Is sevoflurane replacing halothane?

Sir,—Mostafa and Atherton describe the successful use of sevoflurane as an induction agent in three adult patients with difficult airways.1 My practice includes anaesthesia for children with difficult airways.

I recorded some details of the first 30 such children in whom anaesthesia was induced with sevoflurane when it became available in late 1995. My method is to use 8% sevoflurane in 100% oxygen or a 2:1 nitrous oxide–oxygen mixture delivered from a T-piece by a bare hand. Sevoflurane is discontinued when the patient loses consciousness, halothane or isoflurane being introduced.

Anaesthesia was induced in 30 children (15 boys, 15 girls). Mean age was 3.3 yr (range 1 month to 12 yr); 11 children were aged 3 months or less. The diagnoses are listed in table 1.

Table 1 Diagnoses of children with “difficult airways” who received sevoflurane for induction of anaesthesia

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cleft lip and palate</td>
<td>9</td>
</tr>
<tr>
<td>Laryngeal papilloma</td>
<td>6</td>
</tr>
<tr>
<td>Subglottic stenosis</td>
<td>5</td>
</tr>
<tr>
<td>Laryngeal web</td>
<td>2</td>
</tr>
<tr>
<td>Micrognathia</td>
<td>1</td>
</tr>
<tr>
<td>Tracheocutaneous fistula</td>
<td>2</td>
</tr>
<tr>
<td>Beckwith’s syndrome</td>
<td>1</td>
</tr>
<tr>
<td>Choanal atresia</td>
<td>1</td>
</tr>
<tr>
<td>Laryngomalacia</td>
<td>1</td>
</tr>
<tr>
<td>Mandibular fracture</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
</tr>
</tbody>
</table>

Mean time to loss of consciousness was 34.1 (so 9.23; range 21–62) s. In all of these inductions, the only untoward event was one child who coughed twice. No patient suffered breath-holding or desaturation.

My previous calculations2 confirm Mostafa and Atherton’s impression that sevoflurane is not especially expensive when used as an induction agent. Sevoflurane is now my routine inhalation induction agent for children with difficult or “easy” airways.

I. BARKER
Sheffield Children’s Hospital
Western Bank
Sheffield

Propofol in paediatric intensive care

Sir,—I was pleased to see another rational study of propofol in a small group of children in intensive care, together with a supportive editorial.3 2 It always seemed to me that propofol was the ideal induction agent for children with difficult or “easy” airways.

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I. BARKER
Sheffield Children’s Hospital
Western Bank
Sheffield


combined spinal–extradural analgesia to those women who request regional analgesia in later labour (so as to benefit from the rapidity of onset of analgesia); those who require instrumental assisted delivery in the absence of a pre-existing regional block; and those who at any time in labour are excessively distressed (so as to rapidly achieve control of the situation).

L. MCLoughlin
Department of Anaesthesia
Queen Charlotte’s and Chelsea Hospital
London


Sir,—I thank Dr McLaughlin for his interest in my letter. The high rate (2.3%) of post-dural puncture headache (PDPH) in the original series of 300 combined spinal–extradurals for analgesia in labour, reported by Collis and colleagues,1 was the only published data available when we made our decision to use low-dose extradural alone rather than combined spinal–extradural analgesia. Subsequently, Morgan and Kadim2 reported a much lower rate of PDPH after combined spinal–extradural analgesia for labour. Recently, cases of both aseptic3 4 and bacterial4 5 meningitis have been associated with increased operative and instrumental delivery and mode of delivery.124

I believe combined spinal–extradural techniques should be used with caution for analgesia in labour.

J. G. Jenkins
Department of Anaesthesia
Royal Surrey County Hospital
Guildford


Sir,—We agree that the evidence that combined spinal–extradural analgesia increases the incidence of post-dural puncture headache (PDPH) is conflicting. This expands the assertion in our original letter.1 Combined spinal–extradural analgesia has proved extremely popular in centres in which it has been introduced, including Queen Charlotte’s Hospital.2 It has been suggested that conventional extradurals with bolus doses of plain bupivacaine are associated with increased operative3 and instrumental delivery and long-term backache.4 It is possible that combined spinal–extradural analgesia in labour has a different profile of side effects.5 It would be prudent to await the results of large scale, randomized controlled studies with long-term follow-up to establish the role of combined spinal–extradural analgesia in labour. We agree that where it is necessary to achieve rapid onset of profound analgesia, particularly where pain is of sacral origin, combined spinal–extradural analgesia has advantages.6 However, the fact that anaesthetists at Queen Charlotte’s Hospital experienced a greater incidence of side effects when combined spinal–extradural analgesia was introduced compared with subsequent studies2 would imply that the use of an unfamiliar technique in such fraught situations may not be desirable.

C. D. Elton
A. E. May
Department of Anaesthesia
Leicester Royal Infirmary
Leicester


Transposition of rotameter tubes

Sir,—With the publication of the new checklist for anaesthetic apparatus from the AAGBI4 by the anaesthetists of Great Britain and Ireland (AAGBI)4 the anaesthetist who was working in that theatre. Nothing untoward was noted at this time. Anaesthesia was carried out safely in the first patient in the anaesthetic room before transfer to theatre. As soon as the anaesthetist turned on the gas flows on the machine in the operating theatre he realized that the flowmeter above the nitrous oxide flow control valve was only calibrated to 2 litre. On closer inspection, this flowmeter was noted to be colour coded and labelled for carbon dioxide. The flowmeter above the carbon dioxide flow control valve was conversely colour coded and labelled for nitrous oxide (fig. 1). The anaesthetist administered 100% oxygen and a volatile agent to the patient while the anaesthetic machine was changed. The patient came to no harm.

How did the flowmeters get swapped? It is standard practice for the service engineer to remove, clean and replace each flowmeter in turn. On this occasion one of the flowmeters was broken on cleaning and then the next was removed, cleaned and replaced before a replacement for the broken one was found. This led to the swap.

Why was this swap not detected during the anaesthetic machine check? The checklist for anaesthetic machines4 states to “set a flow of approximately 5 litre min−1 for the flowmeters. The new trainee anaesthetist knows that this is approximately 50–66% up the oxygen and nitrous oxide flowmeter, without reading the scales. Therefore, the flowmeters were not read accurately to 5 litre during this check. Extra vigilance is needed in checking the anaesthetic machine to exclude a crossover of flowmeters after a machine service. How might such a potentially serious fault be avoided in future? Flowmeter design could be changed so that it would not be possible for them to be interchanged. Clearly the procedures being followed by the service engineer were not foolproof. Perhaps after each anaesthetic machine service the

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machine should be double checked by an independent technician. A much more comprehensive checklist than the one currently described by the AAGBI is possibly required after a machine service. Currently, we reinforce to our trainees that they must set the bobbin height to exactly 5 litre when doing their anaesthetic machine check to guarantee that the flowmeter calibrations are read accurately.

The second question to be asked is what difference to the outlet gas mixture would a switch of this sort make? The two possibilities are of an hypoxic mixture (picked up by the oxygen analyser) or an oxygen-enriched gas mixture leading to the possibility of awareness. Afterwards, oxygen and nitrous oxide concentrations from the faulty machine were measured, setting both flowmeters to read 2 litre as this was the maximum the “nitrous oxide” flowmeter could read. Equal concentrations of each gas were emerging from the common gas outlet. If the flowmeters are calibrated for individual gases, why was the flow of nitrous oxide through a carbon dioxide flowmeter inaccurate? With the bobbin type of flowmeter, the physical property determining flow is gas viscosity at low flow rates and gas density at high flow rates. The viscosities of carbon dioxide and nitrous oxide are 13.9 and 13.5 Pa.s, respectively. The density of both is 1.98 kg m⁻³.

In summary, although service procedures are set and followed by companies, mistakes can occur. No checklist can ever be foolproof. Extra vigilance in checking anaesthetic machines is strongly recommended, particularly after servicing. The importance of following the AAGBI checklist for anaesthetic apparatus cannot be overemphasized. However, a more comprehensive checklist may be required after an anaesthetic machine service.

A. J. WALMSLEY
J. HOLLOWAY

Eastbourne District General Hospital
Eastbourne E. Sussex

2. Chadwick DA. Transposition of rotameter tubes. Anesthesiology 1974; 40: 102
3. Slater EM. Transposition of rotameter bobbins. Anesthesiology 1974; 41: 101

Effect of hydroxyethyl starch on coagulation is difficult to assess in vitro

Sir,—We have read with interest the in vitro studies carried out by Egli and colleagues on the effect of haemodilution with hydroxyethyl starch (HES), gelatin and albumin on blood coagulation.

However, we cannot agree completely with the conclusion that HES compromises blood coagulation more than other substances because in vitro studies of the effect of HES on blood coagulation are, unfortunately, of limited value.

HES leads to coagulation disorders, particularly through impairment of factor VIII/von Willebrand factor. In 1985, Stump and colleagues were able to show that this effect only occurs in vitro, not in vivo. They speculated that the reason for this could be either reduced release or impairment of the synthesis of factor VIII/von Willebrand factor is the cause for the observed coagulation disorders. It is difficult, therefore, to simulate through in vitro studies the complex interaction of HES with the clotting system and in particular elimination via the reticuloendothelial system.

These difficulties are enhanced by the fact that the chemical composition of HES changes markedly in vivo. In recent studies, we were able to show that HES is metabolized in vivo in complex ways, and that impairment of factor VIII/von Willebrand factor depends exclusively on the in vivo molecular weight of HES. This explains why haemorrhagic complications have almost exclusively been observed with high and medium molecular weight HES, which is not easily metabolized by the body, resulting in high in vivo molecular weights of the HES molecules. HES with a medium or low molecular weight which is easily metabolized does not lead to a reduction in factor VIII/von Willebrand factor beyond the dilution effect, and rarely leads to haemorrhagic complications. In vitro studies do not take into account this new finding, because HES is not metabolized in vitro, resulting in unrealistically high molecular weights of the starch molecules which are not observed in vivo.

Nevertheless, we believe that the experimental model of Egli and colleagues is innovative and interesting, particularly as the pathological mechanism of impairment of the clotting system through HES and the mechanism of the reduction in factor VIII/von Willebrand factor have not yet been elucidated. Future in vitro studies that incorporate the recent results should be able to yield interesting new insights.

M. T. GRAUER
J. TEBBE

Neurologische Universitätsklinik
Homburg/Saar
Germany

Sir.—Drs Grauer and Treib were concerned that the in vivo finding of an exaggerated effect on blood coagulation on hydroxyethyl starch (200 0000/5) (HES) compared with gelatin and albumin solutions would not be completely applicable to the in vitro situation. This concern was based on two findings: first, an apparent lack of a decrease of factor VIII/von Willebrand in vitro with impairment of blood coagulation; and second, on the observation that the mean in vivo molecular weight of HES decreased during long-term haemodilution in the treatment of cerebrovascular disease with associated decrease in blood coagulation.

Stump and colleagues described stable VIII/von Willebrand factors in their in vitro experiments assessing mixtures of HES and plasma. These results were achieved in citrated plasma to prevent clotting. Interestingly, when blood coagulation is not inhibited, as in our study using thrombelastography, HES and dextran solutions may precipitate coagulation factors such as factor VIII/von Willebrand in vitro also. The effect of HES in decreasing factor VIII/von Willebrand thus would not have been missed in our in vitro studies. In addition, if we were only able to partially detect the effect of a decrease in factor VIII/von Willebrand in our in vitro study, we would have underestimated the in vivo difference between the blood coagulation effect of HES compared with albumin and gelatin solutions. Thus we cannot agree with the concerns of Grauer and Treib that our results would not substantiate a more exaggerated effect on blood coagulation of HES than albumin or gelatin solutions during surgery.

Treib, Haass and Pindur have recently suggested an interesting new concept to predict the effect of long-term haemodilution therapy using various types of HES on blood coagulation. In patients treated for cerebrovascular disease, they found that the mean molecular weight of HES solutions decreased after infusion in vivo and that the effect on blood coagulation of these different HES solutions was better related to the mean in vivo molecular weight measured after 5–10 days than to the mean in vitro molecular weight (i.e. the mean molecular weight of the HES solution in the bottle before infusion). During surgery, however, HES is administered for relatively short periods of time according to surgical blood loss and the volume replacement requirements of the patient. Thus additional HES solution of the original in vitro molecular weight is infused continually and therefore HES molecules with a molecular weight similar to in vitro molecular weight exert their effect on blood coagulation. The concept of the (long-term) in vivo molecular weight of a particular HES solution affecting blood coagulation is of limited clinical relevance for the intraoperative and early postoperative period.

In vitro testing of the effect of various volume expanders on blood coagulation using thrombelastography is considered adequate. Nevertheless, in vivo findings have to be confirmed in vivo before final conclusions regarding clinical management can be deduced.

G. A. EGLI

A. ZOLINGER

T. PASCH

D. R. SPAIN

Institute of Anaesthesiology

University Hospital Zürich

Zürich

Switzerland


Haemostatic changes during total knee arthroplasty with different anaesthetics

Sir.—The study by Sharrock and colleagues on haemostatic changes in total knee arthroplasty with different anaesthetic techniques reflects the continuing search for a better understanding of the mechanisms of postoperative deep vein thrombosis in major joint arthroplasty. There are three comments I would like to make.

While they are partially correct in their discussion that in total hip arthroplasty we did not confirm using spinal anaesthesia Modig and colleagues' findings of suppressed fibrinolysis with extradural block, this was largely a reflection of haematological methodology. There were demonstrable differences between general anaesthesia and spinal anaesthesia in some measured haemostatic variables. When comparing spinal anaesthesia with general anaesthesia in the immediate perioperative period, platelet count did not increase to the same extent, thrombin generation index (a measure of activation of coagulation) and factor IXIIRa did not increase and there was less activation of fibrinolysis (as demonstrated by euglobulin clot lysis time).

These changes were consistent with suppression of the neuroendocrine response to surgery with spinal anaesthesia, as demonstrated by changes in plasma concentrations of cortisol in our patients and were similar in the general anaesthesia group to those seen with exercise and exogenous infusions of epinephrine.

In our study, no exogenous catecholamines were used and it is possible that any differences between general and extradural anaesthesia in the study of Sharrock and colleagues were hidden by the use of epinephrine in the extradural local anaesthetic solution and i.v. ephedrine for cardiovascular control.

With respect to their comments on limb blood flow, based on our impedance plethysmography study during hip arthroplasty, the enhanced blood flow seen in the surgical limb after reloca- tion of the prosthesis and into the postoperative period with spinal anaesthesia compared with general anaesthesia may not necessarily apply to knee arthroplasty. In the latter case, maximal vasodilation would be expected in the exsanguinated limb after tourniquet release, at least initially, irrespective of the anaesthetic technique used. It will be difficult to follow through this hypothesis with flow studies on patients undergoing knee arthroplasty.

Given the New York group's very large clinical experience in knee arthroplasty, I would be interested in their thoughts on correct exsanguination of the limb before tourniquet inflation and its importance for the occurrence of deep vein thrombosis. Not all orthopaedic surgeons use a compression Esmarch bandage, and when they do it is not always applied efficiently. This could well influence coagulation–fibrinolysis changes in the surgical limb.

M. DAVIS

Tai Tapu

New Zealand


Sir.—We appreciate Dr Davis' comments on our article, as he was perhaps the first person to highlight the role of regional anaesthesia in the genesis of venous thrombosis.

The first question relates to his observation of increased thrombin generation during operation and decreased fibrinolysis after operation in patients receiving general anaesthesia during total hip replacement (THR). We noted no increase in thrombin generation (measured by thrombin-antithrombin complexes) or in...
the effect of thrombin formed (fibrinopeptide A), nor did we note any difference in fibrinolysis, although our study did not extend into the postoperative period. These differences most likely reflect the different operations and possibly the differences in blood loss between spinal and general anaesthesia during THR. There is no effect of anaesthetic technique on blood loss with total knee replacement (TKR).

The issue of the use of catecholamines during anaesthesia is an important point. Although epinephrine and exercise increase fibrinolysis in young people, we were unable to detect any augmentation during THR in elderly patients.\(^1\) In contrast, we have noted that infusions of epinephrine are associated with less thrombogenicity during TKR performed with a tourniquet.\(^2\) These are preliminary observations and whether or not this represents a suppressant effect of epinephrine on coagulation or a variable effect is unclear.

With a tourniquet, there is little opportunity for extradural anaesthesia to augment flow in the operative limb and thereby alter venous thrombotic tendencies. This was one of the reasons we chose to study TKR, as flow should not be a confounding variable. As we were unable to see any difference in fibrinolysis or markers of thrombin generation in our patients, we hypothesized that augmentation of flow after surgery may account for the decreased rate of deep vein thrombosis (DVT) noted with extradural anaesthesia after TKR. Whether or not this is true is unknown.

The final point relates to how well the leg is exsanguinated before tourniquet inflation. I suspect this is a significant determinant of the degree of venous thrombosis. Any blood remaining in the leg will be static and a potential nidus for DVT. For this reason, our surgeons assiduously attempt to exsanguinate the leg with an Esmarch bandage before inflating the tourniquet. However, to the best of my knowledge, this has not been studied.

I thank Dr Davis for his comments, as a fuller understanding of factors contributing to intraoperative thrombosis will help reduce morbidity after surgery.

N. E. SHARROCK
Department of Anaesthesiology
Hospital for Special Surgery
New York NY USA


**Extradural anaesthesia in elective Caesarean section**

Sirs,—We read with interest the article by Morton and colleagues\(^1\) and would like to raise several points. First, we were concerned that the results of nine women from a study containing only 38 patients were not reported for analysis of efficacy or pharmacokinetics, or both. This is a drop out rate of approximately 25%. Second, one patient who apparently had sensory block to T6 after receiving 0.75% ropivacaine 20 ml and increments of 2% lidocaine had spinal anaesthesia for Caesarean section because surgery was delayed. Analysis of table 3 shows that in this patient, surgery started 114 min after extradural injection. Analysis of table 2 shows that the median duration of sensory block at T6 was 3.4 h (range 1.3–5.8 h). Although the results of this patient were not included in table 2, this patient should still have had a significant sensory block. Why was this patient then given a spinal anaesthetic? If the height of the sensory block had receded it could easily have been extended by injecting more local anaesthetic via the extradural catheter. Furthermore, there are several reports in the literature where very high or total spinal blocks have occurred when spinal anaesthesia has been performed after extradural anaesthesia.\(^3,4\)

Third, two mothers received accidental i.v. injection of ropivacaine 75 mg and 150 mg, respectively. The second of these patients developed mild signs of i.v. injection after a dose of 75 mg but symptoms resolved, and therefore the authors gave another 75 mg at which time the symptoms recurred. Surely the authors should have stopped injecting after the initial 75 mg, aspirated the extradural catheter and waited a few minutes to see if any signs of sympathetic, sensory or motor block developed. If no signs of extradural block had developed the catheter should have been assumed to have been i.v. Furthermore, were there any signs of i.v. catheterization at the time of sitting and were the catheters aspirated before injecting the test dose? It is fortunate that the study was not double-blind with ropivacaine!

Fourth, the authors failed to mention which form of anaesthetic was administered to the mother who received ropivacaine 150 mg i.v. The authors did not give the results of neonatal outcome in the two mothers who had accidental i.v. injection. Were their NACS scores normal?

Finally, as the authors mentioned in their discussion, the open design of the study had limitations, namely it prevented true comparisons with other agents or even with 0.5% ropivacaine. The conclusions of the study were somewhat predictable in that 0.75% ropivacaine should provide better analgesia compared with 0.5% ropivacaine or bupivacaine.\(^5\) However, what is interesting is that 21 of 31 neonates in this study had a 2-h NACS score of less than 35 while in the study of Griffin and Reynolds,\(^6\) where 0.5% ropivacaine was used, only five of 31 neonates had a 2-h NACS score of less than 35.

M. PATEL
A. BAXTER
Department of Anaesthetics
Royal Women's Hospital
Melbourne, Australia


5. Griffin RP, Reynolds F. Extradural anaesthesia for Caesarean section: double blind comparison of 0.5% ropivacaine with 0.5% bupivacaine. British Journal of Anaesthesia 1995; 74: 512–516.

Sir,—We thank Drs Patel and Baxter for their interest in our article. The reasons for the results from seven women being invalid for efficacy analysis were technical difficulty in siting the extradural catheter or failure to establish sensory block. Blood samples from another two women (with satisfactory extradural blocks) were not available for analysis for reasons stated in the article. Many authors do not report the fate of patients who are excluded from a study after recruitment (i.e. after giving informed consent) but we believe it is good scientific practice to do so. Furthermore, because ropivacaine was then an unlicensed drug, we felt it important to report the results from all patients who received it, including four of the above seven women (2 with satisfactory extradural blocks).

Drs Patel and Baxter question our choice of subarachnoid anaesthesia for the woman whose Caesarean section was delayed, inferring from the pooled data that she should either have had adequate block or block could have been extended. First, a median duration of sensory block at T6 of 3.4 h does not imply that this patient’s block would have been adequate at the time of surgery. Second, we do not agree that her block could have easily been extended. The reason that she had received additional local anaesthetic was because of inadequate block. In addition, we were concerned about the possibility of systemic local anaesthetic toxicity. We are aware of the reports of high block when subarachnoid anaesthesia is used after inadequate extradural block but felt that the balance of risks still favoured this technique.

The second of the two mothers who received accidental i.v. injections of ropivacaine underwent Caesarean section under subarachnoid block. She received 0.75% ropivacaine 20 ml because...
her symptoms (not signs) were inconclusive. The injection was given slowly, which we believe is vital for safety, and therefore even if bupivacaine had been used, warning symptoms should have occurred before serious systemic toxicity developed. All extradural catheters were placed and all clinical decisions made by the consultant in charge of the patient’s anaesthetic care. This person was not the investigator. As stated in the article, all catheters were aspirated.

NAC scores at 2 h and 24 h for the two women who received accidental i.v. injections were 30, 31 and 33, 36, respectively. None of the neonates in the study gave clinical cause for concern except that, as discussed in the article, one developed transient tachypnoea of the newborn.

C. P. J. MORTON
J. H. MCCLURE
S. BLOOMFIELD
Department of Anaesthetics
Royal Infirmary of Edinburgh NHS Trust
Edinburgh

The expanding role of simulators in risk management

Sir,—We read with interest the recent correspondence by Dr Byrne and Professor Jones in response to Professor Spences’ editorial on the expanding role of simulators in risk management. We are also aware of the excellent work that is being done with the ACCESS simulator.

We admit that we are fortunate in Bristol to have acquired one of the two commercially available American high-fidelity simulators. The METI version that we purchased cost approximately $200 000. The rest of the project money was used to house the simulator in a purpose-built education centre with additional computer-assisted learning (CAL) and conference facilities. The METI high-fidelity simulator can be used by single-handed operators for individual or group tutorial style teaching. It can also be used for the more glamorous scenario-based “anaesthesia crisis resource management” training. It is true that this type of training is time consuming, requires many trainers and benefits only a few trainees at a time.

In Bristol, we are encouraging the involvement of committed medical teachers (not just anaesthetists) to use our centre. Medical students and trainee anaesthetists attend the centre for tuition on a regular basis. Geographical location does mean that those in the South West of England are more likely to benefit from informal teaching programmes but we hope the commercial courses will appeal to many around the country. The manikin is immobile at present but it is our intent that teaching and training by simulation be available to a wider audience in the future. Pilot projects in the USA involve moving the simulator from one hospital base to another. This is potentially damaging to expensive equipment but mobile units may be available in the future. Alternatively, teledicine links may provide opportunities for simulation teaching to a wider audience.

If high-fidelity simulators are to be used for trainee assessment and continuing medical education in the future, several points need to be considered. First, access to simulator facilities will need to be readily available. Second, trainers using simulators will need to cooperate and develop teaching and assessment programmes together, and third, the benefits of simulation as a teaching tool or in improving clinical performance need to be validated.

We are sure that the future success and developments of simulation lies in the collaborative efforts of those working in this exciting field of education.

F. FORREST
S. MATHER
M. TOOLEY
Bristol Medical Simulation Centre Management Board
Bristol