Hypoxia caused by a faulty Steri-cath closed suction device and use of continuous suction

Editor—A 52-yr-old man with severe pneumonia was ventilated artificially using a Siemens Servo 300 ventilator, in pressure regulated volume control mode, with a high $F_{\text{I}O_2}$ (0.6–0.7), high mean airway pressure and an inverse I:E ratio. A Steri-cath closed suction device (SIMS Portex) was used and changed every 24 h. This device enables regular bronchial suction by insertion of an in-line suction catheter down the tracheal tube without breaking the ventilation circuit. It prevents loss of positive airway pressure and subsequent alveolar collapse that may occur using standard suction catheters that are inserted by breaking the ventilation circuit. Continuous suction devices are used particularly in patients with high or critical oxygen requirements. Such patients usually require high mean airway pressures to recruit lung volume and standard suction may cause hypoxia.

The condition of the patient was unchanged on these settings for 5 days, then suddenly he became hypoxic, with a decrease in $S_a O_2$ to 80–85% without obvious cause. The ventilator alarm indicated a low expired minute volume. There was no change in delivered $F_{\text{I}O_2}$. Secretions were minimal, and clinical examination of the chest was unchanged. Arterial saturation returned to 100% on bagging with an $F_{\text{I}O_2}$ of 1.0 but decreased repeatedly when the patient was placed back on the ventilator. The Siemens Servo 300 ventilator digital readouts of inspired and expired tidal volumes were equal when attached to the dummy lung (during bagging), but there was a loss of 30% of the expired tidal volume on reconnecting the patient, demonstrating a leak in the system. There was no obvious tracheal cuff leak. The leak disappeared when the Steri-cath was disconnected from the suction tubing and the proximal end of the suction catheter was occluded with a finger, and also when the closed suction device was removed entirely. There were no adverse sequelae from this event.

The Steri-cath closed suction device had been changed only 1 h before the event. On closer inspection, the manual control valve, which is depressed to open the catheter to suction, had failed to return to the closed position. This had allowed continuous suction to be applied to the patient’s lungs as the suction had been left on continuously ‘for convenience’, contrary to the manufacturer’s instructions, causing loss of lung volume and hypoxaemia. Correspondence with SIMS Portex confirmed that the problem had arisen because of (1) a faulty manual control valve (which had operated normally when re-tested) and (2) use of suction continuously rather than as required for clearing of tracheobronchial secretions.

Unrecognized leaks from the ventilation circuit may reduce airway pressure below a critical level resulting in potentially life-threatening alveolar collapse. Digital displays of inspired and expired tidal volumes may be useful in identifying the leak. When using continuous suction device it would seem sensible to ensure that the manual control valve has reverted to the closed position after use and that suction is applied only when required, as the instructions suggest!

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Placement of double-lumen endobronchial tubes

Editor—I was interested to read about the fibreoptic-guided method of placement of left double-lumen endobronchial tubes described by Cheong and Koh.1 I agree that this method is indicated particularly when isolation is crucial in the first instance. In my experience of several cases of bronchopleural fistulae, it is possible to secure the airway quite rapidly after rapid sequence induction. However, I have found that putting the bronchoscope through the bronchial lumen in the first instance allows unequivocal identification and cannulation of the correct main bronchus (right or left). The double-lumen tube is thus railroaded into position, and checked in the conventional manner with minimal delay in assisted ventilation. I have been teaching this technique to trainees during elective cases, as practice is required in order to achieve the necessary speed when the real need arises.

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Editor—We thank Dr Ip-Yam for his interest in our study. We agree that placing the fibreoptic scope through the bronchial lumen allows for correct identification and cannulation of the main bronchus and we are well acquainted with this technique. For optimum positioning of a double-lumen tube, the secondary bronchial bifurcation should not be occluded by the tip of the endobronchial tube and
the endobronchial cuff should seal the bronchus without herniating into the tracheal lumen. While the former can be determined with the scope in the bronchial lumen, the latter is readily apparent during endobronchial intubation with our method (i.e. having the bronchoscope in the tracheal lumen). This assumes further importance in cases of pulmonary haemorrhage or empyema where rapid isolation of the lung is critical to minimize contamination and allow unimpeded ventilation of the non-affected lung. In addition, since some practitioners advocate routine intubation of the left bronchus, regardless of the operative side, passing the scope through the bronchial lumen into the left main stem bronchus may result in a view obscured by blood or pus and is of limited use if the left lung is the affected side. One further advantage of our method is that we are able to identify problems related to the size of the double-lumen tube. A double-lumen tube which is too big may fail to cannulate the bronchus, or even cause bronchial rupture.1 Too small a tube carries a risk of misplacement and damage to the bronchus.2 There is no ideal method of isolating a lung quickly. We only hope that our method is a safe option for our patients.

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1 Hannallah M, Gomes M. Bronchial rupture associated with use of double lumen tube in a small adult. Anaesthesiology 1989; 71: 457-9

Fibreoptic orotracheal intubation

Editor—Hakala, Randell and Valli state in their article about the difficulties in railroading, ‘How impingement can be avoided during fibreoptic orotracheal intubation is not known’.1 I described previously a technique for avoiding this problem in 1985.2 The anatomical and geometrical factors influencing success or failure of railroading a tube along a flexible guide are the same, whether bougie or fibrescope. Railroading has been shown to be easier with a thicker guide3 or with a thinner tube.4,5 Therefore, it would be expected that the use of a gum elastic bougie, being thinner than a fibrescope, might cause more difficulty. However, Dogra, Falconer and Latto6 found a success rate of 100% when they used a –90° rotation of the tube on the bougie while keeping the laryngoscope in the mouth. In my experience, this always works.

Similarly, Hughes and Smith7 reported a success rate of 93% and 100% at the first attempt when railroading along a nasotracheal fibrescope, by using a –90° and a –180° +90° = –90° rotation, respectively. The quarter-turn anti-clockwise rotation was shown to be helpful in guided blind intubation (variously called ‘retrograde intubation’).8

The secret of success in railroading is to keep the tip of the tube (e.g. 8.0- or 9.0-mm cuffed Portex) in close contact with the anterior surface of the guide. To achieve this, the –90° rotation must be used. The neck should be moderately flexed so that the guide has a single curve, with the concavity facing forward. Failure may occur if the neck is extended, as this would cause the guide to develop a double curve, and the tip of the tube would no longer be in close contact with the bougie if the concavity is facing posteriorly near the larynx. Lubricant jelly should be applied to the tip and the lumen of the tube before threading it onto the guide. It also helps if the tongue is pushed forward by the laryngoscope blade during the railroading.

If Hakala, Randell and Valli had used this technique, they might well have reported good success with railroading.

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3 Hakala P, Randell T. Comparison between two fibrescopes with different diameter insertion cords for fibreoptic intubation. Anaesthesia 1995; 50: 735–7
4 Marsh N. Easier fibreoptic intubations. Anaesthesia 1992; 76: 861
7 Hughes S, Smith JE. Nasotracheal tube placement over the fibreoptic laryngoscope. Anaesthesia 1996; 51: 1026–8

Editor—Thank you for the opportunity to reply to Dr Cossham. Impingement of the tracheal tube is a well known problem with fibreoptic intubation. The incidence of impingement during orotracheal intubation is 5–90% in prospective, randomized, controlled studies.1–7 Dr Cossham suggests that the problem can be overcome by rotating the tracheal tube by 90° anti-clockwise. We have used similar manipulations on resistance to intubation, but we still have experienced failed intubations in our studies.4,7 Impingement of the tube during orotracheal fibreoptic intubation remains an unsolved problem, not only because it can be caused by a variety of factors, such as the anatomical features of the patient6 or the discrepancy between the diameters of the intubation tube and the fibrescope,4 but also because the likely sites of impingement can be either the right arytenoid cartilage,8 the epiglottis, the posterior pharyngeal wall or other laryngeal structures.1
The arguments presented by Dr Cossham are based on experience gained with the gum elastic bougie. The flexibility of the gum elastic bougie is, however, different from that of fibreoptic bronchoscopes or intubating fibrescopes. The results with these intubation aids may also be different, at least as far as impairment is concerned. We are not aware of any study comparing intubation guides of varying flexibility, although such results may be useful in the further development of intubation fibrescopes.

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4 Hakala P, Randell T. Comparison between two fibrescopes with different diameter of the insertion cord for fibreoptic intubation. Anaesthesia 1995; 50: 735–7
5 Randell T, Valli H, Hakala P. Comparison between the Ovassapian and the Berman intubating airway in fibreoptic intubation. Eur J Anaesthesiol 1997; 14: 380–4
8 Schwartz D, Johnson C, Roberts J. A maneuver to facilitate flexible fibreoptic intubation. Anesthesiology 1989; 71: 470–1

Is anaesthesia evidence-based?

Editor—Myles and colleagues\(^1\) reported that it was highly likely that anaesthesia was evidence based. However, I would like to take issue with some aspects of their article.

The authors chose to use the number of patients, rather than interventions, as the denominator in their calculations. They did not explain why they chose to do so, but went on to refer in the summary, text and title of Table 4 to their results as ‘percentages of interventions’. Ellis and colleagues\(^2\) used the same denominator (number of patients) and explained it as a means of providing a more appropriate clinical perspective and avoiding bias from the effect of rarely used treatments. However, Ellis and colleagues had only one intervention per patient. Clearly, while the results of this study would contrast significantly with the quoted ‘10–20%’\(^3\) of interventions being non evidence-based, having allowed for more than one intervention per patient they were not accurate in quoting their results as they did. If one patient had received both an evidenced and a non-evidenced intervention, it would have biased their results to have counted them in the positive or the negative; and if all their patients had received two interventions, one evidenced and the other not, by their reckoning they would have had to conclude that 100% of interventions were evidence-based.

The definition used for level IV evidence is stated in Table 1 and includes the ‘opinions of respected authorities’ among possible evidence required. It is later stated that ‘interventions supported by level IV evidence were presented to three randomly selected anaesthetists from our department’ for ratification. What was the purpose of this further scrutiny of interventions already supported? References for level IV evidence were not provided, so it is not possible to know whose opinions were responsible for such evidence being attributed or not to those interventions.

The authors stated that evidence may ‘be found that contradicts our evidence’ and commented how ‘the evidence-base is expected to evolve’. Are we to understand that the evidence they quoted is the most evolved? If not, or if evidence of equal quality exists which is contradictory, what then is the value of their quoted evidence and should it not be discounted? A similar concern arises when considering the use of textbooks as sources of evidence, as quoted work reflects any bias of an individual author.

The authors made several references to ‘routine anaesthesia’. In their summary and introductory paragraphs, they stated that their interest was in ‘interventions in routine practice’. They then contradicted this in the methods section where they described how they chose interventions for scrutiny as those used in cases that did not fall within their definition of ‘routine anaesthesia’. So, in fact, they have considered interventions performed outside the ‘routine practice’ of their department.

They also stated that ‘routine practice should be regarded as having level IV evidence’. I would contest this statement. The purpose of a study of this nature is to examine the scientific basis of actions undertaken as part of administering anaesthesia—essentially a quality assurance type assessment. Their statement that ‘anaesthesia per se is evidence-based’ because of what history tells us is unhelpful. The issue here concerns the methods used to achieve and maintain safe anaesthesia. The authors judged some interventions to be without evidence even though they may ‘be considered reasonable practice by most anaesthetists’ and yet attributed level IV evidence to ‘routine’ practices not necessarily even supported to that extent. While, as they commented, description and analysis would have been more difficult, if they were to have been as stringent as they asserted then some assessment should have been made. Furthermore, with there being a variety of ‘routine’ practices between individuals and centres, is it possible to generalize about the attributed level of evidence?

Finally, it is important to consider the relevance of their quoted evidence to actual anesthetic practice. For example, they quote evidence not supporting the position of avoiding propofol in epileptics. Recently, an editorial in this journal\(^4\)
suggested that ‘our use of this agent in epileptics may reasonably be guided by pragmatism in addition to science’ and that on this basis it might be avoided in a given situation. If it is pragmatic to adopt such an approach in regard to this or any other of the studied interventions, what then is the value of the quoted evidence?

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Editor—Some of the issues raised by Dr Barnardo were addressed in our original article. Similar concerns have been raised previously, in response to the original article addressed in our original article. Similar concerns have been raised previously, in response to the original article addressed in our original article. Similar concerns have been raised previously, in response to the original article addressed in our original article.

2 Ellis J, Mulligan I, Rowe J, Sackett D. Inpatient general medicine is evidence based. Lancet 1995; 346: 407–10
3 Smith R. Where is the wisdom . . .? The poverty of medical evidence. BMJ 1991; 303: 798–9

Do not rely on a laryngeal mask in major periglottic pathology

Editor—Wakeling, Ody and Ball described a patient with a large goitre whose trachea could not be intubated blindly or fiberoptically via an intubating laryngeal mask airway (ILMA). Their conclusion from this well documented case was that successful blind intubation may not be achieved reliably via an ILMA when major cervical pathology is present.

As early users of an ILMA, we would like to add some comments. First, there are three more reports of patients with major cervical pathology whose tracheas were intubated with variable success via an ILMA. In a series of 19 patients who were known to be difficult to intubate, Cros and Colombani ventilated and intubated blindly all but two tracheas via an ILMA. Two of the five patients with fibrosis of the neck in whom blind intubation failed were successfully intubated fiberoptically via an ILMA. Ferson and colleagues presented a series of 28 patients with difficult intubation, including six patients with fibrosis of the neck and one with a large goitre: in all but one who had a laryngeal cancer, the trachea was ventilated.
successfully and intubated blindly via an ILMA. We presented 33 difficult to intubate patients, including 23 who had undergone surgery for facial carcinoma and had fibrosis and scars on the neck, and some patients with mild to moderate swelling of the laryngeal inlet. In all, the trachea was ventilated successfully and intubated blindly via an ILMA except in one patient who had undergone partial resection of the larynx as a result of cancer and reconstruction of a neoepiglottis. Fibreoptic control showed that the mobile neoepiglottis was depressed by the ILMA and also by a conventional laryngeal mask airway (standard laryngeal mask airway, SLMA). The patient’s trachea was ventilated only by withdrawing both laryngeal masks back to the base of the tongue. Blind intubating attempts in this position resulted in oesophageal positioning of the tracheal tube with both masks. Finally, the patient’s trachea was intubated fibreoptically without the use of a laryngeal mask.

From initial experiences with an ILMA in major cervical pathology, blind intubation and ventilation have been shown to be successful, but by no means guaranteed. Warnings to use a SLMA in cervical pathology are as old as the device but reports of its use are limited. In spite of the warning of Wakeling, Ody and Ball, we believe that in a cannot ventilate–cannot intubate situation, the ASA warning of Wakeling, Ody and Ball, we believe that in a cannot ventilate–cannot intubate situation, the ASA American Society of Anesthesiologist (ASA) Task Force on management of the difficult airway. Practice guidelines for the management of the difficult airway. Anesthesiology 1993; 78: 597–602

H. Langenstein
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2 Cros AM, Colombani S. Preliminary study of intubation with a new laryngeal mask for difficult intubation. Anesthesiology 1997; 87: A481
4 Langenstein H, Möller F. Der Stellenwert der Larynxmaske bei schwieriger Intubation und erste Erfahrungen mit der Intubationslarynxmaske. The role of the laryngeal mask in difficult intubation and initial experiences with the intubating laryngeal

Editor—I thank Drs Langenstein and Andres for their interest in our case report. Since our case report, several series have been published using the intubating laryngeal mask (ILM) in patients with proved or expected difficult intubation. Success rates for ventilation and intubation via the ILM were high, with 120 of 126 difficult airway cases intubated successfully (95%). I agree that the ILM could be substituted for the standard laryngeal mask (LMA) in the ASA difficult airway algorithm. The ILM has the advantage over the LMA in that the rigid handle allows manipulation of the mask position relative to the larynx, ensuring the ability to ventilate and allowing a standard length 8.0-mm internal diameter tracheal tube to be inserted reliably without fibreoptic bronchoscopy. As Drs Langenstein and Andres point out, there is no guarantee of success and therefore thought should always be given to further steps which may be required to secure the airway.

Finally, as they correctly state, the neutral head position is important for reliable ILM function. Incorrect head position may have contributed to the intubation failure in our case report. The importance of the neutral head position was therefore emphasized in our discussion.

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4 Basket PJJ, Parr MA, Nolan JP. The intubating laryngeal mask.
EEG indices and heart rate variability as measures of depth of anaesthesia

Editor—I read with interest the article of Sleigh and Donovan on comparison of bispectral index, 95% spectral edge frequency and approximate entropy of the EEG, with changes in heart rate variability during induction of general anaesthesia.1 The authors concluded that bispectral index was superior to other EEG indices of depth of anaesthesia. I feel that this conclusion cannot be drawn from their investigation as several issues, which might not be familiar to readers not involved in EEG studies, need to be addressed. First, bispectral index and spectral edge frequency were calculated after artefact rejection whereas when approximate entropy was calculated, all artefacts were apparently included. Most artefacts appear during the awake state as a result of body and particularly eye movements. In this study design with repetitive verbal commands to open the eyes, artefacts were even more likely to occur.

Second, bispectral index and spectral edge frequency were calculated from 5-s epochs whereas approximate entropy was calculated from 2-s epochs without any reason for the difference. The fast Fourier transform requires an epoch length of 2^n data points, for example 2 s (\(= 512 = 2^9\) data points) or 4 s (\(= 1024 = 2^{10}\) data points) but not necessarily 5 s. Possibly the 5-s epochs were caused by the internally (inside the Aspect monitor) averaged overlapping epochs and computation of bispectral variables, as bispectral index requires averaging of several epochs.2 3 In contrast, approximate entropy was calculated from shorter epochs without averaging.

Third, for calculation of approximate entropy, the level at which noise was filtered was fixed at 5 \(\mu V\). This is not in accordance with the recommendation to calculate the noise filter level as a percentage of the standard deviation of each epoch.4 5 This issue is highly relevant as fixed noise filter levels lead to different approximate entropy values depending on the EEG amplitude. Therefore, inter-individual variability and even intra-individual variability is unnecessarily increased because of factors such as skin impedance. In summary, the study design clearly favoured bispectral index. This may explain the disagreement with our findings, reported previously,6 that bispectral index and EEG approximate entropy were equivalent for assessment of anaesthetic drug effects.

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Editor—we thank Dr Bruhn for his interest in our article. We agree entirely that a major factor in the success of the Aspect EEG monitor and the bispectral index is the intensive attention to detail of signal pre-processing and artefact rejection. However, we were trying to compare different techniques in the ‘real world’ of patients undergoing routine anaesthetics. The more favourable results that Dr Bruhn reported were almost certainly because he was performing a very controlled pharmacodynamic study, varying the dose of a single potent inhalation agent in an already anaesthetized patient. It was of interest to us that the values of approximate entropy that he obtained were very similar to those from our study, even though he had used longer data segments and presumably an adaptive value for the noise threshold.

Although bispectral index apparently has some merit, we believe that there is still much room for improvement in the use of EEG measures of anaesthetic depth, and hope that scientific method may triumph over commercial muscle in the end.

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Regional anaesthesia for carotid endarterectomy

Editor—We read with interest the excellent review by Stoneham and Knighton.1 We agree that regional anaesthesia is an attractive option and one which we expect will become increasingly popular. A significant proportion of the mortality and morbidity associated with the procedure is cardiac,2 undoubtedly exacerbated by the requirement during general anaesthesia to maintain a high arterial pressure with vasoactive drugs.3–5 Some authors have recommended the use of transoesophageal electrocardiography to monitor myocardial function if arterial pressure is being augmented pharmacologically.5 Our experience suggests that the requirement for vasoactive drugs is significantly reduced with regional anaesthesia as the awake patient can confirm the adequacy of their cerebral perfusion pressure. The anaesthetist is no longer required to try to maintain arterial pressure at an arbitrary predetermined value.

However, we would like to take issue with the suggestion that sedation is best confined to a small dose of a benzodiazepine. Carotid surgery may be prolonged and patients can find the experience uncomfortable. We have developed considerable experience with the additional use of target-controlled propofol infusions for this procedure. This is a highly controllable method of sedation that can be increased as required for regional block insertion, local infiltration, initial surgical dissection and wound closure, yet be reduced rapidly on demand for neurological assessment and during carotid cross-clamping. We believe this maintains all the benefits of regional anaesthesia while making the procedure less stressful for the patient and surgeon. We hope to have data available for publication shortly.

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1 Stoneham MD, Knighton JD. Regional anaesthesia for carotid endarterectomy. Br J Anaesth 1999; 82: 910–19

Editor—Having a major interest in regional anaesthesia, and having spent 15 yr trying to answer the anaesthetic challenge posed by carotid endarterectomy, I read the recent review by Stoneham and Knighton with great interest.1 They present a much better outline of the problems than I have read previously, but I must express some anxiety at their enthusiasm for a large, randomized, comparative study of regional vs general anaesthesia. Such studies always beg many questions and I would like to raise two of them here.

First, precisely what technique of regional anaesthesia will be compared with which technique of general anaesthesia? Several regional techniques are possible, and the variations on general anaesthesia are almost infinite.

Second, and probably more important, is the question of whether the anaesthetists involved will have equal competence and experience of managing this difficult procedure using both anaesthetic techniques? This aspect has all too often been ignored in similar ‘outcome’ studies in the past. It is my view that surgeons and anaesthetists working in these difficult areas should define their criteria, agree management protocols and pursue a standard practice. They should then audit their results, compare them (in all respects) with those of others, and see what improvements might be made in their own approach. I am fairly certain that attention to detail (particularly in regard to control of respiration and circulation) is much more important in the management of carotid endarterectomy than is the choice of one anaesthetic technique over another. All too often in medicine, the existence of more than one approach means that neither is ideal, and that the wrong question is being asked when a comparison is proposed.

Finally, I would ask if the authors have considered my preference, the combined approach? With regional plus general anaesthesia, the anaesthetist, surgeon and patient get the best of both techniques.

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Editor—We thank Drs McCrirrick and Williams for their comments about sedation during the procedure. We agree with them that a target-controlled propofol infusion is a good alternative to a benzodiazepine for providing perioperative sedation. We did mention in our article that a low-dose propofol infusion was an alternative to benzodiazepines. Target-controlled infusions (TCI) were not available to us at the institution in the USA where we obtained most of our experience. However, one of us (M.D.S.) now routinely uses TCI during awake carotid surgery to allay anxiety and provide amnesia during placement of the block and dissection. He is currently investigating the use of patient-controlled sedation using propofol in this context. We would like to reiterate, however, that sedation is no substitute for
adequate regional anaesthesia. During carotid endarterectomy, we believe that verbal communication must be maintained with the patient at all times.

We agree with Professor Wildsmith that it is important for surgeons and anaesthetists to audit their results for this procedure on a regular basis and alter their techniques accordingly. However, we take issue about the requirement for a randomized, controlled study to compare regional with general anaesthesia. The non-randomized, often retrospective data analysed by Tangkanakul, Counsell and Warlow\textsuperscript{1} suggested that there is a 50% reduction in mortality. Warlow\textsuperscript{1} suggested that there is a 50% reduction in mortality. We believe that such a study is important to determine the ‘best’ anaesthetic to us, just as the surgical indications for carotid endarterectomy have been refined further by the publication of large, randomized, controlled studies, such as the North American\textsuperscript{2} and European\textsuperscript{3} ones.

Finally, we cannot see that a combination of regional plus general anaesthesia offers any benefits over a regional technique alone. On the contrary, administering general anaesthesia nullifies the principal reason for keeping the patient awake, namely monitoring adequate cerebral perfusion. However, if a patient requires general anaesthesia for this operation, we believe that a combination of general anaesthesia using remifentanil and a superficial cervical plexus block offers the best combination of techniques to provide a fully awake, yet comfortable patient after operation.

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Central nerve block and thrombophrophylaxis

Editor—We read with interest the review article on central nerve block and thrombophrophylaxis\textsuperscript{1} which has prompted us to consider our current practice. There is an increasing trend in our hospital to use an Iloprost (trometamol) infusion, a prostacyclin analogue, in patients with critical limb ischaemia who are awaiting re-vascularization surgery. The drug has an anti-aggregatory effect on platelets, resulting in a dose-related decrease in thrombogenesis. We thought that if a patient was receiving an Iloprost infusion, this represented a relative contraindication to epidural catheterization. Although the evidence suggests that other antiplatelet drugs are not a contraindication to epidural analgesia,\textsuperscript{2} Iloprost has a different mechanism of action and the risks of epidural bleeding and haematoma formation are not comparable. We considered that i.v. infusion of an antiplatelet aggregation agent had significant potential to cause epidural haematoma formation after central neural block when balanced against the benefit to the patient.

We could find no reference to this risk in the literature and were unsure if our assumptions were correct. Basic science research provides some relevant information. The pharmacodynamic and pharmacokinetic properties of Iloprost have been reviewed.\textsuperscript{3} Iloprost has the same spectrum of activity as prostacyclin. Its site of action is probably multifactorial but involves reversible binding to a specific platelet receptor, possibly on the Gs subunit of GTP. This increases cAMP formation and reduces platelet activation. The effect is short-lived. At the end of an infusion of 2 ng kg\textsuperscript{-1} min\textsuperscript{-1}, the recommended maximal rate, the elimination half-life of Iloprost is 31 min with any inhibitory effect on platelets disappearing by 2 h. Iloprost does not interfere with the clotting cascade and unwanted bleeding associated with its use has not been reported.

However, if used concurrently with heparin, there is an additive effect and bleeding becomes more of a potential risk. This interaction is important in clinical practice as patients undergoing vascular surgery receive heparin in the peri–operative period via a variety of different routes and regimens. Importantly, systemic heparin is given during surgery, often within 1 h of epidural catheter insertion which, independent of the action of Iloprost, carries an increased risk.\textsuperscript{2} Patients are also restarted on Iloprost and heparin after operation and therefore have altered platelet and clotting cascade function when the epidural catheter is removed on the ward.

The risk of epidural bleeding with Iloprost remains unquantified. Concurrent administration of Iloprost with heparin would appear to be a relative contraindication to central regional block. If Iloprost is used alone and provided it is discontinued 2 h before epidural or spinal anaesthesia, the risk of epidural haematoma formation would appear to be very small and should not preclude their use. Similarly, the infusion should be discontinued for 2 h before removal of the epidural catheter. We hope that this would be regarded as safe practice.
Editor—Thank you for the opportunity to respond to Drs Dunnet and Pittman. To date, there have been no reports of vertebral canal haematoma associated with the use of Iloprost infusions in patients who have had central neuraxial block. Iloprost inhibits platelet aggregation in addition to being a vasodilator and has a cytoprotective effect on endothelial walls. It may also have some fibrinolytic activity.¹ The duration of action is short with platelet anti-aggregatory effects undetectable 2 h after stopping the Iloprost² infusion. As long as no other anticoagulant drugs have been administered and Iloprost has been stopped for at least 2 h, the risk of central neuraxial block or catheter removal is small. However, we feel that some caution should be exercised when considering central neuraxial block in patients who are receiving or who are scheduled to receive Iloprost infusions in the perioperative period. The great majority of these patients receive heparin concomitantly in the course of the operative procedure, which increases the risk of haemorrhagic complications. We agree with Pittman and Dunnet that simultaneous Iloprost and heparin infusions are a contraindication to central neuraxial block, but in our experience both drugs are stopped in advance of surgery. If an epidural catheter is already in place and the surgeon requests Iloprost–heparin infusions, it should be removed only when the antiplatelet and anticoagulant effects have resolved.

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Organ Preservation is one of the quarterly issues in the International Anesthesiology Clinics series. My only criticism with the volume, which on the whole I enjoyed reading, is the title. I would have expected a book called organ preservation to specifically deal with issues of organs used for transplantation. This is particularly so, since the editor of this issue is based at a well known UK transplant unit. In fact the volume covers aspects of preservation of organ function. Perhaps organ protection may have been a more accurate title.

Many of the chapters relate to issues of patients undergoing surgery involving cardiopulmonary bypass. Even in the chapters covering more general issues, such as ‘the role of the gut’ and ‘preservation of renal function’, many of the references are from work undertaken in patients undergoing cardiac surgery. Perhaps this is the target readership for this issue of the series. Certainly, reference to this potential bias is made in the preface. However, the point is well made that the changes seen during cardiac surgery can also be apparent during other forms of surgery, and also that the treatment options will be similar if not the same.

Having said that, although the pathophysiological basis underlying the response to major surgery is well described, there are few suggestions as to how such changes can best be prevented and treated. The average reader will therefore have a better understanding of the process of organ dysfunction but probably no new ideas on how to prevent the dysfunction occurring in the first place. Perhaps this is because, as yet, there are few options open to us. However, there is only passing comment to one technique which is said to result in gastric mucosal acidosis in chapter 5 as OH– (which is the hydroxyl ion). In addition, doxepamine is said to result in gastric mucosal acidosis in chapter 5 which may leave the reader a little confused as it is suggested in chapter 4 as a useful agent to prevent gut dysfunction.

Given the bias of the volume, this issue will probably be of most interest to the cardiothoracic anaesthetist who I am sure will learn from its contents. Perhaps the one organ which should have been included and does not feature at all is the brain. This is somewhat surprising in view of the advances made recently in understanding the pathogenesis of dysfunction which occurs in other disease states in this particular organ.

N. R. Webster


This short book belongs to an ever-growing list that I refer to as the ‘How to’ series, in which senior authors are reputed to provide sagacious advice to trainees about the bits of their professional life that they find difficult. The authors are distinguished research workers, two surgeons and a cellular physiologist, who remain extremely active academically. Not surprisingly, the text is full of useful information, is well written and has a clear layout. It is also enlivened by the cartoons of David Langdon.

The emphasis throughout is on undertaking the work required for a thesis. Chapters include reading for a research degree, applying for research positions, and research supervisors and projects. The stages of the work are covered in chapters entitled the first phase, getting started; the second phase, frustration; the third phase, as results arrive; and the fourth phase, writing the thesis. The most interesting chapter for me was a very brief one trying to evaluate the reasons why people do research. Reasons advanced by the authors include the challenge, becoming a better clinician and even fame. A short paragraph on lifestyle merits direct quotation as it encapsulates many of the current problems in recruiting to academic anaesthesia: ‘If you want continual reassurance and direction, research is not for you. Self assurance, independent thought and the willingness to take a chance are valuable in research. . . . The distinction between work and rest is less clear and there is great pressure on you to work at home and on weekends. Monetary rewards while in research are generally less than in clinical medicine.’

I disagree with the authors over some details about planning, undertaking and writing a research project, but this may simply reflect the small differences between anaesthetic and surgical research. It does not detract in any way from the general principles advanced by the authors. I remain uneasy, however, that the authors may have underestimated the skills in research methodology gained by current undergraduates. I recently helped assess research projects undertaken by second year medical students. The oral presentations and written work of some were outstanding and it is salutary to acknowledge that these 20-yr-olds have already grasped many of the basic principles advanced in this book. Although it may be appropriate for current trainees, it is unlikely to remain so in the future.
In summary, this book is a solid achievement. My tentative recommendation for the next edition would be to start at a more advanced level and recruit a young co-author whose thesis has been completed recently.

G. M. Hall

Near Misses in Pulmonary and Cardiothoracic Critical Care. J. Varon, G. Walsh and R. Fromm jr. Published by Butterworth Heinemann, Oxford. Pp. 120; indexed; illustrated; Price £35.

This little book is a new title in the ‘near misses’ series which describes scenarios in pulmonary and cardiothoracic critical care. Previous issues have covered paediatric anaesthesia and cardiac surgery. It has been written by three authors, all of whom have a critical care interest. Two are professors of medicine from Baylor College of Medicine, Houston, and the other is a professor of surgery from the University of Texas.

This is an interesting and easily read text which, the authors claim, is written for ‘everyone engaged in critical care medicine, pulmonary medicine, cardiology and cardiothoracic care’. Many will not agree however, with the statement found in the preface, ‘the definition of critical care is, in fact, a near-disaster or near-catastrophe’! Although obviously directed at the American reader, the omission of unnecessary and confusing jargon will aid its export from that continent. The authors aim to illustrate clinical situations in which ‘errors in diagnosis or treatment or unusual presentations led to potentially serious consequences’.

The subject matter is divided into 30 brief chapters, each of four pages of text at most. Each deals with one case history, the diagnosis and clinical management, followed by suggestions for further reading. Most chapters are illustrated by an EEG or x-ray, which are well presented. Each chapter has a ‘tongue in cheek’ title, hinting at the diagnosis to be made (e.g. ‘The Canadian corpse who wriggled her toes’ or ‘A tall man with thick glasses and chest pain’). The latter deals with a young man presenting with chest pain who is treated with a thrombolytic agent after a suspected myocardial infarction; he collapses and immediately undergoes repair of his aortic arch dissection. This man, of course, has Marfan’s syndrome, and one may wish to ponder his prognosis in a district general hospital without a cardiothoracic unit.

The patient histories are easy to follow, and have been written with a touch of humour. Every topic is therefore somewhat entertaining. Cynical or suspicious readers will suspect the diagnosis before reaching the end of the case presentation, but the style and brevity of the description will sustain their interest. Each discussion is succinct and gives a diagnosis, differential diagnosis and a short review of the condition with some historical perspective. The treatment options are explained, avoiding complex surgical descriptions, and are followed by clinical points with further reading references. Although each diagnosis is described clearly, a more detailed examination of the differential diagnoses and their elimination would markedly strengthen the teaching value of this book. Indeed, this would have filled the excessive amount of white space found between each chapter.

Although this is not a didactic text from which could be gleaned all of the knowledge required for an examination, it is a useful practical guide to acute clinical management. Descriptions of the consequences of breaking basic rules are well elucidated. For example, the management of a patient is detailed in whom a chest drain inserted with a trocar, without digital examination of the pleural cavity, transfixes the left ventricle, and requires immediate operative repair.

This book will be useful reading for senior house officers or new residents in anaesthesia, critical care or accident and emergency medicine for whom I would have added the title ‘All you wanted to know but were afraid to ask’. As a post-fellowship trainee in anaesthesia, I feel that I could have learned from this book when I was a senior house officer. In addition, consultants may well identify with some of the episodes and find the scenarios useful teaching aids. At £35, this book is expensive, although the quality of the x-ray reproductions probably justify its price. It would be a useful addition to a departmental library or provide lighter reading during examination preparation.

E. W. Moore