Mortality of medical oncological patients admitted to intensive care

Editor,—We were interested to read the abstract by Martin, Quintin and Hinds concerning the prognosis of patients admitted to intensive care with life-threatening complications of haematological malignancy.1 We recently conducted an audit on our intensive care unit with similar results.

We audited all patients admitted to the intensive care unit from the Regional Cancer Centre over a 5-yr period (Jan 1993–Dec 1997). All admissions from medical oncology were included. We calculated the mortality in this group and compared it with the background mortality of the unit. We collected data on age, sex, tumour type, duration of stay, mechanical ventilation, extracorporeal renal support, APACHE II score and white cell count. Significant prognostic factors were identified using Fisher’s exact test ($P<0.05$).

Twenty-two patients were identified. Mean age was 46.6 yr, with an equal number of males and females. Haematological malignancies comprised 45% of these admissions; the remainder were solid tumours. The mean APACHE II score was 25 (SD 8.1). The mortality for oncological admissions was 81% compared with a background of 28%, with a relative risk of mortality of 2.9 (95% CI 2.3, 3.6). Neutropenia, renal support and mechanical ventilation were significantly associated with increased mortality (89%, 90% and 93%, respectively, $P<0.0003$), with relative risks of mortality of 3.1 (95% CI 2.4, 4.0), 3.2 (95% CI 2.5, 4.0) and 3.3 (95% CI 2.8, 3.9), respectively. No patient who required IPPV and renal support survived, with a relative risk of mortality of 3.5 (95% CI 3.3, 3.8).

These results confirm the findings in the abstract and in previous studies.2 In view of the very poor prognosis, should patients be given the information required to make an informed decision on whether they wish to have intensive treatment should they become critically ill as a result of their tumour and its treatment?

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factors which have clearly been shown to be associated with a poor short-term outcome relate to progress after admission to intensive care. These include the level of cardiovascular and respiratory support required, failure of the acute illness to resolve, progression to multiple organ failure and persistent neutropenia. As we stated in a recent review... in those with a combination of features associated with a poor outcome it will often be appropriate to limit or withdraw supportive treatment. Once a patient has been admitted to intensive care, the prognosis and treatment strategy should be reassessed at least daily—when making such decisions a consensus must be reached as to the most appropriate plan for management based on an accurate assessment of the prognosis. This should involve close liaison with the oncologists, nursing and support staff, as well as effective and honest communication with the patient and their family and relatives’.

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Sudden airway obstruction following inhalation drug abuse

Editor,—Inhalation drug abuse is a major public health problem world-wide and is on the increase in Britain. We report a case of sudden airway obstruction associated with inhalation drug abuse which required intubation and tracheostomy.

A 37-yr-old white male presented to the accident and emergency (A&E) department disoriented, very agitated and unable to speak. The only history available was from his 15-yr-old son. The patient had been walking along while smoking crack cocaine when he suddenly became breathless, collapsed and ‘stopped breathing’. The attending paramedics confirmed cyanosis and irregular respiration. On examination in the A&E, the patient had a ventilatory frequency of 36 bpm and arterial blood-gas analysis on 100% oxygen revealed: pH 6.99, $PCO_2$ 12.65 kPa, $Po_2$ 44 kPa and base excess −5.2. With the exception of his acute confusional state, other physical examination was unremarkable.

To control his airway and ventilation, and to perform a computerized tomography (CT) scan of the head, the patient was sedated, the trachea intubated and the lungs ventilated. On intubating the larynx only the arytenoid cartilages were seen because of an anteriorly positioned larynx but at this stage no abnormality of the vocal cords was suspected. After intubation and ventilation, blood gases were satisfactory, the CT scan of the head was normal and the patient was transferred to the intensive care unit.

Further questioning revealed a 4-week history of paroxysmal dyspnoea and chest pains. Toxicological analysis was positive for cocaine. On tracheal extubation he immediately developed stridor and upper airway obstruction requiring re-intubation. Microlaryngoscopy revealed normal vocal cord movement but numerous polyps and oedema causing a very narrow laryngeal aperture. Biopsies were obtained and a tracheostomy performed enabling weaning from the ventilator within a few hours. Six days later the polyps had spontaneously resolved leaving a normal larynx. Histological examination of the laryngeal biopsies showed benign polyps and epithelial hyperplasia.

Tracheal polyps after thermal, inhalation injury have been described and the polyposis and epithelial hyperplasia seen in this case could have been caused by chronic tobacco smoking or cocaine. Cardiovascular complications associated with cocaine abuse have been reported and airway complications have been described either as a result of laryngeal burns from inhalation of hot particles of crack or paradoxical vocal cord movement from the local anaesthetic action of cocaine. This could explain the history of paroxysmal dyspnoea in our case.

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Anaesthesia for Caesarean section in severe pulmonary hypertension

Editor.—With regard to the recent case report on anaesthesia for Caesarean section in the presence of severe primary...
pulmonary hypertension. I believe that several points need clarification. O’Hare and colleagues managed the case to the best of their judgement and in the light of available clinical evidence, but the line of treatment cannot be fully justified for the following reasons.

The authors pointed out that the patient’s pulmonary artery pressure was extremely labile and fluctuated markedly with minimal stimulation. But they used alfentanil 1 mg during induction and maintained pain relief throughout the operative procedure with only 50% nitrous oxide. This small dose of alfentanil was probably not sufficient to obtund the stress response to intubation and surgery. To obtund such a response, the patient would have probably needed at least alfentanil 0.3–0.5 mg/kg body weight. Fetal depression after administration of opioid to the mother is not a problem if the paediatrician is informed at the right time. The stress response may have been a contributory factor to the increasing pulmonary artery pressure which could not be controlled with a large dose of GTN.

The use of high-dose GTN also appears to be an exceptional decision which could compromise the systemic circulation and placental perfusion in the presence of coarctation of the aorta. According to the BNF and pharmacology textbooks, GTN is normally infused in the dose range 10–200 µg min⁻¹ or 0.5–1.5 µg kg⁻¹ min⁻¹. When epoprostenol was prepared and a calcium channel blocking drug was available, how could the use of such a high dose of GTN be justified when the authors had decided not to use a regional block to avoid reduction of venous return? Nitric oxide or adenosine would be other choices in these circumstances.

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Editor,—We read with interest the case report of O’Hare and colleagues. They commented on measuring invasive arterial pressure but did not say where they measured it. An upper limb reading would be falsely elevated and give an incorrect impression of uterine perfusion pressure. Systemic arterial pressure may be well maintained in the upper limbs while using vasodilator therapy but be much lower below the coarctation. We disagree with the comment that nitroglycerine has minimal effects on uterine activity. It may be used as a short-acting tocolytic in the presence of severe fetal distress or to abort preterm labour. We understand that this patient presented as an emergency and that transferring the patient would have been extremely hazardous. However, managing these patients in centres with access to nitric oxide therapy and transoesophageal echocardiography has clear benefits when considering the problems encountered in managing this case.

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The intubating laryngeal mask and distorted airway anatomy

Editor,—We read with interest the case report of Wakeling, Ody and Ball on failure to intubate the trachea using the intubating laryngeal mask airway (ILMA) in a patient with an enlarged thyroid and tracheal deviation. The case highlights several important issues on the use of the ILMA, including the requirement for a neutral position of the head and passage of the tracheal tube just beyond the epiglottic elevator bar, lifting it to allow a fibrescopic view of the larynx. Attempted passage of a fibrescope without lifting the rigid epiglottic elevator bar may cause damage to the fibrescope in addition to an obscured view. A learning period of approximately 20 cases has been noted.

Use of the ILMA has been described in more than 850 cases. Of these, 99% involved predicted or actual difficult intubation, of which seven involved use of the ILMA in patients with distorted airway anatomy. In all of these 99 actual or predicted difficult intubations, the ILMA functioned as an effective ventilatory device. The question of blind or fibreoptic aided intubation with the ILMA for the difficult airway has yet to be resolved and it may be that this depends on the cause of the difficult airway.

For patients with distorted airway anatomy, attempted blind placement may cause trauma with swelling, as noted in this case report, which may make subsequent placement
more difficult. Of the seven cases of distorted laryngeal anatomy, five were intubated blind using awake intubation with the ILMA placed under local anaesthetic, one was managed successfully by fiberoptic guided tracheal tube placement via the ILMA and one failure was reported with the ILMA.

Until further experience and clinical studies have evaluated the role of the ILMA in blind placement with distorted laryngeal anatomy, it seems prudent to use the ILMA with a fiberoptic guided technique.

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2 Baskett PJF, Parr MJA, Nolan JP. The intubating laryngeal mask. Results of a multicentre trial with experience of 500 cases. Anaesthesia 1998; 53: 1174–9

Editor,—I would like to thank Drs Patel and Bailey for their interest in our case report. I agree that patients should have their heads in the neutral position as we highlighted in our discussion. They are also correct in emphasizing the importance of distending the EEB without being directed downwards. This results in an obscured view and possible damage from the bronchoscope. This is because the bronchoscope per se is not strong enough to lift the EEB without being directed downwards. This results in an obscured view and possible damage from the bronchoscope. If we had realized this at the time we may have been more successful. This was the seventh time I had used the intubating laryngeal mask airway (ILMA) and so I was still in the 20-case learning period described recently. Drs Patel and Bailey also point out the success of the ILMA as a ventilatory device. In all, 99 described cases of actual or predicted difficult intubation have been ventilated successfully. I have not had difficulty ventilating any patient with the ILMA in my own series which includes rescuing an otherwise ‘can’t intubate—can’t ventilate’ situation. I believe that the ability to oxygenate is one of the most important features of the device whether or not a tracheal tube is placed successfully. There have been no reports of failure to oxygenate with the ILMA to date.

Our patient underwent direct laryngoscopy several times with intubation attempts using a gum-elastic bougie before the ILMA was tried. The ILMA tracheal tube (Euromedical ILM Endotracheal tube, Euromedical) has a particularly soft tip and despite multiple intubation attempts, there was virtually no blood on the ILMA or tracheal tube. It is uncertain to what degree the ILMA contributed to the laryngeal swelling found and how much had been caused by previous intubation attempts or the thyroid goitre itself. We chose not to extubate as a precautionary measure because of laryngeal swelling but also because in the event of postoperative haemorrhage or airway compromise, control of the airway may not have been possible.

Of the seven cases of ILMA use with distorted laryngeal anatomy, our case represents the only failure. We may have been more successful if we had used the tracheal tube through the ILMA before passing the bronchoscope, particularly as intubation was achieved via a laryngeal mask. As more experience has accumulated, distorted laryngeal anatomy does not appear to limit the success of ILMA intubation as much as we thought at the time of our case report. However, I agree with Drs Patel and Bailey that it is prudent to use the ILMA with a fiberoptic guided technique in this patient group, remembering to first pass the tracheal tube to lift the EEB.

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Editor,—We read with interest the case report by Wakeling, Ody and Ball regarding difficult intubation of a patient with a large goitre.1 We wish to report an almost identical case in whom we had to use a different technique to manage intubation.

A 47-yr-old female was undergoing total thyroidectomy. There was no significant past medical history; she had had no previous operations and she was euthyroid. She was not receiving any medications. She had no breathing difficulty, stridor or hoarseness. Examination of the airway showed good mouth opening with Mallampati grade I. There was no limitation of neck–head movement. There was a large mass in the right side of the neck pushing the larynx towards the left and CXR showed tracheal deviation to the left without narrowing. She was premedicated with temazepam 10 mg, 2 h before operation. In case of difficult intubation, propofol 200 mg was given for induction followed by succinylcholine 75 mg. During laryngoscopy, the larynx was found to be pushed over and fixed to the left and there was a space of approximately 3 mm around the epiglottis. It was impossible to lift the epiglottis up and the vocal cords could not be seen. A laryngeal mask (LMA) was inserted and when spontaneous breathing returned, anaesthesia was maintained with isoflurane in an oxygen–nitrous oxide mixture. There was no problem with the airway using the LMA. After removing the LMA, fibrescopic intubation was attempted but we could not get under the epiglottis to see the cords. The LMA was reinserted and the fibrescope was passed through the LMA and into the trachea without difficulty. A gum-elastic bougie was inserted into the trachea along the fibrescope and the position was checked. We then removed the fibrescope and LMA leaving the bougie in place. The trachea was now intubated easily with a size 8.5-mm tracheal tube over the gum-elastic bougie. The operation was performed successfully but during extubation we found no difference in the position of the larynx. The vocal cord could not be seen. The postoperative period was uneventful and the patient and her general practitioner were informed about the difficult intubation on this occasion.

We had no intubating laryngeal mask airway available at this time. We would like to stress that the fibreoptic bronchoscope can be helpful in placing the bougie in the trachea.

L. Hollos
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Editor,—I would like to thank Drs Hollos and Bhala for their interest in our case report. There are many similarities between the case they describe and our own. It is interesting that despite the larynx being fixed laterally and the inability to lift up the epiglottis, the laryngeal mask airway gave a clear airway and directed the fibreoptic bronchoscope to the cords when they could not be found with the bronchoscope alone. This ability of the LMA has been recognized by the American Society of Anesthesiologists and it appears as the fibreoptic conduit of choice in their difficult airway algorithm.1 Placement of the gum-elastic bougie under bronchoscopic control in this case overcame the shortcomings of intubation through the LMA, that is the need for a long and relatively narrow tracheal tube. I suspect that the intubating laryngeal mask would have been successful in their patient as the larynx was clearly seen through the LMA. The ILMA allows a 8.0-mm internal diameter tracheal tube to be placed through it which would make exchanging a narrow tracheal tube (as in our case) or first placing a gum-elastic bougie into the trachea under fibrescopic control unnecessary. The technique described by Drs Hollos and Bhala is clearly useful when an ILMA is not available.

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1 Benumof JL. Laryngeal mask airway and the ASA difficult airway algorithm. Anesthesiology 1996; 84: 686–99

Effect of nitrous oxide on dyspnoea
Editor,—In their interesting observations on the favourable influence of low-dose nitrous oxide inhalation on the discomfort indices of dyspnoeic subjects, Nishino, Isono and Ide1 advanced four possible explanations. However, they have overlooked a fifth, much simpler but more likely possibility.

Viscosity is one of the general physical properties of gases. Viscosity changes with temperature, decreasing as the temperature of the gas increases. In the temperature range 0–150 °C, the curves expressing this relationship are linear. Those for nitrous oxide and oxygen are parallel but widely separated. At 25 °C, the viscosity of oxygen is considerably greater than that of nitrous oxide (of the order of 39%).2 Added to an inhaled atmosphere of oxygen, nitrous oxide will act as a ‘thinner’, making the passage of the mixture through the airways both easier and quicker.

I think that the authors should factor this physical reality into any speculation on the possible causes of their observed effect.

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Editor,—We appreciate Dr Jones’ interest in our article. He raises important issues concerning the effect of gas viscosity on dyspnéeic sensation. As pointed out by Dr Jones, nitrous oxide is less viscous than oxygen. However, this does not necessarily mean that inhalation of a mixture of oxygen with nitrous oxide causes a decrease in airway resistance to breathing, as viscosity is a property of gas which only influences resistance during laminar flow. In addition, in our experiments, subjects breathed only 20% nitrous oxide, and it is unlikely that such a low concentration would cause a great decrease in resistance even during laminar flow. Furthermore, gas flow is largely turbulent in clinical settings when there is difficulty in breathing, and density is more important than viscosity under such conditions. Considering that the density of nitrous oxide is higher than oxygen, inhalation of nitrous oxide may aggravate difficulty in breathing. However, our results showed that inhalation of nitrous oxide reduces dyspnéeic sensation. On balance, we believe that the physical properties of nitrous oxide exert little influence on our observations.

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Inadvertent inhalation anaesthesia during surgery under retrobulbar eye block

Editor,—We read the case report of Smith1 and wish to suggest an alternative method of avoiding inadvertent inhalation anaesthesia. This method was first suggested to us by Miss Helen Seward of the Mayday Hospital, Croydon, and has been our routine practice for at least 5 yr. We simply avoid the use of piped gas and instead cut the surgical drape in such a way that patients undergoing local anaesthesia for eye operations are allowed to breathe room air. Some experimentation with each individual style of drape is required, particularly as to the positioning of the slit which is performed in the drape.

To date, we have found no significant difference in the (low) rate of endophthalmitis among our patients compared with colleagues in the same unit who favour the more traditional ‘oxygen tent’ arrangement of draping for eye procedures. Figure 1 shows the arrangement of the drape.

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Postoperative cognitive dysfunction in the elderly surgical patient

Editor,—Postoperative cognitive dysfunction in the elderly has been discussed widely in the past year.1-3 Hovorka4 has shown that in patients more than 60 yr of age, \( P_{a\text{CO}_2} \) during general anaesthesia is inversely related to duration of recovery from anaesthesia.5 Regardless of age, patients subjected to hypercapnia (mean \( P_{a\text{CO}_2} = 7.3 \) kPa) scored better in recovery tests than patients subjected to normo- (\( P_{a\text{CO}_2} = 5.3 \) kPa) or hypocapnia (\( P_{a\text{CO}_2} = 3.7 \) kPa).5 Even in the younger group (<49 yr), hypocapnia resulted in nearly the same deterioration in scoring in the recovery tests, which in some patients persisted for up to 48 h after operation. Normocapnic ventilation gave better results than hypocapnic ventilation. The proposed mechanism was preservation of normal cerebral circulation with normo- or hypercapnia. Over a wide range, cerebral blood flow is linearly correlated with \( P_{a\text{CO}_2} \),6 and it also decreases with advancing age. As reactivity to carbon dioxide may be diminished in the elderly, hypocapnia in these patients can reduce blood flow to the ischaemic threshold.7
In the study of Møller and colleagues, ‘capnography was required to ensure normocapnia’. There may be large inter-individual differences between \( \varepsilon \text{CO}_2 \) and \( P_{a\text{CO}_2} \), although the gradient is fairly constant during one session in an individual patient.

Thus for research, at least one end-tidal to arterial gradient should be known for each subject. Additionally, ‘normal’ \( P_{a\text{CO}_2} \) is an individual value and differs with age, gender and pathophysiological factors; it is also influenced by the inspired oxygen concentration.

Textbook values are 4.27–6 kPa for females and 4.67–6.4 kPa for males. As the cerebral circulation is extremely sensitive to arterial carbon dioxide tension, we would expect that in some patients, lowering of end-tidal carbon dioxide may have been associated with cerebral vasoconstriction and reduced perfusion in some areas of the brain.

There is still an unfortunate habit in many centres of moderately hyperventilating all patients in order to save on the use of anaesthetics and neuromuscular blocking agents. We recommend anybody to test their ‘normal’ \( \varepsilon \text{CO}_2 \), using a capnometer to determine the effects of slight hyperventilation (to 4.5%) on subjective feelings.

The concept of ‘permissive hypercapnia’ has demonstrated the safety of high carbon dioxide concentrations during anaesthesia and intensive care. Because it is very easy to adjust ventilation to a fixed \( \varepsilon \text{CO}_2 \), we see no reason why the possible effect of low carbon dioxide tension on brain circulation could not be avoided in future research on the effects of anaesthesia on cognitive dysfunction.

We would suggest that the significant influence of the different centres in the study of Møller and colleagues on the incidence of postoperative cognitive dysfunction was caused by different levels of \( P_{a\text{CO}_2} \) during general anaesthesia as a result of different perceptions of ‘normocapnia’ in the participating centres.

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11 Dundee JW. Influence of controlled respiration on dosage of thiopentone and d-tubocurarine chloride required for abdominal surgery. BMJ 1952: 2: 893

Editor.—The issue of the association of cerebral blood flow responsiveness with hyperventilation, and by inference the possible effects on cognitive function, is interesting but possibly tenuous.

The article by Brian clearly identifies that during anaesthesia, a decrease in arterial carbon dioxide tension during hyperventilation reduces cerebral blood flow. However, this decrease is within the normal range seen in awake subjects in the majority of cases. The concept of permissive hypercapnia clearly exists in any anaesthetized patient breathing spontaneously with a potent inhalation anaesthetic, and the decreased carbon dioxide responsiveness often leads to an end-tidal carbon dioxide value of 7–8 kPa. But the incidence of postoperative cognitive deficit in patients who are ventilated artificially and those who are not, does not appear to differ in studies that have compared the two.

While at present it is impossible to be dogmatic, what evidence there is to implicate hypocapnia as a cause of postoperative cognitive deficit, as opposed to delayed recovery, is limited. However, I do agree that there is no indication for routine hyperventilation, especially in the elderly.

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Logbooks and the ‘millennium bug’

Editor,—As interest in the approaching change of millennium increases, there has been widespread speculation as to the extent that the ‘millennium bug’ is going to affect our everyday lives. Some are worried that as midnight passes we may lose electricity, telephone systems or even the balance in our bank accounts. There is, however, one problem that may affect anaesthetists to a greater degree than those in other specialities, namely the loss of data kept as part of a training logbook. With a large percentage of trainee anaesthetic staff now keeping their logbook in electronic form, either on a PC or hand-held computer such as a Psion, it is likely that at least a few anaesthetists will find their data inaccessible in the year 2000.

Any computer containing a motherboard older than 3 or 4 yr is at risk of having fundamental hardware problems and may refuse to boot up at all in the New Year. Even if the computer does boot up, it should be noted that Microsoft have recently highlighted at least 10 problems that even Windows 98 (previously thought to be completely ‘millennium bug’ proof) may encounter as the change of date occurs.1 How operating systems on older PCs and hand-held computers will fare remains to be seen.

Assuming the computer and operating system can cope, one then needs to ensure the integrity of whichever logbook program is being used. Indeed, if one looks at the PC version of the College approved logbook by Hammond and McIndoe,2 it becomes clear that all dates are entered in the form dd/mm/yy, ignoring the century. A workaround in this case enables the ages of patients to be entered separately from their date of birth. Other dates collected by this program remain unclassified and problems may arise in the future if one wishes to analyse these data.

I would therefore like to urge caution among those keeping their logbooks in electronic form and would suggest that they print a paper copy of their data before leaving the house to attend that infamous New Year’s Eve party!

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1 McCauley J. The countdown has begun. PC Format 1999; 92: 6–7
Book reviews


In the syllabus for the Primary FRCA, tucked between the g-protein and Sherringtonian physiology, exists a Stygian backwater, obscure, murky and seldom visited: this is clinical measurement, in particular the principles of clinical measurement. When the old Part 3 of the Primary Examination was extant, candidates in 1979 were expected to write an answer to the question: ‘Distinguish between random and systematic errors. Give an account of how these may be minimized in any system of measurement’.

Even earlier, written answers in essay form to questions on statistics were required. In the intervening period, corporate amnesia seems to have obliterated such knowledge from the minds of many candidates and teachers. It may be argued that one of the main reasons for this is that no anaesthetist writes authoritatively on the subject, despite the fact that a good proportion of medical students possess a proven ability in mathematics to ‘A’ level. However, an anaesthetic Charon has arrived, belatedly, to row us across the Styx.

Dr Cruickshank divides his text into three parts. The first concerns physiological and pharmacological modelling, dealing with such diverse subjects as alveolar ventilation, the Fick principle and drug–receptor interaction. The second part gives a mathematical background directly answering many of the subjects in the less-often visited parts of the current syllabus, moving from a basic treatment of numbers, dimensions and units to exponential, logarithmic and sinusoidal functions. The final part deals in some depth with probability and statistics.

The book has been most attractively designed and printed. The often difficult (to me) contents are copiously illustrated with line diagrams and cartoons. The text, which in this type of book can be immensely turgid, is broken into bite-sized sections and cleverly boxed. The mathematics, although difficult to those trained in an earlier era, is presented simply and 5–10 min spent on a section will usually promote sufficient recall of memory, painfully achieved some 35 yrs ago, to tackle the various subjects. Anaesthetists with much more recently acquired knowledge of mathematics, to a higher level, should have few problems. One advantage of writing this type of book is that errors are likely to go undiscovered by the vast majority of readers.

Most of us set the task of writing such a book would act merely as editor and farm out individual topics to non-medical co-authors. This book has been written by a single, practising, anaesthetist; it is a tour de force. I have frequently toyed with the idea of writing a book called something like: Anaesthesia: the tricky bits. I no longer need to think about it. Dr Cruickshank has done it in trumps. Taking, as he does, subjects from all over the anaesthetic syllabus and drawing them into a single text, he has single-handedly advanced this aspect of the science as opposed to the art of anaesthesia. I think he should write another book taking the topics he has covered here and deal with them rather more simply. This would allow all anaesthetists, not particularly examination candidates, to visit the subject without fear. The current text would become a teacher’s text and act as a target for all. This book should be owned by all teachers of subjects in the Primary syllabus. Whatever is being taught will be covered in some aspects by this book. It should be present in all anaesthetic departments and candidates wishing to understand rather than just learn the subjects should also have it in their possession. It is a remarkable work.

D. Saunders