CORRESPONDENCE

Malignant hyperthermia and compartment syndrome

Sir,—We were interested to read the report by O'Donnell and colleagues [1] of a patient with compartment syndrome occurring as a complication of malignant hyperthermia (MH). We recently managed a similar case. A 29-yr-old, previously healthy man presented for repair of a fractured tibial plateau. After induction of anaesthesia with propofol, midazolam and fentanyl, he was allowed to breathe isoflurane and 66% nitrous oxide in oxygen. After 1.5 h he developed stridor which persisted despite attempts to manipulate the laryngeal mask airway. He was then given suxamethonium which resulted in the rapid onset of miaseter and generalized muscle spasm. His temperature, measured nasally, increased rapidly to 40.3 °C, arterial Pco2 reached 12.1 kPa and he developed a metabolic acidosis with a base deficit of 12.3 mmol litre⁻¹. Serum potassium was 6.8 mmol litre⁻¹ and creatinine kinase increased to greater than 190 000. Despite the severity of this fulminant attack of MH, the patient made a rapid response to dantrolene (total dose 3.5 mg kg⁻¹). Within a few hours his metabolic derangement was corrected and his trachea was extubated on the following morning.

Over the next 12 h he developed marked swelling of his right forearm, right calf and foot. This was associated with restricted movement, pain exacerbated by passive extension and decreased sensation to light touch. A diagnosis of compartment syndrome was made and he underwent fasciotomies to the relevant muscle compartments under general anaesthesia using propofol, fentanyl and atracurium. This and subsequent anaesthetics for wound closure were uncomplicated and the patient made a full recovery with no loss of function in either limb.

With greater awareness among anaesthetists and the availability of dantrolene, patients with fulminant MH are much more likely to survive than previously [2]. Such patients are at risk of developing compartment syndrome and we emphasize the importance of clinical signs in the early diagnosis of this potentially serious complication of MH. When indicated, fasciotomy should be performed and we suggest that the risk of anaesthesia after recovery from fulminant MH is small, and not sufficient to justify avoiding surgery.

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Sir,—O'Donnell and colleagues described bilateral forearm swelling complicating an episode of malignant hyperthermia [1]. Compartment syndrome of the forearm can lead rapidly to irreversible tissue damage and Volkmann ischaemic contracture [2]. In this case the patient recovered after forearm elevation.

Compartment syndrome is defined as “a condition in which increased pressure within a limited space compromises the circulation and function of the tissues within that space” [3]. The perfusion pressure of the tissues within the compartment is defined as compartment mean arterial pressure — “tissue pressure”, and this determines oxygen delivery. Elevation of the affected compartment reduces compartment arterial pressure. Venous pressure of the compartment is also reduced by elevation, but not below tissue pressure [4]. If compartment tissue pressure is increased sufficiently to compress veins, then venous pressure does not influence blood flow in the compartment. Thus compartment syndrome should not be managed by elevation.

Elevation of the lower limb after closed fracture of the femoral shaft in children resulted in compartment syndromes leading to ischaemic contractures [5]. Elevation of limbs causes a significant reduction in compartment perfusion pressure [4] and tissue oxygenation [6, 7].

Another important aspect of this case concerns the diagnosis and subsequent monitoring of the patient’s compartment syndromes. I agree with O'Donnell and colleagues that the diagnosis is mainly clinical, and I note that they mention that “direct percutaneous measurement of intracompartmental pressures has been proposed.” I contend that there was a strong case for direct monitoring in the case they describe. Matsen, Wingquist and Krugmire [3] advise that measurement of tissue pressure is indicated in “(1) the evaluation of a compartment syndrome in which the diagnosis cannot be made or ruled out with certainty and (2) the prospective evaluation of a compartment syndrome at high risk, as in a patient with a severely swollen limb but with no neural deficits”. The second scenario occurred in this patient, as the swelling at the wrist itself necessitated release of her watchstrap, yet sensation was preserved clinically. The severity of complications is related inversely to the promptness of surgical intervention [8]. Information on the perfusion of affected compartments can be gained readily by the relatively simple bedside procedure of catheter insertion under local anaesthesia. This gives valuable and continuous information on tissue viability within the measured compartment and would have allowed more exact diagnosis and management in this case.

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Sir,—Ball has criticized the management of our patient [1] on the grounds that elevation of a limb may result in reduction in compartment arterial pressure, with a resultant decrease in local perfusion pressure if tissue pressure exceeds venous pressure. He cites the occurrence of ischaemic contractures in children with closed fracture of the femoral shaft who were managed by elevation [2] as evidence that compartment syndrome should not be managed in this way.

However, we remain unconvinced that it is appropriate to apply conclusions from compartment syndromes associated with limb fractures to the type of case which we described. We contend that, although the article by Thompson and Mahoney [2] reported ischaemic contractures in children with femoral shaft fractures
treated by elevation, it cannot be concluded that elevation per se was the cause of compartment syndromes. During the 17-yr period of their study (1931–1947) several practices which might be regarded today as less than optimal were used, for example application of adhesive splinting to the limb “after half to three quarters of an hour when the patient is warm and drowsy and out of shock”. The limb was then elevated in traction applied over the end of the cot frame; although no details of the degree of elevation were quoted, the photographic illustrations in the article indicate that this was in the order of 35–45° from the horizontal. Two patients had a severe head injury (but were presumably not undergoing ventilation) “which prohibited a head-down position”, but the others were managed in the Trendelenburg position (degree not stated). With one exception, all were children less than 6 yr of age in whom normal mean arterial pressure (MAP) would be in the region of 50–75 mm Hg, and in whom this degree of elevation of the limb above the level of the heart would have caused a significant reduction in compartment perfusion pressure. We cannot assume that arterial oxygenation, fluid resuscitation or local pressure area care were ideal in these patients and it is perhaps surprising that the incidence of ischaemic contracture was not higher than reported.

Nevertheless, we concede that the points raised by Ball are valid, and perhaps we did not provide sufficient detail of posture in our original article [1]. In our patient the forearms were maintained in a moderately elevated position by resting them on two pillows with the patient in a semi-recumbent position. This effectively produced elevation of the forearm compartments approximately 10 cm above the level of the heart, which would correspond to a reduction in MAP in the region of 7 mm Hg. Although Ball describes it as a “relatively simple bedside procedure”, there is no consensus among orthopaedic surgeons as to its role in the management of compartment syndrome. The equipment required to perform it is not routinely available, and the interpretation of information from a pressure transducer may be misleading unless the technique is familiar to those using it. We also have concerns about the potential infection risks associated with introducing a catheter into a muscle compartment where tissue viability may be already compromised.

We believe that in our patient, who was awake and fully co-operative, the combination of careful attention to arterial pressure, hydration, oxygen therapy and regular clinical assessment by a consultant orthopaedic surgeon provided safe treatment and would have allowed early detection of the requirement for decompressive fasciotomy. If she had required sedation and mechanical ventilation this would not have been the case. We fully agree with Steele and colleagues that fasciotomy should be performed if clinically indicated, and would not have hesitated to arrange surgery and anaesthesia for our patient had it been necessary.

Another complication of the combined extradural–subarachnoid technique

Sir,—Following the case reports of Drs Harding, Collis and Morgan [1], reporting meningitis after combined extradural–subarachnoid anaesthesia in two obstetric patients, we wish to present a further case which highlights an additional clinical point. A 27-yr-old primigravida presented at term plus 5 days for induction of labour for twin pregnancy. She requested extradural analgesia and this was sited at the level of L1–2 using the Carpent technique, that is, mask, gown, gloves and alcoholic chlorhexidine solution to clean the skin. The neurological block produced was unilateral despite re-positioning the patient, withdrawal of the catheter and another dose of local anaesthetic being given. A second extradural was inserted in an adjacent interspace. This produced an improved block, however, analgesia still remained incomplete on the left side.

Approximately 6.5 h later a decision was made to perform lower segment Caesarean section for failure to progress and the patient was keen to be awake for the procedure. The anaesthetist decided to perform a subarachnoid block in view of the continuing inadequacy of the extradural block. This was performed with the patient in the left lateral position using a 25-gaugeatraumatic spinal needle and 0.5% heavy bupivacaine solution 2 ml. Although it is not recorded, marks on the skin suggest the same space as the extradural catheter was used for this procedure. Full aseptic precautions were taken with alcoholic betadine solution being used to clean the skin.

The immediate peroperative period was unremarkable and the patient appeared well when seen on the following morning’s anaesthetic ward round. Approximately 18 h after operation the patient became acutely confused, pyrexial, exhibited both receptive and expressive dysphasia and ignored her left side.

Clinical examination was unremarkable and there were no focal neurological signs. An initial differential diagnosis of acute meningitis was made and i.v. antibiotics were started. Investigations included full blood count: this showed an increased white cell count (13.5 x 10^9 litre^1), differential neutrophil 87% and lymphocytes 10.8%. Lumbar puncture revealed a raised opening pressure of 26 cm H2O, CSF glucose 0.1 mmol litre^1 (patient’s venous sample 5.3 mmol litre^1) and CSF protein 3.89 g litre^1. No organisms were seen on Gram stain or grown in subsequent cultures. A diagnosis of acute meningitis was made and i.v. antibiotics were continued.

She made an uneventful recovery but on day 4 after operation the patient developed a headache consistent with post-dural puncture headache. Conservative management with simple oral analgesics and copious oral fluid intake was ineffective. By day 5 postpartum the patient was requesting further treatment for her headache, posing a clinical dilemma of how to treat the post-dural puncture headache in an immuno-compromised patient. We gave our patient sumatriptan 6 mg s.c. with good clinical effect; the headache completely resolved within 1 h.

This case has many clinical features in common with the first case described by Harding, Collis and Morgan—multiple invasions of both extradural and subarachnoid spaces, acute confusion with dysphasia and no growth of organisms from CSF or blood cultures. It was felt that bacterial meningitis was more likely than chemical meningitis because of the very low CSF glucose concentration in association with neutrophilia. The lack of growth from CSF or blood cultures could have been caused by i.v. antibiotics being started before blood cultures or lumbar puncture.

This case raises two important points for discussion. First, what were the possible mechanisms for introduction of infection, and second, how should a suspected post-dural puncture headache in a patient with recent meningitis be treated? The possible mechanisms for introduction of infection were: use of contaminated local anaesthetic solutions; infection after bacteraemia immediately after the spinal anaesthetic was given; or superficial colonization of the extradural tract with pathogens which were then inoculated into the subarachnoid space by using the same path to perform the spinal component.

It was felt that the first two hypotheses were unlikely; ampicillin from the same batch had been used at induction and ampicillin had negative effects and although meningitis secondary to bacteraemia and central block has been reported [3], it is extremely rare. It would seem that the third hypothesis was the most likely.

Also, how should a patient with presumed post-dural puncture headache unresponsive to conventional therapy in whom where blood patch is contraindicated be managed? In our case a single dose of sumatriptan proved successful.


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3. No organisms were seen on Gram stain or grown in subsequent cultures. A diagnosis of acute meningitis was made and i.v. antibiotics were continued.

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6. Also, how should a patient with presumed post-dural puncture headache unresponsive to conventional therapy in whom where blood patch is contraindicated be managed? In our case a single dose of sumatriptan proved successful.
As a result of this case we propose that if a subarachnoid block is performed after prolonged extradural cannulation that it be performed in a separate “clean” space. We also agree with the comments of Dr Wee [4], that the possible increased risk of infection may outweigh the benefits of immediate analgesia using a combined technique. We have therefore suspended the use of the mobile technique in our unit until further evaluation of this technique is undertaken.

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Ovarian hyperstimulation syndrome

Sir,—We read with interest the editorial on ovarian hyperstimulation syndrome by Williamson and Mushambi [1], who emphasized that with increasing popularity of assisted reproduction techniques, complications such as ovarian hyperstimulation syndrome (OHSS) may be encountered more frequently by anaesthetists and intensivists. During the past 18 months we have treated three patients with severe OHSS in our intensive care unit (ICU).

Although it is a rare complication of exogenous gonadotrophin-induced ovulation, severe OHSS is a life-threatening condition, and ICU admission is appropriate to enable careful monitoring, meticulous fluid therapy and abdominal paracentesis. Occasionally, ascites production may fail to decrease with conservative management. In such patients i.v. re-infusion of ascitic fluid (which has been used primarily for the treatment of diuretic-resistant ascites caused by liver cirrhosis and malignancy) has recently been reported [2]. Various techniques have been described, the common principles of which involve paracentesis, ultrafiltration and i.v. re-infusion of the concentrated, protein-rich ascitic fluid [2, 3]. Febrile reactions, thrombocytopenia and coagulopathy are common complications which may limit the success and safety of these techniques.

We successfully used a continuous ascitic recirculation technique for the treatment of a patient with severe OHSS, in whom ascites production exceeded 5 litre per day, which avoided these potentially serious complications [4]. At the time of ICU referral, ascitic abdominal distension, bilateral pleural effusions and recurrent episodes of hypovolaemia had caused significant respiratory and renal compromise. Ultrason-guided paracentesis and i.v. fluid therapy failed to improve the rate of ascites production and continuous ascitic recirculation was therefore commenced, using a haemofiltration pump (Hospal BSM 225C), plasma filtration filter (Asahi Plasmalof OP-05) and an endotoxin-bacteria retaining re-infusion filter (Pall Biomedical CPS-02) (fig. 1). The procedure produced rapid improvement in renal and respiratory function and was continued for a total of 15 days. No coagulation disturbances or adverse haemodynamic effects were observed, and minor febrile episodes responded on each occasion to replacement of the re-infusion filter. Although further evaluation of this technique is necessary, we have also used it successfully in a patient with refractory ascites secondary to hepatic disease [5], and consider continuous ascitic recirculation a safe and effective treatment option for complicated severe OHSS.

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Figure 1 Continuous ascitic recirculation system incorporating sump (A), haemofiltration pump (B), plasmapheresis filter (C), infusion pumps (D, G), filtrate sump (E), Pall biomedical filter (F) and synthetic colloid (H).
Sir,—Thank you for the opportunity to respond to the letter from Dr Beck and colleagues. We are grateful for their comments and the detailed description of the technique of i.v. re-infusion of ascitic fluid in severe ovarian hyperstimulation syndrome (OHSS).

Aboulghar and colleagues described successful autotransfusion of ascitic fluid in only three patients with OHSS [1] and it may prove to be a method of treating ascites in this syndrome. However, as stated in our editorial [2], a controlled randomized study should be undertaken before it can be recommended as a safe and effective treatment of severe complicated OHSS.

The suggestion made by Beck and colleagues and by us [2] that OHSS may be encountered more frequently with increasing popularity of assisted reproduction techniques is supported by two recent publications which may not have been readily available to anaesthetists. First, the issue of guidelines by the Royal College of Obstetrics and Gynaecology on “Management and prevention of OHSS” [3] and second, a recent commentary in the British Journal of Obstetrics and Gynaecology by Jenkins, Mathur and Cooke [4] on the management of severe OHSS.

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Postoperative analgesia for arthrolysis of the elbow joint

Sir,—We read with interest the case report of Parikh, Rymaszewski and Scott on prolonged postoperative analgesia for arthrolysis of the elbow joint [1], detailing the successful use of continuous suprascavicular brachial plexus block. We strongly commend their use of a regional technique for this operation, and the excellent analgesia achieved justifies their choice.

However, we were curious as to the reason for tracheal intubation and the resulting use of suxamethonium. The duration of surgery was 60 min and the usual position for this procedure does not require intubation. As the plexus block was so successful (“full sensory and motor block of the limb was achieved”), the doses of enflurane and supplementary fentanyl required were therefore such that the patient would tolerate tracheal intubation. Because of the recent furore over the use of suxamethonium in children and adolescents [2], we feel that its use should be avoided where possible, even in adults. Perhaps a laryngeal mask airway, allowing the patient spontaneous respiration, would have been a more appropriate choice of airway management, reducing enflurane requirements and eliminating the need for further opioid.

Shoulder surgery is performed routinely in our institution using a combined technique of intracavalar block, together with general anaesthesia with the laryngeal mask airway and this avoids the use of suxamethonium or other neuromuscular blockers. Review of almost 200 cases, we have found supplementary opioids unnecessary, and the requirements for halogenated agents of the order of 0.4 MAC. The technique could apply equally well to this otherwise excellent case report.

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Sir,—We are encouraged by the remarks of Drs Frizelle and Zetlaoui regarding the management of postoperative pain relief of our patient.

The duration of surgery for this operation often exceeds 60 min at our hospital. The patients are frequently placed in a lateral position during surgery. The anaesthetic agents used and the method of general anaesthesia undertaken during operation may indeed be altered to take account of local circumstances.

Further studies to establish the safe use of this method of pain relief in orthopaedic wards are in progress at our institution.

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Animal toxins

Sir,—Karalliedde stated that there is no specific remedy for poisoning by ciguatoxin [1]. Palfax and colleagues reported a series of 24 patients with ciguatera; they exhibited neurological and neurosensory manifestations, including coma in two cases, and they responded dramatically to 20% mannitol, to a maximum dose of 1 g kg

Sir,—As there is no specific antidote, management of ciguatera with mannitol, particularly within the first 24 h of exposure, is of concern to many clinicians. Palfax and colleagues’ report is of interest and allows us to discuss the possible mechanisms of action for mannitol as treatment in ciguatera.

In animal toxins, such as scorpion and spiders, it is known that treatment with mannitol increases the excretion of toxins. There appears to be little effect on the biochemical basis of such action, or on any other intervention, and the improvement in symptoms is postulated to be due to mannitol improving the patient’s fluid balance.

However, there is evidence of a direct mechanism for mannitol in the treatment of ciguatera fish poisoning. In a series of 24 patients with ciguateromegaly, Palfax and colleagues’ reported that the patients responded dramatically to 20% mannitol, to a maximum dose of 1 g kg

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Broadgreen Hospital
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Sir,—As there is no specific antidote, management of ciguatera fish poisoning consists of symptomatic treatment. Mannitol was found to be useful in altering the clinical course of the neurological symptoms but had no effect on the gastrointestinal symptoms and the less frequent cardiovascular effects. Most reports on the use of mannitol have been from uncontrolled studies and of an empirical nature. I agree with Dr Whitehead that at present mannitol therapy will be useful in the management of the neurological symptoms of ciguatera poisoning.

L. KARALLIEDDE
Queen Elizabeth Military Hospital
Woolwich, London


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Correspondence

Retrograde cannulation of the jugular vein

Sir,—We read with interest the report by Fumagalli and colleagues [1] on the erroneous positioning of the jugular venous bulb catheter in the subarachnoid space. While we sympathize with the authors, we do not support their recommendation of radiographically examining all retrograde catheters with contrast medium using lateral and anteroposterior views. Moreover, this would not have prevented the potential risk of herniation, as suggested by the authors.

The authors admit that the catheter was placed by a person inexperienced in antegrade but not retrograde jugular cannulations. We feel that this may have been the problem. In our experience with more than 400 catheters placed at our institutions (some of which have been reported), the only complication we encountered was a 1–2 % rate of carotid artery puncture which was controlled by firm pressure [2]. Apart from defining the anatomical landmarks with great care (the two heads of the sternocleidomastoid muscle), the needle should not be advanced more than 3 cm below the skin, because the internal jugular vein is normally very superficial at that level. We can only assume that the needle must have been advanced too far and must have passed through the vertebral foramen for the catheter to terminate in the subarachnoid space.

Jugular venous bulb catheters have been shown to play an important role in the management of comatose and head injured patients [3–5]. They have also been shown to be beneficial in the intraoperative management of patients undergoing neurosurgery [2]. Provided the correct technique is followed, the placement of these catheters carries no more risk of complications than the placement of antegrade central venous catheters.

Localization and confirmation of the placement of catheters necessitates aspiration during localization of the vessel. Subsequent radiographic confirmation with contrast medium would not prevent aspiration of CSF and possible herniation if the catheter had been placed in the subarachnoid space. Rather, careful technique and recognition of free flow of venous blood are of paramount importance. Plain lateral or anteroposterior radiographs of the neck should be sufficient to confirm correct placement, after all we do not confirm the position of central catheters using contrast media! We recommend that a person inexperienced with retrograde catheterization should only perform this technique under supervision.

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Sir,—The technique of retrograde jugular cannulation described by Matta and Lam is very similar to the one used in our ICU. In the case we reported, we believe that the guidewire and not the needle lacerated the posterior wall of the jugular vein (as shown by blood aspiration during placement) to finally lie within the subarachnoid space.

In our ICU we have experience with more than 150 retrograde jugular cannulations performed by senior intensivists and we reported six carotid artery punctures and one erroneous positioning in the subarachnoid space. We are surprised that authors with much more experience than ours do not report major complications other than carotid artery puncture. In fact many cases of complications after jugular catheterization have been reported. Zullo, Wallerson and Lang [1] for example described the formation of a fistula between the common carotid artery and internal jugular vein. Robinson, Jewkes and Kendall [2] noted a vertebovertebral arteriovenous fistula as a complication of internal jugular catheterization. In difficult cases these and other authors [3–5] suggest the use of two-dimensional imaging echography coupled to colour Doppler examination to guide central venous cannulation. McGee and Mallory [6] included spinal cord cannulation and neck haematomas causing severe compression and obliteration of the trachea.

In conclusion, we believe that the possibility of rare but dramatic complications exists and we should be able to prevent and recognize these rare complications. We suggest radiographic examination and, when it is useful for doubtful catheter positioning, even contrast media.

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Comparison of ropivacaine and bupivacaine for extradural analgesia

Sir,—The results of McCrae, Jozwiak and McClure [1], comparing ropivacaine and bupivacaine, were predictable. There is no doubt that local anaesthetics work, and if given in sufficient volume, in a concentration that corresponds to the upper, flatter part of the dose–response curve, an 80–90 % success rate might be expected when given extradurally to women in labour. No difference in analgesic efficacy would have been anticipated between these groups.

Given that there would be no difference in analgesia, it might seem reasonable to compare variables such as motor block and hypotension. However, although the two local anaesthetics appear to have equivalent analgesic effects, because their doses lie on the flatter part of the dose–response curve, there is no reason to believe that they are comparable. If given in sufficient volume, both drugs produce a high level of analgesia which is comparable to that produced by intravenous opioids. Nevertheless, the ability to identify comparable doses of extradural local anaesthetics now exists, rendering studies of this nature obsolete. Critical differences between these two local anaesthetics are unlikely to emerge unless comparable doses are used, preferably on a steeper part of the dose–response curve.

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M. DRISNER
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Sir,—We would accept that dose–response curves exist for bupivacaine and ropivacaine as measured by sensory, motor and sympathetic block. Most mother volunteering for a study would wish to be assured that in terms of sensory block they would sit on the upper flat part of the dose–response curve. The response of motor block is more difficult to measure clinically and we would re-emphasize that a quantitative measure of motor block by isometric contraction of a single muscle group is more valid than the quantitative assessment of spread by the Bromage scale.

We would not accept that studies of the nature of our own are obsolete, as only when clinicians are assured that the desired effect is obtained and the drug is safe, can they then address the issue of minimum local anaesthetic concentration (MLAC), as proposed by Lyons, Drener and Wilson. The concept of MLAC is flawed however, as it is not only the dose (mass) of local anaesthetic drug but also spread which influences “response”. A. F. McCrae
H. Joziwak
J. H. McClure
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Mivacurium and prolonged neuromuscular block
Sir,—The two recent case reports [1, 2] describing prolonged recovery after administration of mivacurium can only be part of a growing trend of such problems with the use of this drug. In his editorial, Bevan [3] predicted that because of abnormal plasma cholinesterase, there could be a prolonged block occurring at a rate of 1 in every 3000 patients after a full dose of mivacurium but did not commit himself to quantifying how long this would last. I would venture to suggest that the 3 h given by Fox and Hunt [2] as a typical time to full recovery after administration of mivacurium to a homozygote for the atypical or silent gene, or a heterozygote for the atypical and silent gene, is on the conservative side. The Danish Cholinesterase Unit [4], investigating mivacurium in known atypical homozygotes, administered 0.03 mg kg$^{-1}$ (one-sixth of the dose of Fox and Hunt) to five such patients. They found extensive prolongation of the block even at these low doses, with one patient showing no return of T1 for more than 2 h. A case report by Goudsouzian, d’Hollander and Viby-Mogensen [5] showed that a healthy young man may have a block of 8 h duration after mivacurium if he is an un diagnoed abnormal homoygote for the plasma cholinesterase gene. The treatment that Bevan recommended included support of ventilation and sedation until antagonism can be attempted when there is some evidence of spontaneous neuromuscular recovery. Whatever the length of the prolonged apnoea, it is likely to be at least a few hours and if mivacurium is to be used in such situations as day-case anaesthesia, the inconvenience of finding a patient to be an unexpected homozygote for any of the abnormal genes, thus needing sedation, ventilation and admission to an intensive care following mivacurium, would be maximized.

On a point of nomenclature, the method of abbreviating genotypes, as used by Fox and Hunt (EF/E$^+$), is based on the mistaken belief that there were two loci for the plasma cholinesterase gene. A more appropriate method of abbreviating the genotype of such individuals is suggested by La Du and colleagues [6] whereby the genotype on each DNA strand is ascribed a letter or letters and each strand is separated by a slash. If the case of Fox and Hunt truly had one atypical and one silent gene then the new abbreviation would be AS. However, as La Du and colleagues point out, most atypicals also contain another mutation and the new system could take this into account. For example, an atypical phenotype could actually be caused by a AK/AK, A/AK or AK/S genotype. This is a truer representation of what mutations have been inherited and although these nomenclature changes may not always make a difference to the clinical anaesthetist, it should help when family studies are performed.

L. Davis
Anaesthetics Unit
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Sir,—We thank Dr Davis for pointing out the errors in nomenclature of abbreviating the genotypes of patients with abnormal plasma cholinesterase. Dr Davis ventures to suggest that we give 3 h as a typical time to full recovery after administration of mivacurium to a homozygote for the atypical or silent gene or a heterozygote for the atypical and silent gene. We did not infer and nor do we imply that 3 h was or is a typical time to full recovery; it just happened to be the time for full recovery in our patient. In discussion we also drew attention to the work from Denmark [1], suggesting a time to full recovery of the order of 6–8 h in adults.

M. Fox
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Peterborough

Preoperative fasting for paediatric anaesthesia

Sir,—We were interested to read Dr Weaver’s comments on preoperative starvation times in children in light of recent reappraisals of current practice [1, 2]. We have also audited practice, albeit in a different hospital setting. Data from 531 patient were collected over a 6-week period during 1993, which represented 66% of all patients anaesthetized during this period at Birmingham Children’s Hospital. Median fluid fasting times are shown in table 1, stratified by child’s age.

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Median</th>
<th>Mean</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>0—1 (n = 75)</td>
<td>5.0</td>
<td>6.0</td>
<td>2—16</td>
</tr>
<tr>
<td>1—5 (n = 196)</td>
<td>6.0</td>
<td>8.6</td>
<td>1—20</td>
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<tr>
<td>&gt;5 (n = 260)</td>
<td>8.0</td>
<td>9.2</td>
<td>2—24</td>
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Table 1: Median, mean and range of starvation times (h) from last drink, in children according to age.

These median starvation times are in broad agreement with the first audit of Weaver before revised guidelines to parents were introduced [1]. In our audit, prolonged starvation occurred more often in day-cases than in inpatients. The most striking feature of our data was the bimodal distribution of fasting times in day-case children over the age of 1 yr. The probable cause is the timing of surgery, morning cases being started overnight. In general, the older the child the longer the fasting time because the youngest children tend to be scheduled early on the operating list as staff are more aware of the consequences of prolonged starvation in young children, and because of a smaller sample of children aged less than 1 yr.

We believe there are two distinct problems. First, our unit, in common with many others, admits day-case patients by 08:30, which is often too close to the scheduled time of anaesthesia to allow a clear fluid drink to be given to the child. Revising the guidelines to parents, either by letter or pre-admission clinic, is one method of addressing this and overcoming the natural reluctance to wake a sleeping child to give him/her a drink. However, as Weaver found in his second audit, there are still some children who fast for unduly long periods. The second problem occurs in hospital where natural caution by all staff leads to many inpatients also having prolonged starvation times. Surgical colleagues compound the problem by altering the order of the published lists at short notice, or by using day-cases as “fillers”.

Ideally, the time of surgery for each patient should be known in advance and adhered to. Realistically, however, we must continue to emphasize the importance of this issue. Recent evidence suggests that a clear fluid drink, 2 h before operation in elective cases, is safe [3]. We believe that, wherever practicable, a drink of clear fluid 3 h before operation should be prescribed by the anaesthetist as part of the preoperative assessment. This allows some leeway for variations in the timing of the operating list and effectively focuses the attention of all concerned, doing much to relieve preoperative distress in children.

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Hypothermia during liver transplantation

Sir,—The recent study by Russell and Freeman [1] documented the changes in core temperature which can occur during orthotopic liver transplantation. They demonstrated that the forced warm air convective heating blanket appears to be more efficient than the electric under blanket or forced warm air undermattress, in maintaining peroperative body temperature. Since the Scottish Liver Transplant Unit opened in November 1992 there has been the clinical impression that hypothermia has been less of a problem than was anticipated from data from other centres. We have reviewed retrospectively the anaesthetic records of 20 patients who had elective liver transplantations in our unit. We found that mean blood temperature (from a pulmonary artery catheter) was 35.6 (± 0.69) °C at the start of surgery, and the lowest temperature was 35.0 (1.03) °C. Mean duration of surgery was 8.1 (1.7) h. Mean core body temperature at the end of surgery was 36.9 (1.4) °C, almost identical to the value obtained with the warm air convective heating blanket, although the mean minimum temperature was closer to that seen with the two other methods. A feature of our data however is the greater variability, as reflected by the higher SD (1.4 compared with 0.3 in the study).

Our routine method of temperature conservation involves wrapping the patient’s arms and legs in gangee surrounded by polythene. A circle breathing system, and heat and moisture exchanger are used routinely and all fluids are given via two “level one” rapid infusion systems. A heated water blanket under the patient is maintained at 40 °C throughout the procedure. In addition, the theatre temperature is generally much higher than average, often to the point of being uncomfortable. This is not through choice but rather because surgery is undertaken in a converted theatre built 125 years ago. The heating system consists of heated plates on the walls and ceiling. While very effective, there is no scope for fine adjustment such that ambient temperature can vary significantly from day to day. It is not uncommon for patients to be hyperthermic towards the end of operation (as in nine of the 20 cases selected). While our data are not directly comparable with those collected prospectively by Russell and Freeman, they do suggest that simple measures may be effective, particularly if ambient temperature is kept high. However, specifically designed devices may give more predictable results and also make conditions more tolerable for surgeons and other theatre personnel.

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Sir,—Morris has previously demonstrated that it is possible to maintain normothermia by manipulating the temperature of the operating theatre [1, 2]. If the temperature was between 24 and 26 ºC, all patients remained normothermic; however on 13 and 24 ºC, 70% of patients remained normothermic and below this level all patients became hypothermic (below 36 ºC oesophageal). These were patients undergoing a variety of intra-abdominal and extra-abdominal procedures and with a variety of anaesthetic techniques.

It would be interesting to know what the ambient temperature of the operating theatre was in the study of Walsh, Philips and Armstrong and to compare it with the results reported by Morris. There would seem to be considerable variation in patient temperature at the end of surgery and presumably some were hyperthermic. The large variability may in itself be of some concern; it is obviously preferable to have patient temperatures closely grouped within the physiological range than to have nine patients hyperthermic and possibly a similar number hypothermic. The increased precision of temperature control allowed by patient-specific heating devices must be preferable.

It would be interesting to see if final temperature correlated with room temperature for each patient, as the authors mention that this varied considerably from day to day. It may be that a threshold level can be identified, above which normothermia is maintained in all patients, as Morris has suggested [2].

The authors refer to their measures as “simple” but this is really more of a reflection of familiarity with a particular technique. The application of a warming blanket is probably no more time-consuming than wrapping all four limbs in gangee, and although it is currently necessary to cut a hole in the blanket...
and tape the edges up, it is hopefully only a matter of time before manufacturers perceive a need and supply appropriately modified blankets for major abdominal surgery. The equipment required for a heated water mattress is comparable with a forced air convective system.

Cost is another factor of increasing importance. It is worth remembering that the expense of heating an operating theatre is not negligible. This is obviously dependent on many factors, such as local climate and size of room, but in some circumstances the extra cost required to increase the temperature by a few degrees may exceed that of specifically designed patient warming devices. The cost of disposables for a second level 1 rapid infusion system (two of these devices are used for each patient in the study described) also considerably exceeds the cost of the disposable mattress.

In conclusion, it would seem that in the circumstances described the theatre personnel have no choice but to tolerate the conditions. If a choice were offered, very few would choose a high ambient temperature, and the advantages of a specific warming device in allowing a cooler working environment for theatre staff, while keeping the patient normothermic, are impossible to quantify, but probably very real.

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Neurotoxicity of 5 % lignocaine

Sir,—In a recent article [1], an incorrect reference was made to studies published from our laboratory. The authors stated that “Synergistic neurotoxic effects of lignocaine and glucose have been demonstrated in vitro in frog sciatic nerve....” The reference cited [2] provides no evidence and makes no claim that hyperbaric glucose acts synergistically with 5 % lignocaine. Rather the conclusion is that 5 % lignocaine alone, in solutions otherwise physiological in salt content and pH, produces rapid, irreversible and total conduction block in isolated frog myelinated nerve. Subsequent studies from our laboratory on frog nerve have shown that these irreversible effects occur partially but significantly in frog nerve with lignocaine as low as 1.5–2 % [3] and that in isolated, desheathed mammalian nerve, conduction in both myelinated and non-myelinated fibres is blocked irreversibly and fully by 5 % lignocaine alone and partially by 2 % lignocaine alone [4].

These results support the authors’ discouraging advice in reference [1] concerning 5 % lignocaine, whether or not hyperbaric glucose is present.

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2. Lambert LA, Lambert DH, Strichartz GR. Irreversible conduction block in isolated nerve by high concentrations of local anesthetics. Anesthesiology 1994; 80: 1082–1093.

Sir,—We apologize for the incorrect reference to Strichartz and Lambert’s study [1]. Our mistake was based on a preliminary report on the same study published as an abstract [2].

Clinically, in our opinion, hyperbaric glucose may be a contributing factor to neurotoxicity. When injected intrathecally with a local anaesthetic, especially with the patient sitting, a hyperbaric solution may be poorly distributed, resulting in a high concentration of local anaesthetic. Our conclusion was based on our clinical trial, in which only hyperbaric 5 % lignocaine was used.

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Psychological characteristics and patient-controlled analgesia

Sir,—In their study comparing the use of papaveretum in patients after total abdominal hysterec- tomy, in which the drug was given either by i.m. injection or by a patient-controlled analgesia system (PCA) [1], the authors concluded that patients who received PCA used significantly less analgesia and were discharged earlier than patients who received an i.m. injection. While their findings have important implications for anaesthetists involved in the management of acute postoperative pain, I would like to mention some points which I believe need to be clarified.

The anaesthetic technique was said to have been standardized, yet the analgesic component consisted of either fentanyl or papaveretum, together with an opioid premedicant (either pethi- dine or papaveretum). Previous work has shown that opioid premedicants can significantly reduce the consumption of post-operative analgesics [2, 3] while other work has shown that the timing of administration of opioids in patients undergoing total abdominal hysterectomy may reduce the consumption of post-operative analgesics by 27 % [4]. The authors failed to state when the analgesic drugs were given and if both groups were given these drugs at comparable times.

After operation these patients were treated with either papaveretum 15–20 mg with a minimum dosing interval of 4 h or the same dose of papaveretum can be given safely when administered on an hourly basis as required [5]. The authors have thus chosen to compare i.m. injection which has been shown to be less than adequate, with an efficient form of i.v. administration. It is hardly surprising that the former was shown to result in less efficient pain control. Surely it would have been more appropriate to have given the i.m. injection with a shorter minimum dosing interval?

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Expanding the applications of patient-controlled techniques

Sir,—The study reported by Thomas and colleagues [1] on the effectiveness of patient-controlled analgesia (PCA) is valuable in confirming that anxious individuals with poorly developed coping styles achieve greatest benefit from this method of pain relief. Pain can be defined as “an unpleasant sensory or emotional experience associated with actual or potential tissue damage, or described in terms of such damage”, thus a technique which restores an element of control is likely to attenuate emotional distress.

I suspect that it is this improvement in psychological well-being that is mainly responsible for the apparent analgesic benefit resulting from self-administration of opioid compared with conventional methods of postoperative pain relief. The findings of Wheatley and colleagues [2] revealed that the use of PCA led to a significant reduction in postoperative pain, yet the two matched groups consumed similar mean doses of morphine: 54 mg (i.m.) vs 51 mg (PCA) over 24 h.

Thomas and colleagues [1] also identified that the total analgesic dose consumed was directly and significantly correlated with state anxiety, and with coping style. It is therefore possible that much of the inter-individual biological variation that the patient-controlled method so usefully deals with, also results from psychological factors. Therefore, we should be making greater use of patient-controlled techniques. The same psychological advantages should be obtained theoretically from controlling one’s own extradural.

I have also used patient-controlled anxiolysis (sedation) during regional anaesthesia for hip and knee replacement, and for insertion of central venous catheters. Propofol is an ideal agent for this purpose, as it is usually possible to produce anxiolysis without sedation. A typical regimen for an adult would be a bolus dose of 8–12 mg with a lockout of 1–2 min.

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