Correspondence

Rapid sequence induction: suxamethonium or rocuronium?

Drs Cadamy and Booth condemn the use of rocuronium at rapid sequence induction (RSI) and advocate a propofol/alfentanil regimen as an alternative to thiopentone/suxamethonium (*Anaesthesia* 1999; **54:** 817).

Although a number of studies involving healthy subjects have shown that tracheal intubation can indeed be performed without neuromuscular block, there is no evidence that such propofol/opioid techniques are safe in those elderly and unfit patients undergoing RSI who will be less tolerant of the likely concomitant hypotension. I believe that the emergency patient whose trachea proves difficult to intubate runs a greater risk of hypoxaemia and aspiration from waning suxamethonium block as opposed to continuing profound nondepolarising block with rocuronium. A partially paralysed patient bucking and straining will consume large amounts of oxygen. Huge increases in intragastric pressure have been measured [1]. I suggest that partial recovery from suxamethonium has the potential to turn a difficult intubation into a failed intubation, and 'can't intubate, can ventilate' into 'can't intubate, can't ventilate'. The timecourse of recovery from suxamethonium block is of course unpredictable [2]. Benumof and colleagues have warned against relying on recovery from suxamethonium to enable a patient to breathe adequately before critical desaturation occurs [3]. The lack of concordance in the practice of RSI and management of failed intubation has been highlighted recently [4]. Adoption of rocuronium at RSI means that the anaesthetist is committed to maintaining oxygenation by positive pressure ventilation. However, sustained optimal conditions will be afforded for unhurried successful tracheal intubation or placement of a Combitube/LMA. The guaranteed absence of pharyngeal and laryngeal reflexes must surely increase the likelihood of safe airway management without provocation of vomiting/regurgitation.

Rocuronium is an immunologically clean drug, devoid of the catalogue of potential complications peculiar to suxamethonium. Randomised trials of suxamethonium vs. rocuronium in elective and emergency patients undergoing RSI have confirmed that rocuronium 1.0 mg.kg^{-1} provides intubating conditions clinically equivalent to suxamethonium 1.0 mg.kg^{-1} after 60 s [5, 6]. Although there are as yet no published comparisons of morbidity following unanticipated difficult tracheal intubation at RSI with the two agents, I believe that continued unquestioning faith in the 'safety' of suxamethonium, based on its (unpredictable) spontaneous offset, is unwarranted.

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A reply

Our objection to rocuronium for rapid sequence induction (RSI) is its prolonged duration of paralysis. The papers quoted by Dr Levy have shown that rocuronium provides intubating conditions similar to suxamethonium 1.0 mg.kg^{-1}. The doses compared were 0.6 or 1.0 mg.kg^{-1} rocuronium and suxamethonium 1.0 mg.kg^{-1} [1, 2].

The relatively rapid recovery of

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suxamethonium although, perhaps, unpredictable is usually more rapid than the 60–70 min described for such doses of rocuronium [3]. Prolonged paralysis is not a problem in the face of a difficult intubation providing the airway is easy to maintain. If the anaesthetist is struggling to maintain the airway, then the more rapid the recovery of airway reflexes, the better.

The postulated risks of hypotension with the propofol/alfentanil technique need to be balanced with the known morbidity and mortality following the indiscriminate use of muscle relaxants in patients who cannot be intubated. To attempt to solve some of the concerns relating to suxamethonium with a drug whose effect persists for over an hour is illogical and dangerous. We stand by our original criticisms of this technique.

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Use of the airway exchange catheter for the patient with a partially obstructed airway

The July editorial (*Anaesthesia* 1999; **54:** 625–8) provided useful guidance and a rational approach to the obstructed airway. We completely agree with the de-emphasis on fibreoptic techniques in this situation. However, even after gaseous induction, dilemmas still exist. A patient may maintain an airway throughout induction only to lose it at the point of attempted intubation when friable tumour may bleed or completely obstruct the airway if disturbed. Thorough pre-oxygenation will not necessarily protect the patient if complete obstruction occurs. We would like to describe a case in which we used a technique that was not mentioned in the editorial to reduce the risks in this situation.

A 79-year-old woman presented with a 7-month history of progressive stridor. Pre-operative assessment suggested severe upper airway obstruction, requiring her to sit up to maintain airway patency. Minimal pressure on the front of her neck resulted in total airway obstruction. A CT scan and flexible bronchoscopy performed elsewhere had shown a large subglottic tumour. The ENT surgeons were planning to perform a tracheostomy.

An inhalational induction was performed with halothane in oxygen with the patient in the semisitting position. Laryngoscopy gave a good view of the laryngeal inlet and confirmed a large haemorrhagic subglottic tumour with a narrow posterior airway. Tracheal intubation with a size 5.0 mm microlaryngeal tube could not be performed because of airway distortion and the risks of haemorrhage or tumour dislodgement. A Cook Airway Exchange Catheter was instead passed gently through the airway opening, the ability to ventilate via the catheter confirmed, then the tracheal tube was passed carefully over the catheter. The patient was placed supine and an uneventful tracheostomy performed.

The Cook Airway Exchange Catheter is a narrow, semirigid, catheter that has a blunt tip and resembles a long gum-elastic bougie but is hollow with distal sideports. A variety of sizes are made between 2.7 mm and 6.3 mm external diameter. Oxygenation can be maintained via the catheter itself, which has a removable 15-mm or Luer lock connector (Fig. 1). Although specifically designed to allow oxygenation during single- or double-lumen tracheal tube exchanges, it can be used to assist the initial placement of a tracheal tube. Used as an adjunct to an inhalational technique, it allows the anaesthetist the security of knowing that the patient with a severely narrowed airway can be

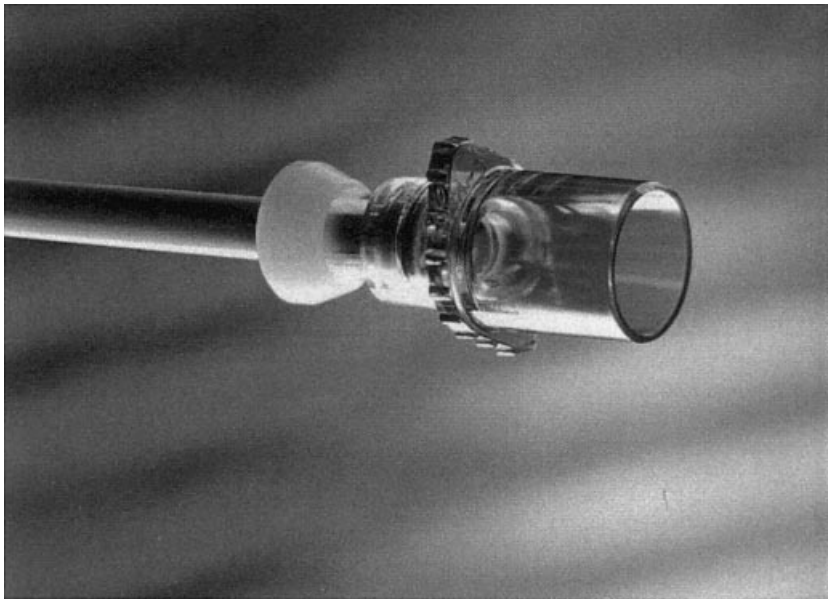

Figure 1 Cook Airway Exchange Catheter with removable 15-mm connector. Photograph provided by Cook UK Ltd.

oxygenated when definitive tracheal intubation is difficult or hazardous.

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The use of an oxygen source attached to the nonventilated lung during one-lung anaesthesia

We read with interest the recent article by Pfitzner, Peacock and Daniels describing an ambient pressure oxygen reservoir apparatus for use during one-lung anaesthesia (*Anaesthesia* 1999; **54:** 454–8). We believe that their technique with four clamps is unnecessarily complex. Our unit has been employing a similar technique for 5 years now. We use a C-breathing system with a 5-cm H_2O PEEP valve attached instead of an APL valve. We connect this to the nondependent lung having clamped off the double-lumen connector, and ventilate with 100% oxygen shortly before the pleura is opened. We then maintain $1\,l.min^{-1}$ of oxygen flow into the system, and allow the lung to deflate via the PEEP valve when the pleura is opened. We have had no incidences of arterial desaturation necessitating re-expansion of the nonventilated lung. We also question the authors rationale for clamping off the double-lumen connector at end-expiration to initiate one-lung anaesthesia. We believe this encourages early atelectasis.

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A reply

The letter of Drs Latter and Mulvey touches upon several interesting issues. Yes, the four clamps do look cumbersome, but not so in use [1]. Clamp 1 initiates and maintains single lung ventilation, as is standard practice. Clamp 2 enables the double-lumen tube on the side of the nonventilated lung to be occluded for the short time it takes to connect the ambient pressure oxygen reservoir. Clamp 3 serves to contain the oxygen in the reservoir until such time as it is connected to the nonventilated lung. Clamp 4 can be omitted, if desired, as described in a separate correspondence [2].

The use of Clamp 2 is a simple, practical method of excluding nitrogen from the nonventilated lung, which is after all the object of the exercise [1]. If the airway of the nonventilated lung is momentarily left open to air as the reservoir is being connected, it is possible for more than 250 ml of ambient air to enter the nonventilated lung in the course of a single ventilation to the dependent, ventilated lung [3].

Drs Latter and Mulvey report that, once single-lung ventilation to the dependent lung is initiated, they separately ventilate the nondependent lung with 100% oxygen presumably by hand 'shortly before the pleura is opened'. This practice will progressively wash out any nitrogen that may have entered the nondependent lung at the time their C-breathing system was connected. With nitrogen successfully excluded or eliminated, I suggest they try connecting an oxygen reservoir at ambient pressure rather than oxygen CPAP. The former practice offers several additional practical advantages [1], while the latter is contraindicated or counterproductive in many thoracoscopic procedures.

I apply Clamp 1 at end-expiration to minimise the increase in inflation pressure in the ventilated lung over the short period before the nonventilated lung is opened to the oxygen reservoir. Whilst this is not of any importance in most cases, it may well be of relevance in patients with respiratory disease and high levels of intrinsic PEEP. Of greater importance, however, is the need to ensure that Clamp 2 is not left inadvertently applied for longer than is necessary [1].

As regards the apparent concern about 'early atelectasis', I hold the view that prompt passive and then absorptive collapse of the nonventilated lung does not prejudice patient wellbeing either intraoperatively or postoperatively. It does, on the other hand, serve to improve surgical access and operative conditions in many thoracic operations, especially those performed thoracoscopically.

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Death in the dental chair

I read with interest the recent views for [1] and against [2] the subject of providing in-chair dental anaesthesia in facilities outside hospitals and community dental services. At first glance, one is inclined to accept the validity of various opinions and suggestions made by all concerned. However, what we should be aiming for is access to professional Anxiety Dental Management Clinics which are capable of providing a step system from reassurance and local anaesthesia through relative analgesia sedation to general anaesthesia of the highest quality to those patients whose clinical need dictates its use. Such facilities are not readily available in the majority of hospitals and community dental clinics. Hence, we should try to provide similar facilities in those dental practices that are currently complying with measures recommended in the Poswillo Report [3], the new guidelines laid down by The General Dental Council in November 1998 [4] and The Society for the Advancement of Anaesthesia in Dentistry (SAAD) in July 1998.

We as anaesthetists and dentists equally have the ethical and moral obligations to avoid the use of general anaesthesia in

dentistry whenever possible and to ensure that general anaesthesia will be provided only when all other measures have failed and when there are positive clinical criteria for the use of general anaesthesia rather than solely as a result of preference, economic considerations or time factors.

I have been involved in providing paediatric and adult in-chair dental sedation and anaesthesia for nearly 4 years in a dental practice that is situated in a small town close by a district general hospital in the North-east of England. Over the past 2 years, we as a group of anaesthetists and dentists made a combined effort to transform this unit into a state-of-the-art Anxiety Management Clinic where the providers have gone further than they are required in providing quality dental services by imposing a strenuous effort on all occasions to avoid the use of general anaesthesia and in achieving its prime objective of substituting alternatives to general anaesthesia. As a result, the rate of general anaesthesia to adults has sharply fallen to a mere trickle, if any. Equally, the rate of paediatric general anaesthesia has been dramatically reduced. For those who require general anaesthesia, propofol, sevoflurane and use of laryngeal mask airways and closed circle system has been in use since late 1998.

The report on the annual inspection of our unit concluded 'The facility puts many hospital operating theatres to shame. This is far and away the best facility I have seen. The service surpasses that in any community clinic, hospital or dental hospital that I have seen'. I should like to think that there may be similar facilities currently operating in the UK and would encourage Dr Cartwright and others who share his views to visit our unit; they may be convinced that under the present circumstances this approach may be the safest and only way forward.

Finally, in order to regulate the current standard of in-chair dental anaesthesia practice and to ensure the compliance of the practising dentists and anaesthetists with the present recommendations, I suggest that The Royal College of Anaesthetists Dental Committee and The General Dental Council should combine their joint efforts with the various Dental and Medical Defence Societies in imposing restrictions or refusal to legally cover any practising member who fails to comply with current regulations and recommendations whilst engaged in providing dental services.

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Target-controlled infusions

We read with interest the problems encountered by Drs Jenkins and Lawton with a patient having target-controlled infusions (TCI) during right pneumonectomy (*Anaesthesia* 1999; **54:** 610–11). We fully agree with the authors that TCI clearly relies upon successful drug delivery and are puzzled then why they chose a dependent limb for both TCI and intravenous infusion (IVI).

It is not uncommon to insert a central venous line prior to major thoracic surgery to provide a route for rapid transfusion [1]. A central venous line could be placed in the right internal jugular vein for right pneumonectomy so that any potential complications of insertion such as pneumothorax or haemothorax would be revealed at surgery and the dependent ventilated lung would not be adversely affected. Both the TCI and IVI could be successfully used if either a triple-lumen or two single-lumen central venous lines would be inserted. Moreover, the central venous pressure could be measured, if necessary.

The authors used a 20G cannula for TCI and a 14G cannula for IVI on the same dependent arm, which might have been compressed by the patient's body weight. Both the cannulae were in the superficial venous system [2] and the bag of infusion was empty. We presume that blood and propofol tracked up the bag because of a higher pressure created in the dependent upper arm veins due to compression.

We also wonder how long the delay could be to restart the infusions, because the authors first recognised the problem and then arranged a camera to take the photograph, which they produced as Figure 1. The blood levels of remifentanil and propofol may drop below the therapeutic levels in such situations, causing awareness during operation.

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In defence of target-controlled infusion

Dr Walder's letter (*Anaesthesia* 1999; **54:** 818–19) berating target-controlled infusion (TCI) verges on the polemic and we feel correction and clarification is needed. In no way does TCI claim to be 'achieving an ideal anaesthetic regimen' nor can it be considered to be a closed loop delivery system. The anaesthetist retains clinical responsibility and control throughout the conduct of a TCI case. It is folly to believe otherwise, a fault of the user not the equipment. We agree that a calculated target concentration is in essence 'virtual', based on programs derived from pharmacokinetic

data sets. It serves as a *guide* during conduct of anaesthesia. We also agree that there are wide variations in individual pharmacodynamic and pharmacokinetic activity. These criticisms apply equally to all other techniques including volatile-based anaesthesia, which Dr Walder appears to champion.

We learn through training and experience that anaesthesia must be individually tailored to the patient, their problems and their procedure. Therein lies the skill in delivery of quality anaesthesia of whatever nature. Techniques based on propofol infusion, be they manual or target controlled, allow us to administer alternative forms of general anaesthesia that may be superior to orthodox volatile-based regimens for many patients. TCI is a drug delivery system for propofol; it is not perfect but it is progress.

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Simple remifentanil infusions

We present an easy dilution technique to simplify the administration of remifentanil by infusion to supplement anaesthesia during cardiac surgery. Remifentanil is available as 1-mg, 2-mg and 5-mg vials for reconstitution for intravenous administration. Dilutions to concentrations of 20–250 $\mu g.ml^{-1}$ are recommended [1]. To aid this, the summary of product characteristics contains four tables to clarify administration rates for given concentrations as titrated to weight. Although of note, none of these tables includes weights less than 30 kg. We believe the below calculation is simpler and results in a more responsive and titratable infusion.

We use the algorithm of

3333/body weight in kg = ml of dilutant in to which to place 2 mg of remifentanil.

Thus 10 $ml.h^{-1} = 0.1\ \mu g.kg^{-1}.min^{-1}$. An example for a 68-kg patient is thus

3333/68 = 49.0 ml of dilutant in which to place 2 mg of remifentanil.

The dose range that we use for supplementing anaesthesia is 0.05–0.2 $\mu g.kg^{-1}\ min^{-1}$ during cardiac anaesthesia. The above method of administration is preferred for a number of reasons:

The extra dilution of our technique allows for more exact administration of boluses with minimal loss of effective dose by dead space. This is inherent to maintaining cardiovascular stability in the pre-bypass period.

By using a higher infusion rate, it is more apparent whether the syringe driver is functioning correctly, as the volumes infused can be more easily observed.

Accurate drug delivery may be more likely as there is less reliance on the necessity of accurate infusions of fractions of a millilitre.

There is less wastage, as the entire contents of each vial are drawn up for administration.

We have used the above for the last 12 months and find in practice the standardisation of the delivery rate in this manner aids simplicity of understanding in clinical areas and is practically useful.

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Reference

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Iatrogenic postoperative peripheral neuropathy is more common than generally realised

Dr Henderson (*Anaesthesia* 1999; **54:** 919) cited a case of permanent foot drop following a gynaecological procedure under combined general and epidural anaesthesia, stating that this illustrated the dangers of performing a regional block after the induction of general anaesthesia. I think that the foot drop was much more likely to have been due to an iatrogenic peripheral neuropathy, the common peroneal being the most frequently affected major motor nerve in the lower limb [1]. Warner and colleagues [2] reported 55 cases of peripheral neuropathy (43 common peroneal, eight sciatic and four femoral neuropathies) in patients who had had surgery in the lithotomy position. In 45% of cases, the foot drop or leg weakness persisted for at least 1 year. Sciatic and femoral neuropathies due to compression during intra-abdominal surgery have also been reported [3]. The incidence of femoral neuropathy following abdominal hysterectomy may be as high as 11.6% [4]. These neuropathies are associated with the use of self-retaining retractors; in a prospective study the incidence of femoral neuropathy fell from 7.45% to 0.07% when no retractors were used during abdominal hysterectomy [5]. Neurological symptoms occurring after an epidural or spinal anaesthetic are not necessarily due to that epidural or spinal block

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Combined spinal–epidural techniques and the awake/asleep debate

I suspect that I am not alone in using different techniques of combined spinal–epidural depending on the circumstances. I am becoming increasingly uneasy, however, about methods that involve inserting a spinal needle first, administering the dose, and then inserting the Tuohy needle. The ongoing debate about the advisability of attempting central neural blockade in anaesthetised, paralysed patients assumes that the anaesthesia is general anaesthesia. However, the profound block produced by a spinal dose of local anaesthetic such as that used for Caesarean section or even late labour will render the patient, though awake, into an anaesthetised paralysed state within seconds of the injection.

Although the 'difficult back' of a distressed labouring woman seems to 'cry out' for a quick spinal to give immediate relief and make insertion of the Tuohy needle easier, we should be aware that we have obtunded that reaction which might protect the patient (and the anaesthetist) from subsequent nerve injury.

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Profound motor blockade with epidural ropivacaine

We read with interest the article by Buggy *et al.* (*Anaesthesia* 1999; **54:** 895–8) and should like to present a similar problem possibly related to ropivacaine. An 85-year-old woman was admitted to hospital to undergo an elective right total knee replacement. She had suffered with severe osteoathritis of both knees, right hip and lower lumbar spine for the past 19 years. At the time of operation she was wheelchair-bound, only able to walk a few steps with the aid of a stick. Other problems included hypertension and ischaemic heart disease. Previous surgery included a cataract operation and removal of a thyroid cyst. There was no history of anaesthetic complications. At pre-operative assessment she was considered to be ASA 3.

Induction of general anaesthesia was with fentanyl and propofol; a size 3 laryngeal mask airway was inserted. The patient was then placed in the left lateral position and a lumbar epidural inserted at the L_{2-3} level by a consultant anaesthetist. Loss of resistance to saline was used and an epidural catheter with a filter was inserted. An aspiration test was negative; a test dose of 5 ml of 0.5% bupivacaine was injected. There were no problems with the insertion of the epidural catheter and the patient had not received any thromboprophylaxis.

The intra-operative course was uneventful. Anaesthesia was maintained with oxygen, nitrous oxide and isoflurane. Epidural boluses were administered to a total of 20 ml bupivacaine 0.5%. A tourniquet was applied to the right leg at a pressure of 400 mmHg for 98 min. During the operation, the patient was positioned supine with her arms across her chest. There were side supports on the operating table and foam was applied bilaterally to the elbows.

She had an uneventful immediate recovery period and a continuous epidural infusion was started with ropivacaine 0.2% infused at 8–10 $ml.h^{-1}$ for 32 h postoperatively. During the epidural infusion, the patient had remained haemodynamically stable with a pain score of 0 (0–3 scale). Sedation score was 1–2 (0–3 scale) for the immediate 14 h postoperatively. The day after stopping the infusion (third postoperative day), mobilisation was attempted with the physiotherapist but the patient was noted to have some weakness in the left leg which caused her to fall. A more thorough examination revealed decreased sensation in the left leg corresponding to the L_{1-4} dermatomes. On further questioning, the patient admitted to having paraesthesia in these areas whilst the epidural had been infusing. There was also weakness (power 2/5) of left hip flexion and knee extension and mild weakness of left foot dorsiflexion (power 4/5). Plantar flexion was normal. The knee jerk was present and both plantars were downgoing. There were no neurological signs in the right leg. Perianal sensation and tone were tested and found to be normal. By the evening of the same day, the symptoms had started to improve.

On the fifth postoperative day, the urinary catheter was removed and the patient was able to pass urine normally. By the sixth postoperative day, full sensation and movement had returned to the left leg and it was decided that no further investigations were needed. She was discharged from hospital on the 11th day after surgery at which time she was able to walk with the aid of appliances and ascend stairs unaided.

We wonder if this patient's unilateral neurological symptoms could be attributed to the slow reversibility of ropivacaine on the nerve roots in the region of L_{1-4}?

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Buggy *et al.* (Anaesthesia 1999; **54:** 895–8) conclude that an interaction between bupivacaine and ropivacaine may have been responsible for the prolonged lower limb motor block that they saw in two patients who had received a spinal anaesthetic with bupivacaine for Caesarean section followed by patient-controlled epidural analgesia with ropivacaine. I feel that they have looked a little too closely at the pharmacodynamic aspects in trying to explain their observations and not considered the more practical clinical aspects. Differential nerve block and frequency-dependent block are both phenomena that can only be acutely observed in specialised laboratory preparations. They may well have clinical implications, but clinical explanations must be eliminated before they are implicated in explanations of clinical observations. A number of clinical explanations are missing from the paper:

1 Bupivacaine is an agent that may, in an individual patient, produce an extremely long duration of effect. The epidural ropivacaine might just have been quite irrelevant.

2 The epidural catheters in both patients were placed at the lumbar level, but the

wounds were in the lower thoracic dermatones. The delivery of small boluses of local anaesthetic to the epidural sections of the lumbar nerves would produce a profound effect on them, particularly if their intrathecal courses were already affected by bupivacaine. The two effects might simply be additive, with no need for theories about interactions. Both patients also had poor quality pain control because the small bolus doses of local anaesthetic that they self-administered would not spread physically from the lumbar region to affect the lower thoracic nerves to a degree sufficient to produce pain relief.

3 The final factor that may be of some relevance is the well-known greater sensitivity of nerves to local anaesthetic block in pregnant patients at term. This might well explain how a concentration of ropivacaine not particularly associated with a great degree of motor blockade would have an exaggerated effect in these patients.

Any or all of these factors might have been relevant, although I suspect the most important one is the position of the epidural catheter. Epidural catheters must be placed at a level appropriate to the surgical wound if they are to be used effectively and safely for postoperative pain relief.

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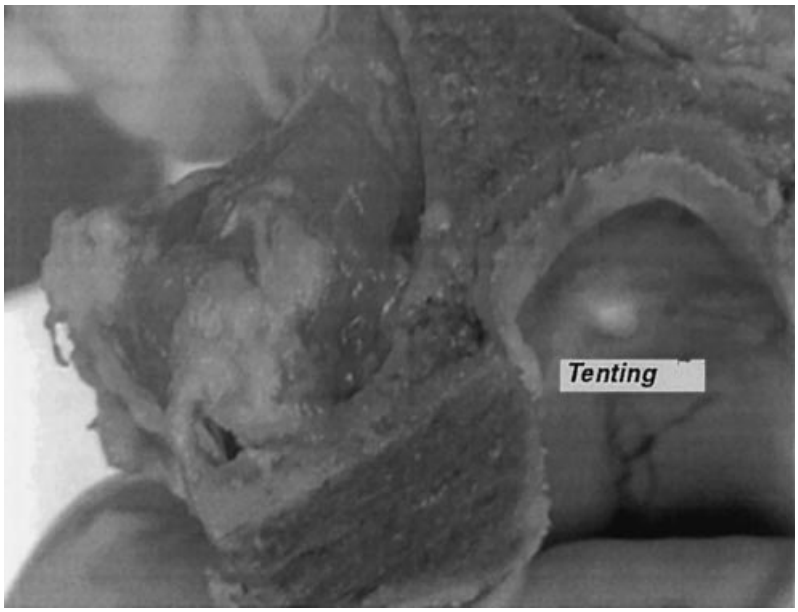

Figure 1 Tenting of the ligamentum flavum by an epidural catheter seen in a dissected pig cadaver.

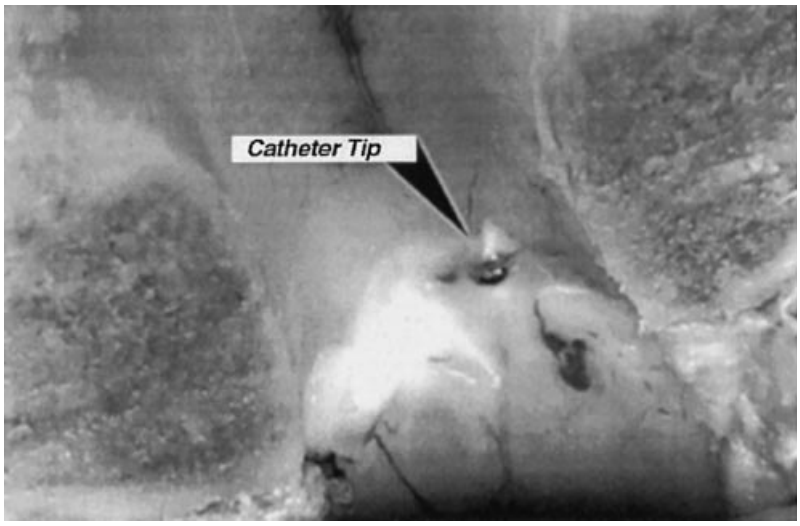

Figure 2 Penetration of the ligamentum flavum in the same specimen as Fig. 1.

A new method of identifying the epidural space

I should like to describe what I believe to be a novel method of identifying the epidural space. The most commonly used techniques for identification are loss of resistance to either air or saline. Both techniques require equipment, usually a purpose-built low-friction syringe, and the introduction of a fluid into the epidural space. The injection of air into the epidural space has been suggested to be associated with both inadequate analgesia and life-threatening complications [1]. The new technique involves using loss of resistance to the passage of the catheter itself as the means of identifying, and subsequently catheterising, the epidural space.

A standard Tuohy needle is inserted into the interspinous ligament in the usual fashion. The trochar is removed, the feeding guide is placed in the hub of the needle and the catheter is passed through it down the needle. The catheter stops against the tissue at the bevel of the needle. The catheter is now grasped with the thumb and index fingers about 5–10 mm from the feeding guide, i.e. at approximately the 12-cm mark on the catheter. The needle is advanced in increments of 1–2 mm and, at each stop, an attempt is made to advance the catheter. The pressure applied is limited to the first sign of buckling of the catheter. When the catheter passes through the ligamentum flavum, a distinct loss of resistance is felt and the catheter can then be advanced into the epidural space.

Bench tests have shown that a 16G Portex epidural catheter is able to exert a pressure of up to 100 mmHg at the end of the needle before buckling when used in this fashion, while an 18G catheter can exert a pressure of up to 70 mmHg. Studies with pig dissections have confirmed that these pressures are sufficient to allow the catheter to pierce the ligamentum flavum that has been scored by the needle, but not the dura mater (Figs 1 and 2 on previous page). Microscope inspection of used catheters has revealed no damage to their tips.

I have successfully used this technique in over 400 obstetric and surgical patients without a single incident of accidental dural puncture. I am currently embarking on a study to determine the success and complication rates when the technique is used by anaesthetic trainees. I would welcome the comments of readers on this technique.

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Postanaesthetic shivering

Few anaesthetists have not witnessed patients suffering from postanaesthetic shivering. Piper *et al.* (*Anaesthesia* 1999; **54:** 695–9) are to be congratulated on their interesting study into the prevention of this common problem using clonidine or nefopam. However, we feel that a number of issues relating to their study are worth raising.

The authors quote from the literature an incidence of postanaesthetic shivering of 5–70%. Postanaesthetic shivering can be classified as being either thermoregulatory or nonthermoregulatory. Most thermoregulatory shivering has been shown to be secondary to core hypothermia [1]. Such shivering can be prevented by maintaining intra-operative normothermia. Indeed, in one study no shivering was observed in volunteers who underwent normothermic anaesthesia without surgery [2]. In current anaesthetic practice, patient warming during major surgery is routine. Hypothermia can be avoided after almost all types of surgery with appropriate use of effective fluid warming devices, circle breathing systems and warm air blowers.

Nonthermoregulatory shivering certainly does occur. Volatile anaesthetic agents and postoperative pain have both been implicated as contributory factors [1].

Piper *et al.* studied 60 patients undergoing abdominal or orthopaedic surgery. During the surgery, neither the patients nor their intravenous fluids were warmed in any way. Patient temperatures were recorded but the results were not included in the paper. For analgesia, the patients were given fentanyl $3\,\mu g.kg^{-1}$ at induction of anaesthesia and then piritramide was titrated to pain levels in recovery. With such a study design it is inevitable that patients undergoing abdominal surgery will wake up hypothermic and in significant pain. The degree of hypertension recorded in the recovery room in both the placebo and nefopam groups (mean MAP > 105 mmHg) would seem to validate this conclusion. Given such conditions, it is not surprising that 60% of the control patients (12/20) woke up shivering. This study looks at the prevention of shivering in hypothermic patients in pain and therefore cannot be extrapolated to apply to nonthermogenic shivering in well-analgesed patients. It is this patient group in whom drug treatment of postanaesthetic shivering might be considered most appropriate.

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A reply

We were pleased that Wasson *et al.* were so interested in our study as to find it worthy of comment. However, their assertion that our patients were inadequately warmed and without proper analgesia and thus shivering was more frequent cannot go unchallenged.

Firstly, we did indeed not actively warm our patients. Wasson *et al.* find this unacceptable for major surgery and thus question our protocol. However, our study was not undertaken on patients undergoing major surgery (unless one considers laparoscopic cholecystectomy, hernia operations or ankle osteosynthesis as major operations). We apologise for not explicitly stating the types of operations in our paper, but operations lasting on average about 90 min are surely not major. For such operations, we do not have the equipment to warm patients actively as part of routine practice, so there was no difference between study and routine in this respect. The lowest intra-operative temperatures, which were not stated in our paper, were: $35.4 \pm 0.5\,^{\circ}C$ in the nefopam, $35.2 \pm 0.5\,^{\circ}C$ in the clonidine and $35.3 \pm 0.6\,^{\circ}C$ in the placebo group. Temperatures 15 min after extubation (i.e. on arrival in the recovery room) were slightly higher than lowest intra-operative temperatures.

The second concern of Wasson *et al.* is inadequacy of our analgesia regimen (fentanyl $3\,\mu g.kg^{-1}$ given intravenously prior to induction of anaesthesia) with 60% nitrous oxide and about 0.4% to 1.6% end-tidal isoflurane intra-operatively, followed by piritramide on demand in recovery. This concern probably also stems from the assumption of the subjects undergoing major operations, but, as already explained, only patients undergoing minor operations were studied. In any case, the patients received as much piritramide in recovery as needed. However, only 40% of the patients in the nefopam group and 30% of the patients in the clonidine and placebo groups needed any piritramide, the highest dose being 22.5 mg. Consequently, it seems reasonable to assume that our fentanyl

dosage was adequate in spite of slightly elevated mean arterial pressures after extubation, which were well tolerated in our ASA 1 and 2 patients.

Finally, Wasson *et al.* consider our incidence of shivering in the placebo group to be excessive as a result of the above discussed hypothermia and pain. It should be noted that although 60% of the patients in the placebo group had a shivering score of 1 or higher, this included three patients with a shivering severity of 1 (piloerection or vasoconstriction without visible muscle activity). Without these patients, who would not have been considered as shivering using less stringent criteria, the incidence would have been 45% (well within the range reported in the literature).

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Hyperchloraemia causes metabolic acidosis by reducing strong ion difference

Dunn *et al.* (*Anaesthesia* 1999; **54:** 566–8) describe the management of a patient with hypokalaemic, hyperchloraemic metabolic acidosis requiring ventilation. As the title of the case report suggests, hyperchloraemia was the cause of the metabolic acidosis. Why, then, did the patient receive so much chloride during the course of his treatment? He received 4 l of normal saline containing 616 mmol of chloride in the first 12 h. In addition, he received 1860 mmol of potassium presumably as potassium chloride in 42 h. The patient was hyperchloraemic even at the time of discharge. The brisk diuresis following suprapubic catheterisation should have corrected the hyperchloraemia effectively but failed to do so in this case presumably because of continued administration of chloride.

Currently there appears to be a lack of appreciation of the primary role of the strong electrolytes like sodium and chloride in acid–base homeostasis. The analysis and interpretation of acid–base using the Henderson–Hasselbalch equation is frequently misleading because it gives the wrong impression that hydrogen ion concentration is controlled by the partial pressure of carbon dioxide and the bicarbonate concentration. This is a valid mathematical equation based on the law of mass action but no physiological cause and effect between hydrogen and bicarbonate can be deduced from it. To understand the cause of metabolic acid–base disturbances, the main players or the independent variables, namely strong electrolytes and total albumin, must be brought into the picture. Both the hydrogen ion and bicarbonate are dependent variables and they merely respond to changes in independent variables. The chemical laws of mass conservation, mass effect and electrical neutrality are the controlling forces of the interaction between the independent and the dependent variables. This comprehensive and new approach to acid–base has been described by Stewart [1].

Based on Stewart's concept, the primary cause of the acidosis in this case report is reduced strong ion difference as a result of increased chloride (an independent variable). The reduced strong ion difference forced secondary changes on hydrogen ion and bicarbonate (both dependent variables) through the application of the above mentioned chemical laws.

The composition of the resuscitation fluid must be taken into consideration in order to prevent iatrogenic acid–base disturbances. Ringer-Lactate would have been better than normal saline because of the lower chloride content. Potassium may be given in the form of its acetate instead of chloride. To treat the hyperchloraemia more aggressively, the rehydration fluid may be given as isomolar sodium bicarbonate or sodium lactate. Lactate, being negatively charged like chloride, has the potential to cause acidosis but does not [2], presumably because of its removal by the liver.

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A reply

We wish to thank Dr Dorje and colleagues for their letter in which they brought to our attention Dr P. A. Stewart's interesting paper. We are not aware of the existence of potassium acetate, as it does not appear in the British National Formulary. Our Pharmacy Department has ascertained that it can be obtained in the UK with difficulty.

We reported the case to illustrate how quickly a patient with this type of bladder reconstruction can get into trouble in the absence of an adequate bladder voiding mechanism.

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Self reports of postoperative cognitive dysfunction

I read with interest the paper by Rödig and colleagues (*Anaesthesia* 1999; **54:** 826–30) evaluating self reports of cognitive dysfunction before and 2 months after cardiac or major vascular surgery. The similar increases in reported postoperative 'cognitive failures' in the two surgical groups led the authors to conclude that factors other than cardiopulmonary bypass affect self-assessed postoperative cognitive function.

The apparent lack of a significant correlation between self reports of postoperative cognitive dysfunction and formal neuropsychological testing is a curious observation that seems to be true for both cardiac [1] and noncardiac surgical patients [2]. Newman *et al.* found that patients reporting greater levels of cognitive dysfunction after coronary

artery bypass surgery tended to have higher levels of depression and state anxiety, suggesting that self reports of cognitive dysfunction are 'mood sensitive' [1]. Could postoperative mood and/or anxiety have contributed to the findings reported by Rödig *et al*.? The lack of postoperative measures of mood and state anxiety in their study makes it difficult to draw any conclusions.

Objective measures of cognitive function following cardiac surgery, however, do not appear to be 'mood sensitive' [3]. Furthermore, the long held belief that depression is a common sequel of cardiac surgery also appears to be incorrect. At 6–9%, the incidence of new onset depression after cardiac surgery is remarkably low [3, 4].

In the final analysis, which is the greater success – the patient with no measurable impairment who complains of cognitive impairment or the patient with few complaints who is blissfully unaware of his impaired cognition?

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A reply

Thank you for giving me the opportunity to reply to the comment on our article 'Evaluation of self-reported failures in cognitive function after cardiac and noncardiac surgery' (*Anaesthesia* 1999, **54:** 826–30). The objective of our study was to evaluate the subjective perception of the patients with regard to their cognitive abilities, both after cardiac and after noncardiac surgery. The discrepancy between the patients' self-reported failures and the cognitive dysfunction assessed by neuropsychological testing is a well-known phenomenon. Our study, however, was not designed to further elucidate this issue. Actually, we assessed state anxiety and mood before hospital discharge, data that were not part of this study. These results (the mean (SD) state anxiety score in the coronary artery group was 41 (12) compared to 40 (10) in the vascular surgery group, the mood score was 18.5 (8) and 15 (9), respectively) suggest that there were no differences in either state anxiety or mood scoring between both groups early after surgery. We did not assess state anxiety and mood 2 months postoperatively as we had decided to send only a second Cognitive Failures Questionnaire, along with some minor questions, to avoid putting too much pressure on the patients' compliance, and to avoid nonresponders wherever possible. Finally, we believe that both subjective complaints and measurable deficits may affect the patients' quality of life.

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Hemispheric-synchronisation for nociception control

I read with interest the paper by Kliempt *et al*. (*Anaesthesia* 1999; **54:** 769–73) describing the use of hemispheric-synchronisation for nociception control. The authors claim to demonstrate that patients who were played hemispheric-synchronisation tapes through headphones whilst undergoing surgery under general anaesthesia had greater intra-operatve nociception control than patients who were played either classical music tapes or blank tapes. However, several points in the paper need to be clarified:

1 One of the patient exclusion criteria was knowledge of hemispheric synchronisation. However, for informed consent to have been obtained they would have been given information about it.

2 Patients were given intravenous fentanyl if intra-operative heart rate or arterial blood pressure were 20% above pre-operative baseline values for more than 5 min. Fentanyl requirement then served as a surrogate marker for adequacy of nociception control. The authors do not describe the fentanyl-dosing protocol. If a standard protocol was not used then this is a potential source of bias.

3 The subjects underwent a range of operations, e.g. lump removal, haemorrhoidectomy, vaginal hysterectomy. There are obviously differences in the potential magnitude of surgical stimulation and hence nociception. However, there are no data regarding the distribution of the patients in the hemispheric-synchronisation group and the two control groups in terms of the surgery received. Nonuniform distribution is another potential source of bias. Instead, the results are broken down and analysed using the estimated length of stay – less than and greater than 2 days, this estimate being determined by the surgeon pre-operatively. The number of surgeons who participated in the trial is not stated. Nor are we told if the estimated length of stay proved to be correct.

4 The study is supposedly double-blind; however, in their discussion, the authors cite evidence that patients may in fact continue to hear under general anaesthesia.

5 The information that would be most interesting is the patients' pain scores postoperatively in the recovery unit. Pain and nociception are not synonymous. When considering these two phenomena, the overall aim of the anaesthetist is to reduce pain. Until these points are clarified and the possibility of bias ruled out I do not think larger studies should be undertaken as the authors suggest.

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A reply

We read with interest the letter of Dr K. Ahmad and we would like to reply to the points raised in his letter as follows:

1 In order to obtain informed consent, the patient was explained the nature of Hemi-Sync sounds, how they work and how they might influence him/her intra-operatively. The statement in our publication that patients were excluded from the trial if they 'knew the Monroe Institute or knew about Hemi-Sync' refers to the fact that they were excluded if they had prehospital knowledge about the Monroe Institute, Hemi-Sync or both. This measure was taken to prevent any bias in favour of Hemi-Sync because it could not be ruled out for sure that such patients listened to Hemi-Sync sounds in the past. Hemi-Sync sounds can induce altered states of consciousness [1], and have a learning effect according to the Monroe Institute. Through the learning effect of Hemi-Sync, a person can over time acquire the skill to enter altered states of consciousness without actually listening to Hemi-Sync sounds, in the same way as a person learning a foreign language with the help of audiotapes has no more need for such as soon as he/she becomes proficient in the foreign language.

2 If a patient's heart rate, systolic blood pressure or both exceeded the pre-operatively measured values by about 20% for more than 5 min, fentanyl was given intravenously according to a standard protocol. The protocol required fentanyl to be given in increments of 25 μg in periods of at least 5 min, in order to allow its effects on the heart rate and/or the blood pressure to be assessed until pre-operative recorded values were reached again.

3 The distribution of the patients into two subgroups of estimated postoperative stay of up to 2 days and of more than 2 days was done to prevent nonuniform distribution for later statistical analysis. Otherwise, the nociception control required by patients undergoing, for instance, major orthopaedic surgery would have been compared with the nociception control required by patients undergoing minor gynaecological procedures. Table 1 above shows the number of operations in these two groups and a subgroup analysis.

Table 1 Number of operations in the two subgroups

Operations	Blank	Classic	Hemi-Sync
Postoperative stay up to 2 days	15	13	16
Postoperative stay less than 2 days	11	12	9
Total	26	25	25

When doing a Kruskal Wallis test for the operations requiring a postoperative stay of up to 2 days, the Hemi-Sync group still required significantly less fentanyl than the blank and classic groups (Chi-squared = 21.06, $p < 0.001$). The same held true for the operations requiring a postoperative stay of more than 2 days, the Hemi-Sync group requiring significantly less fentanyl (Kruskal Wallis, Chi-squared = 13.61, $p < 0.001$).

It is difficult for us to understand why Dr Ahmad thinks that the number of surgeons performing the operation included in the study would be of any importance to the outcome. Since our publication states that the operations consisted of general surgical, orthopaedic and gynaecological procedures, it is self-evident that a number of surgeons were involved. In a *Medline* search (1960 to present), there is neither a publication to be found which claims that the intra-operative nociception caused by the incision performed by one surgeon is greater or smaller than the one performed by another surgeon, nor is there any publication retrievable claiming that the number of surgeons ever had any influence on intra-operative nociception in any trial.

The estimated postoperative length of stay was solely used in order to distribute the patients for the intra-operative period into the appropriate nociception subgroup. Since the Hemi-Sync trial focused only on the possible intra-operative nociception control of the tapes used, a verification of the correctness of the estimated postoperative stay would have been of no consequence to the results, and was therefore not performed. The estimated postoperative stay can be influenced by a wide variety of well-known postoperative complications such as wound infections, pneumonia, pulmonary embolism, myocardial infarction, etc., none of which is of any consequence to the results of the intra-operative nociception control.

4 The theory of the double-blind approach in clinical trials is to prevent knowledge of treatment conditions from influencing the outcomes of the trial and its assessment [2]. The possibility to gain knowledge of treatment requires a conscious perception of the treatment on both sides, that of the patient and that of the doctor. For the patients' part, this is not possible in general anaesthesia where the patient is asleep to such a degree that he/she can undergo surgery. In our paper, we described the procedures employed in the study to ensure that the anaesthetist was blind to treatment condition. The Hemi-Sync trial was therefore fully double-blind or perhaps more appropriately double-deaf. That, according to the results of our study and other studies cited in our publication, patients under general anaesthesia might nevertheless be able to hear, does not change this fact. The patient under general anaesthesia hears on an unconscious level. A similar phenomenon takes place when people deliberately play subliminal tapes of various contents during their sleep [3–5].

5 The *Surgical Support Series* tapes of the Monroe Institute consist of six audiotapes with Hemi-Sync sounds, three of which are dedicated to surgical procedures (PreOp, IntraOp and PostOp tape). Because of manpower and resource constraints, we were unable to utilise all three tapes in a trial so that it was decided to undertake a pilot study testing the IntraOp tape only. Since our trial, utilising only the intra-operative tape, yielded positive results, it can be expected that the utilisation of all three tapes will also yield positive results. Reports of individual American doctors using Hemi-Sync sounds for their patients suggest this. However, this has to be proven in a

proper study, and we therefore suggest that larger trials using Hemi-Sync sounds should now be undertaken.

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Carbon dioxide narcosis caused by midazolam in a patient with myotonic dystrophy

We should like to report a case of hypercapnia caused by administration of small doses of midazolam in a patient with myotonic dystrophy. A 44-year-old female weighing 50 kg who developed myotonic dystrophy in her childhood was scheduled for abdominal total hysterectomy. Her pulmonary function testing showed a low vital capacity and normal forced expiratory ventilation in 1 s (32.3% and 90.4% of the predicted normal values, respectively). Arterial blood gas analysis showed severe hypoxia and normocapnia with pH of 7.35, P_aO_2 of 59.2 mmHg, and P_aCO_2 of 39.6 mmHg whilst breathing room air.

An epidural catheter was inserted via the L_2–L_3 interspace. After a test dose of 2 ml, followed by 12 ml of lignocaine 2%, anaesthesia was maintained with epidural administration of 5 ml of lignocaine 2% about every 40 min during surgery. Oxygen 5 l.min^{-1} was given via a facemask. After confirming the anaesthetic level, which stretched from T_7 to S_4, the operation was commenced. The patient became anxious during the surgery, and midazolam 0.25 mg was administered intravenously on two occasions.

At the end of surgery, the patient was calm but unrousable. Her spontaneous respiration was maintained at an adequate rate, but her tidal volume was considered to be decreased based on the movement of the chest wall. The end-tidal carbon dioxide level increased to 70 mmHg, and the arterial blood gas analysis showed a P_aCO_2 of 99.2 mmHg and a P_aO_2 of 92 mmHg. Carbon dioxide narcosis caused by midazolam was diagnosed and flumazenil 0.2 mg was given. After 5 min, the end-tidal carbon dioxide had decreased to 45 mmHg, and the patient woke up. Immediately, she became alert and her vital signs were stable in the recovery room. Her postoperative course was uneventful and she was discharged 10 days after the operation.

According to several reports [1, 2], regional anaesthesia can be safely used in patients with myotonic dystrophy. However, there is a controversy about sedation during regional anaesthesia in myotonic dystrophy. Ravin *et al*. [3] reported that there is increased sensitivity of the central nervous system to the effects of opioids and sedative drugs in myotonic dystrophy. Others have reported that mental stress can affect the blood pressure in this disease [4]. We considered that minimal sedation with small doses of midazolam was required in this case. In addition to the effect of midazolam, oxygen administration could have a harmful effect on the patient with hypoxic restrictive lung disease. Also, muscle relaxation caused by the epidural could also influence respiration. It is possible that what the patient felt as anxiety during surgery might in fact have been a symptom of respiratory depression as a result of these factors. Careful attention is required in giving sedative drugs, even small doses, in such cases.

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Anaesthesia for MRI angiography in a patient with Williams syndrome

We should like to report the anaesthetic management of a 21-year-old male with confirmed diagnosis of Williams–Beuren syndrome undergoing magnetic resonance imaging (MRI) angiography. This procedure required three prolonged breath-holding sequences. Williams syndrome (1 in 20–50 000 live births) is due to sporadic elastin gene mutation or deletion. It results in supravalvular stenosis of the aorta (SVAS) and other blood vessels and various other features such as: facial dysmorphism (elfin facies), mental retardation associated with a friendly loquacious personality, growth impairment and disturbed calcium homeostasis.

The anaesthetic technique chosen had to provide a stress-free haemodynamic state since the condition of the coronary

vessels was unknown, and many cardiovascular abnormalities can occur in Williams syndrome, including: coronary tree involvement (including myocardial infarction in a 3-year-old child with no associated SVAS) [1], blood pressure discrepancies between left and right arms, and a 40% incidence of hypertension in a population with a median age of 6.5 years [2].

The scan was being undertaken to investigate the multiple murmurs detected on physical examination. There were murmurs over his aorta, carotid arteries, posterior thorax and abdomen. These were considered to represent possible aortic, carotid, pulmonary or renal stenoses, all of which may occur in Williams syndrome [3]. Previous echocardiography had demonstrated a small proximal aorta but there was no definite evidence of stenosis. A poor echo window and the patient's restlessness made a complete scan impossible and an MRI was therefore organised, with an anaesthetic requested due to the prolonged duration of the procedure and the patient's mental state.

Anaesthetic reports are scarce; one report recommended the use of a transoesophageal echo (TEE) for a child with proven supravalvular aortic stenosis undergoing aortoplasty, who subsequently required coronary revascularisation [4]. Gene linkage studies following a case of masseter spasm [5] have concluded that the malignant hyperpyrexia locus is outside the deleted elastin region [6].

Our patient was of small stature and weighed only 39 kg. He had limited exercise tolerance but was able to walk unaided at a moderate pace, although he tended to be peripherally cyanosed at times. On examination, he had a resting tachycardia of 120 beat.min^{-1} and small pulse volumes in both arms with auscultatory blood pressure recordings impossible to measure. By palpation, the systolic blood pressure in the right arm was 70–80 mmHg and that in the left arm 80–85 mmHg. He was not taking any medication. His ECG had voltage criteria for left ventricular hypertrophy and chest X-ray was normal.

Temazepam 10 mg and ranitidine 150 mg were given as a premedicant and amoxicillin was given as prophylaxis against endocarditis. Induction consisted of remifentanil 1 μg.kg^{-1}, etomidate 12 mg and atracurium 25 mg. The patient was intubated and ventilated and anaesthesia maintained using sevoflurane in nitrous oxide and oxygen. The patient's blood pressure remained at approximately 90/45 throughout (measured on the right arm) with a pulse rate of 80 beat.min^{-1}. Further 0.5 μg.kg^{-1} boluses of remifentanil were administered on three occasions for patient repositioning, together with boluses of atracurium. Maglife C monitoring (Bruker, France) included ECG, pulse oximetry, gas analysis, and non-invasive blood pressure measurement. The patient could not be allowed to breathe spontaneously for the scan, since three breath-holding sequences of up to 70 s were required, and the procedure was envisaged to take about 2 h.

The high magnetic fields in the MRI suite constitute a suboptimal environment for a high-risk case such as this: ST segment analysis of the ECG is not possible owing to the graphite electrodes being placed very close together to avoid current induction. Neuromuscular blockade cannot be monitored since all peripheral nerve stimulators contain ferrous components. Invasive monitoring such as TEE was also impractical for the same reason. A remifentanil infusion could have been used with a long infusion catheter and a pump outside the room, but we found that by giving boluses prior to each repositioning of the patient, haemodynamic stability was maintained. Following a 2-h scan, muscle relaxation was reversed with neostigmine and glycopyrronium and the patient's trachea was extubated once he was awake.

MRI angiography showed an abnormal arborising pattern of the pulmonary arterial tree due to stenoses of the origins of both upper lobe pulmonary arteries and stenoses of several segmental arteries arising from the lower lobe arteries. The descending aorta was small in calibre but there was no evidence of supravalvular aortic stenosis. Carotid and vertebral arteries were normal. There was an absent left renal artery and kidney. The condition of this patient's coronary arteries remains unknown.

This case highlights the administration of a haemodynamically stable anaesthetic in an MRI suite to a patient with many potential problems.

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Latex allergy – further comment

We read with interest the article by Dakin and Yentis (*Anaesthesia* 1998; **53:** 774–81) concerning the management of latex allergy. Since the publication of this article we have been working on a protocol for the management of latex allergy. This problem was further highlighted recently when we anaesthetised a

patient, herself a healthcare worker, suffering from a severe latex allergy for two laparotomies over a period of 10 days. From an anaesthetic point of view, we found the advice contained in the article to be very helpful. Anaesthesia and surgery were conducted without incident. However, the case highlighted potential problems on the ward that could have serious implications for a patient with latex allergy:

1 There was a lack of awareness of latex allergy amongst general medical and nursing staff and other ancillary workers, e.g. phlebotomist not wearing latex-free gloves.

2 The lack of latex-free resuscitation equipment on the ward.

3 The requirement for frequent handwashing to ensure no trace of latex on hands prior to contact with the patient.

4 The need for more information regarding CVP lines, surgical appliances and catheters, i.e. suitability for use in latex allergy.

5 Large notices were required and staff needed constant reminding during the postoperative course.

Whilst we appreciate that it is impossible to cover every aspect in an article, it is important to highlight the problem of caring for these patients outside the operating theatre. Further education is needed to increase awareness and understanding of an ever-increasing problem. Happily our patient suffered no serious consequences.

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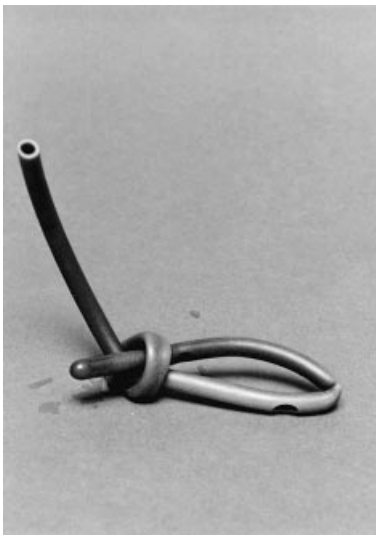
Knotting of a nasogastric tube

We report a complication encountered during attempted removal of a nasogastric tube. A 33-year-old woman had a lower segment Caesarean section for placenta praevia under general anaesthesia. The immediate postoperative period was complicated by postpartum haemorrhage, which required a 6-unit blood transfusion. The next day, she complained of abdominal distension. An abdominal ultrasound scan was normal. At this stage, a nasogastric tube was inserted for paralytic ileus or possible partial small bowel obstruction. A 12 FG/CH plastic nasogastric tube (RT 2021 Ryles Tube, Pennine Healthcare) was inserted uneventfully and free drainage was ensured. This remained *in situ* for 5 days until her abdominal distension disappeared.

It was not possible to remove the nasogastric tube without the patient complaining of severe retropharyngeal pain with traction on the tube. Consequently, she was taken to the operating theatre for examination of the nasopharynx under general anaesthesia. On laryngoscopy, a knotted nasogastric tube was found in the pharynx. The proximal end of the tube was cut and the knotted end removed via mouth. The unusual type of knot is shown below. At 3 weeks postpartum, the patient was readmitted and underwent laparotomy to relieve a small bowel obstruction.

Although nasogastric tube coiling and knot formation is a known complication, it is more commonly seen with small-diameter tubes or in patients with a small stomach (e.g. following gastroplasty) or when an excessive length of the tube is left in the stomach [1–5]. Unusual types of knot have previously been reported with traumatic complications [5, 6]. The possibility of a true knot in the nasogastric tube should be kept in mind if a stiff resistance is felt during its attempted removal.



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Training in fibreoptic intubation

Skilful use of a fibreoptic laryngoscope is a necessary skill in modern anaesthetic practice [1]. Unfortunately for many reasons the acquisition of this skill can be difficult. Training models offer the opportunity for trainees to learn how to set up, handle and obtain good control of an endoscope before ever coming in contact with patients, thereby enabling the safer and more efficient use of clinical training opportunities.

Training models range from anatomical figures to other devices designed purely to teach and develop manual dexterity [2–4]. Many of these are unrealistic or insufficiently challenging, leading to boredom and dissatisfaction amongst trainee endoscopists.

We have developed a training aid – The Artificial Throat – which can be

varied in difficulty, and which is also easy and cheap to construct. The Artificial Throat consists of a 16-cm segment of 40-mm plastic drainage pipe with a 60° angle connection at the proximal end, the distal end being left open. Plastic partitions inserted into the tube provide a course for the endoscopist to negotiate. Transverse cuts are made at intervals across the underside of the pipe to the halfway (widest) point to accommodate the plastic partitions. These are constructed from old X-ray films, cut into strips slightly smaller than the inside diameter of the pipe and doubled over. The two opposing surfaces are glued together, the edges rounded off at one end and a hole is bored through each sheet before it is inserted into the tube. The model is then secured to a mounting board by standard fittings (see figure above).

The plastic plates are arranged and rearranged to create a variable course for the endoscopist to follow. Holes may be of differing sizes, in various locations, the sheets may be reversed, or the order of the plates may be altered to make a model more difficult to negotiate. The plastic partition sheets are easily removable even when the pipe is fixed to the mounting board.

This model offers several useful features, it is cheap (£4 vs. £2000 for a training mannequin), easy to build and can be constructed by any hospital maintenance department. We have 10 different models, ranging from green through to black 'runs'; these have proved extremely useful and very popular training devices in our hospital.

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Opioid-induced breathing patterns disappear from view

Leino and co-workers (*Anaesthesia* 1999; **54:** 835–40) are correct in saying that little is known about changes in breathing pattern caused by opioids. There is some [1], and we would have appreciated being cited, but it is not really the authors' fault that they could not find our work. In *Medline* (Silver Platter^{TM}) there are MESH terms 'respiratory-control' and 'opioids', but our paper is filed only as 'alfentanil–pharmacology' and 'respiration–drug–effects'. Unless authors include the word 'opioid' in the title of their papers, research on individual drugs seems likely to sink without trace.

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Multiple choice examinations

I read with interest Dr McGuire's letter regarding MCQ examinations (*Anaesthesia* 1999; **54:** 720) where he suggested the abolition of negative marking. Having also sat many MCQ papers in the past, I cannot agree with the call to drop negative marking. Indeed, there is evidence that MCQs are the least-favoured assessment method among examination candidates [1] but it has been shown that negatively marked MCQ scores correlate well with success or failure in examinations [2].

What is more important with MCQ examinations is to ensure that they do not contain undefined, imprecise terms that only serve to confuse candidates. A recent survey of Final MB and Part 1 MRCP examinations revealed that imprecise terms occur commonly as do construction errors leading to a disproportionately large number of 'true' branches [3].

As with all examinations, it is technique as well as knowledge that is important, as it has been shown that candidates who know a subject well may still fail a negatively marked MCQ examination [4]. However, in my opinion, it is important to retain negative marking to discourage guessing in clinical practice and

to encourage an informed decision-making process.

It is therefore important to help examination candidates with both knowledge acquisition and examination technique. Losing negative marking would only serve to devalue the information gained about a candidate's knowledge and ability to make informed decisions.

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Side-effects of cardiac output measurement

The most common way to measure cardiac output is by thermodiluton [1] using a balloon-tipped pulmonary artery catheter (PAC). The catheter carries a fast response thermistor at its tip which when properly placed lies in the distal pulmonary artery. The injection port opens into the right atrium. An accurately predetermined volume of cold solution is injected through the catheter using a double-skinned (insulated) automatic filling syringe. This injection has to be made within 4 s to obtain the best curves for cardiac output measurement [2]. Thus, a volume of 10 ml would be injected at the rate of at least 3 ml.s^{-1}. This rapid injection through the narrow lumen of the PAC generates a large pressure on the thumb of the clinician.

I lost the sensation of touch and pain on the distal part of my right thumb while working as a registrar in a 12-bedded intensive care unit (ICU). The cause of this problem was not immediately apparent and I went through a series of referrals and tests. The orthopaedic consultant felt it was a localised injury and that it would recover in a few months time. I had meanwhile moved to a paediatric unit as part of my rotation and after about 4 months my sensations returned and I made a complete recovery. The following year, however, I was posted again to the ICU and the thumb after a few weeks became numb again. It was then I realised that the loss of sensation of my thumb was due to the localised pressure, created while performing cardiac output studies using a 10-ml syringe through a pulmonary artery catheter. On an average, I must have performed about 30 measurements per day.

Intrigued, I calculated the pressures required to empty the cardiac output syringe using the Hagen–Poiseuille equation [3]. According to this equation,

$$Q = P\pi r^4/8\eta L.$$

This can be rearranged to read

$$P = Q8\eta L/\pi r^4$$

where P is the pressure required to empty the cardiac output syringe and is expressed in Pa, Q is the flow per second in cubic metres (in this case 10 ml in less than 4 s, say 3.3 ml.s^{-1} or 0.33×10^{-5} m^3.s^{-1}), η is the viscosity of the cold injectate (the value used for this calculation is that of water and it is 1×10^{-3} mPa.s^{-1} at 20 °C), L is the length of the PAC from the injection port to the opening (1.1 m), r is the radius of the proximal injectate lumen (its value as obtained from the Baxter Healthcare Ltd is 0.03312 inches or 0.4191 mm).

Substituting the above values in the equation we get the value for P which is a surprising 299 kPa or 2242.50 mmHg.

This theoretical evaluation of pressure was further confirmed by an *in vitro* study. A pressure gauge designed to read high pressures was attached to the side arm of a three-way tap in continuity with the PAC. The syringe was emptied in the quickest possible time. The pressures were well above 260 kPa even when different volunteers performed the test. The pressure required to empty a cardiac output syringe through a PAC is more than 20 times the average mean arterial pressure. This could well explain the pressure effects on the thumb. I now use my palm to empty the syringe; the pressure is distributed over a larger area on the cushioned part of the hand and therefore there are no damaging effects.

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Harmless herbs: a cause for concern?

There is extensive use of the herbal medicine St John's Wort in many western countries. It is used to treat anxiety, anorexia and depression and is readily available over the counter in most High Street chemists. Its active ingredient, hypericin, causes irreversible inhibition of both monoamine oxidase (MAO) A and B with higher activity towards type B [1]. Therefore, hypericin (like MAOIs) may also have the potential to interact with anaesthetic drugs.

A 37-year-old female was scheduled for removal of silicone breast implants. She was fit and generally well with no significant past medical history. On direct enquiry, she denied any regular medication. She was noted to be anxious pre-operatively and so was offered premedication. However, she declined this and instead took one of her St Johns Wort tablets that she had found to ease her anxiety in the past. It transpired that she had been taking St Johns Wort

regularly and therefore her elective operation was postponed due to the potential risk of a serious reaction to opioids and sympathomimetics, as discussed below.

There are no reported cases of interactions between St John's Wort and anaesthetic drugs, but there have been cases of adverse reactions in patients undergoing anaesthesia while taking MAOIs. For example, a 21-year-old on an MAOI, developed hypotension and bradycardia while under spinal anaesthesia. This was treated with ephedrine and atropine but she subsequently developed agitation, dyspnoea, severe headache, hypertension and tachycardia [2]. Churchill-Davidson advised checking patients' sensitivity to MAOIs and opioids by giving a test dose of 5 mg of pethidine and monitoring vital signs regularly [3]. There are also reports of MAOIs causing an increased predisposition to the development of a malignant hyperthermia syndrome [4] and prolongation of suxamethonium block [5]. Thus, the recommendation is that MAOIs should be stopped 2 weeks prior to anaesthesia because of the potential for serious drug interactions [6].

From 1983 to 1989, the National Poisons Unit in London received 1070 inquiries following exposures to herbal preparations of which 25.2% were symptomatic [7]. Increased public use has made the medical community begin to seek accurate information about their therapeutic safety and side-effects. Medicinal herbs are not considered as drugs, are not regulated by the FDA and are exempt from the stringent testing procedures that licensing under the Medicines Act 1968 requires.

The Pharmaceutical opinion is that excessive use of St John's Wort should be avoided. It would be wise for patients taking this drug to observe precautions appropriate for conventional MAOIs and the same applies to anaesthetic practice. In addition, there should be an increased awareness of self-medication with herbal adjuncts in the anaesthetist's pre-operative assessment.

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Removal of the laryngeal mouse airway

We support the view that the laryngeal mask airway may safely be removed when the patient has awoken from anaesthesia [1, 2] but would like to report an unpredicted and unusual complication following this practice. A 38-year-old woman was scheduled for elective removal of a submental lipoma under general anaesthesia. Current medication included venlaflaxine, a serotonin and noradrenaline reuptake inhibitor used in the treatment of depressive illness. She received temazepam and diclofenac pre-operatively. Anaesthesia was induced with propofol 2.5 $mg.kg^{-1}$ supplemented with fentanyl 2.5 $\mu g.kg^{-1}$ and maintained with a propofol infusion. A size 4 flexible reinforced laryngeal mask airway (FLMA) was inserted and the lungs ventilated with oxygen and nitrous oxide. Muscle relaxants were not given. The wound was infiltrated with bupivacaine 0.25% with adrenaline.

Surgery was uncomplicated, completed within 30 min and anaesthesia was then discontinued. Upon wakening, the patient became very agitated and struggled to remove the FLMA, whereupon she exclaimed that she had 'swallowed a mouse with a wriggly tail'. She subsequently required intravenous midazolam in the recovery area to treat emergence delirium. Further recovery was uncomplicated. During our postoperative visit, she remained amnesic for all peri-operative events. Upon prompted questioning she volunteered that as a child she was tormented by her peers and once a live mouse had been forced into her mouth.

We conclude that this patient's stormy recovery was multifactorial with the combination of a childhood memory, awakening with a 'wriggly tailed' FLMA in place and delirious emergence possibly attributable to a type of serotonergic syndrome [3].

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Peanuts or paranoia?

Perhaps anaesthetists at St. Mary's are extra vigilant over the contents of cereal packets, because, in the space of a few

weeks, several interesting ingredients have been discovered on packaging.

Firstly, it was noticed that peanut oil was present in bone cement liquid monomer [1] which seemed to be a relatively new addition to the list of ingredients. Now, however, this very same peanut oil is found to be a constituent of Naseptin cream, frequently used in ENT surgery [2]. Naseptin, containing chlorhexidine and neomycin, is used to clear staphylococci from the nasal vestibule, but an interesting additive is arachis oil (peanut oil). Although labelled by the manufacturers as an inactive ingredient, it is deemed worthy enough to be labelled as a separate constituent and highlighted.

It is interesting to note that there is an association between nasal polyps and staphylococcal infection [3, 4], requiring treatment with, for example, Naseptin cream. Given the rare, but well–known, association between nasal polyps, asthma and NSAID hypersensitivity [5], is it reasonable to give peanut oil (albeit refined) to potentially atopic patients without knowing whether the peanut oil is completely inert?

Nut allergy is a well-recognised problem, and nuts are known to be both potent and potentially fatal allergens [6]. The question should therefore be asked, why if arachis oil is so inactive, do the manufacturers feel the need to highlight the fact that it is an ingredient of their product? Should we be specifically asking patients about nut allergy before administering this medication?

Or maybe we should just stop reading labels and unnecessarily worrying ourselves.

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