Removal of LMA in children

Sir,—The article of Kitching, Walpole and Blogg1 investigating removal of the laryngeal mask in anaesthetized and awake children merits further discussion. At our hospital we routinely use the laryngeal mask airway (LMA) in the general paediatric population for various ear, nose and throat (ENT) procedures.2 Our standard practice is in accordance with those of both the inventor and the manufacturer of the masks.3 The LMA is left in situ until the patient regains protective airway reflexes unless problems arise both with the airway and management. We agree that children have an increased incidence of airway complications when recovering from anaesthesia compared with the adult population4 and appreciate the challenging period this can be. For this reason we have conducted a prospective audit into the clinical use of the LMA at our hospital. As part of this we have examined specifically the recovery phase of anaesthesia with the LMA in paediatric patients. This information should add to the debate which this article will generate.

Currently we have audited 140 paediatric patients aged 1–14 yr old, weighing 5–45 kg, undergoing routine ENT procedures. Anaesthesia was induced with propofol i.v. in 112 cases and sevoflurane in the remaining 28 patients. Each LMA was inserted by the classical method, as described by Brain,5 and only reinforced masks3 sizes 2, 2.5 and 3 were used. All were left in situ at the end of the procedure to allow full return of airway reflexes and any airway complications before or after removal of the LMA were noted. A record was also kept of who removed the mask and depth of anaesthesia on removal.

A total of 126 (90%) children had a totally uneventful recovery period with no airway complications. In those cases where complications occurred, the commonest problem was either coughing or retching associated with impending mask expulsion by the patient. The overall incidence of airway complications with the LMA during recovery was as follows: seven (5%) patients coughed, four (3%) retched and three (2.5%) had laryngospasm. There were no episodes of aspiration or loss of airway. The LMA was removed by the child in 28 (22%) cases, recovery nurse in 89 (70%) cases and the anaesthetist in nine (8%) cases. None of the masks was removed at a deep stage of anaesthesia; the conscious level of the patient on removal of the LMA was recorded as light (i.e. responded to physical stimulation but inappropriate response to verbal command) in 36 (28%) cases and awake (i.e. appropriate response to verbal commands) in 90 (72%) cases. The three cases of laryngospasm occurred in patients undergoing adenotonsillectomy and all presented soon after cessation of anaesthesia, with the LMA being removed by the anaesthetist in two of these cases. In two patients the LMA lodged in the oropharynx on attempted removal but in both cases the mask was soon extricated with gentle manipulation. One case of partial airway obstruction occurred when the child displaced the bite block and consequently bit on the LMA tube; this resolved on awakening. Only three (2%) patients required the LMA to be replaced with an oral tracheal tube for either inadequate airway maintenance or mechanical problems during the procedure.

The results of our ongoing audit on the use of the LMA in the ENT paediatric population shows a lower overall incidence of airway complications than the study by Kitching, Walpole and Blogg. Our values bear more relation to those of Mason and Bingham6 who studied the use of the LMA in 200 paediatric patients. They reported 12 (6%) cases of coughing, 11 (5.5%) of biting, five (4%) of laryngospasm, four (4%) of retching and three (3.5%) of vomiting during the recovery phase. These authors left the LMA in situ in all cases until return of laryngeal reflexes and expulsion of the mask by the patient. Laffon and colleagues7 investigated airway complications after anaesthetized compared with awake removal of the LMA in 60 children. This study demonstrated a lower incidence of both the anaesthetized and awake groups than the study of Kitching, Walpole and Blogg and, more importantly, detected no significant difference between the two groups in individual airway complications. Again, in this study, the LMA remained in situ until the children demonstrated recovery of airway reflexes and had opened their eyes or mouth. Both of these previous studies and our current LMA audit have demonstrated a lower incidence of airway complications with the LMA during recovery than the study of Kitching, Walpole and Blogg. Several reasons may explain this variation in clinical experience with the LMA.

First, the LMA was removed when the child had awakened sufficiently to swallow. We contend that this depth of anaesthesia does not equate with either full return of airway reflexes or an awake state. The presence or return of swallowing in the anaesthetized patient with the LMA in situ is associated with an inadequate or "light" depth of anaesthesia.8 The mask was therefore removed at too early a stage in the recovery process, associated with increased airway reflexes and complications. Second, the authors failed to mention if the LMA cuff was deflated before removal. Brain has stated previously that it is best to leave the cuff inflated until the mask is ejected spontaneously and to monitor cuff pressure via the pilot balloon during recovery.9 A third possible cause of airway stimulation might have been inadequate perioperative analgesia as not all children received a caudal anaesthetic and the number in each group receiving such analgesia was not clearly stated. The study design also included rectal diclofenac on arrival in the recovery room but because of the rapid recovery time of sevoflurane10 the children may have been in a light plane of anaesthesia while the suppository was administered. Any form of stimulating procedure at such a stage of anaesthesia can be associated with reflex airway responses. Finally, the authors failed to mention one of the major advantages offered by the LMA during recovery, namely maintenance of a patent airway thus decreasing the incidence of airway manoeuvres required during this period.2 The importance of this benefit cannot be overstated.

Avoidance of coughing and retching during recovery from ENT and plastics anaesthesia is important in order to prevent venous congestion and the potential for subsequent haemorrhage. ENT practice is associated with a higher incidence of airway complications than other areas of anaesthesia and the need for airway control in this group of patients is critical if such problems are to be avoided. We suggest from our experience that the LMA should be left in situ at the end of operation and remain so until it is expelled spontaneously by the awake child. There is a need for increased education of recovery staff in the correct management of the recovery phase with the LMA and non-intervention, avoiding excessive stimulation and suctioning is recommended if the full potential of the LMA is to be achieved.

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Sir,—We thank Drs Parry, Glaisyer and Bailey for their interest in our article and for raising some points of difference between us.

The patients in our study received a “standardized” anaesthetic. Children who could not have the particular anaesthetic technique were excluded. All children had intubation induction with halothane as we wished to eliminate the different effects on airway irritability possessed by various i.v. induction agents and other volatile agents. Because all patients were undergoing a plastic surgery procedure on the perineum or lower limb, a caudal or local block was performed for intraoperative and postoperative pain relief. We encountered no problems with insertion of the dilcocen suppositories in either the “deep” or “awake” group, probably because of the block. The entire anaesthetic technique was designed to make the time of removal of the LMA the only variable. The cuff was deflated before removal. Sevoflurane was not available.

Although Parry, Glaisyer and Bailey described a lower overall incidence of airway problems than in our study, we noted that two of their 140 patients required tracheal intubation whereas none of our 60 children did. However, patients differed from those reported by Parry, Glaisyer and Bailey in being younger, not undergoing airway surgery and all having had antialagogue premedication (oral atropine). Monitoring in the recovery period differed between these studies as all of our patients had continuous observations by observers who are authors in addition to pulse oximetry. It is regrettable that Parry, Glaisyer and Bailey did not provide objective evidence of continued adequate oxygenation from SpO₂ measurements during recovery.

In our study of young children undergoing plastic surgery to the lower limbs and perineum, those in whom the LMA was removed at a deep plane had fewer coughing episodes compared with those in whom the LMA was removed at a shallow plane. In this regard we echo Parry, Glaisyer and Bailey’s remark that “in the future some kind of grading of the plane of anaesthesia will be required”. The incidence of coughing is an important measure of airway function, especially in the recovery phase of anaesthesia. An accurate measure of coughing may be considered as either easy or difficult with no indeterminate or intermediate categorization options. Second, we have to assume that these defined states are both absolute and universal (i.e. experienced and trainee anaesthetists would recognize the same classification and make the same judgements). Finally, we relate the proposed clinical indices to a non-Calman situation where the “on-call” trainee anaesthetist works without direct supervision and the experienced colleague is available to be called in from home.

For the trainee, clinical responsibility with regard to prediction of difficulty should end when difficulty is diagnosed. We expect the experienced colleague to come from home and deal with all such cases or at least take clinical responsibility for whatever happens. From the trainee’s point of view, continuing responsibility is important in respect of the cases predicted as easy. The trainee anaesthetist’s nightmare (TAN) is the number of cases predicted to be easy but which turn out to be unexpectedly difficult relative to the total number predicted as easy. Referral to standard statistical definitions shows TAN = 1 – NPV (NPV = negative predictive value, the number of easy intubations correctly predicted relative to the total number of intubations predicted to be easy). In a similar manner, the experienced anaesthetist’s tribulation (EAT) is the number of cases predicted to be difficult which turn out to be easy relative to the total number predicted as difficult. This refers to the number of times attendance for allegedly difficult intubations will in fact be inappropriate. Again this is defined as EAT = 1 – PPV (PPV = positive predictive value, the number of difficult intubations correctly predicted relative to the number of intubations predicted to be difficult).

These “worst case” clinical indices may help clarify the clinical relevance of the statistical definitions which obviously came first and were not defined specifically for predicting difficult intubation. Another “aide-mémoire” which clinicians may find useful for remembering the statistical definitions is the graphical square box description in Altman’s book. This text also describes the limitations of each of the statistical indices and the importance of considerations of prevalence for the condition to which they apply.

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Effect of amino acid infusion on body temperature during anaesthesia

Sir,—We were interested to read the study on the effects of preoperative infusion of amino acids in preventing postoperative hypothermia by Selldén, Bränström and Brundin. We wish to comment on their conclusion.

The authors infused amino acid solutions (240 kJ h⁻¹) over 1–2 h before anaesthesia and compared the results with those from control patients who received saline, believing that preoperative administration of amino acids prevents anaesthesia-induced hypothermia. In their previous study, they suggested that peroperative amino acid infusion also prevented anaesthesia-induced hypothermia. In the present study, they found that rectal temperatures increased significantly from the basal state during the first hour of amino acid infusion and decreased with the onset of anaesthesia. In the control group, rectal temperature from the onset of anaesthesia was significantly greater than that in the amino acid-treated group.

We infused 335 kJ of amino acid solution during the first hour of anaesthesia to 25 patients and compared changes in nasopharyngeal and skin surface temperatures and the incidences of postoperative shivering with data from 25 control patients. In our study, nasopharyngeal and skin surface temperatures decreased in both groups during the first hour of anaesthesia. Nasopharyngeal temperatures decreased from 36.0±0.4 to 35.5±0.7°C in the amino acid-treated group and decreased from 36.2±0.4 to 35.7±0.5°C in the control group. Skin surface temperatures...
decreased from 32.2 ± 1.2 to 32.1 ± 2.1°C and from 32.4 ± 1.6 to 32.5 ± 1.7°C in the amino acid-treated and control groups, respectively. At the end of anaesthesia nasopharyngeal temperatures were 35.3 ± 0.9°C in the amino acid-treated group and 35.5 ± 0.7°C in the control group (ns). The incidences of shivering were similar in both groups (nine of 25 and seven of 25 in the amino acid-treated and control groups, respectively). Our results do not confirm those of Selldén and colleagues. Therefore, we believe that the effect of amino acid infusion on thermoregulation and its effect on preventing anaesthesia-induced hypothermia and postoperative shivering need further investigation.

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Sir,—Dönmez, Şekerçii and Arslan reported that infusion of an amino acid solution (composition not reported) during the first hour of anaesthesia did not prevent postoperative shivering or anaesthesia-induced reductions in nasopharyngeal or skin temperatures in 25 patients. The authors argue that these findings do not correlate with two of our published studies in which we demonstrated that i.v. infusion of a mixture of 19 amino acids (Vamin, Pharmacia Upjohn, Stockholm, Sweden) stimulated oxidative heat generation to an extent that effectively prevented whole body hypothermia and postoperative shivering. We have recently published further confirmation of these findings,1 and wish to offer the following comment.

(1) We do not know how nasopharyngeal temperature was recorded or what this temperature represents. The reported basal values, 36.0 and 36.2°C, seem low compared with normal mixed blood or body core temperatures; (2) the observed average anaesthesia-induced reduction in nasopharyngeal temperature did not exceed 0.5°C in any of the groups. If this value represents the whole body temperature change, the observation would indicate that the anaesthetic agent used (not reported) did not induce clinically significant hypothermia in any group; (3) a reduction in body core or mixed blood temperature of 0.5°C would hardly be expected to induce much generalized shivering. Nevertheless 30% of patients shivered in response to this average decrease in nasopharyngeal temperature. Although individual correlations between temperature and shivering were not presented, the finding might further suggest that the changes in nasopharyngeal temperature did not adequately reflect whole body temperature changes; (4) the shivering incidence of nine of 25 in the amino acid-treated patients seems surprising not only because of the small temperature reduction. We have never recorded postoperative shivering in any patient subjected to our mode of intraoperative amino acid treatment; (5) an unchanged skin surface temperature may be a reasonable finding in view of the well known centrifugal redistribution of heat during general anaesthesia.

We appreciate the interest of Dönmez, Şekerçii and Arslan in this field and would like to encourage the authors to continue studies including rectal, oesophageal or mixed blood thermometry.

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Sir,—We read with interest the article by Chung and colleagues1 comparing different volumes of 0.25% hyperbaric bupivacaine for Caesarean section using spinal anaesthesia and wish to make the following points.

(1) Although the authors used different volumes of 0.25% hyperbaric bupivacaine for spinal injection according to patient height, there is no evidence that varying the dose of local anaesthetic in this manner affects the final characteristics of a spinal block.2 If in this study patient height did indeed influence the spinal block, there would have been differences between subgroups given the same volume. For example, did the characteristics of the block with bupivacaine 3.6 ml given to the subgroup of patients in group 1 who were 161–165 cm in height differ from the subgroup of patients in group 2, 156–160 cm in height, given the same volume of local anaesthetic. Unfortunately no analysis of these subgroups was presented.

(2) The results of this study are also not related directly to the effects of volume of 0.25% hyperbaric bupivacaine as suggested, as the same concentration of local anaesthetic was used

Anatomical configuration of the spinal column as determined by magnetic resonance imaging

Sir,—The anatomical configuration of the spinal column significantly affects the distribution of local anaesthetic solutions within the subarachnoid space. Hyperbaric anaesthetic solutions migrating cephalad, pool in the lowest region of the thoracic hollow under the influence of gravity.1 Textbooks of anaesthesia always describe that, in the supine position, the lowest point of the thoracic hollow is located at T5–6, a concept proposed by Barker in 1907.2 However, no evidence exists to confirm this classical anatomical concept. We have demonstrated previously that the lowest point of the thoracic hollow determined by magnetic resonance (MR) imaging is located in the vicinity of T8 in Japanese healthy volunteers3 and these findings do not agree with long-held concepts. However, differences in the curvature of the thoracic hollow may exist in subjects with different racial characteristics. Therefore, we attempted to examine sagittal MR images of the spinal column which are illustrated in MR imaging textbooks published in Europe and North America.4 There were no sagittal MR images to demonstrate that the lowest point of the thoracic hollow is located at T5–6. Of the eight images examined, the lowest point of the hollow was located at T7 in one, T8 in one, T8–9 in one, T9 in two, T9–10 in one and at T12 in two. MR imaging provides precise anatomical information on the configuration of the spinal column in living subjects. Consequently, the lowest point of the thoracic hollow in the supine position is unlikely to be located at T5–6, even in European and North American subjects.


Spinal anaesthesia with 0.25% hyperbaric bupivacaine for Caesarean section: effects of volume

Sir,—The anatomical configuration of the spinal column significantly affects the distribution of local anaesthetic solutions within the subarachnoid space. Hyperbaric anaesthetic solutions migrating cephalad, pool in the lowest region of the thoracic hollow under the influence of gravity.1 Textbooks of anaesthesia always describe that, in the supine position, the lowest point of the thoracic hollow is located at T5–6, a concept proposed by Barker in 1907.2 However, no evidence exists to confirm this classical anatomical concept. We have demonstrated previously that the lowest point of the thoracic hollow determined by magnetic resonance (MR) imaging is located in the vicinity of T8 in Japanese healthy volunteers3 and these findings do not agree with long-held concepts. However, differences in the curvature of the thoracic hollow may exist in subjects with different racial characteristics. Therefore, we attempted to examine sagittal MR images of the spinal column which are illustrated in MR imaging textbooks published in Europe and North America.4 There were no sagittal MR images to demonstrate that the lowest point of the thoracic hollow is located at T5–6. Of the eight images examined, the lowest point of the hollow was located at T7 in one, T8 in one, T8–9 in one, T9 in two, T9–10 in one and at T12 in two. MR imaging provides precise anatomical information on the configuration of the spinal column in living subjects. Consequently, the lowest point of the thoracic hollow in the supine position is unlikely to be located at T5–6, even in European and North American subjects.


Spinal anaesthesia with 0.25% hyperbaric bupivacaine for Caesarean section: effects of volume

Sir,—The anatomical configuration of the spinal column significantly affects the distribution of local anaesthetic solutions within the subarachnoid space. Hyperbaric anaesthetic solutions migrating cephalad, pool in the lowest region of the thoracic hollow under the influence of gravity.1 Textbooks of anaesthesia always describe that, in the supine position, the lowest point of the thoracic hollow is located at T5–6, a concept proposed by Barker in 1907.2 However, no evidence exists to confirm this classical anatomical concept. We have demonstrated previously that the lowest point of the thoracic hollow determined by magnetic resonance (MR) imaging is located in the vicinity of T8 in Japanese healthy volunteers3 and these findings do not agree with long-held concepts. However, differences in the curvature of the thoracic hollow may exist in subjects with different racial characteristics. Therefore, we attempted to examine sagittal MR images of the spinal column which are illustrated in MR imaging textbooks published in Europe and North America.4 There were no sagittal MR images to demonstrate that the lowest point of the thoracic hollow is located at T5–6. Of the eight images examined, the lowest point of the hollow was located at T7 in one, T8 in one, T8–9 in one, T9 in two, T9–10 in one and at T12 in two. MR imaging provides precise anatomical information on the configuration of the spinal column in living subjects. Consequently, the lowest point of the thoracic hollow in the supine position is unlikely to be located at T5–6, even in European and North American subjects.

throughout the study. Therefore, as the volume of the spinal injection changed, so did the total dose of drug administered. The effect of volume independent of the actual amount of drug administered intrathecally was not compared. Many workers have demonstrated that the dose of intrathecally administered local anaesthetic is more important than volume.1,4 Furthermore, there is evidence from the literature that the major factor affecting intrathecal spread of local anaesthetic in pregnant women at term is movement of the patient after spinal injection.3 This is thought to result from extrudural venous engorgement, secondary to veno caval compression while the patient is being turned, decreasing the volume of the dural sac and thereby leading to displacement and spread of the local anaesthetic solution. A recent MRI study appears to support this theory.6
(3) We also question the accuracy of the sensory block assessments. From the data in table 3, it appears that surgery started and ended approximately 16 min and 83 min, respectively, after spinal injection. We deduce from figure 1 that during these 67 min of operating time, pinprick assessments was still continuing. Presumably, according to the dermatomal levels recorded within this period, pinprick testing was continuing within the area of a supposedly sterile operating field. Or, inconceivably, were the surgeons also undertaking intraoperative sensory testing?
(5) Finally, considering the high incidence of hypotension in group 3, we would have expected umbilical cord gas tensions to have been measured.

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Sir,—I thank Drs Bell, Fernando and Parry for their interest in our article.1 Although the subgroups were small (only 10 cases), we analysed the differences in spinal block between all subgroups in groups 1 and 2, and groups 2 and 3, including subgroups given the same volume in different groups. There were no significant differences between all subgroups, except for subgroups with different heights, given different volumes. For example, the characteristics of spinal block with bupivacaine 3.2 ml in the subgroup of patients in group 1 who were 156–160 cm in height, differed from those with bupivacaine 4.0 ml in the subgroup of patients in group 2, 161–165 cm in height. The results support the concept that patient variables, especially height, are not important factors in determining the final characteristics of spinal block.2

Many workers4,6 have demonstrated that the dose of intrathecally administered local anaesthetic is more important than volume or concentration in determining spread of anaesthetic solution in the CSF. However, other workers9 have shown that increasing the volume administered into the subarachnoid space, especially with hyperbaric solutions, resulted in significantly greater cephalad spread. I agree that the effect of volume independent of the dose of drug administered intrathecally was not compared in our study. In the clinical dose range (10–15 mg) of 0.5% or 0.75% hyperbaric bupivacaine for spinal anaesthesia in Cesarean section, the volume does not exceed 3 ml. When 0.25% hyperbaric bupivacaine solution is used, a dose of 10–15 mg exists in a volume of 3.0–6.0 ml. The large volume of hyperbaric solution itself may influence the spread of local anaesthetics in CSF and final block, especially in the narrow subarachnoid space of the term parturient. I think that the volume of hyperbaric spinal anaesthetic solution may be additive to the effects of gravity, position and dose.

As shown in tables 2 and 3, in all patients in groups 2 and 3, the regression times to T10 were more than 100 min and induction to end of surgery times were less than 85 min. Sensory block assessment was made easily in the flanks of patients without disturbing surgery in all patients in groups 2 and 3, but omitted during operation in approximately 50% of patients in group 1. However, the data did not influence the results, that is assessment of segmental spread of sensory analgesia (fig. 1) and regression time to T10 and L5 (table 2).

Umbilical cord blood analysis was not performed in this study and I think in retrospect that it would obviously have been better if these measurements had been made. The aim of our study was to assess the safety and efficacy of 0.25% hyperbaric bupivacaine in glucose for spinal anaesthesia in Cesarean section. We found that if Cesarean section was undertaken with 0.25% hyperbaric bupivacaine for spinal anaesthesia, the volume required was at least 4.0 ml. Considering the quality of anaesthesia and incidence of hypotension, I think that 0.75% or 0.5% hyperbaric bupivacaine for spinal anaesthesia in Cesarean section is preferable to 0.25% hyperbaric bupivacaine.

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Management of rhabdomyolysis

Sir,—Tuckey reported on the management of a patient who developed rhabdomyolysis after bilateral calf compartment syndrome resulting from the lithotomy position.1 Such patients are at great risk of developing renal failure and Tuckey correctly stated that this may be reduced by aggressively promoting an alkaline diuresis with infusions containing bicarbonate and mannitol. While I agree that such vigorous therapy should be given to prevent or limit further morbidity and mortality, therapy itself carries significant risk on three accounts.

First, the recommended volume infusions (12 litre day−1)5) may result in fluid overload in an actual or potential critically ill patient. Second, bicarbonate infusion can produce metabolic alkalosis. Alkalaemia is associated with increased mortality.3 In response to metabolic alkalosis, compensatory hypoventilation may occur. It has been suggested that the magnitude of this response is limited by the imposition of hypoxic drive.4 However, patients have been described with metabolic alkalosis and life-threatening hypoxaemia and hypercapnia.5-6 Metabolic alkalosis also impairs the relationship between ventilation and perfusion in the lung7 and limits systemic oxygen delivery by promoting leftward shift of the oxyhaemoglobin dissociation curve. Third, infusion of large doses of mannitol has been implicated in provoking renal failure.8

Rhabdomyolysis is a critical illness. Its treatment may cause serious complications. Management in an intensive therapy unit is indicated.

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Sir,—I thank Dr Ball for his interest in my case report of a patient who suffered bilateral compartment syndrome after prolonged lithotomy position. The discussion included a description of the treatment of renal failure associated with rhabdomyolysis which should commence with appropriate cardiovascular resuscitation and prompt fluid therapy to encourage urine flow. The renal protective effect of alkalinizing the urine has been demonstrated and results from the fact that urate and myoglobin are more soluble in alkaline urine. In the report I quoted Better,1 who suggested a very aggressive regimen of fluid containing bicarbonate and mannitol, as an example of management guidelines. I acknowledge all of Dr Ball’s comments as valid, but I wish to reply to each point in turn.

1. The aggressive fluid regimen of Better must be tailored to the status and individuality of the individual patient, using appropriate invasive cardiovascular monitoring in the intensive care in the critically ill patient. Better has stated elsewhere that if myoglobinuric acute renal failure has occurred, with reduced renal output, solute load should not be given and regular haemodialysis is indicated.2 3

2. Bicarbonate therapy has been controversial for many years. Bicarbonate is used in two main contexts; first, in the treatment of metabolic acidosis and second, to facilitate alkaline diuresis to enhance excretion of poisons.

Regarding the use of bicarbonate, Arieff proposes that metabolic acidosis with increased anion gap can be classified into those conditions in which tissue hypoxia is either present or absent.4 In general, when there is metabolic acidosis in the presence of tissue hypoxia, available tissue oxygen is not adequate for the individual’s needs. Treatment of metabolic acidosis with sodium bicarbonate tends to limit further available oxygen because of its effect on the position of the oxygen dissociation curve, and this leads to increased production of lactate thereby worsening the metabolic acidosis. Arterial PCO2 is directly proportional to production of carbon dioxide and inversely proportional to alveolar ventilation.5 Thus increased generation of carbon dioxide by administration of bicarbonate increases venous PCO2 if there is impaired cardiac output (resulting in decreased delivery of venous carbon dioxide to the lungs), or decrease in alveolar ventilation. Carbon dioxide readily diffuses into cells such that intracellular PCO2 approaches that of mixed venous blood. In a closed system, carbon dioxide accumulates after administration of bicarbonate thereby decreasing intracellular pH.6 Several workers have demonstrated the negative inotropic effect of bicarbonate on the ischaemic myocardium.7

However, sodium bicarbonate may be beneficial to patients with metabolic acidosis in the absence of tissue hypoxia.8 Bicarbonate dialysis is tolerated better than acetate dialysis without cardiovascular instability, in critically ill patients with cardiorespiratory dysfunction,9 10 possibly because of a direct effect on myocardial contractility11 or through the effects of alkalosis on peripheral resistance.12 Bicarbonate may be used in alkaline diuresis to enhance excretion of toxins, including myoglobin and salicylates. In the treatment of crush syndrome, Better has advocated early volume replacement and, if urine flow commences, he recommends forced solute alkaline diuresis to prevent myoglobinuric acute renal failure.2 3 Noji recommends a similar technique.13 Likewise, in the treatment of salicylate poisoning, alkalization has been shown to enhance excretion of the poison.

3. Mannitol. Some workers who have described mannitol nephrotoxicity syndrome have found that it occurs only after high doses of mannitol (e.g. > 200 g day−1) but not at lower doses.10 20 It may occur at slightly lower doses if patients have pre-existing impaired renal function.20 It appears that at a low dose mannitol acts as a renal vasodilator, while in high doses it is a renal vasoconstrictor.19 21 Mannitol-induced acute renal failure responds promptly to haemodialysis with rapid resolution of anuria and recovery of renal function.19 21

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J. TUCKEY
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Sir—Regardless of the presence or absence of space-occupying intracranial lesions, suxamethonium in humans and animals produced significant increases in intracranial pressure (ICP) in many investigations. Lanier, Milde and Michenfelder found that the increase in ICP with suxamethonium was accompanied by EEG arousal and an increase in cerebral blood flow (CBF). The increase in CBF was disproportionately greater than that expected from a concomitant increase in P\textsubscript{A\textsubscript{\text{CO}}\textsubscript{2}}. In their subsequent study, they demonstrated that an increase in muscle spindle afferent activity paralleled the increase in CBF. Fasciculations in the muscles of the neck, causing strain in the muscles, might also be a factor contributing to increased ICP with suxamethonium. Pretreatment with paralysing doses of pancuronium did not block afferent muscle activity or the CBF response. The increase in ICP was also blocked by pretreatment with thiopentone.

In our institution we also studied the effects of suxamethonium on middle cerebral artery flow velocity using transcranial Doppler during intubation of the trachea in 10 head injured patients and found a significant increase in flow velocity after an i.v. bolus of suxamethonium (unpublished data until now). The mechanism of how suxamethonium influences ICP and CBF in humans remains uncertain. In contrast, there are as many studies concluding that ICP is not influenced by suxamethonium. Kovarik and colleagues found that in brain-injured patients, suxamethonium did not alter cerebral blood flow velocity, cortical electrical activity or ICP. The literature on the effect of suxamethonium on ICP in animals and in humans has not provided firm indications that suxamethonium per se increases ICP. Furthermore, inclusion of other drugs, differences in anaesthetic states and concurrent stimuli such as tracheal intubation further complicates analysis of the effect of suxamethonium on ICP. Increases in ICP seen with suxamethonium may also be caused by autoregulatory decreases in cerebral vascular resistance secondary to systemic hypotension. Braun and colleagues have made a valuable contribution to the literature, but despite many studies, including those of Braun and colleagues evaluating the effects of suxamethonium on ICP, controversy remains.

Respiratory problems and pulmonary infections often occur after head injury because of neurological dysfunction and laryngeal incompetence. Parr and Manara stated in their letter that suxamethonium may have an onset of action approaching that of suxamethonium but its long duration of action makes it unsuitable for use as the first choice neuromuscular blocking agent in the management of patients with severe head injury. I cannot imagine any situation where early extubation of the trachea of a patient with severe head injury is planned and after intubation the clinical duration of action should be of minor concern as the effect of suxamethonium on cerebral dynamics is not controversial. The only reason for the statement of Parr and Manara, therefore, is difficult airway management. Obviously we agree with them regarding patients with ileus, but those with severe head injury are at risk of aspiration with or without neuromuscular block. After intubation of the trachea of head injured patients using either vecuronium or suxamethonium, Redan and colleagues concluded that paralysis and tracheal intubation in the emergency department is safe and may be a potentially life-saving manoeuvre in combat trauma patients. Nevertheless, a short onset of neuromuscular blocking agent is preferable as ventilation of the lungs of patients without intubation is risky. Intubation of the trachea of head injured patients in the emergency room should not be delayed as it may be for elective surgery or even for the patient with ileus. If intubation is impossible any other methods of airway access are required, for example emergency tracheotomy is indicated. Is suxamethonium superior because its duration of action lasts only a few minutes? The use of additional boluses of suxamethonium because of multiple intubation attempts may be accompanied by bradycardia, dual block and the other side effects of this drug.

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Intraoperative heat administration

Sir,—The study by Beck and colleagues1 claimed that warming with a water perfused oesophageal heat exchanger was significantly more efficient at preventing intraoperative cooling than warming using ventilation with warm humidified gases, which was statistically ineffective compared with no heating. However, the study design leaves this conclusion in some doubt.

Approximately 75% of the benefit of ventilation with warm humidified gas (airway warming (AW)) arises from prevention of loss of heat and moisture which occurs during respiration, with the remainder comprising condensation of hot water vapour and a very small amount from the increased gas temperature.2 These last components are very small using a temperature of 39°C, and the system used provides only approximately 85% of the thermal gain possible with the temperature recommended for use in the treatment of accidental hypothermia (i.e. 45°C).2 Unfortunately, in the two other groups the patients’ lungs were ventilated with cool (room temperature) humidified gas. This would provide approximately 65% of the benefit achievable through AW. The study therefore compared the effect of 85% AW with 65% AW and 65% AW plus the oesophageal water warming system. A fair evaluation of the thermal value of AW would have required the two other groups to undergo ventilation with dry gas.

The gas temperature of 45°C was selected for use during treatment of accidental hypothermia to avoid thermal damage to the mouth, pharynx and larynx when the system is used with a face mask. Because of its ample blood supply the respiratory tract below the larynx is very resistant to thermal damage. For example a patient at 24.3°C undergoing ventilation for 24 h with saturated gas at 80°C suffered only mild, reversible damage to the respiratory tract.3 Temperatures of 350°C with dry gas or steam at 94–104°C are needed to cause burning to the trachea.4

Finally, while the oesophageal water circulating system has been used effectively in the treatment of accidental hypothermia, it has disadvantages in that the equipment is suitable only for hospital use; there is a danger if the thin rubber balloon bursts or tears and the GI tract is flooded with water; insertion of the tube may be impossible if the patient’s teeth are clenched; and there is the risk of trigging insertion of the tube. Airway warming can be used in the field and does accelerate the rate of rewarming to a statistically significant degree.5

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Sir,—We thank Dr Lloyd for his interesting comments on our recently published study.1 As clearly stated both in the title and in the text, our goal was to evaluate the efficacy of two warming methods in a clinical intraoperative setting, under standardized conditions. For this reason we chose a temperature level (39°C) that may be judged too low, but which is higher than the usual temperature (30–34°C) used during clinical anaesthesia. Moreover, we had to modify the Kontron Pearl humidifier that in its standard configuration for human use cannot exceed the 37°C limit. We were definitely not willing to use the high temperature used by some for treating severe accidental hypothermia.

According to Hendrickx, Trahey and Argentieri2 heat lost by one of our pigs (34°C) undergoing ventilation at a Vt of 4 litre min⁻¹ in dry air–oxygen at room temperature (24°C) would be approximately 24.9 kJ h⁻¹. Humidification (100%) of inspired gas at room temperature (24°C) would reduce heat loss to approximately 12.1 kJ h⁻¹ thus supplying (through condensation heat) less than 50% of heat loss. Humidification (100%) of inspired gas at 39°C would provide approximately 20 kJ h⁻¹ (4.5 by increased gas temperature and 15.5 by condensation heat) which is 80% of the theoretical heat loss, approximately 8 kJ h⁻¹ (66%) more than humidification at room temperature. Therefore, humidification alone at room temperature would supply approximately 60% of the heat supplied by humidification at 39°C.

However, the humidifier that was used in our experiment at 24°C provided 80% relative humidity. In this situation, humidification of inspired gas at room temperature (24°C) would reduce heat loss to approximately 14.6 kJ h⁻¹ thus supplying (through condensation heat) approximately 40% of heat loss or approximately 50% of the heat supplied by 39°C humidification.

We agree with Dr Lloyd’s conclusions; our experiment design did not allow comparison of warm humidification with a true control or with oesophageal warming alone. Our study compared the effect of ventilation with warm (39°C) humidified (100%) gases, which is the 100% airway warming (AW) in this experiment, with 50% AW and dry gases and an oesophageal warming system. A strict “in vitro” procedure should have supplied 0% AW during the warm humidification period.

We do not agree with Dr Lloyd’s conclusion that our study is invalid. We planned to compare two heating systems in a controlled, standardized clinical condition; ventilation with dry gases does not exist now in clinical practice. Moreover, the gas from the hospital supply system has a relative humidity at room temperature of approximately 40% this makes it impossible to use dry gas. In any case, by avoiding the use of a cold humidifier in the warm humidification period, we would have compared the effect of 100% AW with 28°C AW and with 28°C AW plus oesophageal warming.

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Extraction of nitric oxide and nitrogen dioxide using molecular sieve 5A

Sir,—We read with interest the article by Poulton and colleagues1 demonstrating the efficacy of a molecular sieve in absorbing both nitric oxide and nitrogen dioxide. However, we have some reservations with regard to the clinical applicability of this bench test. We have recently used a molecular sieve 5A, pellet size 1.5 mm (SW Scientific, Bristol, UK) in a vertically mounted Waters canister (id 6.5 cm, length 10 cm) for the purpose of scavenging nitric oxide and nitrogen dioxide from the expired gas of patients receiving inhaled nitric oxide during mechanical ventilation. However, after using this method in two patients we have failed to demonstrate any efficacy.

Both patients were given inhaled nitric oxide via a Servo 900C ventilator (Siemens Elba AB, Sweden) and humidification of the inspired gas was provided by a heat and moisture exchanger. At each dose of nitric oxide (0, 2, 10, 20 and 0 ppm) both nitric oxide and nitrogen dioxide were measured in the expired gas immediately before and after the Waters canister using a calibrated electrochemical analyser (Micro Medical, Kent, UK). There was no measurable nitrogen dioxide with any dose of nitric oxide (table 1). As can be readily seen, we could not repeat the results of Poulton and colleagues. There are two possible explanations for this. First, we did not heat the zeolite to 400°C for 24 h as we had been informed this was not necessary for efficacy (personal communication, SW Scientific). Second, the gas flowing through the Waters canister in our case not only contained nitric oxide and oxygen but also nitrogen, carbon dioxide and water. It is likely that...
that nitrogen, carbon dioxide and water alter the performance of the sieve and although this was mentioned by Poulton and colleagues the effect was not quantified. If it is really necessary to dry the zeolite by prolonged exposure to high temperature then the effect of expired water vapour on the function of the sieve may be significant. Further work is necessary to clarify these points.

In conclusion we agree that efficient scavenging of nitric oxide and nitrogen dioxide from a dry carrier gas comprising oxygen only can be achieved using a molecular sieve 5A but we have been unable to reproduce this result in clinical practice. We feel that extrapolation of this bench test result to clinical practice should be undertaken with caution.

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Sir,—Thank you for the opportunity to respond to the comments of Findlay, Swart and Smithies relating to the use of molecular sieve 5A as a scavenging device in nitric oxide therapy. We feel that these authors have been misinformed about the crucial importance of dehydrating the sieve before use. As we have outlined in our article, the internal structure of the sieve is charged and therefore intensely hygroscopic; 125 g of dry sieve may be expected to take up 40 g of water. When the internal pores are saturated, the zeolite loses its ability to function as a sieve. Water vapour entering the sieve also reacts with nitric oxide to form nitric acid which leads to degeneration of the sieve. Clearly if our bench top experiment is to be applied clinically, all possible precautions must be taken to exclude water from the sieve. We have already alluded to the possible use of a bed of silica gel. Condensation trapping devices could also be used.

We do not believe that any other technical errors are to blame for the large discrepancy between our own observations and those of Findlay, Swart and Smithies. The sieving capabilities of the zeolite depend on its existence in the dehydrated state.

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