{-2} and past medical history included a thrombophilic tendency having suffered from two previous deep vein thromboses. Her first child had been delivered by LSCS 18 months previously under a combined spinal-epidural anaesthetic. Throughout the whole procedure she had felt extremely short of breath and distressed despite nebulised β agonists.

Prior to her second LSCS she received intravenous magnesium sulphate 2 g in 10 ml normal saline over 10 min. A combined spinal-epidural was then performed in the sitting position and 0.5% heavy bupivacaine 2.5 ml and diamorphine 0.3 mg were injected into the subarachnoid space. She was placed in the supine position with left tilt and received nebulised β agonists with oxygen 4 l.min^{-1} once the operation started. There were no signs of tachypnoea or respiratory distress and her oxygen saturations remained greater than 98%

throughout the duration of surgery (50 min). Her PEFR increased from 200 to 275 $l.min^{-1}$ following administration of the magnesium. She reported no adverse side-effects and the surgeon reported no increase in blood loss.

Magnesium plays a vital homeostatic role within the body via transmembrane and intracellular modulation of electrical cellular activity. It causes smooth muscle relaxation by altering extracellular calcium influx and intracellular phosphorylation reactions. These properties are utilised in a range of pathologies including pre-eclampsia, cardiac arrhythmias, ischaemic heart disease, migraine and alcohol withdrawal [1].

Magnesium was first reported anecdotally for the treatment of asthma 50 years ago [2]. Its use has been further supported in recent years following several published randomised clinical trials. Skobeloff *et al.* [3] reported improved PEFR and discharge rates from emergency departments in adults with severe asthma. Two further trials [4,5] have highlighted its effectiveness in moderate to severe asthma in both paediatrics and adults at a dose of 20–25 $mg.kg^{-1}$ and 2 g, respectively. Its use in pregnancy for the treatment of acute bronchospasm has also been documented in a case report [6].

Pregnancy is often associated with exacerbations of asthma especially as gestation increases. This is multifactorial due to an increase in prostaglandin levels, raised upper airway resistance, decreased expiratory reserve volume and a decrease in cell-mediated immunity resulting in an increased susceptibility to upper respiratory tract infections and inflammation. These factors, together with a splinted diaphragm, anxiety and the supine position, make it likely that asthmatic symptoms are exacerbated during Caesarean section. The use of magnesium in this case is further supported because pregnancy can significantly decrease serum magnesium levels, especially in white patients [7]. Although our patient's measured serum magnesium was only just outside the normal range (0.69 $mmol.l^{-1}$; normal range 0.70–1.10 $mmol.l^{-1}$), serum magnesium levels do not necessarily correlate with total body stores [8].

Magnesium may be a useful adjunct in the brittle asthmatic undergoing Caesarean section and we would be interested to hear if others have used it in this situation.

J. Bromilow
A. McCormick
Poole Hospital NHS Trust,
Poole, UK
E-mail: brom@doctors.org.uk

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Diprifusor – things are not always what they seem

Computerised equipment is frequently used in modern anaesthesia. In clinical practice, we do not necessarily pay attention to their engineering details. 'Diprifusor' a Target Controlled Infusion (TCI) pump for use with propofol is a perfect example of this. We discovered that by operating a latch on the right hand side, the pump could be flipped open as seen in Figs 5 and 6. We performed a medline search to resolve the mystery of what looked like two pumps.

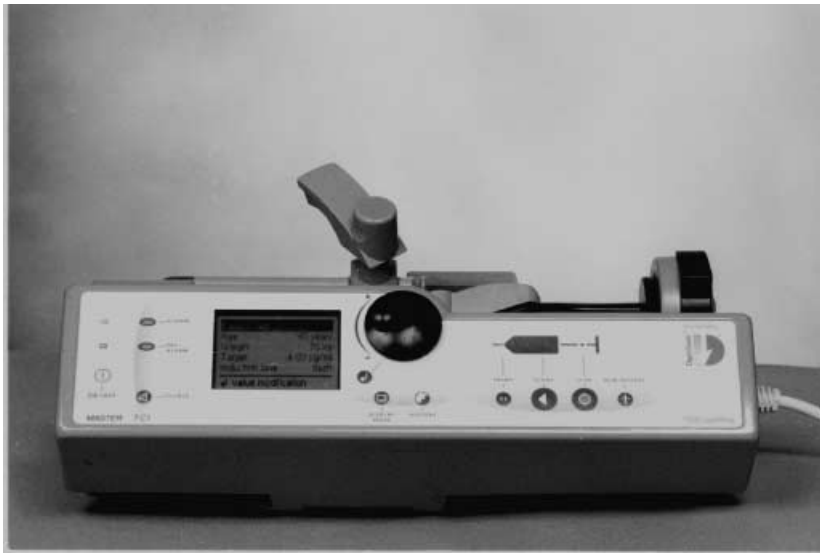

Figure 5 The 'Diprifusor' pump façade

Figure 6 Behind the façade of the 'Diprifusor' pump is a syringe driver.

It became clear that behind the mask of the TCI (Fig. 5) technology is the face of a simple syringe-driver (Fig. 6). The TCI software enables the syringe-driver to deliver a desired target concentration, the pharmacodynamic response being influenced by pharmacokinetic parameters incorporated into the system.

The pharmacokinetic programme selected for the 'Diprifusor' consists of a three-compartment model with modification for distribution to the central compartment as described by Marsh *et al.* This model tends to produce a 16% greater blood concentration than predicted by the system. This improves predictive performance of the system to provide acceptable clinical anaesthesia.

We think that for the casual TIVA users like us this is an interesting finding. We realise that anaesthetists with a special interest in TIVA may be already aware of this.

M. Vanarase
G. Biswas
Wycombe Hospital,
High Wycome HP11 2TT, UK
E-mail: hgargi@aol.com

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Pulmonary mechanics and volatile anaesthesia

The paper about the pulmonary mechanics during isoflurane, sevoflurane and desflurane anaesthesia (Dikmen *et al. Anaesthesia* 2003; **58**: 745–8) investigates an interesting topic namely the bronchodilator effects of the different inhalation anaesthetics. However, I have some questions about the methodology, results and conclusions of the authors?

The authors use the concept of MAC, but in the methods we read two important facts: First, they used fentanyl. As we know, the MAC value is defined for anaesthesia without morphine derivates. The MAC with morphine derivates is always lower than the well-known and used MAC value. On the other hand, the authors used the term 'equivalent doses… a concentration of approximately one MAC'. What does approximately means? Knowing that the pharmacokinetics of the different inhalation anaesthetics are different, can we prove that effects were measured at a stable steady state value of inspiratory fraction (Fi) or expiratory fraction (Fe). What does approximately in 'steady state' mean? Second, we have to evaluate the effects of fentanyl on the bronchial smooth muscles. If we could accept that this effect is similar for each inhalation agent, then this paper could be of value. If not, what are the conclusions? Considering the half time of fentanyl, we have to keep in mind the effects of this drug in this study.

Last but not least, the authors conclude that the effect of desflurane at 2 MAC results in an increase in airway resistance. If we look at the results, the respiratory resistance base-line value of desflurane is 19.9 (4.0) and at 2 MAC 19.7 (3.4), 19.7 being statistically not significant compared to the baseline. In mathematics (19.9–19.7)/19.9 = 0.01 or only 1%. Is it clinically significant? Please also take into consideration the variability of the method of measuring the respiratory resistance. Can we conclude that we really have bronchoconstriction? What is its clinical significance? All we can say is: if you go abruptly from 6% to 12% desflurane for only 5 min, there is no bronchodilation. But it doesn't necessarily mean bronchoconstriction. This questions whether the well known pungency of the drug can explain this and according to the paper of Ebert [1] about the cardiovascular effects, if this effect is equally short lived (± 7 min) or not? Finally how can they write that at T 2 the PIP decreased 6.7% (13.8) with isoflurane, when the reported values are 20.7 (5.9) and 18.8 (4.2)? In mathematics (20.7–18.8)/20.7 = 0.09 or 9% and not 6.7%. If you look at the other results you will have the same surprises. If you consider not the absolute mean value but the dispersion around it, there could be questions about the validity of the results and the conclusion. Why should we compare T3 with T2 and not with the baseline T1?

J.-L. Demeere
Clinique Générale St Jean,
1000 Brussels, Belgium

Reference

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A reply

Dr Demeere has pointed out some interesting points. First is the comment on MAC and fentanyl. It is widely accepted that morphine derivatives may decrease the need for inhalation agents; but it is impossible to find good evidence how these three agents are influenced by the fentanyl dose used in our study. The MAC values we used were the concentrations in oxygen/air mixture. We believe that giving fentanyl 1 $\mu g.kg^{-1}$ on induction should have a minimal effect and should be in the same proportion for all agents. The same inhalation doses of isoflurane, sevoflurane and desflurane with fentanyl had been used in other studies, without the need to discuss this issue [1]. The use of MAC gives the opportunity to compare the effects of different inhalation agents, accepting that these concentrations are equipotent. The application of inhalation doses for 5 min was considered sufficient to reach a steady state and to measure the effects of these agents on bronchial tone, although longer periods such as 10 min were used in other studies for different end points [1]. As all agents are highly lipid soluble and have a potent action, and as they directly interact with bronchial mucosa to exert their effects, 5 min should suffice.

Another point is the effect of fentanyl on bronchial tone and its duration of action. Fentanyl is a potent morphine derivative with fast onset and intermediate duration of action (0.5–1 h) [2]. It may cause rigidity of the muscles of the thorax, but this effect may be inhibited by the use of muscle relaxants. Even if it had an effect on bronchial tone, this would be the same for all patients as the same dose was used in all. That's why we don't consider the use of fentanyl as a confounding factor.

When you compare the respiratory resistance values at T1 and T3, namely without inhalation anaesthetic and at 2 MAC inhalation, the values are quite similar with no statistical difference. But the decrease from base line at 1 MAC inhalation is significant and it would definitely indicate a decrease in bronchial tone (given the other variables which effect respiratory resistance, such as flow rate, tracheal tube size, are held constant). Also the increase in respiratory resistance after switching to 2 MAC is statistically significant when compared to 1 MAC, which would indicate an increase in bronchial tone. It is true that it returns to baseline and it is not clinically significant. There would be only one explanation for this increase, and that is bronchoconstriction. This may be explained by the pungency of desflurane, when you increase the concentration, there may be an increased reaction to this agent.

When comparing the effects of different agents on different variables, simple mathematics can't be applied. You either compare the individual values obtained from every patient or you calculate the difference (i.e. percentage change) induced by the application (i.e. increasing the concentration of agent) in each patient. To my knowledge, it is not applicable to compare the difference in means in this context.

We compared T3 with T2 and also with T1 with the use of ANOVA for repeated measures and Tukey tests. This is because we aimed to see that whether increasing doses of inhalation agents causes an increased effect. We observed a continuing bronchodilating effect with 2 MAC sevoflurane, no change with isoflurane and an increase in respiratory resistance with desflurane.

Y. Dikmen
Medical School of Cerrahpasa
34303 Istanbul, Turkey

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Fetal distress and the 30-minute rule

The Editorial (Yentis. *Anaesthesia* 2003; **58**: 732–3) highlighted the problems with the diagnosis and management of 'fetal distress'. The tool with which this diagnosis is often commonly made, namely the cardiotocograph (CTG), is imprecise; therefore, it is not surprising that the diagnosis would often be incorrect. A normal outcome following fetal distress is a retrospective finding; therefore, it is understandable why obstetricians, midwives and obstetric anaesthetists become 'distressed' on account of the fetus in the face of unsatisfactory CTG patterns given that is could possibly indicate fetal hypoxia, the severity of which may be impossible to estimate.

There are attempts by obstetricians to improve this situation by the presence of senior obstetric cover on the Delivery Suite as well as a new classification system for Caesarean section. Despite these, the need to deliver babies at very short notice will remain. What is required is an agreed protocol for response in these situations.

There is evidence that the 30-minute interval for delivery of a baby that may be compromised is arbitrary and unscientific [1]. It is also illogical that this same time interval holds in both large and small units where staffing issues are clearly not the same.

Until we are better able to decide whether fetus is compromised or not, agreed protocols for rapid response by all concerned, communication and teamwork are the ways forward.

U. I. Esen
South Tyneside District Hospital,
South Shields NE34 0PL, UK

Reference

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Syntocinon and 'epidurals' in labour – which comes first?

Numerous studies have been published that show an association between the use of regional analgesia and the need for augmentation of labour [1–3]. We have found that great emphasis is given to this when non-anaesthetists discuss regional analgesia with women. How-

Table 1 Syntocinon requirements. Values are number (proportion).

	Total $n = 500$	Spontaneous labour $n = 326$	Induced labour $n = 174$
No syntocinon	198 (40%)	167 (51%)	31 (18%)
Syntocinon before analgesia	187 (37%)	70 (22%)	117 (67%)
Syntocinon after analgesia	115 (23%)	89 (27%)	26 (15%)

ever, little mention is made of work that suggests that this association may not be *causal* [2,3]. We had the impression that regional analgesia is often requested when contractions become painful *as a result of* syntocinon- being started, or the woman is advised to 'have an epidural *before* the drip which makes the contractions painful'.

We carried out a simple audit to determine which came first, augmentation (or the perceived need for augmentation) of labour or the use of regional analgesia. We recorded whether a syntocinon- infusion was already in progress, or had been planned, at the time of requesting regional analgesia. At the postnatal follow-up, we noted if a syntocinon-infusion had been used prior to delivery. Data were collected on 500 consecutive women requesting regional analgesia. Of these, 61% were nulliparous and 65% had a spontaneous onset of labour. Median gestation was 40 weeks (IQR 39–41 weeks) and the median cervical dilatation at the time of request was 3 cm (IQR 3–5 cm).

Of the women who received regional analgesia, 302 (60%) also had intravenous syntocinon to augment labour. Of these, however, 187 (62%) were already receiving syntocinon or its use had been planned by the time regional analgesia was requested. In total, 115 women (23%) required augmentation of labour following regional analgesia (Table 1). Of the 326 women in spontaneous labour, 89 (27%) required augmentation following regional analgesia (Table 1). This compares with an overall augmentation rate of 22% for spontaneous labours in our unit over the same time period. A survey of intrapartum care for low risk primigravidas in the UK [4] found that 28% of spontaneous labours (range 13–46%) were augmented with syntocinon. In conclusion, in our unit, 60% of women with regional analgesia required augmentation of labour. However, in the majority of cases (62%), this was either planned or already in progress before the regional analgesia was commenced. Therefore, regional analgesia cannot be held solely responsible for high rates of augmentation of labour. A policy for active management of labour is likely to be associated with a higher demand for epidurals [2].

N. L. Lewis
F. Plaat
A. M. Qureshi
Queen Charlotte's and Chelsea Hospital, London W12 0HS, UK
E-mail: FPlaat@hhnt.org

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Ethical considerations in obtaining consent under anaesthesia

We read with interest the report (Kuehn *et al. Anaesthesia* 2003; **58**: 725–6), describing the management of a woman who was actually planned for evacuation of retained products of conception but finally ending in delivering a baby. We would like to comment on one of the statements made in the article about obtaining consent. The authors mentioned that the patient's mother could not be traced for consent. This implies the team had alternatively sought to seek consent from the mother, which is not inline with the General Medical Council's (GMC) advice statement. The GMC document 'Seeking patients' consent: the ethical considerations' [1] states that:

'At age 16 a young person can be treated as an adult and can be presumed to have capacity to decide'.

No one may make decisions on behalf of a competent adult.

The patient mentioned in the article was 16-year-old and according to the GMC statement [1], can be treated like an adult and no one can make decisions on behalf of a competent adult.

Under general anaesthesia, if a procedure other than that for which the patient has consented needs to be performed, it may be done only in situations where the treatment is intended to save life or avoid significant deterioration in the patient's health [1]. My question to the author is whether they would have done the Caesarean section if they were able to get the parental consent. We think it would have been inappropriate to perform Caesarean section on this patient even after parental consent for the following reasons:

The clinical diagnosis was incorrect. Proceeding to performing a Caesarean section, even with parental consent, is inappropriate since the patient was not informed of the misdiagnosis and offered an option.

Medical consent to perform Caesarean section in this instance would be inappropriate because that was not the only option available for the patient, the other being normal delivery as it had happened.

The GMC 'best interest' principle also states that in a patient who lacks capacity to decide, one may take views of the patient from a third party (like a parent). However, one should also consider the options that least restricts the patient's future choices where more than one option (including non-treat-

ment) seems reasonable in the patient's best interests. Hence, it would have been inappropriate to do a Caesarean section anyway.

M. Ranganathan
G. Raghuraman
Birmingham Heartlands Hospital,
Birmingham B9 5SS, UK
E-mail: Raghulatha@aol.com

Reference

1 General Medical Council Seeking patients' consent the ethical considerations. London: 1998, GMC.

A reply

Thank you for the opportunity to respond to the comments made by Drs Ranganathan and Raghuraman on our report.

The consent for Caesarean section from the mother was sought in the consultant gynaecologist presence's. This was thought to be in the best interest of patient and her baby under the circumstances (she was fully dilated and about to go into the second stage of labour). With hindsight and after a normal delivery, we agree it might not have been the best possible option for the patient.

As regards to the misdiagnosis, another fact came to light following the delivery. For social reasons, she had deliberately misled the medical staff by concealing the date of her last menstrual period.

All we now can say is that perhaps the inability to find the mother perioperatively was a blessing in disguise.

H. N. Saxena
B. Kuehn
G. Menon
Mayday Hospital,
Croyden, UK
E-mail: harshnsaxena@hotmail.com

Whoosh test 2 and confirmation of lumbar epidural space

Loss-of-resistance [LOR] to injection of air or saline as the tip of the epidural needle penetrates the ligamentum flavum is a popular means of identifying the epidural space [1]. Operators use either a glass syringe or a disposable plastic syringe to elicit LOR. We have observed that the LOR syringe, especially a glass syringe, occasionally elicits a false positive LOR to air injection. This may be due to unrecognised air leak secondary to poor connection of the syringe to the hub of the needle, leak between the barrel and the piston, and at times leakage between the track made by the larger introducer and the epidural needle. Since the LOR syringe is usually attached after the epidural needle has been introduced to a depth of 2–3 cm, any false positive LOR raises the apprehension of proceeding any further. To overcome this dilemma, when suspicion of false positive LOR exists, we perform the Whoosh Test-2 originally suggested for identifying the caudal epidural space [2]. We now follow a protocol wherein an injection of 1 ml saline with 0.5 ml air is made through the epidural needle. An assistant auscultates for the Whoosh – 2 sound two dermatomes above the injection site. To elicit an audible Whoosh – 2 sound, this injection of saline and air should be made rapidly as the Tuohy needle is of a wide bore and its spread is in either direction unlike the small gauge needle used for caudal injection where the spread is predominantly upwards. A negative Whoosh – 2 sound clearly identifies the false positive LOR and the operator either proceeds afresh in an adjacent epidural space and/or with a new LOR syringe. To date, we have never had a failed epidural following a positive Whoosh Test – 2.

R. M. Khan
J. Chabra
M. T. Alam
M. Ashraf
D. Jain
J.N. Medical College, A.M.U.,
Aligarh – 202002 [U.P.], India
E-mail: seeras_alig@rediffmail.com

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Diagnostic tattoo in a parturient with 'ecstasy' use

A 17-year-old woman at 38.5 weeks gestation was in labour and requested analgesia. When interviewed by the anaesthetist, she denied any medical and/or social problems (including substance abuse). However, preanaesthetic physical examination of her lower back revealed an unexpected 'diagnostic' tattoo in the sacral area (Fig. 7). Upon

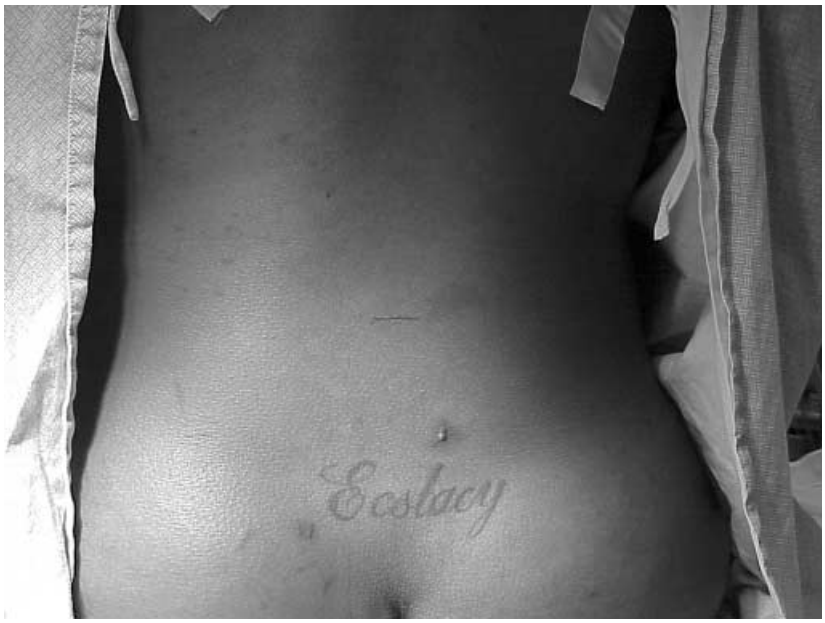

Figure 7 Lower back 'Ecstasy' (misspelled) tattoo encountered in our patient

further questioning, the patient admitted that she had her 'Ecstasy' tattoo inscribed a few weeks after engaging into recreational 3,4-methylenedioxymethamphetamine (MDMA, i.e. ecstasy) use.

Tattoos are an increasingly prevalent form of self-expression, especially for adolescents and young adults [1]. Roberts and Ryan studied the behavioural risk factors associated with tattooing in adolescents and young adults and concluded that permanent tattoos are strongly associated with high-risk behaviours including substance use, unprotected sexual intercourse, and domestic violence [2].

In summary, in the clinical setting, the presence of a tattoo noted during physical examination of a young parturient can alert obstetricians and anaesthetist to the possibility of other risk-taking behaviours.

K. M. Kuczkowski
UCSD Medical Center,
San Diego, CA 92103–8770, USA
E-mail: kkuczkowski@ucsd.edu

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Announcement

The journal would like to inform its readers that the 'Deaths of members reported to Council October 2002 to September 2003' will appear in the published January 2004 issue.