Acupressure and prevention of PONV

Editor,—I read Harmon and colleagues’ article on the use of acupressure in the prevention of postoperative nausea and vomiting (PONV) with great interest. A cheap (and presumably re-usable) device without side effects and with such demonstrable efficacy must surely inspire great enthusiasm, and I look forward to seeing further such research. However, I was somewhat concerned by the process by which patient consent was obtained for this study. Patients were told that ‘a form of acupuncture (using wrist bands ... ) may reduce the incidence of postoperative sickness and (that) we were investigating the most appropriate site for (these) to be placed’. This is somewhat disingenuous. The stated purpose of the study was to determine the efficacy of acupressure compared with placebo (or sham). There was no intention on the part of the investigators to determine the optimum site of placement. The active group received acupressure at the P6 site (considered to be the effective site) and the controls received it in any other sham site (known to be ineffective). While I appreciate the difficulties in the design of acupuncture research, there can be no excuse for misinforming patients in this way, even if, as in this case, the substance of the ‘misinformation’ is trivial.

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Editor,—Thank you for the opportunity to reply to Dr Farmery. Our method of obtaining consent is questioned. This is an important issue, and I would like to clarify our statement in this regard. The substance of his assertion that we misinformed patients as to the purpose of the study is incorrect. Before the study, it was explained clearly to patients that we were not aware of any benefit of either of the two sites we proposed to use (P6 or sham acupressure) or indeed the efficacy of acupressure in this setting. Our description of patient consent consisted of one sentence in the patients and methods section. Had we expanded further, I am confident that the matter would not have been open to misinterpretation by Dr Farmery.

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Vertebral canal haematoma is a hazard of spinal–epidural anaesthesia in patients treated with low-molecular weight heparins

Editor,—We read with interest the case report by Yin and colleagues highlighting the hazards of administering low-molecular weight heparins (LMWH) soon after epidural catheter removal. It has become increasingly apparent that LMWH should be considered a potential risk factor for vertebral canal haematoma in patients undergoing spinal or epidural procedures (needle placement, catheter insertion or removal). Tryba and Wedel suggested that LMWH presented an even greater risk of clinically important haemorrhage because of vascular lesions during central neuraxial block compared with unfractionated heparin.

When first introduced in the UK in 1987 and in the USA in 1993, one of the main selling points for LMWH was a perceived advantage over unfractionated heparin in terms of less bleeding complications with a similar or better antithrombotic effect. It was suggested that less platelet inhibition and a greater anti-Xa/anti-IIa ratio compared with unfractionated heparin were responsible. Greater bioavailability as a consequence of lower protein binding and lower endothelial uptake was another obvious benefit. Once-daily administration has been shown to provide appropriate venous thromboembolism prophylaxis and dose adjustment or monitoring of drug effect was not necessary.

In spite of the perceived benefits, an ever growing number of cases of vertebral canal haematoma in patients undergoing spinal or epidural instrumentation and receiving LMWH have been reported, prompting the North American Food and Drug Administration (FDA) to release a Public Health Advisory Notice. The larger recommended dose for thromboembolism prophylaxis with LMWH in the USA may be one factor contributing to this unexpected development. Checketts and Wildsmith in their editorial also suggest greater rather than less platelet inhibition by LMWH compared with unfractionated heparin, and furthermore emphasize the fibrinolytic potential of LMWH.

Was this change in the perception of a relatively new group of drugs predictable? Possibly, as initial dose–response studies showed an increased bleeding frequency after larger doses of LMWH compared with equipotent doses of unfractionated heparin. What would currently constitute best clinical practice? Undoubtedly venous thromboembolism prophylaxis with either low-dose heparin or LMWH saves lives and reduces morbidity. When planning an epidural in these patients, guidelines regarding timing of the procedure and subsequent monitoring have been suggested and should be adhered to. If in doubt about
the patient’s ability to form an effective clot after injury to epidural or spinal blood vessels we produce a thrombelastograph beforehand. This technique has been shown to have outstanding sensitivity to low-dose heparin treatment1 and allows us to decide whether we can proceed safely with the planned procedure.

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S. V. Mallett  
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2 Tryba M, Wedel DJ. Central neuraxial block and low molecular weight heparin (enoxaparin): lessons learned from different dosage regimes in two continents. Acta Anaesthesiol Scand 1997; 41: 100–3  
4 Lumpkin MM. FDA Public Health Advisory: Reports of epidural or spinal haematomas with the concurrent use of low molecular weight heparin and with spinal/epidural anaesthesia or spinal puncture. Anaesthesia 1998; 88: 27–8A  

Editor,—We would like to thank Drs Wilkes, Mallett and Peachey for their interest in our case report and their comments regarding thrombelastography (TEG).1

There is a general consensus that there is a higher risk of epidural haematomas associated with low-molecular weight heparin (LMWH) than with unfractionated heparin in patients with epidural catheters. The safe practice of epidural anaesthesia in patients given LMWH depends on several factors which have been highlighted in recent studies.2–4 These include: appropriate dosing adjusted for body weight; timing of dosing (once-daily dosing being safer than twice-daily dosing); clear procedures for the timing of epidural catheter removal in relation to LMWH administration; and an increased awareness of this problem among doctors and nurses.

While there is no evidence that monitoring LMWH reduces complications of the therapy, there may be a case for dose–effect monitoring in some select patients (e.g. the elderly patient with renal insufficiency). Calculation of dosing based on weight may not give the correct dose in every patient. Correct dosing can really only be achieved by dosage adjustments based on the measured pharmacodynamic effect in the individual patient. One of the problems with LMWH is the difficulty in assaying the effects of the drug. Anti-Xa and IIa assays are relatively difficult and expensive and not widely available to the clinician.

Drs Wilkes, Mallett and Peachey have suggested that TEG may be a useful technique4 for monitoring the pharmacological effects of LMWH. One of the theoretical advantages of TEG is that it enables a rapid and full evaluation of the global clotting process, and the physical characteristics and stability of the clot, while other laboratory tests examine only one part of the clotting cascade. The use of TEG, as suggested, may lie in the assessment of effective clot formation when contemplating epidural insertion or removal. While it may assist in the timing and removal of the epidural catheter in relation to LMWH administration in a particular patient, the extent to which TEG helps prevent epidural haematomas is unknown. One problem with TEG is that blood samples must be analysed immediately which may be difficult in a hospital ward as this would require a TEG machine to be immediately accessible. In addition, TEG is not a widely available technique.

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Dry soda lime degrades sevoflurane during simulated inhalation induction

Editor,—We would like to comment on the article by Funk and colleagues on dry soda lime and degradation of sevoflurane during simulated inhalation induction.1 The authors note differences with previous work on this topic,2,3 which in our opinion do not exist.

They point out that they could not reproduce the complete absence of sevoflurane for the first 20 min in their experiments and that they could not detect compounds B–D when the compounds of the gaseous phase were collected with cool traps.2 We also observed the absence of sevoflurane for this period using a Datex Capnomac,3 the device they used for detection of sevoflurane. Funk and colleagues believe that more complete drying of the soda lime is the reason for this phenomenon but we do not agree.
To make these experiments highly reproducible and to achieve standardized conditions, fresh soda lime (Drägersorb 800) was dried until no further weight loss was observed for 24 h. With heat treatment or vacuum desiccation, a further weight loss of less than 1% wet weight was achieved.

The experimental settings were almost comparable. However, Funk and colleagues delivered sevoflurane 8 vol% in oxygen 6 litre min\(^{-1}\) into the lime, whereas we used sevoflurane 5 vol% in oxygen 2 litre min\(^{-1}\). Thus their sevoflurane input was 4.8 times higher. We reported previously\(^3\) that increased sevoflurane input led to faster movement of the heat zone through the absorber. This had little effect on the heat generated, regardless of whether the change in input was caused by increased sevoflurane concentration or carrier gas flow.

We assume that degradation of sevoflurane with dry soda lime is a stoichiometric reaction of sevoflurane with the compounds of the lime rather than a reaction catalysed by the lime. Therefore, the 4.8 times higher sevoflurane input accounts for the 5–6 times faster appearance of IR absorption on the Datex Capnomac. We believe that Funk and colleagues’ findings are in good agreement with our experiments, which is confirmed by the comparable temperature change with both limes.\(^3\)\(^4\)

Nevertheless, we are concerned about measuring sevoflurane concentration and recovery by IR absorption using the Datex Capnomac under these conditions. As we demonstrated with such a device in Figure 7 of our article,\(^3\) after 18 min IR absorption started with an initial peak of about 5 min duration, which exceeded the vaporizer setting by a factor of almost two. Therefore, we suggested that measurement using non-specific IR absorption may not be reliable under these conditions. This suggestion was confirmed by the cool trap experiments\(^2\) performed under the same conditions. In this period, several degradation products, including large amounts of methanol, were found which may affect IR absorption of sevoflurane. This may account for the remaining minor differences in the time delay before the appearance of sevoflurane.

Although during the degradation of sevoflurane there was the typical smell of formaldehyde, we were unable to detect formaldehyde definitively in our laboratory. In cool trap experiments with GC–MS, the typical spectra for formaldehyde were not detected. In recent investigations, dimethoxymethane, which forms formaldehyde in water, was detected and quantified in addition to methanol and compound A.\(^5\) In Funk and colleagues’ experiments, less methanol and dimethoxymethane were detected with Sofnolime than with Drägersorb 800. However, methanol reduction in these experiments was about twice as high as in Funk and colleagues’ results.

The authors emphasized that they could not detect compounds B–D or other intermediates of sevoflurane degradation. They are correct in that collecting the gaseous phase acts as a focusing process and allows detection of compounds at low concentration. In addition, the degradation products of sevoflurane, especially the intermediate compounds, are highly reactive. Even when the condensed phase is stored for a few hours at –20°C in a sealed vial, the GC–MS spectra change in a manner that cannot be explained by evaporative loss of compounds. Thus beside differences in the collection of the samples, the high reactivity of these compounds may additionally account for the minor differences in the findings.

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Editor,—We agree with Wissing, Kuhn and Warnken that the reaction of sevoflurane with soda lime is of a stoichiometric nature depending on the strong alkalinity of the lime. The higher sevoflurane input in our experiment might be part of the explanation for the early appearance of sevoflurane in the outflowing gas. The much more complete drying of the lime in their experiments (many days to weight constancy) might be another. The water content of our Drägersorb was 1.8–2.9%, while in Wissing’s study it was less than 1%. With extremely dry lime, even halothane is not detectable in the outflowing gas for more than 1 h.\(^1\)

Sevoflurane degradation has been shown to be strongly dependent on water content.\(^2\) The earlier appearance of sevoflurane in our experiments which were set up to mirror clinical conditions, can be interpreted as lower sevoflurane degradation.

To answer the question, if and to what extent the Capnomac might be affected by degradation products, one might look at the sevoflurane data from the gas chromatograph (GC). The similarity of the peak areas for sevoflurane in the GC and IR absorption throughout the experiment was excellent. Interference is theoretically possible as the Capnomac measures sevoflurane at one
wavelength only (3300 cm\(^{-1}\)), but this is of minor quantitative importance.

Concerning compounds B–D, we stated that we were unable to detect them with our experimental set-up. The reason is probably the large sevoflurane peak in our gas chromatograms which may hide small peaks, even if the substances have retention times substantially greater than sevoflurane. Little is known of the toxicity or toxic doses of these compounds which are not very volatile. Compared with potent toxins such as methanol, formaldehyde and compound A, they are probably of minor importance.

In contrast to the Frankfurt group, our method provides quantitative data on compound A and methanol in situations which are likely to occur in clinical practice.

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Sevoflurane anaesthesia with an Oxford Miniature Vaporizer

Editor,—The study reported by Liu and Dhara\(^1\) was encouraging for those who wish to use sevoflurane effectively and economically. The authors made several suggestions for further study. To prevent unnecessary work on some of these, I should like to comment on their findings, conclusions and suggestions for such studies. I have recently studied different means of inhalation induction with sevoflurane, based on systematic observations over many years. I believe that nitrous oxide causes excitation, rather than suppressing it, as Liu and Dhara hope it may. I have observed excitation when nitrous oxide was used with halothane, more commonly than when halothane was used in oxygen, when this was the agent of choice for inhalation induction.\(^2\) This is so consistent an observation that I have not bothered to formally study and report it, although in the interests of equipoise\(^3\) and to prevent others going up a blind alley, I suppose I should.

I agree with Liu and Dhara that a gradual increase in concentration of sevoflurane is likely to reduce airway reflexes such as cough. With a vaporizer out of circuit, a logarithmically increasing concentration setting (0.5%, 1%, 2%, 4% and then 8%) causes minimal upset (most subjects do not notice) and has the advantage of rapidly achieving ‘overpressure’ concentrations. This is superior to an inspired concentration of approximately 3% with the vaporizer in the circle, and makes induction more prompt.

Finally, Liu and Dhara report breath-holding and movement as complications. I would define ‘breath-holding’ as an inspiration followed by glottic closure. I suspect that their patients were not ‘breath-holding’ during induction. Although breath-holding can occur during inhalation induction, it is far less common than end-expiratory apnoea, which coincides with loss of consciousness and is frequent in hypocapnic subjects.\(^4\) The subjects in the study of Liu and Dhara were encouraged to breathe deeply for 1 min and thus would be hypocapnic to some extent, although patient compliance with the instruction to breathe deeply varied considerably. Perhaps if hyperventilation is to be encouraged, the carbon dioxide absorber should be switched off so that normocapnia is ensured. After all, since the gas composition is being monitored, hazardous concentrations of carbon dioxide would be detected promptly, if they occurred. This is most unlikely if the absorber is only switched off for the duration of induction. We have reported a similar strategy using a Mapleson D system (Guracha Boru and Drummond, submitted for publication) that can eliminate respiratory disturbance during inhalation induction.

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Editor,—We would like to thank Dr Drummond for his comments and his references. While nitrous oxide may cause excitation, it is still used widely together with other anaesthetic agents, including sevoflurane, for inhalation induction of anaesthesia. The concentrating effect in particular and also the increased inspiratory ventilation effect (second-gas effect) of nitrous oxide would be expected to hasten VIC induction as sevoflurane has a relatively low solubility in blood, but whether these effects and the anaesthetic effect of nitrous oxide itself are useful in sevoflurane VIC induction still need to be ascertained.

We agree that sevoflurane VOC induction used in the manner prescribed by Dr Drummond is likely to be superior to VIC induction, but propose that low-flow sevoflurane VIC anaesthesia with the OMV is a feasible and economical alternative when sevoflurane VOC systems are unavailable.

We agree that end-expiratory apnoea is a more accurate
description of what was observed in our patients. However, the word ‘breath-holding’ is not necessarily limited to the phase of ‘inspiration followed by glottic closure’. It is a good suggestion to turn off the carbon dioxide absorber during induction to reduce the incidence of apnoea. This was not possible with our anaesthetic machines, and in none of the patients was end-tidal carbon dioxide partial pressure less than 4 kPa before apnoea was observed.

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Induction of anaesthesia in the lateral decubitus position in morbidly obese patients

Editor,—Airway management during induction of anaesthesia and tracheal intubation in morbidly obese patients is often difficult because movement of the cervical spine and atlanto-occipital flexion may be limited by numerous layers of fat around the chin. Standard laryngoscope handles may also be obstructed by upper thoracic fat pads in the supine position. In morbidly obese patients, the risk of regurgitation of gastric contents is also high. We speculated that such risks and difficulties may be caused by induction of anaesthesia in the supine position. Thus we investigated if airway management in the lateral decubitus position would be easier in such patients.

After obtaining informed consent and approval from the Ethics Committee, we performed induction of anaesthesia in eight morbidly obese patients (six females) aged 14–30 yr, ASA II–III, weighing 90–160 kg (median 125.5 kg), height 155–172 cm (median 166 cm) and body mass index 35–50 kg m⁻² (median 45 kg m⁻²), undergoing elective surgery. No patient was premedicated. Monitoring during anaesthesia included electrocardiography, arterial pressure and percutaneous oxygen saturation (SpO₂) in addition to end-tidal carbon dioxide concentration (PetCO₂).

Patients positioned themselves easily in the lateral decubitus position (left side up) before induction. Anaesthesia was induced in three patients with halothane and in five patients with sevoflurane in 100% oxygen by face mask. The inspired concentration of each agent was increased by 0.5% every three breaths up to 5% halothane and 7% sevoflurane. After 5–10 min of each inhalation agent with the patient breathing spontaneously, 4% lidocaine 3–4 ml was sprayed onto the oropharynx, epiglottis and trachea. The trachea was then intubated without a neuromuscular blocking drug.

In all cases, the airway was managed easily by neck flexion alone. Using a standard laryngoscope handle and blade, the trachea was easily intubated. During induction, SpO₂ was 98–100% and PetCO₂ immediately after completion of intubation was 5.5–6.4 kPa. There were no adverse events in any patient. After induction of anaesthesia and intubation, three or four anaesthetists and nurses pushed the patient gently into the supine position for surgery. This procedure was done without difficulty in all patients.

Induction of anaesthesia in the lateral decubitus position has several potential benefits for airway management of the morbidly obese patient.

1. Neck flexion can be achieved more easily than in the supine position. The atlanto-occipital joint is more extended so that the oral, pharyngeal and laryngeal axes are brought nearly into a straight line. These conditions provide easy airway maintenance and make direct laryngoscopy easy.

2. The chin can be freed from upper thoracic fat pads because the neck can be flexed almost freely in the lateral decubitus position. Standard laryngoscope handles are not obstructed.

3. The tongue does not move posteriorly even in the unconscious state.

4. The patient’s airway is protected from inhalation of vomit in the lateral position.

The number of patients in our study was small, but our results suggest that in morbid obese patients, the lateral decubitus position can make airway management easier.

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2 Benumof JL. Management of the difficult adult airway, with special emphasis on awake tracheal intubation. Anaesthesia 1991; 75: 1087–110

The electronic anaesthetic logbook and the millennium bug or Y2K problem

Editor—Dr Lee is right to advise caution in the use of electronic logbooks during the 1999–2000 period. Backups of computer files (‘logbook.rea’ in the case of the anaesthetic logbook) should always be maintained as computer system crashes can occur at any time. While most of the year 2000 problems are likely to be hardware and operating system dependent and will need to be independently assessed, there are some that specifically relate to the application software itself which can be minimized by appropriate action now.

The electronic logbook for desktop and laptop computers released by the Royal College of Anaesthetists/SCATA was written using FileMaker Pro v3.05. Dr Lee is incorrect in his assertion that all dates are entered in the form dd/mm/yy. In fact it allows users the convenience of using either four-digit explicit (dd/mm/yyyy) or two-digit assumed (dd/
mm/yy) dates. The potential problems arise from the interpretation of dates entered using the assumed (dd/mm/ yy) format. Trainees should not be concerned as the logbook will handle dates up to the year 3000 and correctly handle the leap years in the year 2000 and beyond.

In general, software programs such as the logbook are less likely to encounter problems when dates have been entered using the full four-figure year notation. Thus when the operating system is set to display four-digit years and all dates are entered into the logbook in the format dd/mm/ yyyy, date-related calculations and functions will perform as expected and there will be no ambiguity or problem with the year 2000. This is by far the easiest solution.

For those who have already entered date directly into the logbook using the short date format (**/**/98, **/**/99 etc.), rest assured that the logbook interprets any two-digit data already present in a database as ‘19xx’. However, perhaps now is the time to start using four figures to identify the year and century for all future entries into data fields that may be used in calculations.

Users who import their cases from the SCATA Psion logbook need not be concerned. The SCATA Psion logbook uses the long date format (dd/mm/yyyy) exported as a text string and is interpreted appropriately when imported to the FileMaker logbook. Both logbooks are therefore millennium compliant provided users recognize the problem of assumed two-digit dates and use the appropriate explicit four-digit dates.

Those wishing to continue to use the two-digit assumed date format, either for direct data entry or by importing it from sources using the short date format, should be aware of the potential problems this may create at the turn of the century. The logbook will interpret newly entered or modified two-digit dates according to a strict set of rules and expand the result to a four-digit date if it is anything other than ‘19xx’. This ensures users will be immediately aware of how the date is being interpreted. Further information on the use of two-digit dates can be found on the FileMaker web site at http://www.filemaker.com/about/ year2000.html.

In order to avoid any problems and subsequent headaches, we strongly recommend that the four-digit explicit date format be used for ALL entries into ALL data fields in the logbook and that a separate backup copy of the ‘logbook.rca’ file is maintained at all times. We have changed the display format of dates in the new version of the logbook, which will be available shortly. This will ensure that users see exactly how the program is interpreting dates entered in either format.

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Editor,—I welcome the response of Drs Hammond and McIndoe and am glad that in the next version of their logbook program any confusion over the entering of dates will be resolved. I am grateful to them for clarifying how dates are stored in the current version of the logbook and will certainly be entering four figure years from now on.

I do hope that provision will be made for those of us who have entered several thousand records in the form dd/ mm/yy to change all existing unspecified records to dd/ mm/yyyy before January 1, 2000. This would solve any potential problem upon upgrade to the new version.

I would also like to acknowledge the time and effort that Drs Hammond and McIndoe are putting into the production of the logbook program that is rapidly becoming the recognized standard for logbook storage in anaesthetics across the country.

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Low-dose nitrous oxide and dyspnoea

Editor,—I read with interest the recent article by Nishino, Isono and Ide1 on the beneficial effects of low-dose nitrous oxide on the sensation of dyspnoea, and the subsequent correspondence by Jones2 concerning the physicochemical properties of that gas. Jones is correct in stating that the viscosity of nitrous oxide is less than that of oxygen, but he goes on to say that gas viscosity decreases with temperature. While this may be true for liquids, the opposite is true for gases.3

The kinetic theory of gases relates gas viscosity coefficient \( \eta \) (eta) to molecular weight, molecular diameter and temperature by the complicated formula:

\[
\eta = \frac{m}{3\sqrt{\pi}} \frac{8kT}{\pi m} \sqrt{\frac{1}{\pi m}}
\]

where \( m \) = molecular weight, \( \sigma \) = collision cross section of the molecule (and is equal to \( \pi d^2 \), where \( d \) is the molecular diameter) and \( T \) = temperature in degrees Kelvin. Thus it can be seen that \( \eta \) is proportion to \( T^2 \), that is gas viscosity increases as temperature increases.

The reason behind the different behaviours of gases and liquids is that they have different frictional mechanisms at the molecular level. While viscosity in liquids is a result of inter-molecular attraction between adjacent molecules, viscosity in gases is caused by molecular collisions leading to the transfer of momentum from faster molecules to slower molecules. An increase in temperature leads to an
increase in molecular collisions and an increase in the transfer of momentum, thus viscosity increases.

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Tears at bedtime

Editor,—I very much support the sentiments offered by Dr Wolf in his editorial ‘Tears at bedtime’, particularly those regarding underestimation of the efficacy of simple analgesics such as paracetamol. However, I would like to add a few points regarding the doses quoted.

He refers to a loading dose of paracetamol of 30–40 mg kg–1, but does not state by which route. I thought it important to clarify this point as very different plasma paracetamol concentrations are attained after the same dose is administered by different routes. The widely used oral dose of 15–20 mg kg–1 achieves therapeutic concentrations but loading doses as large as 40 mg kg–1 orally have been studied in children, with the authors claiming improved analgesic effect.2

However, I assume from reading the reference quoted3 that Dr Wolf is referring to rectal loading doses. The work that exists on rectal loading doses of paracetamol demonstrates that doses of 30 and 35 mg kg–1 are insufficient4 5 and doses of 40 and 45 mg kg–1 achieve therapeutic concentrations in the vast majority of paediatric patients,6 7 with most authors recommending rectal loading doses of 40 mg kg–1 or greater.4 6–8

With regard to maintaining therapeutic concentrations, to my knowledge there are no studies demonstrating that any of these loading doses followed by doses of up to 90 mg kg–1 day–1, by any route, ‘maintain therapeutic concentrations’. Indeed, computer simulation studies have shown that a rectal dose of 15 mg kg–1 4-hourly does not maintain therapeutic concentrations,9 although this was not preceded by a loading dose.

As one of Dr Wolf’s main points was that these analgesics are highly effective but only when used correctly, I thought my comments worthy of note.

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8 Anderson BJ. What we don’t know about paracetamol in children. Paediatr Anaesth 1998; 8: 451–60

Editor,—The editorial by Wolf1 highlights problems with analgesia in paediatric day-case surgery. Children are good candidates for day-case surgery as they generally recover well from intermediate and minor surgical procedures. Furthermore, Campbell, Scaife and Johnstone2 found that children have significantly less psychological and behavioural changes after day-case surgery. Adequate analgesia is one of the key factors involved in the success of day-case surgery for children. Pain has been reported to be one of the major causes of readmission,3 and is associated with a higher incidence of nausea, vomiting and late behavioural problems.4 5

We conducted a telephone survey in 33 hospitals in the UK (11 teaching hospitals, 11 district general hospitals and 11 children’s hospitals) to determine the facilities and analgesic programmes for paediatric day-case surgery. A total of 20 hospitals (60.6%) (eight teaching, four district and eight children’s hospitals) had a paediatric day-case facility, either a day-case unit or a day ward. Another 20 hospitals (60.6%) (six teaching, seven district and seven children’s hospitals) had specific theatre lists for children undergoing day-case surgery; some of these hospitals also had some mixed day-case surgical lists with either adults or inpatients.

Interestingly, only 11 hospitals (33.3%) (three teaching, three district and five children’s hospitals) had guidelines for postoperative pain management. Advice on duration of analgesia ranged considerably between hospitals from 24 h to 7 days or until required. Follow-up in the community was performed by a paediatric district nurse in only six of the 33 hospitals (18.1%) and by a general district nurse in 11 hospitals (33.3%), and 16 hospitals (48.4%) asked the parents to telephone either the hospital ward or their general practitioner if their child had any problems.

The results of this pilot telephone survey show that there
is tremendous room for improvement. There is a need to improve facilities, guidelines and follow-up for day patients in many hospitals. We agree with Wolf on the need for clear, simple instructions. However, these instructions must be consistent between different members of the team (e.g., medical and nursing). The way forward is locally agreed programmes for pain management which include the appropriate drug(s) to be prescribed for each subgroup of patients, and the correct dose, frequency and total duration of drug administration. Information to parents must be backed up by written leaflets and they must have an emergency telephone number. Wolf suggests extra provision for problem cases and extending nursing input from the day unit to the home. However, in this age of reducing numbers of hospital beds and financial constraints, these solutions may not be practical. We reiterate that the simplest and most effective way forward is implementation of local day-case procedures which must include the supply of appropriate analgesics in the hospital and good information for parents on how best to use them.

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2 Campbell IR, Scaife JM, Johnstone JMS. Psychological effects of day case surgery compared with inpatient surgery. Arch Dis Child 1988; 63: 415–17

Editor,—In a recent editorial, the importance of providing suitable and safe analgesia for children undergoing day-case surgery was emphasized. Various forms of regional analgesia are useful for day-case surgery but as Dr Wolf pointed out, their duration is limited to 4–9 h and the availability of preservative-free solutions of ketamine, 0.5 mg kg\(^{-1}\), for example, is also limited. The result is, in the title of your editorial, ‘tears at bedtime’ and an uncomfortable night for the patient and parents.

While the editorial highlighted the efficacy of paracetamol, ketorolac and diclofenac, these agents have the disadvantage that onset of analgesia is delayed after oral administration until adequate serum concentrations and receptor occupancy are achieved.

Recently, tramadol paediatric oral drops have become available in some countries. This centrally and peripherally acting analgesic has mu receptor affinity with few side effects. While not yet licensed in the UK for use in children less than 12 yr of age, several European countries allow its use in children more than 1 yr of age. We recently completed an efficacy–safety study in 60 children aged 4–7 yr undergoing dental extraction of six or more teeth. Tramadol oral drops 1.5 mg kg\(^{-1}\) provided good analgesia with minimal side effects. No respiratory depression was seen, no increased nausea (vs placebo) and no time delay in recovery or discharge home.

A pharmacokinetic study of tramadol at 1.5 mg kg\(^{-1}\) in 24 children aged 4–7 yr, showed that the oral drop formulation provided peak serum concentrations within 30 min. These concentrations remained above the analgesic level of 100 ng ml\(^{-1}\) for 6.8±0.9 h.

For paediatric day-case surgery, tramadol oral drops offer effective analgesia with minimal side effects. The pharmacokinetic pattern of rapidly achieved therapeutic serum concentration should be advantageous as children and parents will not have to suffer the time delay inherent in cessation of regional analgesia and the slower commencement of action of other oral analgesic agents.

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University of Stellenbosch, South Africa
E. A. Shipton
Faculty of Health Sciences
University of the Witwatersrand, South Africa

3 Payne KA, Roelofse JA, Shipton EA. Pharmacokinetics of oral tramadol drops for postoperative pain relief in children aged four to seven years—a pilot study. Eur J Anaesth (submitted)

Editor,—We would like to congratulate Dr Wolf on his excellent and timely editorial entitled ‘Tears at bedtime: a pitfall of extending paediatric day-case surgery without extending analgesia’ and wish to share with your readers our experience from this side of the Atlantic Ocean. Healthcare reforms in this country have exerted pressure on physicians to decrease costs. One way of achieving this goal is to reduce duration of hospital stay. This has resulted in a marked increase in the number of surgical procedures being performed on an ambulatory basis, and paediatric surgery is no exception. We shared the concerns of Dr Wolf and recently undertook a survey to assess pain relief in children after outpatient surgery.

We collected prospectively patient and intraoperative data on 460 consecutive children undergoing outpatient surgery known to be associated with pain and conducted a telephone
interview with a parent or guardian 24 h later. We were particularly interested in the child’s pain relief, use of analgesics and ability of parents to care for their child at home. All surgical subspecialities with the exception of neuro- and cardiothoracic surgery were included. Our results showed that 97% of children had adequate, good or very good pain relief during the first 24 h and 90% of parents reported no difficulty in caring for their child at home. Six parents contacted a healthcare worker because of inadequate pain control, but none of these children required readmission. Poor pain control was associated with genitourinary surgery in children who had not received a regional block. Not surprisingly, we found that parents had greater difficulty in caring for their children if they experienced either poor pain control or postoperative nausea and vomiting (PONV). Postoperative prescriptions are written by the surgical services and we found a wide variation in analgesics prescribed, frequently in recommended doses.

These were generally encouraging results, given the increase in paediatric outpatient surgery, but clearly there is room for improvement. These results emphasize the need for careful parent education regarding the potential for pain and its anticipated severity, likelihood of PONV and management of these problems. In addition, parents must be reassured that advice is a telephone call away. With the potential for more complex surgery being performed on an outpatient basis, continued audit is vital to ensure that there are no ‘tears at bedtime’.

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Editor,—The responses to my editorial add further evidence that postoperative pain after discharge from the paediatric day surgery unit remains a significant problem which needs to be addressed. The severity of the pain is such that if the child were still in hospital rather than at home, active treatment would be used. The solution to this problem is not simple but is an area which presents rich possibilities for multidisciplinary research and development.

The appropriate paracetamol dosing regimen for neonates, infants and children remains a subject for further debate. The excellent review by Anderson1 on paracetamol use in paediatric practice focuses on the issues of pharmacokinetics, pharmacodynamics, drug toxicity and appropriate drug regimens in some detail. A total daily dose of 90 mg kg–1 day–1 appears by consensus to be the maximum, irrespective of whether the drug is given orally or rectally. But cumulative toxicity is a significant but as yet undefined risk in the neonate and therefore a maximum daily dose of less than 90 mg kg–1 day–1 should be adhered to in this age group. Loading doses should be used and these should be given at an appropriate time to ensure adequate plasma concentrations of the drug have been attained before conscious pain perception. Therefore, if a child has had an effective local block at the time of surgery, paracetamol needs to be given after surgery but before the local block has worn off. In contrast, if a local block is inappropriate, it may be necessary to give paracetamol as premedication before surgery. Oral loading doses appear preferable to the rectal route because absorption is more rapid and less variable. The guidelines from the Royal College of Paediatrics and Child Health recommend loading doses of 20 mg kg–1 orally and 30 mg kg–1 rectally (20 mg kg–1 in the neonate). Others have suggested that 30 mg kg–1 orally and 40 mg kg–1 rectally are more effective,2 and oral doses as high as 40 mg kg–1 have been used for children undergoing tonsillectomy.3

New analgesic drugs suitable for use at home in combination with paracetamol and other non-steroidal anti-inflammatory drugs are becoming available. Payne, Roelofse and Shipton found that tramadol is an excellent drug in this respect and I look forward to their forthcoming publications on this subject. However, I disagree with their view that the slow onset of oral paracetamol is necessarily a problem. It is simply a case of anticipation and administration of the drug at the appropriate time.

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1 Anderson BJ. What we don’t know about paracetamol in children. Paediatr Anaesth 1998; 8: 451–60

Evidence-based anaesthetic practice

Editor,—I was interested in the stimulating article by Myles and colleagues1 but I would challenge their conclusion that 96% of anaesthetic interventions are evidence-based. My major criticism is with their definition of evidence. Of the four categories of so-called evidence, only two included information from well-designed, randomized, controlled trials. The authors stated that some anaesthetic interventions can never be supported by such evidence. This is true; although they are the best method of examining the effect of therapeutic interventions, there are situations where other study designs are more appropriate.2 However, a claim that information obtained from other types of experimental studies, reviews of expert committees and opinions of respected authorities, or colleagues, constitutes evidence is precisely the viewpoint that evidence-based medicine
(EBM) seeks to challenge and change. When using such information, the authors point out that ‘it may be that studies can be found that contradict our evidence’ whereas my understanding of EBM is that only when all the relevant published information has been sought out and assessed can a reliable conclusion be reached about a clinical question. As Goodman indicates, many randomized studies are too small to provide definitive answers and ‘where previously cases were chosen to make a point, trials are now chosen the same way’. In their book on how to practice and teach EBM, Sackett and colleagues give specific examples of how expert opinion, using rational knowledge of disease processes and pharmacological mechanisms, can advise therapeutic interventions which are subsequently shown by a randomized, controlled study to be positively harmful. They also make a clear distinction between information and evidence, the latter being a distillation of the former by the process of a systematic search and critical appraisal. I suggest that Myles and colleagues have failed to make this distinction and their conclusion should be that most anaesthetic interventions are not supported by clear evidence of benefit although some are. This is not a way of saying that most such interventions are inappropriate but simply that clear evidence is unavailable.

It is also noticeable from this study that even if the evidence were available, not all anaesthetists are rigorous in their application of it; this criticism applies to myself in addition to the subjects in the study. As examples, in 354 anaesthetics, only one smoker was advised to stop before surgery and only one diabetic patient was given antacid prophylaxis, which implies either a remarkably healthy hospital population or that most anaesthetists ignore what the authors choose to call evidence for these interventions.

Using the book by Sackett and colleagues, I have attempted to incorporate EBM into my practice and have found it difficult and time consuming. Framing an appropriate clinical question based on specific patients and problems is usually simple but performing a literature search, tracking down original articles and attempting a critical appraisal of the results is a slow process, at the end of which no clear answer may have emerged. Being based for most of the working week in the operating theatre, remote from the library or electronic information resources, is a further limitation to the practice of evidence-based anaesthesia but ‘good doctors use both individual clinical expertise and the best available external evidence and neither alone is enough’.4

In summary, I would suggest that while most anaesthetic practice may be informed by research and honed by experience, many of our therapeutic interventions cannot be supported rigorously by explicit evidence and that Myles and colleagues have failed to demonstrate otherwise.

M. Foley
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Editor.—We are pleased that our study of evidence-based anaesthetic practice has generated interest. As Foley points out, the definition of evidence is an important determinant of our findings. We believe that the definition we chose, adapted from the US Preventive Services Task Force, is consistent with that used by others in previous reports from other disciplines. We simply identified and rated the best available evidence using an up-to-date and well established approach. Our approach also allowed readers to form their own conclusions regarding which interventions were evidence-based. For example, some (such as Foley) may choose to accept only level I and II evidence (i.e. that based on results of randomized, controlled trials (RCT)). We took a less stringent, more pragmatic view and accepted lesser levels of evidence, given that contemporary practice should still be guided by laboratory studies, case series and expert clinical opinion. This does not contradict the teachings of evidence-based medicine (EBM), where the aim is to find the latest and best evidence so that the clinician can then consider the applicability of the findings for individual patients. This process demands clinical judgement; some of this is derived from previous experience and consideration of established practices that may never have been subjected to assessment by RCT. This point is acknowledged by Ellis and colleagues from a highly regarded EBM teaching centre, when they chose to use the classification ‘convincing non-experimental evidence’ to represent a lesser level of evidence-based practice.

We agree that interventions based on lesser levels of evidence should be scrutinized closely. Such interventions can also be considered by anaesthesia researchers for evaluation in future clinical studies (to improve the evidence base). This is another positive aspect of EBM. Current and future anaesthetic practice should always be re-evaluated to consider the latest available evidence. As stated in our article, this is a dynamic process which inevitably results in a changing evidence base, and this will continue to guide rational changes in practice.

Regarding the practice of smoking cessation advice and/or diabetes-associated antacid prophylaxis in our article, we asked anaesthetists to identify the most important clinical problem (to them). In other cases, the anaesthetist concerned presumably nominated another (perhaps) more pressing concern. Again, this approach was used in previous studies and we set out to duplicate their approach as much as possible. As stated in our discussion, we felt that multiple
Dealing with expected difficult intubation

Editor,—We read with interest the article by Wakeling, Ody and Ball. They reported a method of dealing with a case of difficult intubation. However, we believe that other considerations should be made.

As they stated ‘patients with large goitres are considered to be more likely to present difficulty at intubation, particularly if the goitre has produced tracheal deviation or has retrosternal extension’. The patient’s chest x-ray showed deviation and narrowing of the trachea. She had prominent incisor teeth and restricted neck extension. Therefore, difficult intubation was not unexpected. The first question to ask is why general anaesthesia was induced, considering that a large goitre, in addition to difficult intubation, can cause airway obstruction when pharyngeal reflexes are abolished. After establishing the degree of intubation difficulty, the authors considered several options, but they did not mention awake intubation. In addition, vecuronium 8 mg was given after induction of general anaesthesia before attempting laryngoscopy. We believe that when difficult intubation is expected and general anaesthesia cannot be avoided, neuromuscular block should be delayed until after laryngoscopy. Also, if intubation has to be facilitated, succinylcholine seems to be more appropriate than vecuronium as it allows more rapid recovery of spontaneous ventilation.

The second question is why they chose a blind intubation technique through the intubating laryngeal mask when they could have used the fibreoptic bronchoscope (FOB) from the beginning. Several attempts were made before using the FOB technique. At the end of the procedure, the laryngeal inlet was swollen where the trachea was intubated. The laryngeal mask airway is part of the ASA algorithm for difficult intubation. However, when the location of the laryngeal mask over the larynx is not central, the chances of successful blind intubation are poor and ‘the unsuccessful (off-centre) insertion of a rigid object through an already off-centre blindly inserted LMA may result in laryngopharyngeal injury’. It appears to us that more attention was given to assessing the usefulness of a device, such as the ILMA, in a particular case, than establishing a robust plan for difficult intubation. We believe that the lesson for next time is that persisting with a strategy which has already failed more than once is not appropriate to deal with expected difficult intubation when other more reliable techniques are available.

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Editor,—I would like to thank Drs Della Puppa and Pittoni for their interest in our case report and appreciate the chance to reply. General anaesthesia was induced carefully and vecuronium was given only after demonstrating easy airway maintenance with a face mask. I do not believe that succinylcholine would have been a better choice because its short duration allows little time for manoeuvre if difficulties arise.

The intubating laryngeal mask (ILMA) was used initially without the fibreoptic bronchoscope (FOB) because it had been reported to be useful in patients with predicted difficult intubation. The tip of the Euromedical ILMA tracheal tube was off-centre blindly inserted LMA may result in laryngopharyngeal injury.

1 Wakeling Hj, Ody, A, Ball A. Large goitre causing difficult intubation and failure to intubate using the intubating laryngeal mask airway: lesson for the next time. Br J Anaesth 1998; 81: 979–81
Correspondence

...laryngoscopy to laryngeal swelling is uncertain as is the degree of oedema caused by the goitre itself.

One of our ‘lessons for next time’ related to putting the tracheal tube through the ILMA to lift up the epiglottic elevating bar before passing the FOB. We were unable to lift the FOB tip anterior enough to see the laryngeal inlet because the epiglottic elevating bar was pushing it caudally. While I agree that persisting with a ‘strategy that has failed more than once’ is not good, continuing with sensible adjustments to a strategy such as changing ILMA size and rotation were fully justified. Drs Della Puppa and Pittoni suggested that we should have used ‘more reliable techniques’. However, with the accumulation of ILMA experience, the reliability of the device in difficult and expected difficult intubation is high (44 of 45 patients (97.7%) successfully intubated1–7). Is there a more reliable technique available for intubating this group of patients?

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Despite vascular anaesthesia being increasingly recognized as a specialty area in its own right with the formation of societies such as the Vascular Anaesthesia Society of Great Britain and Ireland, there are few books written specifically on the subject. Those books which are currently available tend to come from North America and as such do not reflect fully the practice of vascular anaesthesia in the British Isles.

The book is written by three anaesthetists from one vascular centre and aims to provide guidance to trainees in the practice of vascular anaesthesia. It consists of nine chapters, the quality of which is somewhat variable as is often the case with such books. The opening chapter covers cardiovascular physiology and pathophysiology and is reasonably well written, although I am not so sure as to the relevance of the pathophysiology section. The second chapter on the pharmacology of drugs is long. The authors attempted to cover all possible anaesthetic and cardiovascular active drugs which is over ambitious for a chapter in such a small book.

The third chapter covers preoperative assessment. Such a chapter is a cornerstone to any book on vascular anaesthesia. The chapter includes use of cardiovascular risk indices and these are explained well, particularly those by Goldman, Detsky and Eagle. The authors also cover various individual risk factors and discuss further tests aimed at assessing the patient’s cardiovascular status. Within the chapter, a lot of references are quoted, most of which are from North America. However, there is no real sense of giving the reader a true idea of how the information should be used in an individual patient and this is a bit disappointing.

The subsequent chapters in the book are of better quality. Chapter four on monitoring during surgery is excellent and this chapter alone makes the book worth buying for trainees. The ECG is explained in a simple and understandable manner, something which is lacking in many large textbooks of anaesthesia. This should be particularly useful as I have yet to find a trainee who fully understands what he is looking at when using an ECG monitoring system. In addition, invasive monitoring is also well covered and includes sections on transoesophageal echocardiography and oesophageal Doppler.

Chapters five, six, seven and eight cover anaesthesia for carotid endarterectomy, abdominal aortic aneurysm including stenting, lower limb revascularization and miscellaneous vascular operations. The introduction to each chapter gives an overview of each condition and the indicators for surgery. In general, these chapters contain a good overview of the area and valuable advice and tips, particularly to the trainee. There is discussion as to the risks and benefits of regional anaesthesia for each type of surgery. In addition, there is important information on the use of regional anaesthesia in the presence of anticoagulants. However, the amount of space given to the anaesthetic management of emergency aortic surgery is disappointing and the authors also spend some time describing the conduct of epidural–spinal anaesthesia which seems unnecessary. There are some curiosities, for example the authors suggest that an oesophageal stethoscope should be routine monitoring for aortic aneurysm surgery!

The final chapter deals with postoperative care pointing out to the trainee the particular postoperative complications which can occur in such patients and giving some ideas of how these can be avoided in the early postoperative phase.

Overall, I feel that this book is a good attempt by the authors to produce a British book in a recently neglected area of anaesthesia. The chapters, although varying in quality, are generally well written and the book certainly gives a reasonable overview of the current practice of vascular anaesthesia this side of the pond. The book is obviously well researched and there is a lot of information in all areas of vascular anaesthetic practice. I would certainly recommend it to trainee anaesthetists, but more experienced anaesthetists may find it of less value.

N. Edwards


This excellent, readable volume will be of value to all anaesthetists and non-anaesthetic physicians who provide sedation for diagnostic and therapeutic procedures. It will also provide valuable insight into clinical sedation for the trainee in anaesthetics, surgery or medicine.

In the introduction and first chapter there is a highly relevant discussion of the division between conscious sedation and general anaesthesia. Deep sedation, or sedation which results in loss of patient response to command, is indistinguishable from general anaesthesia and should therefore be within the realms of the trained anaesthetist.

Part one of this text provides a comprehensive overview of the pharmacology of currently used oral, parenteral and inhaled drugs to provide anxiolysis, sedation or sedoanalgesia. It includes an excellent in-depth discussion of the pharmacokinetic and pharmacodynamic properties of benzodiazepines, in particular midazolam, the drug
Book reviews

recommended for sedation by non-anaesthetists. For the more discerning, there are concise reviews of the ventilatory responses to benzodiazepines and flumazenil and of drug interactions between benzodiazepines and other sedative agents.

The relevant discussion of the indications for flumazenil, the benzodiazepine antagonist, includes the controversial issues of routine administration after i.v. sedation for ambulatory procedures and treatment of benzodiazepine tolerance. The authors comment that in the former situation, in order to avoid the risk of resedation (or residual sedation), administration of flumazenil is advisable only when low-dose midazolam has been given.

A variety of sedative drugs and techniques are alluded to in the chapter on ‘Techniques for conscious sedation’, including target-controlled infusions and patient-controlled sedation with midazolam, propofol and/or alfentanil, the majority of which are currently only recommended for administration by anaesthetists. Paradoxically, the discussion of i.v. midazolam administration by titration of a bolus dose and associated patient monitoring is relatively limited. I would like to have read more of the use of non-parenteral benzodiazepines, for example oral temazepam, to provide anxiolysis and amnesia.

In the section on procedural safety, it is assumed that the reader has a working knowledge of ASA status and severity of medical illness, in order to determine whether patients should be managed as inpatients, outpatients or even sedated at all! There is an excellent and comprehensive chapter on cardiopulmonary resuscitation but a discussion of how to deal with the more common complications of sedation, such as upper airway obstruction and hypotension, is noted by its absence.

In the second part of this book, non-anaesthetic sedationists were invited to discuss the use of anxiolysis and sedation in their own specialties, namely gastroenterology, urology, cardiology, accident and emergency medicine, neurosurgery, interventional radiology, paediatrics, dentistry, and intensive care. The editors appropriately stress that these physicians have described their personal applications of anxiolysis and sedation, rather than given an evidence-based review of the use of sedation in their individual specialties. By this means the editors have achieved their goal of presenting a wide range of clinical practice in the ‘real world’. This book provides an ideal opportunity for one sedationist to read how workers in a totally different specialty ‘do it’. On reading these chapters, it becomes evident that midazolam is probably the most commonly used drug for sedation in medicine and dentistry in the UK. As a teacher of i.v. sedation, I particularly enjoyed reading the concise chapter on sedation in the accident and emergency department. It will provide a useful overview of the subject for undergraduate and postgraduate students alike.

I would have been interested to read the editors’ views on training and accreditation for the sedationist of the future. They draw attention to the fact that there is neither any legal standard for the provision of sedation nor any formally recognized training scheme in sedo-analgesic techniques for non-anaesthetists, despite a growing demand for their use. Recent legislation from the General Dental Council and the Royal College of Anaesthetists, restricting the provision of general anaesthesia in dental practice, will inevitably increase the demand for alternative sedative techniques in dentistry alone. In other specialties it is clear that, with increasing use of minimally invasive diagnostic and surgical procedures, training of non-anaesthetists in the practice of anxiolysis, sedation and sedo-analgesic techniques is vital for future patient safety and comfort. In the concluding paragraph of the pharmacology chapter, which stresses that accruing pharmacological knowledge of a drug is vital before its administration, I would add that where possible the trainee sedationist should gain supervised expert tuition in the administration of a different sedative drug or technique.

The text format of this book is clearly laid out, although some of the tables are lengthy and in a small font, making them difficult to decipher.

Overall I feel that this book has much to commend it to the non-anaesthetically trained practising sedationist, namely differentiation between conscious sedation and general anaesthesia, the pharmacology of midazolam and potential dangers of sedative drug combinations, the ‘hands-on’ presentation of sedation by non-anaesthetic physicians working in various fields, cardiopulmonary resuscitation and the clear explanation of pulse oximetry. Other areas of discussion, including the use of propofol, target-controlled infusions and sedation of the critically ill, will have greatest application for anaesthetists.

S. Atkinson


There are now several texts on cardiac anaesthesia, particularly from North American authors. Some are both very extensive and very expensive; thus a book such as this, some 283 pages long, including references, needs to be focused and well written in order to succeed. Authors need to concentrate specifically on ‘current issues’ with minimal recycling of old data and old arguments. So how well does this small book succeed in its task?

In my opinion, very well indeed. As in any multi-author book, some chapters are better than others but the standard throughout is high. The first chapter by Weiner and Wiklund describes preoperative assessment of cardiac patients for non-cardiac surgery and concentrates on the recent joint guidelines from the American College of Cardiology and
the American Heart Association. This chapter is informative and well written, and the problem-orientated case discussion at the end is particularly useful, putting the subject into a practical context. It will be interesting to see whether these assessment guidelines from the USA effectively cross the Atlantic Ocean.

The chapter on myocardial ischaemia and infarction during coronary artery bypass surgery is another good chapter; Uday Jain is a well recognized expert in this field. There is a lot of valuable detail on ECG diagnosis, and although data regarding changes in troponin I and T values may be a little light, that is not too surprising given the recent explosion of interest in these biochemical markers.

Davy Cheng is clearly an authority on the subject of early tracheal extubation after cardiac surgery, and his short chapter is excellent, including numerous recent references. This subject is always bedevilled by two problems: how fast is ‘fast track’ management, and are cost savings made in one health care system more widely applicable?

Balser’s chapter on the perioperative management of arrhythmia is my personal choice for the best in the book, but my decision may be biased by my own interests. However, this chapter is particularly valuable in that it combines sections on theory and mechanisms of arrhythmogenesis with a later section on practical management. It also has some excellent diagrams.

Given the large number of elderly patients undergoing procedures under sedation and/or regional analgesia, a review of the effects of sedation and analgesia in heart disease is timely, and Afifi provides a good one. The concluding guidelines for safe conscious sedation are of value. Other notable chapters include Vannier on pulmonary hypertension and Shore-Leserson and Konstadt on blood conservation. The final chapter on digital echocardiography gives us a glimpse of the future: incorporation of echocardiographic images into the patient’s electronic medical record, in addition to better quality images and a more objective and structured method of image analysis.

The Problems in Anesthesia series is published quarterly by Lippincott-Raven, and the cost for a personal yearly subscription outside North America is about $150 for four issues. Single copies, and hence the cost of this single volume, are approximately $60 each. Is it worth it? Personally, I have long subscribed to the series and I think that books such as this serve as a valuable interface between the original peer-review articles and the new editions of the major textbooks which inevitably take longer to produce. Their success depends on the contributors being experts, and on the contributions being timely and relevant. On that basis this book definitely wins.

Who should buy it? There is much more information here than is required for the FRCA, although final FRCA candidates would certainly find some of the chapters valuable. Each cardiac unit should doubtless have one, and many cardiac anaesthetists will prefer to keep a personal copy. Compared with some of the major texts, it provides up-to-date information in an easily accessible format, and represents very good value for money.

R. O. Feneck

A History of Critical Care and Hyperbaric Oxygen Therapy. International Anesthesiology Clinics, Vol. 37, No 1, Winter 1999. T. W. Feeley (editor). Lippincott, Williams and Wilkins, Philadelphia. Pp. 173; indexed; illustrated. This issue of International Anesthesiology Clinics contains reprints of seven articles devoted to intensive care topics. All except one were originally published in the 1960s. Having started training at that time, they give me a feeling of nostalgia for what were exciting days. But they also emphasize a number of vital points that can get overlooked in the even higher technology units of today.

The first article is a classic account by Bjorn Ibsen. It describes his application of basic anaesthetic principles to the management of bulbar poliomyelitis in Copenhagen in 1952 and later to the management of tetanus. There is the plea for the creation of centres for the treatment of respiratory insufficiency. He also makes the important point that therapy has two aspects. One is curative. The second, where intensive care comes in, is supportive. He enunciates the principles of such support very well. He also includes the remarks we still hear ‘Why was the patient allowed to become so ill?’.

The next article, also from 1964, is the proceedings of a session held at the First European Congress of Anaesthesiology in Vienna in 1962. Chaired by Professor Mushin and Dr van Weerden, it reports on the need for ‘assisted respiration units’, on their optimal size and the needs of the units in terms of equipment and staffing. The speakers came from round Europe and gave a fascinating picture of the level of activity at that time. Poliomyelitis was on the decline but barbiturate poisoning was common. Many varieties of ventilators were coming into use and blood-gas analysis was just becoming available. The speakers also realized the need for treating chronic respiratory failure in addition to acute episodes.

Next comes the only review from the 1980s. Severinghaus and Astrup give a comprehensive review of the development of blood-gas analysis to the point where it had become a standard tool in all intensive care units. The initial problems were many. Those of us who had to work some of the earlier analysers will remember them only too well. They also discuss the debates as to what was the best marker of metabolic acidosis or alkalosis. It took time to realize that what happens in the laboratory may be somewhat different from what happens in the body. Today, we accept the reliability of modern analysers and the values they produce. The old days of expecting candidates for the measurement section of the anaesthetic fellowships to understand the problems have gone, probably for the better. We need to spend more time with the patients and less with the analysers.
The next three articles are from 1963 and 1964. Elam reviews the development of cardiac resuscitation. He has a selected historical review, including the introduction of electrical defibrillation and what was then new, external cardiac massage. He describes what we now accept as basic cardiopulmonary resuscitation and the more advanced techniques. The next review is by Guyton and Crowell on the progressive nature of cardiac shock. Guyton was involved with building mathematical models of the circulation with numerous feed-back loops. The review makes the point that while the heart and circulation have considerable reserves, there comes a point where these become inadequate and failure irreversible. The experiments were made in dogs subjected to haemorrhage, pulmonary artery occlusion or injection of microspheres into the coronary circulation. The message is still relevant: treat the condition as early as possible and before it deteriorates too much. Frank then briefly reviewed septic shock. From more experiments in dogs, he concluded that normal resuscitation methods such as volume loading and pressor drugs did not work but that recovery could happen if the ‘liver was perfused with arterial blood from a normal donor, or if appropriate antibiotics were given before shock was induced’. He also advocated sympathetic block of the abdominal viscera. For clinical care, he advocated the continuous presence of well-informed physicians, continuous arterial pressure and central venous pressure monitoring, urine output measurement, blood volume measurement and blood-gas analysis. He claimed that this regimen was producing success. We seem to have added the pulmonary artery catheter. But we still meet the same problems. General support on these lines helps but we have not yet solved the problem of septic shock.

The final paper is from Rendell-Baker and Jacobson in 1965. It reviews the state of hyperbaric oxygenation. They start with a historical introduction, well illustrated with the variety of chambers used in the preceding century. There is a list of conditions where hyperbaric therapy was thought to help, including the successes with carbon monoxide poisoning and gas gangrene. They end with describing the technology available and a list of the dangers of hyperbaric chambers. The idea of hyperbaric oxygen promised much: as Feeley notes ‘it never had its promised future fulfilled’.

Who should read this issue? For the oldies like me, there is much to remind us of the beginning of intensive care and of its promise. For the young, the first paper by Ibsen is a must. His description of the care of a young girl with poliomyelitis makes points that are still relevant today. The other sections give the flavour of what intensive care was like in the 1960s and how the problems were approached. So read it and rediscover the challenges that still face us. Respiratory care has proved itself: what we need now is the curative therapy for heart failure and sepsis.

J. Norman