Histamine release by neuromuscular blocking agents

Sir,—The design of the study by Naguib and colleagues [1] on the histamine releasing potencies of various neuromuscular blocking drugs resulted in cardiovascular changes which were profound, adverse and predictable. For example, the data sheet for mivacurium suggests administration over 5–15 s. The authors chose 5 s, despite quoting references which showed potential adverse haemodynamic changes caused by rapid administration of some benzylisoquinolinium neuromuscular blockers. Three minutes after administration of mivacurium, the control mean arterial pressure (MAP) of 76.0 mm Hg had decreased to a mean of 58.1 mm Hg, 2 SD below which gives an MAP of 43.7 mm Hg. Hypotension and tachycardia represents the worst possible combination of cardiovascular variables for myocardial perfusion, already compromised by covert coronary atherosclerosis at 60 yr of age, the maximum age in the study sample.

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Sir,—The design of our study [1] was not different from that of similar studies. For example, Basta and colleagues [2] and Moss and colleagues [3] administered atracurium and dimethyl-tubocurarine and tubocurarine, respectively, as a single rapid (5 s) bolus. Administration of mivacurium over 5 s is still within the recommendations of the manufacturer. However, slower administration of larger doses of benzylisoquinolinium compounds such as mivacurium 0.25 mg kg⁻¹, for example over 60 s, results in no significant changes in histamine concentrations or haemodynamic variables [4]. Therefore, with slower administration, the inherent differences between the neuromuscular blocking drugs studied could not have been elicited.

The dramatic scenario created by Dr Todd in his letter does not match our data. The patients in the mivacurium group were aged 20–41 (mean 28.4) yr. The ranges of mean arterial pressure and heart rate observed in this group were, respectively, 47–70 mm Hg and 76–93 beat min⁻¹. For those patients who may be compromised cardiovascularly, it is clear that selecting a drug with less histamine release may be an appropriate choice.

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Preoperative fasting for paediatric anaesthesia

Sir,—I was surprised to find that reference No. 46 in the article by Phillips, Daborn and Hatch [1] concerning the osmolality of feeding mixtures during experimental injections of milk into tracheas was a reference more than 40 yr old. I would have hoped that the authors would be aware that feeding mixtures, their contents and specifically their osmolality have changed a little during nearly half a century in order to modify those mixtures to resemble human milk as closely as possible. Surely this modification should be considered before one could conclude that formula feeds “cause a greater degree of pulmonary oedema than human or cow’s milk when aspirated…”. 

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Sir,—The issue of the relative safety of breast milk and formula feeds remains contentious. It is confused by the variety of types of formula available. Whey based feeds are generally thought to leave the stomach more quickly than feeds with a casein base, but mothers find them less satisfying for their babies. The reference referred to by Dr Hopp was the most recent we could find relating to the pathological changes produced by aspiration of milk. We do not dispute that formula milks have indeed changed but we do not have experimental evidence for what occurs when they are aspirated.

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A. K. DABORN
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Use of i.m. temperature probes during cardiopulmonary bypass in humans

Sir,—Benson and colleagues might be forgiven when they write that they could not find reports of i.m. temperature measurements during and after cardiopulmonary bypass (CPB) and that differences between nasopharyngeal and muscle temperatures have not been reported previously [1].

It is probable that recently retired cardiac anaesthetists from the Queen Elizabeth Hospital, Birmingham, would have assured him that i.m. temperature probes have certainly been used for this purpose in the rather distant past. Even my memory, confirmed by my own limited database [2, 3], tells me that such measurements at leg and buttock sites were reported from the Westminster Hospital some years ago and it was common practice in other cardiac units at that time and later. Books published a mere 15 yr ago mentioned placing muscle probes for temperature measurement during cardiac operations [4, 5]. I would expect a more protracted search than my own would reveal many more references to muscle temperature and hypothermic CPB.

Regarding the restoration of muscle temperature to normal body temperature, there is one current view that a degree of hypothermia in the immediate postoperative period after CPB may be advantageous [6] (provided the patient is not allowed to increase muscle tone or shiver). This relates to the fact that subclinical CNS injury is common after CPB [7] and that hypothermia has a protective effect on the brain after any
ischaemic event [8]. Striving to restore muscle temperature to normal results in the brain temperature being normal or even above normal towards termination of CPB and this could aggravate any injury [9]. Thus in these days of cost-effectiveness, the trend for rapid rewarming and rapid progress through the cardiac intensive care unit may not be in the best interest of the patient’s brain.


Sir,—We are grateful for the opportunity to reply to Dr Manners’ letter, some of which we suspect was written rather tongue in cheek. We were unaware of the article by Burton in 1964 and were very interested to read it. Although the other references cited by Dr Manners refer to temperature probes, they in no way invalidate our study or its contribution to the literature. It seems that little, if any, research post-dates that of Burton until the publication of our article and the abstract of Johnson, Desai and Ponte [1]. A repeat detailed search back to 1980 failed to reveal any other relevant research. If any newly retired anaesthetists have more references, data or unpublished research we would be delighted to hear from them!

With respect to the concept of mild hypothermia and cerebral protection, what evidence there is seems questionable and on the basis of current research the jury is most definitely still out. The prevention of shivering, which is known to jeopardize cerebral oxygenation, is amenable to treatment by conventional, accepted means. It was not our practice to overwarm any patient studied. Our observations which compared deep muscle temperatures with those in the nasopharynx were intended to allow a more rational future therapeutic attack on the problem of afterdrop.

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Sir,—We read with interest the recent article by Wilder-Smith, Hagon and Tassonyi [1]. The authors observed arousal effects on the EEG after tracheal intubation in patients anaesthetized with either propofol or thiopentone. They stated that the effect site concentrations of both propofol and thiopentone were not in equilibrium, but argued that this was unlikely to have influenced EEG arousal, as it changed abruptly at the time of tracheal intubation. Without control groups, where patients are anesthetized but the trachea is not intubated, the cause of the arousal cannot be attributed to intubation.

Furthermore, they concluded that propofol depressed nociception more than thiopentone. Without demonstrating the EEG effects with time after boluses of each drug without intubation, it is impossible to draw this conclusion; again a control group is necessary.

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Sir,—We thank Drs Shannon, McGregor and Brunner for their interest in our study. While we would agree that the concept of a control group not undergoing laryngoscopy and intubation is theoretically attractive, we submit that in practice the absence of such a group does not affect the conclusions drawn from the study. Briefly, these were that the nociception of laryngoscopy and intubation is associated with EEG arousal, and that this arousal is attenuated more by hypothetically equipotent doses of propofol than thiopentone, thus suggesting better antinociception by propofol than thiopentone.

The only alternative origin for the EEG “arousal reaction” described is a pharmacological one. While not explicitly stating that the effect site and plasma concentrations of propofol or thiopentone are not in equilibrium, our discussion addressed the issues resulting from that lack of equilibrium, and how unlikely they were to explain the EEG arousal observed. The pharmacological explanation is unsatisfactory for the following reasons:

(1) The EEG is accepted as a good reflection of hypnotic effect site concentrations in the unstimulated subject [1]. In both groups, the EEG time course, and hence the effect site concentration, was similar up to the time of laryngoscopy and intubation. Only in close temporal association with laryngoscopy and intubation does the EEG diverge and change direction abruptly, and this in the context of a previously stable or very slowly changing EEG. This abrupt discontinuity is even clearer in the unprocessed EEG. Such behaviour, distant from the time of injection, is difficult to explain in pharmacokinetic (or pharmacodynamic) terms.

(2) The EEG excitation of the arousal reaction associated with laryngoscopy and intubation is qualitatively and quantitatively different from the EEG excitation known to be of Pharmacokinetic origin (i.e. as a result of the presence of excitatory biophase concentrations of anaesthetic agent) and seen elsewhere in the study. For example, in the thiopentone group, the excitatory EEG arousal reaction with laryngoscopy and intubation was 3–4 times larger and lasted at least three times longer than the earlier EEG excitation of pharmacological origin associated with induction.

(3) The time course of the EEG (and effect site concentrations) during bolus injection of anaesthetic agents in the absence of stimulation such as laryngoscopy and intubation is now well described [1–4] and accessible in the form of modelling programs [5, 6]. An arousal reaction of the type seen in our study, or other types of EEG excitation, did not occur in these models 3–5 min after injection in the absence of stimulation. These models also suggest that effect site concentrations start to decrease from their peak 5 min after injection at the earliest, with excitatory effect site concentrations being reached even later.

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2. Hung OR, Varvel JR, Shafer SL, Stanaki DR. Thiopentone pharmacodynamics. II. Quantitation of clinical and electro-
Correspondence

1. Bullingham A, Strunin L. Prevention of postoperative venous thromboembolism and cardiovascular surgery

Sir,—While reading the excellent review article “Prevention of postoperative venous thromboembolism” by Bullingham and Strunin [1], I noticed they stated that patients undergoing bypass or valve surgery are “usually fully anticoagulated” in the “preoperative” period. In the cardiothoracic unit where I am presently working this is not the case. Many patients present having recently taken aspirin (which is normally stopped) and approximately 10% have received heparin infusions for unstable angina but the majority are not fully anticoagulated. In the peroperative period they are anticoagulated with heparin for bypass and although antagonized with protamine, presumably have a degree of anticoagulation after operation, related mainly to platelet dysfunction. I suggest therefore that the lack of deaths arising from pulmonary embolism in patients undergoing coronary bypass surgery in the NCEPOD study is related to peroperative anticoagulation and not to their preoperative state.

D. S. MCDONALD
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London


Sir—Dr McDonald has drawn attention to a misprint in our review article [1]. We intended to refer to the effect that *peroperative* and not *preoperative* anticoagulation might have on the incidence of venous thromboembolism after cardiac surgery. As Dr McDonald has pointed out, peroperative anticoagulation is routine for most cardiac operations, while preoperative anticoagulation is uncommon. We thank Dr Mcdonald for drawing our attention to this error.

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Prevention of vomiting after paediatric strabismus surgery

Sir,—While I was very interested in the systematic review of prevention of vomiting after paediatric strabismus surgery [1], I suspect that this methodology has fundamental flaws, particularly if the reviewers are not active practitioners in the field they are reviewing. For example, no mention is made in this analysis of the influence of analgesic technique and tracheal intubation on the occurrence of emesis. As paediatric anaesthetists we know that opioids and tracheal intubation are commonly used for this type of surgery in many countries and that if these two aspects of the technique are avoided where possible, the incidence of postoperative vomiting is very infrequent (<10%).

The pain of squint surgery is mostly conjunctival in origin and topical analgesics, drops of alcohol, propofol or NSAID can be used in place of parenteral opioids. It is no longer necessary to intubate the trachea in the majority of children for strabismus surgery with the advent of the laryngeal mask airway.

The conclusion that propofol should not be used in this group is drawn but it is not made clear that this refers to a “propofol infusion for maintenance” technique as opposed to a “propofol for induction, nitrous oxide-oxygen-volatile agent for maintenance” technique.

Therefore, the emetogenic anaesthetic techniques used in the published series which the authors reviewed are in my view obsolete and a move forward to the non-intubation, non-opioid era will have a much greater effect than any antiemetic drug on the prevention of vomiting after paediatric strabismus surgery. Systematic reviews can give a false impression and practising anaesthetists are often “well ahead of the game”. In this regard, I think the ethical window for conducting such studies in paediatric strabismus surgery will soon be closed if enough paediatric and children’s anaesthetists adopt a more modern approach to these cases.

I strongly commend a propofol-laryngeal mask-topical anaesthesia technique with spontaneous respiration of nitrous oxide-oxygen-volatile agent as day-case anaesthesia for the majority of paediatric squint surgery in terms of its efficacy and lack of emesis. We are currently auditing this assertion but will it be eligible for inclusion in the next systematic review?

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Sir,—We thank Dr Morton for his interest in our systematic review [1]. However, some of his comments show that we have failed to convey the methodological rules for performing clinical trials and systematic reviews. He advocates his own clinical approach to postoperative vomiting (POV) in paediatric strabismus surgery. We wish to comment on these two issues.

The aim of our study was to compare both efficacy and potential harm of currently used propylphylactic antiemetic treatments in a well defined subgroup of patients at particular risk of POV. There were three main results of the combined analysis of all relevant published randomized controlled trials (RCT). First, the mean incidence of POV in controls without antiemetic prophylaxis was more than 50% (with a wide range). Second, the overall efficacy of prophylaxis compared with placebo or with no treatment or, in the case of propofol, with another anaesthetic technique, was disappointing. Third, the potential of propofol for harm was alarming. As stated in the text and table, propofol as a maintenance regimen was compared with halogenated inhalation anaesthetics.

Evaluating the benefit of any treatment involves making comparisons. The statement that a particular anaesthetic technique decreases the incidence of POV in paediatric strabismus surgery to <10% sounds good but is, in the absence of any comparison, no more than a personal view. Before a new therapy is introduced into clinical practice it should have proved to be at least as effective and to induce less harm than old treatments. The widely accepted gold standard to define efficacy and to exclude harm with the least possible bias is the RCT. Clinicians have known for a long time that ignoring this rule may lead to overestimation of treatment effect [2, 3].

All 2044 children in the analysed 27 RCT underwent tracheal intubation. In ophthalmic surgery, as in other situations with more difficult access to the upper airways, many active practitioners, paediatric anaesthetists included, prefer to have the upper airway of the patient secured (i.e. to have the trachea intubated). A laryngeal mask may eventually prove to be less emetogenic than a tracheal tube, but is this technique as safe in paediatric strabismus surgery? Being “well ahead of the game” may have a dark side.
Increasing patient comfort is an attractive target. Decreasing the incidence of POV implies relieving an unpleasant but time limited condition. POV never becomes chronic and almost never kills. Therefore, the level of acceptance of any treatment-induced risk may be low for most clinicians (and patients). Well designed RCT (not audits reflecting personal views) will perhaps show in the near future if the laryngeal mask in paediatric strabismus surgery is advantageous, not only new but also beneficial. Unfortunately, even paediatric anaesthetists do not know why a particular patient vomits after surgery and another does not. Life would be much easier if we could avoid opioids and tracheal intubation in all children in order to decrease the incidence of POV to less than 10%. One of the most striking results was the extraordinarily wide range of incidences of vomiting in these studies. However, only six studies reported the use of intraoperative opioids [4–9] and in one the incidence of vomiting was very low [7]. Indeed, the highest incidences of vomiting were reported in studies where opioids were avoided [10–12]. While it is widely believed that opioids increase significantly the risk of POV, there is little evidence to support this. The suggested reported in studies where opioids were avoided [10–12]. While it is widely believed that opioids increase significantly the risk of POV, there is little evidence to support this. The suggested approach with topical analgesia appears original but is not new; recently published experiences from an RCT were not enthusiastic [13].

Clinicians have to balance treatment efficacy and potential for harm. Systematic reviews of relevant RCT with quantitative analysis of extracted data on both efficacy and adverse effects are powerful tools to make this judgement. However, systematic reviews depend on the quality of reports delivering analysable data. Innovative approaches should be subject to trials of appropriate quality. Good practitioners need to understand some methodologically just as systematic reviewers need to have a grasp of clinical realities.


Maternal sequelae of childbirth

Sir,—Professor Reynolds has drawn attention to the serious neurological complications consequent upon spinal anaesthesia which were described by Ferguson and Watkins in 1938, and by Kennedy, Effron and Perry in 1950 [1–3]. Professor Reynolds attributes these complications solely to the neurotoxicity of the spinal anaesthetic solutions used, which is a satisfactory explanation for most of the cases in Ferguson and Watkins’ case series. However, medical opinion has held that the more persistent and serious neurological disabilities such as were described by Kennedy’s team could not all be ascribed to local anaesthetic neurotoxicity and that the problem has yet to be understood fully [4].

The Woolley and Roe case [4] has since demonstrated that spinal anaesthetic solutions can be contaminated with acid from spinal needles and syringes if descaling solutions are not washed out of water boiler sterilizers. As these water boilers would have been used when Kennedy’s patients were given their spinal anaesthetics, acid contamination is a plausible explanation for these tragedies also. Confirmation of symptomatic India-rubber bung (one of the consequences of damage by acid) in four of Kennedy’s patients supports this explanation.

These water boilers remain in use today in parts of the world where medical facilities are less well developed, and the potential risk which they pose would not have become clear without this re-evaluation of an apparently unrelated historical problem. Yet again we see that an awareness of history protects us from repeating earlier mistakes.

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Nottingham

Sir,—I thank Dr Hutter for his interest in my editorial. I did not, however, intend to leave readers with the impression that I attributed neurological sequelae of spinal anaesthesia in the 1930s and 1950s to local anaesthetic neurotoxicity. What I actually referred to were neurotoxic formulations and techniques. Readers might be interested to hear that the formulation of procaine that was used for spinal anaesthesia in 14 cases of cauda equina syndrome reported from Manchester contained 10 % procaine, glycerine and either gludin or gum acacia, in 15 % alcohol [1]. The idea of this formulation, termed heavy duracaine, was to prolong the action of procaine, an odd choice since cinchocaine, a very long-acting drug, was available then. The technique I referred to was the light spinal technique which involved displacement of a large volume of cerebrospinal fluid by a foreign solution. I acknowledge that this solution itself may have been contaminated, although like Dr Hutter I would not subscribe to the phenol-entering-invisible-cracks theory. However, it was not, as Dr Hutter suggests, the Woolley and Roe cases that demonstrated that spinal injections may have been contaminated with acid from sterilizers, but Dr Hutter himself [2]. His article gives a fascinating account of his investigation of these cases and the development of his hypothesis.
The western world may congratulate itself on having left behind the amazing concoctions of the 1930s, the light spinal technique using 10 ml of hypobaric cinchocaine, boiling water sterilizers, soaking ampoules in phenol, etc. Nevertheless, recent accounts of neurotoxicity after accidental total spinal anaesthesia [3, 4] and puddling in the subarachnoid space of apparently respectable 2–5 % lignocaine solutions [5–7] must remind us how easily injections into the subarachnoid space may cause radicular irritation and even cauda equina syndrome. Perhaps we have reason to be thankful that in the UK bupivacaine rather than lignocaine is used for spinal anaesthesia.

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Venous air embolism: is end-tidal oxygen monitoring more informative than end-tidal carbon dioxide?

Sir,—We read with interest the article by Kyttä and colleagues [1] on monitoring lung compliance and end-tidal oxygen content for the detection of venous air embolism and wish to make some comments.

The authors used 100 % oxygen with 0.5–0.7 % isoflurane for maintenance of anaesthesia during posterior fossa surgery. The use of less than 1 MAC of isoflurane in oxygen during maintenance of anaesthesia is likely to be associated with awareness. Although many neuroanaesthetists have abandoned nitrous oxide during intracranial surgery, it is still being used in various centres worldwide and would have decreased the likelihood of awareness. We presume that the authors considered that the use of nitrous oxide would increase the incidence of air embolism, but it has been demonstrated that this is not the case in neurosurgical patients undergoing surgery in the sitting position [2]. Alternatively, a mixture of oxygen in air with an 2OI of 0.3–0.4 and a concentration of isoflurane in excess of 1 MAC or an infusion of l.v. anaesthetic agents can be used for maintenance of anaesthesia during neurosurgical operations.

PEEP was used for the prevention of air embolism in the sitting position, despite the associated reduction in venous return and cardiovascular instability. As a result of an increase in right atrial pressure, PEEP should reduce the pressure gradient between the cerebral venous sinuses and the right atrium. However, it has been demonstrated that PEEP has little value in the prevention of venous air embolism [3]. Moreover, the authors used hyperventilation with PEEP in their anaesthetic technique. Hyperventilation has been shown to reduce the transverse sinus pressure to within the atmospheric to subatmospheric range which could increase the risk of air embolism. Moderate hyperventilation has been recommended during the most critical period of exposing the posterior fossa followed by normoventilation when surgery of the lesion has begun [4].

In this study, the decrease in the difference between inspired and end-tidal oxygen content was 2-2.5 % during induced air embolism in animals breathing 100 % oxygen. This may be consequent on alteration in composition of alveolar gases as a result of a sudden reduction in end-tidal carbon dioxide concentration. This decrease in the difference in inspired and end-tidal oxygen concentration may not be as great when maintaining anaesthesia with oxygen–air or oxygen–nitrous oxide with an 2OI of 0.3–0.4. As air embolism is being reported increasingly after operations other than neurosurgery, the results would have been more applicable to clinical practice if an inspired oxygen concentration of 30–40 % had been used. In this study, the increase in end-tidal oxygen content occurred after the changes in end-tidal carbon dioxide concentration. In clinical practice, when air embolism is suspected because of a reduction in end-tidal carbon dioxide concentration, the oxygen in the inspired mixture is increased immediately to 100 %. Therefore, at that time the end-tidal and inspired oxygen content differences may not be of value in monitoring for detection of air embolism.

The increase in peak airway pressure reflects a decrease in dynamic pulmonary compliance if tidal volume is constant. The authors recorded an average increase in peak airway pressure of less than 1 cmH₂O subsequent to induced air embolism in animals. This is a small increase and it is doubtful if it would be sufficiently reliable to be a useful monitor in clinical practice. Changes in airway pressure of this magnitude are not uncommon during uneventful anaesthesia and surgery. The results of this study confirm that dynamic pulmonary compliance is a less sensitive monitor than end-tidal carbon dioxide concentration in the early detection of air embolism. It has been demonstrated previously that the increase in airway pressure should not be relied upon as a monitoring aid for detection of venous air embolism [5]. The large variability of the changes in airway pressure (s(t) 2.0–3.0 cm H₂O) in the animal study supports this argument.

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2. Lozasso TJ, Muzzi DA, Dietz NM, Cucchiara RF. Fifty percent nitrous oxide does not increase the risk of venous air embolism in neurosurgical patients operated upon in the sitting position. Anesthesiology 1992; 77: 21–30.


Sir,—Thank you for the opportunity to reply to the correspondence on our recent study on the detection of venous air embolism [1].

Our standard regimen in maintaining anaesthesia during posterior fossa surgery includes high-dose vecuronium and pancuronium as needed, in addition to an end-tidal concentration of 0.5–0.7 % of isoflurane in oxygen. Awareness has not been reported by any of our patients. Higher concentrations of isoflurane may cause haemodynamic lability in patients undergoing surgery in the sitting position.

The use of nitrous oxide in neuroanaesthesia is controversial. In patients at risk of venous air embolism (VAE) it is not recommended as it may increase the size of the air bubbles resulting in more pronounced consequences of air embolism [2]. The incidence of VAE cannot be expected to decrease on abandoning nitrous oxide, the occurrence of VAE being dependent mainly on the surgical technique and positioning of the patient.

There is no evidence of possible detrimental effects of low PEEP in neurosurgical patients. In our experimental study, no PEEP was applied. In the study of Zentner, Albrecht and Hassler [3],...
hyperventilation was recommended for the prevention of VAE during posterior fossa surgery. However, in the editorial comment by Roy F. Cucchiara, it was pointed out that hyperventilation compromises surgical exposure. We agree with the latter, and do not hyperventilate the lungs of our patients.

Changes in the differences between inspired and expired oxygen content were significant in this particular experimental setting. We agree with the correspondent that the changes do not follow a similar pattern if oxygen in air or oxygen in nitrous oxide are used as inspiratory gases during maintenance of anaesthesia [Kyttä, Randell, Tanskanen, unpublished observation, manuscript in preparation].

We did not recommend the use of changes in peak airway pressure for the detection of VAE. However, the decreases observed in dynamic lung compliance were more pronounced and followed VAE consistently. For calculation of dynamic lung compliance, changes in airway pressures during the respiratory cycle and changes in tidal volume are used. We still hold to our conclusion that monitoring of inspiratory to expiratory oxygen concentration, and dynamic lung compliance are valuable supplements to other monitoring techniques for the detection of pulmonary VAE [1].

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Spinal anaesthesia for Caesarean section in women with incomplete extradural analgesia

Sir.—The requirement for operative delivery in the event of inadequate extradural block is not an uncommon problem in obstetric anaesthesia, requiring an alternative anaesthetic technique. Spinal anaesthesia would appear to offer many benefits in obstetric anaesthesia, requiring an alternative anaesthetic technique. Spinal anaesthesia would appear to offer many benefits in obstetric anaesthesia, requiring an alternative anaesthetic technique. Spinal anaesthesia would appear to offer many benefits in obstetric anaesthesia, requiring an alternative anaesthetic technique.