

Correspondence

Is excess intensive care mortality in the United Kingdom concealed by ICU mortality prediction models?

The comparative performance of intensive care units (ICUs) is measured with casemix adjustment systems such as APACHE, SAPS and MPM [1]. These systems calculate predicted hospital mortality based upon reason for ICU admission, degree of physiological derangement, chronic health status, age and medical intervention. Predicted hospital mortality is calculated using data collected shortly before and after ICU admission [1].

There are considerable limitations to casemix adjustment systems [2, 3]. However, if the standardised mortality ratio (SMR; observed mortality/predicted hospital mortality) is taken as an indicator of the effectiveness of ICU treatment, outcome is not clearly worse in the United Kingdom compared with elsewhere [4–9]. If the average predicted hospital mortality of admissions, rather than the SMR, is used to compare ICUs then large differences between countries emerge.

In the North Thames region of the United Kingdom a group of ICUs contribute information to a database [10]. The average predicted hospital mortality by APACHE II [11] for 12 762 patients from 15 ICUs in this database is 28.6%. Another British ICU database reports a predicted hospital mortality of 27.2% [4]. In other countries average predicted mortality is generally lower, for example 19.8% [5], 18.8% and 15.1% in the United States [12]. Out of 37 ICUs in

the United States, four reported an average predicted hospital mortality of more than 25% [12] whereas only two of 15 ICUs in North Thames had a predicted mortality less than 25%. Data from over 13 000 ICU admissions in the United Kingdom, eight other European countries and North America showed that the British hospital mortality for these patients was highest at 32.4%, compared with a median of 21% for the other European countries and 19.7% for North America [13]. Preliminary data from the European Consortium for Intensive Care Data (ECICD) using SAPS II to assess severity of illness show that intensive care patients in the United Kingdom are sicker than any of the eight other participating countries (ECICD abstract, not published). A Canadian study reported a predicted hospital mortality of 24.7% [6] and one of 20% was calculated for a group of Brazilian ICUs [7].

Patients already in hospital account for a high proportion of high-risk ICU admissions. In our data, ICU admissions from the ward are 21.7% of total admissions. These have a 52.9% hospital mortality (1466 deaths), compared with 22.3% (1156 deaths) of those admitted from the operating theatre/recovery and 30.2% (1081 deaths) from the accident and emergency department. Of patients admitted to ICUs following external cardiac massage or defibrillation, 42.9% (677 patients) came from the ward. These patients had a 79.5% mortality. In our own hospital 34.8% of ICU admissions of patients who had been in hospital at least 24 h were following a respiratory or cardiac arrest on the ward

[14]. In 1996 there were 142 cardiac arrest calls to the wards following which 33 patients (23%) were admitted to the ICU.

Our research [14] and that of others [15] suggests that it is possible to identify early those ward patients likely to require ICU admission or suffer a cardiac arrest. Early recognition of these patients may allow management to prevent deterioration in physiological values or to prevent arrest. Such intervention is likely to improve outcome. The incidence of cardiac arrest on the ward may therefore be a useful indicator of the quality of care. Compared with the United Kingdom, in some other countries a higher percentage of resources is given to caring for critically ill patients [16, 17] and ICU admissions have a lower average predicted mortality suggesting that patients are likely to have access to appropriate care earlier. If patients are identified early and admitted to the ICU, in particular before a respiratory or cardiac arrest, their predicted mortality will be less but, given appropriate ICU treatment, so will the observed mortality. There will be no difference in the SMRs and no indication of the improved ICU outcome.

The relative lack of critical care resources and the high predicted mortality of patients admitted to British ICUs point to the possibility of an excess mortality compared with better resourced medical systems. Early identification of critically ill patients may help improve care for these patients on the ward or facilitate early admission to an appropriate high-dependency area or ICU. This is likely to decrease the number of deaths

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without altering the SMR. The use of casemix adjustment systems to compare ICU performance will conceal rather than reveal this excess mortality.

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Early death amongst anaesthetists: a statistical howler

Milner and Ziegler stated recently in a letter to *Anaesthesia* (1997; **52**: 797–8) that 'In a review of the obituaries published in the *British Medical Journal*, Wright and Roberts have shown the average age of death . . . of anaesthetists . . . [to be] the lowest of all doctors born in the UK. [This] . . . gives sobering thought to the profession.' Sober thought is indeed required, but before immediately jumping to the conclusion that this difference is due to the stress under which anaesthetists work, it is necessary to ask whether the conclusions of Wright and Roberts [1] are themselves valid. In fact the Editor of the *British Medical Journal* subsequently admitted that the journal had committed a 'statistical howler' and said that 'many people wrote to point out the error' [2] and published two letters [3, 4], explaining the well-known statistical problem, which is technically the absence of valid denominators.

The problem is easily stated. There is no doubt from the data of Wright and Roberts that obituaries of anaesthetists report a younger age at death than that for other doctors. However, this might result from anaesthetists also being younger in life than other doctors, because being an anaesthetist is a marker of being from a later birth cohort. Anaesthetics is one of the younger medical specialities, having developed substantially in the past four or five decades and indeed its practitioners tend to be somewhat younger (as shown in an analysis of 128 417 doctors on the Medical Register [5]). This probably explains entirely the results of Wright and Roberts.

Anaesthetists may be under more stress than other doctors, which may be deleterious for their health, but such a case will not be helped by quoting data which are statistically flawed. Ironically the Editorial in the same issue of *Anaesthesia* noted, 'it is essential that anyone using a statistical technique is completely familiar with it' [6].

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Self-administration of pre-operative analgesic suppositories

I aim to tell all patients pre-operatively about my plans for postoperative analgesia, especially since an anaesthetist was found guilty of serious professional misconduct for inserting a suppository into an anaesthetised patient without obtaining prior consent [1]. Occasionally I realise after induction that I have omitted to mention the suppository. This presents a dilemma: do I insert it anyway because I believe it provides good analgesia (paternalistic attitude?) or sacrifice this quality of care in respecting the patient's autonomy?

Recently, after discussion with the nurses on the day care unit, I have offered day-case gynaecology patients pre-operative self-administration of analgesic suppositories. This has several advantages: the need to obtain specific consent is avoided; the insertion of suppositories is less likely to be overlooked in the rapid turn-around of patients in the operating theatre; patients participate in their own care; analgesia is provided earlier (utilising any pre-emptive effect). The patients have readily accepted the practice of inserting their own

suppositories in spite of an anonymous questionnaire finding that 50% of female day-case patients considered rectal drug administration unacceptable [2]. The very few who balk at the idea of self-administration are offered the choice of rectal analgesia inserted under anaesthesia (for which consent is then obtained) or post-operative oral medication. The additional work for the ward nurses pre-operatively (in dispensing the suppositories and supervising their administration) is compensated by better postoperative analgesia and more rapid discharge home.

This change in practice avoids inserting suppositories into the large majority of unconscious patients and has been well accepted by all involved. Others might consider adopting it.

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Combined introducer for reinforced laryngeal mask airway

Since the introduction of the reinforced laryngeal mask airway (RLMA), many devices have been described to facilitate its insertion. These include a variation of the standard Magill forceps [1], a metal stylet [2, 3], a small tracheal tube [4] and the Bosworth introducer [5]. Disadvantages of these devices include trauma to the larynx with the forceps and metal stylet, displacement of the mask on removal of the stylet and reduced tactile feedback with the Bosworth introducer. With a tracheal tube alone as an introducer we find that it lacks the rigidity necessary for the insertion of the RLMA, which has a tendency to rotate along its longitudinal axis. With these considerations in mind we wish to describe an introducer combining the advantages of a metal stylet and tracheal tube.

The tracheal tube used is essentially of the same length and diameter as described by Asai *et al.* [4]. A metal stylet is then passed through the tracheal tube and the length adjusted so that the distal end does not protrude through the grill of

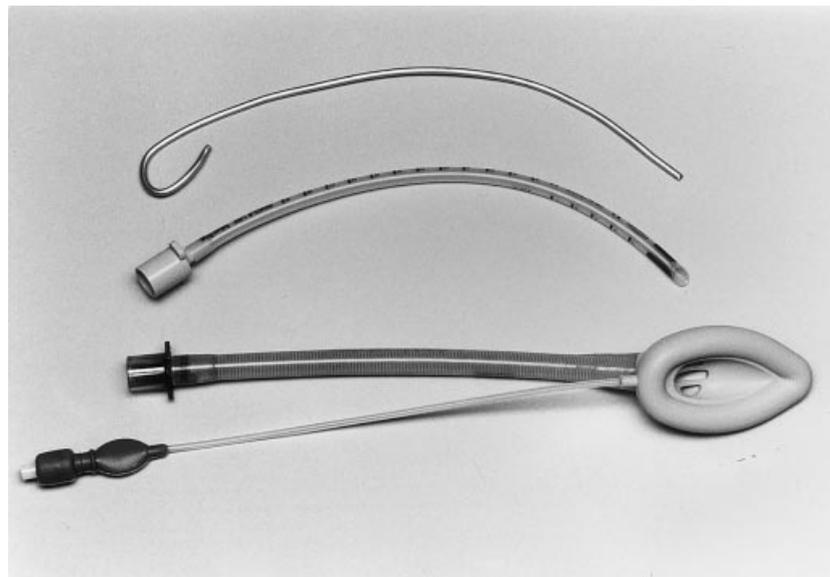


Figure 1

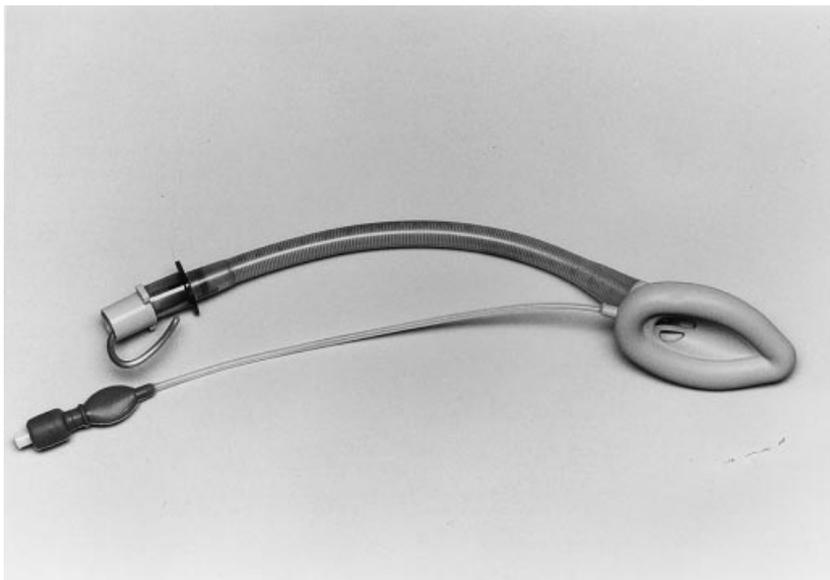


Figure 2

the mask (Figs 1 and 2). This arrangement allows the shaft of the RLMA to be rigid enough to be precurved in order to mirror the oropharynx, thereby making insertion easy and atraumatic. Adequate lubrication of the stylet and tracheal tube allows the removal of the stylet without displacing the RLMA. This is easily achieved by holding the junction of the tube and mask when pulling back the stylet. We feel that this combined approach minimises the potential disadvantages of the stylet and a tracheal tube used alone as introducers for the RLMA yet retaining their individual advantages.

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Cuff inflator for tracheal tubes

The ethos behind the work of Drs Abdelatti and Kamath (*Anaesthesia* 1997; **52**: 765–9) in developing a device for cuff inflation of tracheal tubes is to be commended in principle. The identification of clinical need, the ingenious development of apparatus and the meticulous, accurate testing of new devices has been the cornerstone upon which much of modern-day anaesthetic practice has been established. In their article Drs Abdelatti and Kamath highlight the difficulties that a right-handed assistant may have in inflating a tracheal cuff with their nondominant hand whilst applying cricoid pressure with the other. In addition they list the properties that the 'ideal' cuff inflator device should possess, including 'rapid and consistent inflation of the cuff to produce an air tight seal, measurement of the injected volume of air and safety precautions to prevent over inflation of the cuff.' It has obviously

escaped the authors notice that connection of the syringe to the tracheal cuff inflator *before* the rapid sequence induction has been embarked on will eliminate the assistant's need to carry out this dextrous manoeuvre under stress. Furthermore the preconnected syringe will fulfil all the authors' own criteria for an 'ideal' tracheal cuff inflator device.

Is it really necessary to complicate what is essentially a simple procedure by the development of yet more obsolete anaesthetic equipment? There is an expression about mountains and mole-hills that springs to mind when considering the efforts of Drs Abdelatti and Kamath.

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A reply

Thank you for the opportunity to reply. We would like to reassure Dr Francis that only after using/studying all different options available over the years, was it decided that there was a need for an automatic cuff-inflator. The recent suggestion that 'cricoid pressure' should be performed as a 'two-handed' procedure by the anaesthetic assistant further supports our contention [1]. We would also like to clarify that designing the inflator was only the first stage of development and the facility to measure/regulate cuff pressure is being incorporated presently, to fulfil all the criteria for an ideal device as outlined in our paper. When completed, the device will be left connected to the tracheal tube in the peri-operative period, including during long-term ventilation, allowing continuous regulation of cuff pressure. In present-day anaesthesia where all aspects of patient management are routinely monitored, we believe that we are obliged to devote more attention to one of the essential and sometimes crucial parts of anaesthetic practice, namely tracheal intubation [2].

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Use of a tracheal tube and capnograph for insertion of a feeding tube

A feeding tube can be inadvertently inserted into the tracheobronchial tree and may even penetrate the pleura, causing pneumothorax [1, 2]. In demented or unconscious patients, insertion of the tube into the trachea sometimes does not induce protective airway reflexes and thus the incident may not be detected [1, 2]. Infusion of nutrients through the tube in such a circumstance may be lethal [3]. Therefore, it is crucial to detect inadvertent insertion of a feeding tube in the airway.

Previously, I suggested that the use of a capnograph would be one reliable method for detection [4]: after insertion of a feeding tube, a capnograph is connected to the proximal orifice of the feeding tube; if carbon dioxide is detected, the tube is likely to be in the airway. It is possible to minimise the incidence of migration of the tube into the intrapleural space if the absence of carbon dioxide output is confirmed at the point when the feeding tube has been inserted to a depth of 25 cm. However, this method requires removal of the guide wire from the feeding tube for the measurement of carbon dioxide and re-insertion of the wire for final positioning of the tube into the gastrointestinal tract. This re-insertion of the guide wire may damage the tube or may result in the wire protruding from a side hole of the feeding tube [5].

I now suggest that the use of a tracheal tube will solve this problem. A tracheal tube is inserted into the oesophagus and a sampling catheter of a capnograph is connected to the tracheal tube. When the tube is correctly inserted into the oesophagus, there should be no increase

in carbon dioxide concentration. After this confirmation, a feeding tube is passed through the tracheal tube into the oesophagus and advanced into the gastrointestinal tract. The tracheal tube is then removed. Finally, the guide wire is removed, a capnograph is connected to the feeding tube and the absence of carbon dioxide emission is confirmed.

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Testing regional anaesthesia before Caesarean section

The paper by Bourne *et al.* (*Anaesthesia* 1997; **52**: 896–903) on how the British obstetric anaesthetist tests regional anaesthesia before Caesarean section is timely and appropriate since regional anaesthesia obviously accounts for the majority of anaesthetics for the procedure.

Their figure of 9/555 (essentially obstetric anaesthetists) who preferred general anaesthesia for Caesarean section is encouraging since at the Guildford Obstetric Anaesthetists' Association (OAA) meeting (April 1997) the interactive voting system enabled the 330 anaesthetists in the audience to indicate

their preferred technique for elective Caesarean section: 9% preferred a general anaesthetic, 10% an epidural, 23% a combined spinal/epidural (CSE) and 58% a one-shot spinal. These latter figures should not be compared directly with those provided by Bourne *et al.* since the composition of the audience was unknown, but was probably a mixture of specialist and nonspecialist anaesthetists.

As well as the variation in the recommended level of block for Caesarean section found in textbooks and the lack of description as to which sensation the particular level of recommended block refers, there is a practical confusion about the level of block recorded. Working in a cephalad direction with the patient instructed 'tell me when you feel me touch you' is the level of block the first unblocked dermatome or the last blocked dermatome: the patient's response may indicate touch sensation is appreciated at the T4 dermatome, but is this a block to T4 or T5? This block could be correctly described as a 'block to (i.e. up to) T4' or 'the upper level of block is T5'. It was for this reason that my description [1] of the level of block required for a pain-free Caesarean section was described as 'up to and including T5', i.e. the first unblocked dermatome was T4. It must also be remembered that my results were obtained before the use of spinal and epidural opioids. Clinical experience and the results of many studies indicate that a pain-free Caesarean section is significantly more likely when fentanyl is added to the local anaesthetic (particularly with epidural anaesthesia), although the level of block assessed in the fentanyl and nonfentanyl groups may be the same (i.e. the required level of block for a pain-free section when fentanyl is added is lower than that without fentanyl).

If assessment of the upper level of block is confused, then this is even more so for the lower level. While we all accept that missed segments may occur with epidural anaesthesia it is not true to say 'it is clinically recognised that spinal anaesthesia guarantees complete sensory loss below the most cephalad level'. This will depend on how the block is assessed: if the block is assessed by cold or sharp pin

prick then there may well be significant other sensations below the most cephalad level.

Even with a spinal, many patients are aware of pulling sensations during Caesarean section (although some of these sensations may travel by extraspinal routes) and it is not uncommon to perform a pain-free Caesarean section on a patient who can still move her toes. A retired anaesthetist (E. H. (Ted) Morgan, Australia) drew my attention to the fact that with spinal cinchocaine, it was often very difficult, or impossible, to operate on the sole of the foot (this is confirmed by several of my colleagues with experience in Asia). Over the last few months I have checked the soles of my patients' feet during their Caesarean sections and have found that over 90% can appreciate touch on the soles of both feet! VAS scores for intra-operative pain or discomfort were zero.

My final comment is one of consternation. After Bourne *et al.* discuss the implications of the differential block (between cold, sharp pin prick and touch) and describe how cold and pin prick may be misleading in assessing the adequacy of the block, I am mystified to note the authors' comment 'in our department we use ethyl chloride spray'. Apart from questions about what ethyl chloride is assessing there are also problems associated with environmental pollution by an inflammable chlorinated hydrocarbon.

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Cricoid pressure in chaos

In their letter Brimacombe and Berry (*Anaesthesia* 1997; **52**: 924–6) raise some interesting but controversial issues. The studies that have shown cricoid pressure to be potentially harmful have all applied

excessive force. Forces of 40 N and over can cause: airway obstruction [1, 2], difficulty in passing the tracheal tube [3] and oesophageal rupture [4]. Also, when excessive force is used the view at laryngoscopy has to be improved with neck support [5] presumably because of the head flexing on the neck, but with 30 N of cricoid pressure neck support is not needed; indeed the view is better than without cricoid pressure [6]. It is surprising that these excessive forces continue to be used when 30 N has been shown to prevent the regurgitation of oesophageal fluid with a pressure of 40 mmHg in 10 cadavers [4]. Gastric pressures over 25 mmHg have never been recorded while supine and at rest even during pregnancy; therefore 30 N is probably more than enough to prevent passive regurgitation into the pharynx. After practising on weighing scales 3 kg (30 N) can be reproduced 50 times within safe limits: mean of 3.2 kg, range 2.5–3.7, SD 0.29 [6]. This force should therefore be practised by all anaesthetic assistants.

Light cricoid pressure must be applied to the awake patient during induction of anaesthesia [7]; although 20 N of force is tolerated by most people it is uncomfortable [2] and it must not be exceeded in case retching and oesophageal rupture occurs [4]. If anaesthetic assistants practised 20 N (2 kg) of force on weighing scales a range of forces would actually be applied to patients and therefore 20 N would be exceeded 50% of the time. A safer recommendation would be 10 N (1 kg) when awake increasing to 30 N with loss of consciousness [8]; this has been the practice in our hospital for the past 5 years and we have not seen regurgitation with cricoid pressure.

Never mind cricoid pressure in chaos, what about the failed intubation drill? Brimacombe and Berry suggest that if intubation is difficult, or if oxygen saturation on the pulse oximeter starts to fall during attempts at intubation, then cricoid pressure should be reduced, then released, in order to improve the view at laryngoscopy for another attempt at intubation. Releasing cricoid pressure gradually under direct vision with suction in hand may be relatively safe as cricoid pressure could be increased again if regurgitation occurred which may prevent

aspiration. However, if cricoid pressure is applied correctly, releasing it is unlikely to improve the view at laryngoscopy and will probably make it worse [6]. The philosophy of the failed intubation drill is oxygenation without aspiration and also to admit a failed intubation at an early stage before desaturation starts to occur. Ventilation using a facemask and an oral airway should immediately follow this decision and continue until spontaneous ventilation resumes. Cricoid pressure should be maintained during mask ventilation as regurgitation may be provoked by a rise in oesophageal pressure [9]. Mask ventilation with cricoid pressure maintained was not difficult in 70% of cases of failed intubation in obstetric cases [10]. However, if ventilation is difficult the applied cricoid pressure should then be reduced by half (half of whatever force they happen to be applying); if this does not help a laryngeal mask is inserted. If the laryngeal mask fails to provide an airway it is removed and cricoid pressure can then be released under direct vision, suction in hand, before a second attempt at laryngeal mask insertion, which can be followed by re-application of cricoid pressure. Hopefully by this time the patient is breathing and cricothyroidotomy, which is the next step in the failed ventilation drill, can be avoided.

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The use of CO₂ gap to monitor splanchnic oxygenation

I read with interest the report of Drs Shaw and Baddock on the use of continuous gastric tonometry (*Anaesthesia* 1997; **52**: 709–10). This is an interesting early report of new technology which merits further comment.

The use of intramucosal –arterial CO₂ gap ($P_{iCO_2} - P_aCO_2$) may well hold promise in identifying patients with splanchnic ischaemia. It avoids the assumption that arterial and intramucosal bicarbonate concentrations are equal (a necessary assumption to allow calculation of pH_i) and therefore avoids the potential error of a calculated low pH_i in the absence of splanchnic ischaemia, e.g. renal failure, diabetic ketoacidosis. However the substitution of endtidal CO₂ ($P_{E'}CO_2$) for P_aCO_2 and the use of $P_{iCO_2} - P_{E'}CO_2$ gap may not be valid as the relationship between P_aCO_2 and $P_{E'}CO_2$ may well vary in critically ill patients. This point was mentioned by Drs Shaw and Baddock. However, they then describe a case where $P_{E'}CO_2$ fell and P_{iCO_2} was elevated in relation to occlusion of the inferior vena cava and

presumed fall in cardiac output. During this period of vascular clamping the relationship between $P_{E'}CO_2$ and P_aCO_2 would have been drastically altered. Endtidal CO₂ and, although not measured, P_aCO_2 and tissue CO₂ would almost certainly have risen. It has been demonstrated that alterations in P_aCO_2 are reflected by similar alterations in P_{iCO_2} , although the CO₂ gap remains constant [1]. Therefore the $P_{iCO_2} - P_{E'}CO_2$ gap would have been increased by two mechanisms in this case, neither of which would necessarily infer splanchnic ischaemia. I do not dispute that this operative procedure and decreased cardiac output due to vascular clamping may have caused splanchnic hypoperfusion but the use of $P_{E'}CO_2 - P_{iCO_2}$ gap under these circumstances is inappropriate.

My second reservation is the comment that in the 12 h prior to discontinuation of monitoring the CO₂ gap never rose above 3–4 kPa and that this reflected the patient's excellent condition. Information from the accompanying illustration reveals that the pH_i 90 min prior to this time was 7.19. We have recently shown that a CO₂ gap of > 3 kPa has a sensitivity of 68% and a specificity of 100% in recognising a pH_i < 7.32 [2], which is borne out by the pH_i displayed in the report by Drs Shaw and Baddock. We would be concerned about such a high CO₂ gap and low pH_i as this has been shown many times to be a poor prognostic factor [3, 4].

In summary we feel that although this is promising new technology the use of the $P_{iCO_2} - P_{E'}CO_2$ gap is a noisy parameter due to the nonfixed relationship between $P_{E'}CO_2$ and P_aCO_2 and should be used with caution. A possible refinement for the future would be to combine the digital output from the Tonocap with an intra-arterial CO₂ electrode thus providing a real time $P_{iCO_2} - P_aCO_2$ gap and pH_i if desired. In addition the range for normal CO₂ gap needs to be defined.

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Prediction of outcome by pH_i, pH_a and CO₂ gap

I read with interest the thought-provoking article by Gomersall and colleagues regarding the prediction of outcome by gastric tonometry (*Anaesthesia* 1997; **52**: 619–23). In appraising this paper it is worthwhile considering the derivation of pH_i and the similarity to the derivation of pH_a:

$$pH_i = 6.1 + \log\left(\frac{[HCO_3^- \text{ arterial}]}{[0.225 \times P_{iCO_2}]}\right)$$

$$pH_a = 6.1 + \log\left(\frac{[HCO_3^- \text{ arterial}]}{[0.225 \times P_aCO_2]}\right),$$

where P_{iCO_2} equals intramucosal CO₂ tension. As can be seen from these two equations the derivation of pH_i can be reduced to

$$pH_i = pH_a - \log P_{iCO_2}/P_aCO_2.$$

From this third equation it can be easily appreciated that low pH_i can be attributed to three mechanisms: (1) secondary to low pH_a due to mathematical coupling (low HCO₃⁻ arterial or acute rises in P_aCO_2); (2) elevation of P_{iCO_2} with respect to P_aCO_2 ; (3) a combination of the above two mechanisms.

If one believes that low pH_i as a marker of splanchnic ischaemia is superior in predicting poor outcome than pH_a, as has been shown by previous studies [1, 2], then the only logical

conclusion is that mechanism 2 above is of importance. This has been elegantly demonstrated in a theoretical model by Schlichtig *et al.* [3]. However, in the study by Gomersall *et al.*, pH_a was superior to pH_i in predicting outcome at 0 and 24 h, suggesting that low pH_i may be, in a large part, due to mechanism 1 above. In addition the CO_2 gap was poorer than both pH_i and pH_a at discriminating survivors from nonsurvivors.

Of the three possible explanations of the findings of Gomersall and colleagues they focus on the hypothesis that the $P_iCO_2 - P_aCO_2$ gap is not a marker of splanchnic ischaemia. However, Friedman *et al.* have shown that the CO_2 gap was significantly higher in patients with evidence of tissue hypoxia than those with no evidence of tissue hypoxia and that pH_i correlated better with P_iCO_2 than arterial bicarbonate or base deficit [4]. In addition we have recently shown that a CO_2 gap of ≥ 3 kPa has a sensitivity of 68% and a specificity of 100% in predicting that $pH_i < 7.32$ [5]. It may be that the incidence of splanchnic ischaemia in the patients studied by Gomersall *et al.* was lower than their calculated pH_i may suggest. An analysis of their admission diagnosis reveals that many patients have the potential to have a low pH_i due to mechanism 1 above. Asthma, chronic obstructive airways disease and pneumonia may lead to respiratory acidosis which has been shown to produce low pH_i but unaltered $P_iCO_2 - P_aCO_2$ gap [6]. Similarly, diabetic ketoacidosis may lead to low pH_i due to low arterial bicarbonate. These patients, and the miscellaneous group, constitute over 50% of the study population thus potentially undermining the study.

In conclusion, the finding that pH_a is superior to pH_i in predicting outcome is not in agreement with much of the published literature. This may be due to the heterogeneous study population of small numbers and mathematical coupling leading to low pH_i . Furthermore the statement that $P_iCO_2 - P_aCO_2$ gap is not a measure of splanchnic ischaemia is not supported by the data, as no independent measures of splanchnic oxygenation were performed. Finally this paper does not give grounds for the

abandonment of the use of the $P_iCO_2 - P_aCO_2$ gap.

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A reply

We thank Dr Findley for his interest in our paper. In reply we would like to make the following points. Schlichtig *et al.*'s mathematical model demonstrated that $P_iCO_2 - P_aCO_2$ gap is independent of systemic acid–base state. They did not assess its importance in the causation of low pH_i [1]. In the complex setting of critically ill patients, the relative contributions of pH_a , pH_i and P_iCO_2 are almost certainly not constant either in magnitude or in relative importance with regard to their contribution to

pH_i . It is the clinical setting that we evaluated.

The superiority of pH_a over pH_i was not discussed and we do not believe that our data allow Dr Findley to conclude that pH_a was a superior predictor in our patients. Although not mentioned in our paper, there was no statistically nor clinically significant difference between the area under the receiver operating characteristic (ROC) curves for pH_a and pH_i . We chose not to discuss this issue because our sample size was too small for such a comparison to be meaningful. Very large sample sizes are required for comparisons of the area under ROC curves if the comparison is to have adequate power when the two areas are similar [2]. Friedman *et al.* [3] only showed that $P_iCO_2 - P_aCO_2$ gap was higher in septic patients with raised lactate levels. It must be pointed out that, although frequently raised in sepsis, lactate is a poor indicator of tissue hypoxia [4, 5]. In addition we did not state that $P_iCO_2 - P_aCO_2$ gap is not a measure of splanchnic ischaemia but only that that is the most likely explanation of our results. We are acutely aware that we did not measure splanchnic oxygenation, for the simple reason that this would have been extremely difficult to do. Indeed we know of no human studies in which splanchnic oxygenation has been measured by a validated method.

As stated in our paper Vandermeer *et al.* have shown that pH_i may be low in the absence of tissue hypoxia in an animal study [6].

We agree that a substantial number of our patients may not have had splanchnic ischaemia but disagree with Dr Findley regarding the effect of this on the significance of our study. We chose a miscellaneous group as it is only if a risk factor is present in only some of the patients studied that it is likely to be a predictor of outcome in that particular group – it might be expected to distinguish those with and without mucosal ischaemia and therefore those with worse and better outcome. If it is present in the majority of patients it would be less likely to be predictive.

This paper was not intended to give grounds for abandoning investigation of

the $P_1\text{CO}_2 - P_a\text{CO}_2$ gap, but we do not see to what use it can be put until it is established that it has some clinical significance. Prior to acceptance of our paper only one other published study had examined the clinical significance of $P_1\text{CO}_2 - P_a\text{CO}_2$ gap: the results of this study are addressed in our paper. Since then the results of two small studies have been published. The first described the significance of $P_1\text{CO}_2 - P_a\text{CO}_2$ gap in 20 neonates being weaned from extracorporeal circulation. The results indicated that in this highly specialised group of patients $P_1\text{CO}_2 - P_a\text{CO}_2$ is of prognostic significance [7]. However, we were surprised that the authors of this paper only chose to analyse the results of the single reading taken during the weaning phase despite taking regular measurements from the time of initiation of extracorporeal circulation. The second examined the prognostic significance in 19 young children with severe sepsis or septic shock. In these patients $P_1\text{CO}_2 - P_a\text{CO}_2$ gap was marginally higher (statistically significant) in nonsurvivors at 24 h but not at 0, 12 or 48 h. Even at 24 h the addition of $P_1\text{CO}_2 - P_a\text{CO}_2$ gap to blood lactate and mean arterial pressure did not appreciably increase the predictive power for death suggesting, as our data have, that its clinical significance is limited [8]. In the era of evidence-based medicine we feel it is inappropriate to abandon use of a new technique only when it is proved beyond all doubt to be of no value, rather we believe that new techniques should be adopted only when there are data to support their use. At present we do not believe that there are good data to support the use of $P_1\text{CO}_2 - P_a\text{CO}_2$ gap, except possibly in certain specialised groups.

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Reshaping the Macintosh blade

We enjoyed the paper by Bucx *et al.* on the biomechanics of a reshaped Macintosh blade (*Anaesthesia* 1997; **52**: 662–7). This paper, as well as the previous ones of these workers, greatly enhances the understanding of the biomechanics of laryngoscopy. It is, however, not quite complete in references to blades similar to the one proposed by Bucx *et al.* as they cite only that by Bizzarri and Giuffrida [1].

'Accessories for endotracheal anaesthesia are without end. There is no living anaesthetist who holds the distinction of not having designed one or more.' [2]. Several curved blades with a reduced flange are described [1, 3–9]. Of these, we have tested the Bizzarri and Giuffrida [1], the Wiemers [8] and the RFQ-blades [9] (Fig. 1 below) in mannequins [9, 10]. We found intubation time and subjective handling with the virtually flangeless Bizzarri–Giuffrida to be significantly inferior compared with the Macintosh blade [10]. Like Bucx *et*



Figure 1 The RFQ-blade is another example of a flange-reduced blade (RFQ Medizintechnik, Tuttlingen, D-78532).

al., we observed inadequate mouth-opening with flangeless or flange-reduced blades as well as a tendency to lift the head of the mannequin.

Maybe some head restraint and special training are needed to make flange-reduced blades successful.

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Non-rewireable plugs

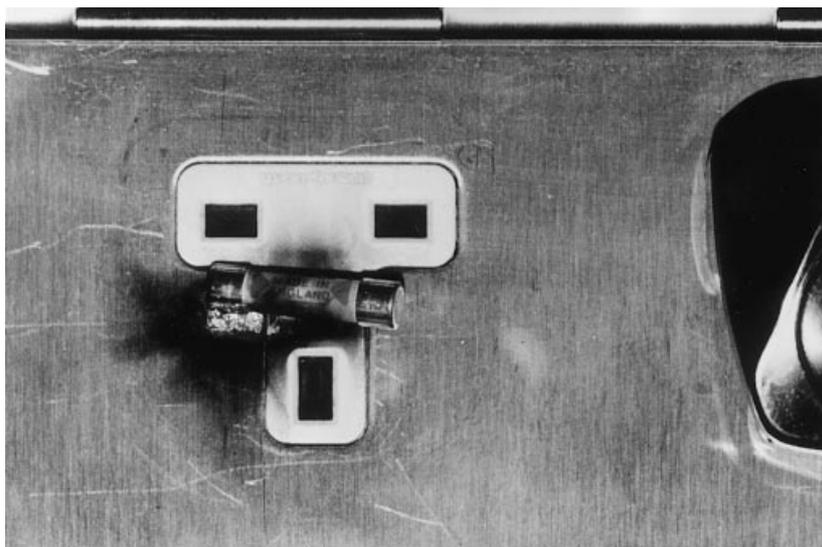
The operating theatre environment has become home to an increasing array of electrical equipment. The presence of wires attached to an ever-growing number of plug sockets from which flourish an abundance of infusion devices, monitors and other equipment makes the operating theatre environment a potentially hazardous place for both patients and staff [1]. The two cases that I report below involve problems with non-rewireable electrical plugs.

The first incident occurred 20 min into a surgical procedure in which the mains supply failed to the anaesthetic machine. The machine was changed and connected to a different power socket in an attempt to identify the source of the problem. However, the mains electric supply failed within minutes and the Residual Current Devices [RCD] located outside the operating theatre were tripped out. It was also noticed that immediately before the power supply failed, the display on one of the syringe drivers continued to show 'mains failure'. Following disconnection of this particular syringe driver from the mains, the power supply to the anaesthetic machine and the rest of the

theatre remained intact. On closer inspection it was found that fluid had ingressed into the fuse holder of the plug of the syringe driver. The fluid had short circuited the fuse causing current to flow between live and the earth points on the plug which caused the RCD to trip. The 20-min delay before the circuit breaker tripped after the faulty pump was switched on at the beginning of the operation was most probably due to the time it took to heat the fluid in the plug. The rise in temperature would have the effect of lowering the electrical resistance of the fluid until a point was reached where sufficient current flowed to trip the RCD.

In a separate incident a non-rewireable plug had been dropped, the fuse holder had fallen out and the fuse incorrectly re-inserted. The plug was then inserted into the electrical socket as shown in the figure. Because the fuse was incorrectly inserted, the electrical plate holding the socket provided the earth path and consequently current flowed from the live fuse to earth. This had the effect of melting the fuse against the earth plate as shown in the figure.

Both of these incidents demonstrate potential problems with non-rewireable plugs. Whilst the second incident was originally the fault of the person inserting the fuse, it would still not have occurred had the plug not been of the type described above. Whilst these plugs



are a convenient means whereby fuses can be changed easily and safely by nontrained personnel, it has been demonstrated that their use can be associated with significant and potentially serious sequelae.

All plugs should conform to the British Standards specification – BS 1363 [specification for rewirable and non-rewirable plugs]. In some situations a variation of this specification, known as BS 1363/A [rough usage], is used to cover equipment which is used in more hostile conditions such as the operating theatre. According to a recent safety action notice [1, 2], a number of other incidents have been reported from outside this institution concerning this particular type of plug. The latest Safety Action Notice specifies that non-rewirable plugs should confirm to BS 1363/A specification, have a moulded one-piece body and electrical connection pins should be solid and not of the folded sheet metal type.

It is now the policy of some manufacturers to supply their syringe drivers with moulded non-rewirable plugs. It must be pointed out, however, that these plugs, while being an improvement on the nonmoulded counterparts, still have an access point for fluid to ingress. There have not been any reported incidents with these plugs to date. It is currently the policy in this hospital to replace all non-rewirable plugs, which do not meet the recommendations of Safety Action Notice [SAN] 97/10 [3], with wireable versions for safety reasons. The minor inconvenience caused by having to dismantle a plug, when changing the fuse, is a small price to pay if one is to avoid problems such as those described above. Although these incidents appear to be few and far between it is probably fair to say that electrical safety problems are generally under reported.

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Over reliance on pulse oximetry

We would like to report an incident whereby a pulse oximeter (Hewlett Packard model number M11908) read a false high oxygen saturation which led to a patient becoming markedly hypoxic through over reliance on this device. The lungs of a 41-year-old man suffering from pneumocystis pneumonia were being ventilated on our ITU. Due to inadequate gas exchange, it was decided to paralyse him with atracurium, whereupon his oxygen saturation by pulse oximetry rose from 92% to 100%. Over the following 90 min it was possible to reduce the inspired oxygen from 86% to 50% and all seemed to be going well, until his blood pressure fell dramatically. It was noted that he had become markedly cyanotic, although the pulse oximeter read 98% and showed a good waveform. Arterial blood gases revealed a P_{O_2} of 4.09 kPa. This rapidly corrected once inspired oxygen was increased and he did not appear to suffer any adverse consequences. The probe has been tested by our 'in-house' medical engineers, who could find no fault, and it was returned to Hewlett Packard for further assessment.

We could find only one report of pulse oximetry giving a false high reading, in hypoxic children using an adult probe; interestingly it was the same model Hewlett Packard probe as in our case. The incident we report occurred in an adult patient using the standard adult probe. We would like to stress the same message, that electronic monitoring equipment is merely an aid, which is liable to error and should not replace vigilant clinical observation.

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A reply

The Hewlett-Packard M1190A Adult Reusable Sp_{O_2} Sensor which was reported as giving an incorrect reading

was returned to Hewlett-Packard as defective. The incoming inspection revealed damage to the sensor housing, which could cause an incorrect reading due to ambient or other light sources shining on the sensor. The sensor was replaced to the customer's satisfaction and no further problems have been reported. Hewlett Packard is continuously endeavouring to improve the strength and reliability of its reusable sensors, but this case illustrates the importance of regular inspection of all pulse oximetry sensors for signs of physical damage.

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The 'non-empty' bottle revisited

I have recently been handed a boxful of used isoflurane bottles by our operating theatre staff. It was noticed that a significant volume of fluid was left in the bottle when the vaporiser had been filled by the key filler method and I was asked how we should dispose of these contaminated bottles. Our pharmacy was unable to help. The basic problem is that the key filler system for isoflurane empties the bottle, but the filler tube does not empty, so that when the filler is disconnected from the vaporiser, a few millilitres of fluid is returned to the bottle leading to wastage of drug and thus NHS money and possible pollution. Further enquiry among members of the South Thames (East) Region Anaesthetic Specialty Committee about their management of this problem demonstrated a wide variation in practice in spite of its having been discussed in correspondence in *Anaesthesia* in 1992 and 1992 [1–3].

Over a period of 2 months in our main operating theatre unit, 250 ml of isoflurane or one bottle BNF price £97.50 is being wasted in this way and being sent off for glass disposal. The quantity of unused drug which must be wasted globally given an annual production in excess of 800 tonnes does not bear

thinking about. It seems to have similarities with Coleman's mustard who were said to have made their money from the mustard left on the side of the plate. I approached the manufacturer (Abbott); individual members of the company were extremely interested and sympathetic, but a corporate solution was not forthcoming. As this correspondence commenced, they increased the size of their bottles, so the proportion of isoflurane wasted was reduced, but there is still no plan to change the filler system to one similar to that for sevoflurane which empties completely. We need some sort of solution to prevent the current wastage; now.

We could copy the brewery industry and call it ullage and then demand that Abbott, like a brewery, put this amount extra into the bottle and take back the incompletely emptied bottles. Not surprisingly, they have rejected this idea. The filler could be modified. Unfortunately, as I understand it, the British Standards and European Committee CEN have completed the standards for the isoflurane filler and did not address the problem of the dregs, but standards are not required for patented devices. It seems unlikely that it will be economically worthwhile for a manufacturer to design a new filler. We probably have to accept that somehow one bottle has to be emptied into the next. I do not accept that this practice would make quality control difficult to enforce [3] because it is universal practice to mix different batches in one vaporiser already. Provided that the bottles are kept in a locked drug cupboard, substitution of the contents should not be a problem, as opened bottles are frequently returned to the drug cupboard following topping-up of the vaporisers at the beginning of a list already. Dr Robin Loveday has suggested a filler with bottle ends at both ends so one bottle could be emptied safely into the next (personal communication). This would seem to be the best solution.

Meanwhile, to avoid waste, can we solve this problem?

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Attenuation of pain on injection of propofol – an unexpected benefit of co-induction with midazolam

Drs Driver *et al.* (*Anaesthesia* 1997; **52**: 698–700) once more highlight the facilitation of insertion of the laryngeal mask airway where a technique of co-induction employing midazolam (0.04 mg.kg⁻¹) 3 min prior to alfentanil and propofol is advocated. I agree that ease of insertion is much improved and have used co-induction routinely since their first publication on this subject [1].

Propofol frequently causes pain on injection and to date the widespread response to this problem is to mix 20–40 mg of lignocaine with 20 ml of the emulsion. In doing so, changes in the nature of the micelles result [2] and the practitioner assumes end-user responsibility from the manufacturer. I soon became aware that the prior administration of midazolam attenuated the pain on injection produced by propofol. The reason for this is unclear, but is not simply due to amnesia of the event, as 0.04 mg.kg⁻¹ does not typically cause sedation and patients only report slight discomfort (if at all) when asked. As a consequence I have not found it necessary to add lignocaine to propofol for the past 16 months, as pain on injection when using midazolam for co-induction is no longer a clinically significant problem. Drs Driver *et al.* do not mention this incidental observation, but appear to have used unmodified propofol in each of their studies.

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Use of heparin in arterial lines

We read with interest the article by Heap *et al.* (*Anaesthesia* 1997; **52**: 640–5) on the effect of heparin in arterial lines on coagulation studies performed on blood sampled from such lines. While this is encouraging in demonstrating little clinical effect, provided at least 4.5 ml of blood is withdrawn first, we question whether heparin is necessary at all. Stimulated by an article in the nursing literature [1] we omitted heparin from 10 consecutive arterial lines in the ITU and could find no observable difference. Following this, we have not put heparin in our arterial lines for the last 3 years and have found no reason to revert to adding heparin. Consequently the question of validity of coagulation studies does not arise.

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Anaesthesia, anaesthetics and anaesthesiology

With regard to spelling: as long ago as 1755 Dr Johnson in his famous dictionary remarked: 'AE or Æ. A diphthong of

very frequent use in the Latin language, which seems not properly to have any place in the English . . .’ This diphthong is an affectation and should be quietly put to rest, as was that in ‘aether’, a form still occasionally used when I was a student.

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Footnote: This correspondence is now closed
Editor.