Use of rocuronium in a pregnant patient receiving magnesium medication

Sir,—The report by Gaiser and Seem1 highlighted some important aspects of the use of non-depolarizing neuromuscular blocking agents in the presence of magnesium infusions. In their patient, the use of rocuronium for neuromuscular block was followed by prolonged paralysis. However, there are several problems with this report. The dose of rocuronium used was well in excess of the usual recommended dose of 0.6 mg kg⁻¹. The prolongation of non-depolarizing neuromuscular block by magnesium is so well known that the decision to use such a high dose of a non-depolarizing blocker needed to be very carefully considered. If it was known at the outset that the case was to last 6 h, then the use of rocuronium in these circumstances is acceptable, but prolongation of its action should have been expected. The validity of the choice of blocker is also open to debate. The evidence against the use of suxamethonium in patients with open eye injuries is, at best, weak. There is little hard evidence that the small increase in intraocular pressure (IOP) seen after the use of suxamethonium poses a real threat to the injured eye and several strategies are available to limit this increase in IOP.2 In addition, magnesium decreases the fasciculations produced by suxamethonium,3 and is likely to limit the increase in IOP consequent on suxamethonium use. The aspiration risk in this patient was clear, and it seems inappropriate to consider the minute risk of ocular injury from suxamethonium ahead of the hazards associated with the use of a non-depolarizing neuromuscular blocking agent.

The plasma concentrations of magnesium referred to in this case report must be wrong. I suspect that the units were erroneously quoted as mmol litre⁻¹ whereas the reported values are consistent with mEq litre⁻¹. It is extremely unlikely that, at the doses of magnesium given, the concentration in plasma would have been 5.8 mmol litre⁻¹. The correct therapeutic range is not 4–8 mmol litre⁻¹ as quoted in this article, but 2–4 mmol litre⁻¹. The quoted values tally with the normally quoted mEq litre⁻¹ range, those for molar concentrations being half the concentration reported when equivalents are used for divalent ions.

The important lesson of this report is that the well known interactions of magnesium and neuromuscular blocking agents should always be borne in mind. Careful selection of appropriate drugs and dosage is vital if this type of complication is to be avoided.

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Sir,—We appreciate the comments of James on our case report, especially those concerning the units for the plasma concentration of magnesium. In the original manuscript, it was mEq litre⁻¹. We apologize for the error in typesetting.

However, we disagree with several of his other statements. After initiating the case, we knew the neuromuscular blocking effects of rocuronium would be prolonged by magnesium; it was the extent with which we were unsure. In determining the risk and benefit of using rocuronium, we felt the benefit of avoiding the increase in intraocular pressure (IOP) outweighed the risk of possible post-operative intubation and ventilation should surgery conclude before its action had terminated.

He also states that the dose of rocuronium, 0.9 mg kg⁻¹ (3×ED₉₅), was excessive. As outlined in the manuscript, this dose, compared with 0.6 mg kg⁻¹, decreases the onset time to complete neuromuscular block by 50%.1 In the pregnant patient who is prone to oxygen desaturation on induction, we felt that rapid securing of the airway to be most important.2 As such, the higher dose was used.

Suxamethonium increases IOP by approximately 8 mm Hg. James stated that magnesium may attenuate this increase as magnesium would decrease the fasciculations produced by suxamethonium. Contraction of the extraocular muscles does not completely account for the increase in IOP. Pretreatment with a non-depolarizing neuromuscular blocking agent to prevent fasciculations does not reliably prevent this increase.3 In fact, in patients scheduled for elective unicerebral who had all of the extraocular muscles detached, suxamethonium still increased IOP.4 Therefore, one would have to question if magnesium would limit the increase in IOP by suxamethonium.

Finally, James suggests the use of propofol and alfentanil to attenuate the increase in IOP. Although these agents may be helpful, it is important to remember that the patient was a 31-yr-old parturient with a 28-week gestation. Although propofol has been used successfully during pregnancy, it is not recommended.5 There are no adequate and well-controlled studies concerning the use of alfentanil during pregnancy. Its use as a continuous extradural infusion during labour has been investigated. Neonatal hypotonia was observed.6

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Hepatocellular integrity after inhalation anaesthesia

Sir,—In their study of hepatocellular integrity after inhalation anaesthesia, Trauner and Rosenberg7 found that GSTA concentrations increased in patients who received isoflurane or halothane, but the increase appeared greater in those receiving halothane. As they point out, this contrasts with previously published studies,2 3 and they speculated on possible reasons for this. We believe the discrepancy between their findings and ours is explained by the use of different modes of ventilation in the two studies, and not from differences in the concentrations of agents administered.

There is little doubt that GST concentration measured by specific radioimmunoassay does not increase in normotensive, spontaneously breathing patients who receive isoflurane. Of the 18 patients who received isoflurane, only two showed an increase in GST concentration (4.1 and 4.3 μg litre⁻¹) after anaesthesia.2 This confirmed results from a previous study in which no patient who received isoflurane showed an increase in GST concentration after anaesthesia.3 In contrast, we have shown that the mode of
ventilation significantly influences GST concentration more profoundly than the use of different volatile anaesthetic agents.\(^5\) We studied 48 patients undergoing controlled hypotension for middle ear surgery who were allocated to one of four groups to receive either IPPV or SV, and halothane or isoflurane. GST concentrations increased at 1–3 h after the end of anaesthesia in all four groups but there was no significant difference in GST concentration between patients who had received halothane or isoflurane, irrespective of the mode of ventilation. Combining the results obtained using the two volatile agents allowed assessment of the effect of ventilatory mode and revealed that GST concentration was significantly greater at 1 h after the end of anaesthesia in patients who had received IPPV (pre-induction 2.3 μg litre\(^{-1}\); 1 h 3.6 μg litre\(^{-1}\)) than in those who had breathed spontaneously (pre-induction 2.5 μg litre\(^{-1}\); 1 h 2.6 μg litre\(^{-1}\)). We do not consider that differences in concentration of volatile agents administered account for the differences observed between the study of Tiainen and Rosenberg and ours. We have never found that the change in GST from preoperative values is related to the use of different anaesthetic agents. We agree with Tiainen and Rosenberg that the likely reasons for this include the relatively insensitive GST assay used and infrequent sampling after anaesthesia. It should also be noted that whereas Murray and Tiainen’s groups measured total alpha class GST (i.e. B1 and B2 subunits), we measured only GST B1 subunits in our studies; measurement of B1 subunits appears more sensitive than B2 at detecting hepatic damage.

We believe that hepatocellular integrity is not impaired in spontaneously breathing patients who receive isoflurane, and that mode of ventilation has a greater influence on GST concentration after anaesthesia than the use of different inhalation anaesthetic agents.

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Sir,—Thank you for the opportunity to respond to the comments by Ray, Beckett and co-workers\(^2\) and by Hussey and co-workers\(^1\) on hepatocellular integrity after isoflurane and halothane anaesthesia.\(^1\) The two differences between our study\(^3\) and that by Hussey and co-workers\(^2\) were: (1) in our study there was no significant difference in GSTA between the isoflurane and halothane groups; and (2) in our study GSTA increased also in the isoflurane group. The latter fact may, indeed, have been caused by the influence of positive pressure ventilation in normotensive patients as seems to be the case in patients operated on under deliberate hypotension.\(^3\) However, the former requires other explanations.

For about 9 yr it has been thought that halothane anaesthesia is usually associated with a disturbance in hepatocellular integrity while isoflurane anaesthesia is not.\(^2\) Our study challenged this “truth”\(^3\). We cannot totally reject our speculation that the former difference between the studies is caused by previous exposure to halothane or by differences in concentration of anaesthetics. Halothane was probably more commonly used in Scotland in the 1980s than in Finland in the 1990s. The change in GSTA seems not to be related to the dose of anaesthetic agent or to duration of anaesthesia, but we have not found any study on GSTA comparing different concentrations of volatile anaesthetics, or comparing different durations of anaesthesia to prove these concepts. Murray, Rowlands and Trinick\(^4\) used a relatively insensitive assay and found a difference in GSTA concentration between halothane and isoflurane at 2 MAC for 10 h. There was also an increase in aspartate aminotransferase.\(^4\) On the other hand, we found no significant difference between these anaesthetics at 1 MAC for 3 h.\(^1\) In the studies in which Ray, Beckett and Allan have been co-authors,\(^3\) the concentrations of volatile anaesthetics and the changes in GSTA concentration seemed to fall between these extremes.

We agree that positive pressure ventilation may be an important cause for the increase in GSTA after isoflurane anaesthesia but we need other explanations for the lack of difference between isoflurane and halothane. The increase in GSTA concentration after sevoflurane anaesthesia is also interesting because it occurs after positive pressure ventilation\(^5\) but also after spontaneous ventilation.\(^6\)


**Safety issues and volatile agent analysers**

Sir,—Dr Strachan and Richmond raised some interesting points in their report concerning the presence of diethyl ether in an isoflurane vaporizer.\(^1\)

Older Datex anaesthetic agent monitors are calibrated to measure levels of halothane, enflurane, isoflurane and methoxyflurane. It has been noticed in this hospital that when using sevoflurane the agent monitor seems to give readings corresponding to the vaporizer settings when set to measure methoxyflurane which is not seen when the agent monitor is set for any other volatile agent. This was purely a chance observation and has certainly not been used clinically as a monitoring method.

It does however seem surprising, given the different physical and chemical properties of sevoflurane (molecular weight 200.1, blood-gas partition coefficient 0.69, boiling point 58.6°C) and methoxyflurane (molecular weight 165, blood-gas partition coefficient 13, boiling point 105°C) and the seemingly closer similarity of sevoflurane to the routinely used volatile agent, that this must reflect similar infrared absorption bands of sevoflurane and methoxyflurane.

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This would seem to indicate that in some circumstances two compounds with quite distinct properties can effect monitoring in similar ways given similar absorption spectra. This shows that advancing the scope of anaesthesia in one area must be paralleled by the ability to use appropriate monitoring techniques and, at best, monitoring is only an adjunct to clinical vigilance.

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Ether in an isoflurane vaporizer and the use of vapour analysers in safe anaesthesia

Sir,—We read with interest the case report by Strachan and Richmond1 and wish to make the following comments.

The ether-filled temperature compensation unit used in the PPV Sigma has been used safely in Penlon vaporizers for more than 40 yr and was one of the world’s first automatic TC units, having been first introduced into the EMO vaporizer.

The design of the now obsolete PPV Sigma vaporizer discussed in the article necessitated a significant TC movement that was not possible to obtain by bimetallic expansion alone. That movement could only be provided by that type of liquid-filled expansion system.

The current range of Penlon Elite vaporizers makes use of significantly different control variables and as a result of these major design changes Penlon vaporizers no longer use liquid-filled TC units but do use a solid state bimetallic TC unit in common with some other manufacturers.

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Non-invasive measurement of cerebral blood flow

Sir,—I read with interest the recent article by Gupta and co-workers.1 It was only when I set out to derive the equations presented within this article that I realized that one of the definitions is wrong. The authors define CLVHR as “the coefficient of the large to small vessel packed cell ratio” whereas it is the inverse, the ratio of cerebral (small vessel) to large vessel haematocrits, as described by Lammertsma and colleagues2 and calculated as 0.69. It is the definition that is misleading, the equations are correct for the value quoted. Unfortunately, Gupta and co-workers have simply compounded the original error in definition given by Ellwell and co-workers.3

My second comment is on the use of $P_{\text{FeCO}_2}$ as equivalent to $P_{\text{FeCO}_2}$ when calculating carbon dioxide reactivity. First, the normal value for $P_{\text{FeCO}_2}$ is 5.3 kPa but this does not equate to an $P_{\text{FeCO}_2}$ of 5.3 kPa. At rest, with quiet respiration, there is a small difference of 0.3–0.6 kPa between $P_{\text{CO}_2}$ and $P_{\text{FeCO}_2}$. Thus data should have been indexed to a normal value for $P_{\text{FeCO}_2}$, namely 4.85 kPa. The difference between $P_{\text{FeCO}_2}$ and $P_{\text{FeCO}_2}$ is important in the context of this study as respiration is altered to achieve different $P_{\text{FeCO}_2}$ values. We are not told how the volunteers hyperventilated to reach a $P_{\text{CO}_2}$ of 3.5 kPa, but most people would increase ventilatory frequency and tidal volume may decrease; conversely, adding carbon dioxide to the gas mixture triggers hyperventilation largely by increasing depth of ventilation in addition to ventilatory frequency. As the difference between $P_{\text{FeCO}_2}$ and $P_{\text{FeCO}_2}$ is dependent on tidal volume, the slope of the CBV vs $P_{\text{FeCO}_2}$ line is no longer identical to the slope of the CBV vs $P_{\text{CO}_2}$ relationship. Physiologically, carbon dioxide reactivity is dependent on $P_{\text{CO}_2}$ not $P_{\text{FeCO}_2}$ and so this source of error in determining carbon dioxide reactivity should be discussed as we are not told if the volunteers were asked to maintain a constant tidal volume for all parts of the study.

The lack of any significant change in packed cell volume in relation to $P_{\text{FeCO}_2}$ is indeed puzzling when the calculated values for CBV are clearly related to $P_{\text{FeCO}_2}$. Although the possibility of following changes in CBV non-invasively may be useful clinically, particularly for head injuries, to do this by altering $P_{\text{FeCO}_2}$ is not acceptable. It seems this point must be addressed before the methodology presented here is acceptable.

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Sir,—I read with interest the article by Gupta and colleague1, but have grave doubts on the validity of the conclusions regarding the ability to non-invasively assess cerebral blood volume, for several reasons.

First, no currently available near-infrared (NIR) spectroscopic system has the ability to completely exclude the scalp signal, which may potentially cause a great deal of artefact. Second, the oxidized and reduced haemoglobin concentrations were derived from NIR absorption spectra by an algorithm, introducing a further potential inaccuracy. Third, calculation of $\text{Hb}_{\text{oxd}}$ involves examining the changes in the derived signals, thereby compounding the error of an algorithm with the influence of the scalp tissue signal. Possibly this helps explain the 25% difference in the calculated cerebral blood volume (CBV) reactivities of 1.25 and 1.06 ml 100 g$^{-1}$ kPa$^{-1}$ when $\text{HbO}_2$ or $\text{Hb}_{\text{red}}$ concentrations were used to perform the calculation. Fourth, CBV calculated in ml 100 g$^{-1}$ assumed a large number of variables to be constant. Haemoglobin was assumed to be 15 g dl$^{-1}$, even though five of 13 subjects were female and of an age likely to be menstruating. The coefficient of large to small vessel packed red cell ratio is assumed to be constant throughout, even though the authors noted that flow, as measured by transcranial Doppler, changed with moderate differences in oxygen content indicating vasoreactivity, which must surely change the large to small ratio.

Lastly, there is no “gold standard” with which the authors have compared their results, and although they may have measured changes in cerebral blood volume, one cannot be sure from this study.

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Sir,—We thank Drs Hill and Williams for their comments. In response to Dr Hill, we agree that the definition of CLVHR is the small to large vessel haematoctrit ratio, as described by Lammertsma and colleagues.2

The study aimed to evaluate non-invasive methods of assessing cerebral blood volume. While we accept that there is a small difference in $P_{\text{FeCO}_2}$ and end-tidal carbon dioxide ($P_{\text{FeCO}_2}$), arterial blood-gas monitoring was not used as described in the article. The data were indexed to the calculated CBV at and $P_{\text{FeCO}_2}$ 5.3 kPa. Although this does not equate to a $P_{\text{FeCO}_2}$ of 5.3 kPa, the regression equation of CBV reactivity should not be affected.

Further, the expression of CBV reactivity as a percentage of baseline CBV compensates for individual variations in baseline CBV. End-tidal carbon dioxide manipulation is a recognized method of measuring carbon dioxide reactivity2 and is used clinically to assess carbon dioxide reactivity. However, we would not alter $P_{\text{FeCO}_2}$ to measure CBV in head injured patients. This
is not implied in the study; we have used $P_{CO_2}$ variations in volunteers merely as a paradigm of CBV alterations in volunteers and these changes would result from spontaneous pathophysiological mechanisms in disease.

To Williams' comments, it is well recognized that there is extracranial contamination in the near infrared signal. It has been reported that there is approximately a 15% contribution of skin changes to the $HbO_2$ signal during carbon dioxide challenge, and much of this change is related to changes in systemic arterial pressure. Both $\beta$-adrenergic blockers showed no significant change in MAP. It therefore seems unlikely that changes in skin blood flow contributed significantly to the carbon dioxide reaction of the NIR signal. The use of an estimated $Hb$ value and the approximations involved in the calculation may have biased the absolute level of CBV measured, but are unlikely to have affected reactivities.

While there is as yet no direct comparison of NIR measurement of CBV and CBV reactivity against the gold standard of positron emission tomography (PET), we have discussed our results in the context of data obtained by other authors and different techniques, including PET.3

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Sir,—We thank Drs Jerwood and Willis for their interest in our investigation but their letter deserves comment on two points: first, preoperative preparation, and second, effect of phaeochromocytoma.

We agree that no controlled, randomized study has demonstrated that preoperative preparation for phaeochromocytoma reduces perioperative morbidity and mortality. It is also not established that preoperative treatment should last at least 21 days to improve perioperative hemodynamic stability. Moreover, no preoperative preparation can control all perioperative hemodynamic events. The issue of the need for any preoperative preparation has even been questioned elsewhere.3 $\alpha$-Adrenergic blockade requires time to be fully effective (this sometimes “justifies” up to 21 days of preparation) and has many drawbacks: (a) arterial pressure instability when inducing treatment may delay the time of completing vasodilator therapy, (b) decreased venous tone and cardiac preload, and (c) increased risk of severe postoperative hypotension, especially with long-acting drugs such as phenoxybenzamine. In our experience, dihydropyridines allow short-term preoperative preparation,4 do not induce arterial pressure instability, do not adversely affect venous return and cardiac preload, and do not cause severe postoperative hypotension. All of these features provide many reasons to choose dihydropyridines rather than $\alpha$-adrenergic blocking drugs for preoperative preparation of phaeochromocytoma surgery. Labetolol was used as a $\beta$-blocking drug, and it was added to a preliminary vasodilator therapy; therefore the risk of paradoxical hypertension was avoided.

In the first of the two case reports, peritoneal insufflation induced a severe increase in catecholamine concentration that mimicked adrenal tumour response to surgical manipulation. This is the evidence that insufflation of the phaeochromocytoma may induce direct stimulation of the adrenal tumour. Even 21 days of phenoxybenzamine preparation would not have avoided this catecholamine release from the adrenal tumour, as a $\alpha$-adrenergic blockade does not act on tumour secretion. Therefore, our conclusion is justified and we recommend caution with peritoneal insufflation for laparoscopic phaeochromocytoma resection.

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Airway problems after carotid endarterectomy

Sir,—I read with interest the article by Munro, Makin and Read1 describing four cases of acute respiratory obstruction after development of postoperative haematoma following carotid endarterectomy. They make the point that such patients require continuous respiratory monitoring in order to detect developing respiratory obstruction at an early stage. It is worth emphasizing that the neurological complications they refer to after carotid endarterectomy may include changes in respiratory function and control.2 The presence of hyperventilation or even apnoea3 would make the obstructive effect of postoperative haematoma even more rapid in onset. Gradual onset of respiratory obstruction may also lead to hypcapnia, which by means of the development of cerebral steal syndromes might lead to a decrease in blood flow in already ischaemic areas of the brain, thereby exacerbating the already compromised control of respiration. Inadequate control of arterial pressure in the postoperative period increases this effect in addition to increasing the size of postoperative wound haematomas.

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In vivo and in vitro dose–response curves

Sir,—Our Japanese colleagues1 have made a good attempt in interpreting human anaesthetic dose–response curves at a molecular level. But they admit that not all the values they have derived are plausible. May I offer two suggestions?

They used the method of least squares, however, this has underlying assumptions that the data are Gaussian and homoscedastic, neither of which is true for quantal data. One method of analysing quantal data was described by Waud:2 the appendix provides a simple BASIC program, which will solve equation (2) of the Osaka study. Waud’s programme is translated easily into Microsoft Professional Basic, a language which overcomes most of the objections to old BASIC raised by computer professionals.

Second, the Osaka team suggested that the in vivo curve is the outcome of a normal, or log-normal, array of events at the molecular level. If so then their equation (2) would match reality better as a series, not a single unit:

\[
y = \frac{x^n}{K_1^{n} + x^n} + \frac{x^n}{K_2^{n} + x^n} + \ldots
\]

Adding one unit should reduce residual variance and the experiment repeated until there is no further improvement. With three units the solution would require inverting a 6 × 6 matrix, but that is no problem in Professional Basic, which has an efficient algorithm for any size of matrix.

Lastly, anaesthetics act on the synapse; if 1% halothane reduces transmission across each synapse to 70% of normal, then over a relay of five synapses it is decreased to 17% (0.75). Does it not follow that the m in vivo curve should be steeper than effects at single synapses? This is why a simple reflex, such as withdrawing the foot, can be present when consciousness has been extinguished, as John Snow noticed 150 yr ago. May I wish them good luck with their endeavours.

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Ambulatory extradural analgesia in labour and mode of delivery

Sir,—We appreciate the letter of Dr Cormack which relates to our article on dose–response curves.1 He suggests that assays of in vivo dose–response data have to be treated by the method of analysing quantal data, instead of the method of least squares, as described by Waud.2 According to Waud’s programme, we have re-examined the in vivo data and obtained revised results as follows: ED\(_{50}\) = 1.01%atm (SEM = 0.08%atm), n = 3.59 (SEM = 0.89) for isoflurane, and ED\(_{50}\) = 0.76%atm (SEM = 0.008%atm), n = 29.2 (SEM = 7.6) for halothane. These results are in agreement with those in our articles (ED\(_{50}\) = 1.03%atm, n = 3.6 for isoflurane; ED\(_{50}\) = 0.75%atm, n = 27 for halothane). Therefore, our least square fittings did not differ markedly from quantal assays.

Second, he suggests better fitting of data to the Hill equation (2) in our text by adding another unit. This may be effective for reducing residual variance, but the biological or molecular meaning of the adding term is not clear. Such a modification should be plausible when two or more doses are applied simultaneously,2 or when some integrated effects take place on an organism, such as linkage effects between two or more receptor molecules each of which can bind with a drug molecule. In both cases, however, we have another problem, whether or not simple addition of one unit is suitable, because it depends on an adopted model.

Lastly, he mentioned an effect of a series of synapses. This is an interesting suggestion, but if there were long series of synapses, only a small amount of dose applied would anaesthetize any patient. In other words, it cannot show the existence of a critical dose value above which a patient does not respond; the critical value is different between patients. In a complicated system of neurone networks, information propagates backwards in addition to forwards, and then some integrated response emerges. We suggest that this must be responsible for the existence of the critical dose value mentioned above. Although our MWC model over-simplifies the system, the model inherently has an integrated nature.

We thank Dr Cormack for his valuable suggestions and his interest in our article.

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County Hospital was performed in late 1993. Because of the high rate (2.3%) of post-dural puncture headache after combined spinal–extradural analgesia reported by Collis and colleagues,4 we adopted successfully a technique using extradural boluses of 0.1% bupivacaine 70 ml with fentanyl 2 μg ml⁻¹, without a spinal and without a conventional test dose. In 1996, 1100 (95.8%) of the 1148 extradurals (42.4% of labouring mothers) given for pain relief used this mobile technique, compared with one (0.11%) of 907 extradurals (35.2% of labouring mothers) in 1993.

Unfortunately, there has not been a reduction in the rate of emergency Caesarean section in mothers using extradural analgesia. In 1993, 78 (8.6%) of 907 mothers with extradurals required emergency Caesarean section compared with 146 (12.7%) of 1148 in 1996. This is a statistically significant increase (chi-square = 15.9, P < 0.001) and a significant decrease in the number of instrumental deliveries, from 341 (43.8%) in 1993 to 356 (31.0%) in 1996 (chi-square = 33.5, P < 0.001). In those mothers who did not receive an extradural, the rates of spontaneous vaginal delivery, instrumental delivery and emergency Caesarean section have not changed significantly between 1993 (85.4%, 5.3% and 9.3%, respectively) and 1996 (87.8%, 4.2% and 8.0%, respectively). A randomized study by Collis, Davies and Aveling compared ambulatory combined spinal–extradural analgesia with standard non-ambulatory extradural analgesia found no difference in the outcome of labour. However, Asselineau observed a significantly lower instrumental delivery rate but similar Caesarean section rates in the ambulant group when comparing ambulant and non-ambulant extradurals. Experience at this hospital suggests that the use of mobile, or ambulatory, extradurals may lead to a higher rate of spontaneous vaginal delivery and a lower rate of instrumental delivery.

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The “Dumfries claim”

Sir,—The reference to the first trial of ether anaesthesia at Dumfries and Galloway Royal Infirmary on 19 December 1846 (in your editorial) is dismissive and, as such, deserving of comment. In an allusion to William Scott’s assertion of priority over Robert Liston with regard to the date, Professor Spence refers to the “Dumfries claim”, quoting, I presume, the title of my original paper in your journal.

The “claim”, as Professor Spence correctly stated, was contained in William Scott’s letter to The Lancet on October 19, 1872, one of the no fewer than three reliable references to the validity of Scott’s assertion. While the question of precedence was an important personal issue for William Scott, at no time did he infer that his early application of anaesthesia at Dumfries had any influence on the rapid acceptance of ether. Nevertheless, had James Y. Simpson been in Edinburgh at the time, in which case the news of Scott’s operation would have reached him almost immediately, the Old World would doubtless have been informed of the anaesthetic properties of ether through alternative channels! In that case, Robert Liston might well have had to share the honours with his colleagues in south-west Scotland.

The suggestion by Professor Spence that James Y. Simpson travelled to London “for the specific purpose of learning about anaesthesia” would appear to be incorrect. Simpson’s journey to London in December 1846 was the first of two visits in connection with his appointment as surgeon to Her Majesty Queen Victoria’s household in Scotland and, on the date of the Squire:Liston success, he was either on his way to, or had already arrived in, London. He received the news of ether, therefore, not from London but in London.

The narrative surrounding the Dumfries operation is a fascinating one and has been the subject of several publications by this writer over the past 30 yr. The gentlemen at Dumfries provided the chronicles of anaesthesia with a remarkable story and the fact that particulars regarding the patient are, as yet, incomplete does not lend the right to dismiss the incident with, “the outcome, whatever it was, adds nothing to science or medicine.”

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Allergies and anaesthesia

In their case report,\textsuperscript{1} Rae, Milne and Wildsmith highlighted the importance of obtaining a detailed history, particularly in the area of allergies. I recall a similar case also during a Caesarean section under regional anaesthesia which reinforces this point.

A 35-yr-old ex-nurse, with atopy but no known allergies, underwent a routine combined extradural–spinal anaesthetic for elective Caesarean section. Catherization with a latex urinary catheter was uneventful. Just after delivery and a bolus of i.v. oxytocin she complained of “feeling unwell” and said her tongue “felt swollen”, she developed an erythematous rash on her upper body with visible facial oedema and her arterial pressure rapidly became unrecordable. A diagnosis of anaphylaxis was made; amniotic fluid embolus was part of the differential diagnosis. Treatment with oxygen, i.v. crystalloid and adrenaline rapidly restored her to her previous state. A similar, but less severe, episode occurred before surgery was completed. Recovery was uneventful, the urinary catheter was removed 24 h after operation.

It was only afterwards on direct questioning that the patient described past episodes of facial swelling on blowing up balloons, contact dermatitis to rubber gloves and vaginal irritation to condoms. Skin prick testing proved highly positive for latex, and negative for oxytocin, bupivacaine and Gelofusine.

This case also demonstrates that the risk of latex allergy in medical staff (2.8\%) is greater than that found in patients presenting for skin prick tests (0.8\%) although theatre staff are at greatest risk (7.8\%).\textsuperscript{2} Repeated latex exposure is believed to be responsible for these observations.

Latex-containing products release variable amounts of antigen (water-soluble proteins), examination gloves and urinary catheters releasing far more than surgical gloves. This may explain why anaphylactic reactions associated with the former commonly occur within 2–3 min, as in Rae, Milne and Wildsmith’s case, while those associated with surgical gloves contacting the large absorptive surface of the abdomen (the commonest reported route) often take longer to develop, typically 40–300 min,\textsuperscript{3} as in the above case. Why the two patients reacted so differently I am at a loss to explain.

Since the original case report\textsuperscript{4} latex sensitivity has become recognized as a relatively common cause of allergic dermatitis and anaphylaxis. This may be caused by a combination of change in the manufacturing process, greater antigen exposure, heightened clinical awareness and more frequent use of latex as an antigen during investigations of drug reactions. Whatever the cause it is necessary to inquire about latex sensitivity from all of our patients to avoid similar scenarios.

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