Unexpected high spinal block in obstetrics

Sir,—Elliott, Voyvodic and Brownridge recently reported an unexpected subarachnoid block in a parturient, despite careful aspiration testing and prior administration of local anaesthetic via a catheter, presumably located in the lumbar extradural space.1

We have recently described five similar cases of high spinal block produced by the initial test dose alone after negative aspiration testing.2 We have had three additional cases since our report, two in labouring parturients and one in an elderly woman undergoing ankle surgery. In common with our previously reported cases, patients were seated during insertion and testing of the catheter, and 18-gauge Tuohy needles with 20-gauge open-tip, single-orifice polyamide catheters were used (B. Braun Medical, Inc., Bethlehem PA, USA). After uneventful catheter insertions, carefree local anaesthetic was administered (3 ml of glucose-free 1.5% lignocaine with adrenaline 1 : 200 000) produced abrupt high sensory block and complete lower extremity motor block consistent with subarachnoid injection in each patient. High spinal block was accompanied by profound hypotension in both parturients. One developed a high motor block resulting in ventilatory failure requiring tracheal intubation, followed by emergency Caesarean delivery for persistent fetal bradycardia. In each case, CSF was aspirated easily through the catheter with subsequent negative pressure aspiration testing. To help detect a subdural catheter that becomes subarachnoid after injection, we perform a negative pressure aspiration test before and after injection of 3 ml of saline, as suggested by some authors.3 Injection of 3 ml of saline should be as effective as, and safer than, a local anaesthetic test dose in detecting arachnoid rupture. Furthermore, we have abandoned the lignocaine test dose. Abraham and colleagues proposed hyperbaric 1.5% lignocaine (Xylocaine, Astra, Westboro, MA, USA) as a safe alternative intrathecal marker,4 however, this incurs significant added expense ($6.65 per 2 ml vial in the USA). Instead, we combine adrenaline 15 μg with 2 ml of glucose-free 0.25% bupivacaine to produce a combined i.v. and intrathecal test solution. Bupivacaine 0.25% (2 ml) has been shown to safely provide rapid onset of labour analgesia and sensory block, with little risk of hypotension or high motor block when administered intrathecally.5 We find the inclusion of adrenaline useful because of the 5% incidence of i.v. cannulation associated with this catheter in our institution. These changes in both aspiration technique and test dose should minimize the occurrence of unexpected high spinal block without increasing expense or time.

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Figure 1 While initial placement of the catheter is subdural, injection of fluid results in disruption of the arachnoid mater. After removal of the catheter, non-adjacent dural and arachnoid defects create a flap valve minimizing CSF leak.

4. Palkar NV, Boudreaux RC, Mankad AV. Accidental total Tuohy needle or 20-gauge extradural catheter is unlikely. However, absence of PDPH might be expected if the dural puncture site and site of arachnoid tear do not overlap, as we propose (fig. 1). After removal of the catheter, positive hydrostatic pressure in the subarachnoid space generated in the upright position should effectively seal the dural hole with underlying arachnoid mater. This contrasts with the more common occurrence of inadvertent dural puncture recognized by immediate CSF flow through the needle or catheter, in which the dural and arachnoid puncture sites directly overlap each other. Such cases have a high risk of PDPH.

Because of the high blocks and hazardous associated effects observed, we have altered our testing of extradural catheters. To help detect a subdural catheter that becomes subarachnoid on injection, we perform a negative pressure aspiration test before and after injection of 3 ml of saline, as suggested by some authors.3 Injection of 3 ml of saline should be as effective as, and safer than, a local anaesthetic test dose in detecting arachnoid rupture. Furthermore, we have abandoned the lignocaine test dose. Abraham and colleagues proposed hyperbaric 1.5% lignocaine (Xylocaine, Astra, Westboro, MA, USA) as a safe alternative intrathecal marker,4 however, this incurs significant added expense ($6.65 per 2 ml vial in the USA). Instead, we combine adrenaline 15 μg with 2 ml of glucose-free 0.25% bupivacaine to produce a combined i.v. and intrathecal test solution. Bupivacaine 0.25% (2 ml) has been shown to safely provide rapid onset of labour analgesia and sensory block, with little risk of hypotension or high motor block when administered intrathecally.5 We find the inclusion of adrenaline useful because of the 5% incidence of i.v. cannulation associated with this catheter in our institution. These changes in both aspiration technique and test dose should minimize the occurrence of unexpected high spinal block without increasing expense or time.

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Figure 1 While initial placement of the catheter is subdural, injection of fluid results in disruption of the arachnoid mater. After removal of the catheter, non-adjacent dural and arachnoid defects create a flap valve minimizing CSF leak.


6. Abraham RA, Harris AP, Maxwell LG, Kaplow S. The efficacy of 1.5% lidocaine with 7.5% dextrose and epinephrine as an epidural test dose for obstetrics. Anesthesia 1986; 64: 116–119.


Is Doppler monitoring mandatory in laparoscopic surgery?

Sir,—In the article published recently by Nyarwaya and colleagues1 on carbon dioxide embolism in piglets, the authors observed some disturbances in $P_{\text{CO}_2}$ and $S_{\text{O}_2}$, with few changes in haemodynamic variables, and they concluded that “The presence of Doppler monitoring should be mandatory” in laparoscopic surgery because of the risk of carbon dioxide embolism which may not be detected with usual monitoring (ECG, AP, $S_{\text{O}_2}$, $P_{\text{O}_2}$).

Although it is true that carbon dioxide embolism is infrequent during laparoscopic surgery,2 most occur without any clinical disturbances and are detected only by transoesophageal echocardiography or precordial Doppler, without the need for therapy. For these reasons, in common with other authors, we believe that monitoring of patients without previous pathology should include ECG, AP, $P_{\text{CO}_2}$ and $S_{\text{O}_2}$. Doppler monitoring, pulmonary catheter, or both, is recommended in patients at risk, although the indications for laparoscopic surgery should be assessed carefully and attention paid to the important haemodynamic changes that occur with pneumoperitoneum.3,4

Finally, although carbon dioxide embolism occurs infrequently in laparoscopic surgery, the presence of a patent foramen ovale must be excluded because of the danger of paradoxical embolism.5,6

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Sir,—The letter of Ferrando and colleagues emphasizes at least two important points concerning the risk of gas embolism in anaesthesitized patients undergoing laparoscopic surgery. First, they suggest assessing, before operation, in any patient undergoing laparoscopic surgery, whether or not a patent foramen ovale exists. We agree totally with this proposal because of the potential occurrence of venous gas embolism, and subsequent risk of “paradoxical” systemic embolism. Nevertheless, the value of such a preoperative testing procedure will never reach a safety level of 100%. Second, as reported previously (and supported also more recently experimentally), signs of mild carbon dioxide embolism may be difficult to detect if routine monitoring is restricted to ECG, NIBP, $S_{\text{O}_2}$ and $P_{\text{O}_2}$. For a fixed or even progressively increasing carbon dioxide embolism, the value of $P_{\text{O}_2}$ monitoring for quantification of the magnitude of the carbon dioxide embolism is highly questionable (see fig. 5, upper trace). In any circumstances, carbon dioxide as any other gas embolism is easily detected by more sensitive and specific diagnostic tools, such as pre-thoracic or oesophageal echocardiography.7

For our group, it is not appropriate to expose the patient to the clearly increased risk of gas embolism of changing composition,8 despite the fact that the nature of the injected gas is obviously carbon dioxide at the start, without having specific and sensitive monitoring for early detection of such events.

In the case of gas embolism with peritoneal carbon dioxide insufflation, the difference between a catastrophe9 and an incident may be the promptness of correct diagnosis obtained with sensitive and specific monitors followed by rapid adequate corrective manoeuvres. For these reasons, we believe that Doppler monitoring is mandatory in laparoscopic surgery.

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Fuzzy logic control of inspired volatile anaesthetic concentrations

Sir,—The article of Curatolo and colleagues on fuzzy logic control of inspired isoflurane and oxygen concentrations is in our opinion an important contribution to automatic control of the gaseous composition in the circle system. This seems to be the method of the future to facilitate minimal flow techniques to the majority of anaesthetists. Automatic control, however, should be capable of competing with manually controlled methods.

Our concerns are about the method in which the volatile anaesthetic delivery was calculated. The formula used by the authors seems to take the volatile anaesthetic as a fraction of the fresh gas passing the flowmeters. However, the volatile anaesthetic is added to that gas flow so that total gas flow (100%) consists of the
volatile anaesthetic, as set at the vaporizer (vap%), in addition to the fresh gas passing the flowmeters (100−vap%). Thus volatile delivery has to be calculated as follows:1

$$\frac{\text{EGF}}{100} = \frac{\text{FGF} (\text{ml min}^{-1})}{1 - \frac{\text{vap}}{100}}$$

where FGF = sum of fresh gas set at the flowmeters (ml min⁻¹) and Vap = vaporizer setting (vol%).

Comparison of the results from the two formulae shows that volatile delivery calculated by the formula used in this article is underestimated to a percentage corresponding to the vaporizer setting (vap%).2 While 1–5% may be negligible for traditional volatile agents and when only costs are of concern, the difference becomes relevant with volatile agents used in high concentrations, such as desflurane, and in relevant studies, such as the one considered here. The article compared a “low flow” and a “minimal flow” technique. During minimal flow, the vaporizer is at higher settings than during low flow. Consequently, underestimation of volatile consumption is more pronounced in the minimal flow group than in the low flow group. Thus the data given in the article show a slightly greater difference in delivery between the two groups than is actually there.

Compared with our own experience, volatile anaesthetic consumption reported in this study with respect to only the maintenance period appears to be high. In manually controlled minimal flow techniques we calculated an average isoflurane delivery of only 12.9 ml min⁻¹ in 20 entire anaesthetics for the period from intubation to extubation (thus including the wash-in periods) lasting 149±69 min maintained at an end-tidal isoflurane concentration of approximately 0.8 vol%2. We used an Ohmeda Modulus CD/CV with the MAS circle system, the volume of which is approximately 1.5 litre less than that of the Dräger Cicoer used by Curatolo and co-workers.

The frequent sharp increases in fresh gas flow to remove foreign gas from the circle system are quoted to explain why the net difference in volatile consumption between the two flow rates is relatively small. One may consider that the higher volume of the circle system makes the excess gas during minimal flow and the high flow flush less effective in removing foreign gases from the circle system. Furthermore, the system response to changes in the vaporizer setting are slower with a larger circle system. These factors may have contributed to the lower volatile anaesthetic consumption in our work.

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Sir,—We thank Drs Wissing and Kuhn for their interest in our investigation and for their comments.

We agree that the formula to calculate isoflurane delivery suggested by Drs Wissing and Kuhn gives a more precise estimation. However, as they pointed out, underestimation resulting from our formula is quantitatively insignificant and does not therefore influence the conclusions of the investigation. We agree that a more accurate calculation should be used when performing a similar study on desflurane.

Concerning the difference in isoflurane consumption between our study and the experience of Drs Wissing and Kuhn, we believe there may be a comparison cannot be made without more detailed information on patients characteristics, premedication, co-administration of other analgesics, anaesthetist involved, surgical procedures, etc. All of these factors can greatly affect isoflurane delivery. Furthermore, median fresh gas flow in our study was higher than 0.5 litre min⁻¹ (table 5) because of the occasional activation of the “turbo” mode (fresh gas flow 10 litre min⁻¹) when a rapid change in the inspired isoflurane concentration was desired.

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Hypotension during subarachnoid anaesthesia

Sir,—The article by Drs Critchley and Conway1 investigating the effects of colloid or metaraminol, or both, in patients receiving subarachnoid anaesthesia to facilitate surgery for fractured neck of femur makes interesting reading. However, I should like to make two comments, particularly concerning their conclusion that “hypotension may be treated more effectively by using a vasoconstrictor”. First, elderly patients presenting for fractured neck of femur surgery are, in my experience, almost invariably hypovolaemic, through loss of considerable blood volume into the fracture site and also through inadequate fluid intake in the orthopaedic wards. Performing subarachnoid block in hypovolaemic patients is dangerous2 and therefore I believe that correction of hypovolaemia is of paramount importance in this group of patients before the use of any vasoconstrictor therapy. The statistically significant increases in mean cardiac index and central venous pressure reported in the colloid only group may represent adequate fluid replacement.

Second, we do not know if the use of metaraminol is without risk, particularly when used as an infusion in these elderly patients with potential widespread atherosclerosis. Is the routine use of a vasoconstrictor in the treatment of modest hypotension of any improved long-term benefit to the patient compared with fluid replacement therapy? While I accept that this was a physiological study examining the short-term effects of colloid and metaraminol, I do not believe that one can draw conclusions on which is the better treatment of hypotension without examining the incidences of postoperative complications.

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Sir,—Thank you for the opportunity to reply to Dr Watters’ letter commenting on our recent article on the use of colloids and metaraminol during spinal anaesthesia in patients with femoral neck fractures.1

Normovolaemia is essential before spinal anaesthesia. It is therefore standard practice in our hospital that all patients admitted with femoral neck fractures have i.v. hydration on admission to compensate for blood loss at the fracture site and fasting. Despite this practice, patients with femoral neck fractures still experience a greater decrease in arterial pressure during spinal anaesthesia compared with other groups, such as urology patients.2 We believe that this increase in hypotension is caused by vasodilatation of blood vessels that become abnormally vasoconstricted as a result of the pain and trauma associated with the fracture site.

In our study we measured the effects of treatment with colloid and metaraminol on preload during spinal block. We found that both colloid 8 ml kg⁻¹ and metaraminol by i.v. infusion were equally effective in correcting decreases in preload. However, the
Haemodilution induces a hypercoagulable state

Sir,—We read with interest the article by Ruttmann, James and Viljoen on haemodilution-induced hypercoagulability. While we agree that the reported effect might be a pitfall of non-activated TEG measurements alone, we believe that our observations are not inconsistent with theirs.

In a similar investigation involving 12 volunteers, 10 ml of citrated blood were obtained and native TEG was performed with undiluted blood (control) and with a 20% dilution with 20% saline (native). The volunteers then received 1 litre of the same saline. The volunteers then received 1 litre of the same saline solution i.v. (equal to a 20% dilution) and a TEG measurement was performed. Our results are shown in Table 1.

These data confirm the results of Ruttmann, James and Viljoen showing a hypercoagulable state when saline was mixed in vitro. However, after i.v. infusion of saline, no significant changes in TEG variables were observed. We thus suggest that the reported effect might be an in vitro phenomenon. Our opinion is enforced by observations using activated TEG (activation with Innovin 5 μl) which revealed no significant changes when saline was added in vitro. This indicates that the in vitro effect might be a pitfall of native thrombelastography.

There are no data to explain this phenomenon. Non-activated (native) thrombelastography is a very sensitive method for evaluating factors influencing coagulation. This is seen for example in the long r time of the control in this study (9 min). Conclusions based on the effects seen in non-activated thrombelastographic measurements to the in vitro situation should be drawn with great care as the activation situation in non-activated thrombelastographic measurements does not reflect the in vivo situation. In the body, coagulation takes place mainly after severe extrinsic activation, for example after vascular injury.

The low contact activation of the coagulation system in native TEG measurements is not a physiological situation. With respect to the better clot formation and higher MA in the in vitro saline tests compared with the control, the following mechanism should be excluded: it is known that a high sedimentation rate leads to an artificially high MA. Thus sedimentation processes during the relatively long measurement periods may play a role. Also, clot formation in non-activated TEG is influenced by many factors. A study with non-activated TEG measurements alone might not be sufficient to explain these complex mechanisms.


Sir,—We thank Dr Entholzner and colleagues for their comments. It is of interest that their in vitro studies confirmed our in vitro results, despite the fact that they used citrated blood before performing the TEG studies. We have recently conducted some preliminary tests using immediately sampled blood and comparing it with the same blood citrated and then reconstituted with calcium in a manner identical to that recommended by the TEG manufacturers, that is 250 μl of citrated blood placed in the TEG cup to which is added 100 μl of 0.65% calcium chloride solution. Our observation was that the citrated and reconstituted blood clotted much more vigorously (shorter r and k times, steeper angle and larger MA) than the non-treated controls. We are in the process of validating this observation at the present time. We are, therefore, sceptical that citrated blood truly represents the native state and that the results of Entholzner and colleagues may have reflected this.

The comment that the effect may be an in vitro phenomenon is of interest and we had already considered this possibility. A subsequent study of in vivo haemodilution has been performed by us and is currently under consideration for publication and we cannot, therefore, publish the data. However, in essence, this study showed a similar but slightly smaller phenomenon in the in vivo haemodilution compared with our in vitro experiments. We suspect that the difference observed by Entholzner and colleagues may be a result of their use of citrated blood.

As regards the difference between native and activated thrombelastography, we find their comments difficult to understand. First, the comment regarding the long r time of the control group in the study (9 min) makes no sense whether or not it refers to their letter or to our own data. It is true that the non-activated TEG measurement reflects only the activity of the intrinsic pathway. Whether or not the activated TEG truly reflects the activity created by the addition of tissue thromboplastins, such as occurs with tissue injury, is speculative and not supported by our knowledge.

While it is true that we speculated on several possible mechanisms for this phenomenon, in our subsequent in vivo study, we have also demonstrated a decrease in antithrombin III, thus confirming the possibility raised by Monkhouse that this may be at least part of the mechanism. Therefore, we cannot accept the suggestion of Entholzner and colleagues that our results are an in vitro effect. The article published by Ng and Lo confirms that not only is this effect demonstrable in vivo but is demonstrable in the presence of major surgery where, presumably, activation of coagulation by tissue thromboplastins has taken place. We believe that our observations reflect a real clinical entity and not simply an in vitro aberration.


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Table 1 Mean (SD) TEG values before and after in vitro and in vivo dilution with 20% saline (n = 12). *P < 0.05 compared with control (Wilcoxon)

<table>
<thead>
<tr>
<th>Control</th>
<th>In vitro dilution</th>
<th>In vivo dilution</th>
</tr>
</thead>
<tbody>
<tr>
<td>r (min)</td>
<td>7.9 (2.3)</td>
<td>5.8 (1.9)</td>
</tr>
<tr>
<td>k (min)</td>
<td>4.8 (1.9)</td>
<td>2.6 (1.4)</td>
</tr>
<tr>
<td>MA (mm)</td>
<td>51.9 (6.1)</td>
<td>56.7 (6.5)</td>
</tr>
</tbody>
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Haemodynamic and neuroendocrine responses after pneumoperitoneum during cholecystectomy

Sir,—O’Leary and colleagues1 investigated haemodynamic and neuroendocrine responses after pneumoperitoneum and body position changes during cholecystectomy.

Direct mechanical compressive effect on the abdominal aorta is unlikely to contribute to the increases in cardiac afterload and arterial pressure observed during laparoscopic surgery. Indeed, in human studies, haemodynamic alterations associated with laparoscopic surgery persisted for more than 20 min after pneumoperitoneum had been evacuated.2 Moreover, an acute 9 mm Hg increase in abdominal pressure reproduced in a dog model the haemodynamic changes associated with laparoscopic surgery in humans.3 Such a low pressure applied to the aortic high pressure system is unlikely to have induced the reported cardiac output redistribution towards the upper part of the body.4

Hypercapnia is unlikely to contribute to the haemodynamic changes associated with laparoscopic surgery. Indeed, during prolonged intraperitoneal carbon dioxide insufflation, carbon dioxide diffusion into the body has been demonstrated to be limited.5 It results in a slight increase in Pco2, accounted for by the Chassard and co–diaphragmatic impairment induced by increasing intraperitoneal pressure.6 In the study of O’Leary and colleagues,1 end-tidal Pco2 was easily maintained at 0.5–0.7 kPa.

Hormonal responses described during laparoscopic surgery have been elicited by mechanisms to reflect the degree of surgical stress in “conventional” open surgery.7 Vasopressin release paralleled changes associated with surgery. Indeed, during infusions, there was a close correlation between changes in serum sodium and albumin-corrected serum calcium concentrations. After infusion, however, serum calcium concentration was restored more rapidly than serum sodium concentration. Dr Chassard and colleagues also found a close correlation between serum sodium and free (ionized) calcium concentrations during the first 20 min of there study. Thereafter, serum calcium remained essentially unchanged, although the glucose infusion continued for another 40 min. All of these findings suggest that calcium, but not sodium, is involved in this process.8

The principal difference between the correction of diluted concentrations of sodium and calcium is that the latter ion can be mobilized easily from loosely bound bone stores, and equilibration has been claimed to occur with a half-time of 70 min.9 The relatively poor perfusion of bone certainly explains some of this delay.10 I enjoyed reading the article by Dr Chassard and colleagues on the effect of glycine solution on calcium homeostasis and would like to suggest an interpretation of their results. I have measured serum calcium concentrations during i.v. infusion of 1000 ml of glycine solution over 20 min in young men and in prostatectomy patients.1 A review of my original data shows that, during infusion, there was a close correlation between changes in serum sodium and albumin-corrected serum calcium concentrations. After infusion, however, serum calcium concentration was restored more rapidly than serum sodium concentration. Dr Chassard and colleagues also found a close correlation between serum sodium and free (ionized) calcium concentrations during the first 20 min of there study. Thereafter, serum calcium remained essentially unchanged, although the glucose infusion continued for another 40 min. All of these findings suggest that calcium, but not sodium, is involved in this process.8

Dilution of serum sodium during glycine infusions is governed mainly by the volume of irrigant absorbed. Correction occurs by diffusion of glycine and irrigant water from the extracellular to the intracellular fluid compartment, and marked, but delayed, cellular oedema develops.11 Urine excretion also plays a part, but the urine contains large amounts of sodium,12 particularly when a large volume of glycine solution is given.13 The principal difference between the correction of diluted concentrations of sodium and calcium is that the latter ion can be mobilized easily from loosely bound bone stores, and equilibration has been claimed to occur with a half-time of 70 min.9 The relatively poor perfusion of bone certainly explains some of this delay.10 I enjoyed reading the article by Dr Chassard and colleagues on the effect of glycine solution on calcium homeostasis and would like to suggest an interpretation of their results. I have measured serum calcium concentrations during i.v. infusion of 1000 ml of glycine solution over 20 min in young men and in prostatectomy patients.1 A review of my original data shows that, during infusion, there was a close correlation between changes in serum sodium and albumin-corrected serum calcium concentrations. After infusion, however, serum calcium concentration was restored more rapidly than serum sodium concentration. Dr Chassard and colleagues also found a close correlation between serum sodium and free (ionized) calcium concentrations during the first 20 min of there study. Thereafter, serum calcium remained essentially unchanged, although the glucose infusion continued for another 40 min. All of these findings suggest that calcium, but not sodium, is involved in this process.8

Some degree of “self-treatment” of the hypocalcaemia can be expected as the TURP syndrome is associated with metabolic acidosis, although this is usually fully compensated until circulatory disturbances occur.10


Glycine absorption and hypocalcaemia

Sir,—I enjoyed reading the article by Dr Chassard and colleagues on the effect of glycine solution on calcium homeostasis and would like to suggest an interpretation of their results. I have measured serum calcium concentrations during i.v. infusion of 1000 ml of glycine solution over 20 min in young men and in prostatectomy patients.1 A review of my original data shows that, during infusion, there was a close correlation between changes in serum sodium and albumin-corrected serum calcium concentrations. After infusion, however, serum calcium concentration was restored more rapidly than serum sodium concentration. Dr Chassard and colleagues also found a close correlation between serum sodium and free (ionized) calcium concentrations during the first 20 min of there study. Thereafter, serum calcium remained essentially unchanged, although the glucose infusion continued for another 40 min. All of these findings suggest that calcium, but not sodium, is involved in this process.8

Some degree of “self-treatment” of the hypocalcaemia can be expected as the TURP syndrome is associated with metabolic acidosis, although this is usually fully compensated until circulatory disturbances occur.10

3. Hahne R, Hahn RG. Glycine absorption and hypocalcaemia


Sir,—We are grateful to Dr Hahn for his help in the discussion of our recent findings on the effects of glycine infusion on calcium homeostasis. A readily exchangeable reservoir is responsible for 500 mmol of calcium exchange per day from bone to extracellular fluid. As he suggested, this could have contributed to the absence of further effect of glycine on ionized calcium after 20 min of i.v. infusion. However, the pH-normalized ionized calcium remained unchanged throughout the study, despite acute absorption of glycine (equivalent to 2000 ml for humans). As we concluded, changes in plasma sodium concentration rather than changes in calcium homeostasis are probably more important in the development of the TURP syndrome. As previous studies and Dr Hahn suggested, we encourage further clinical studies, including monitoring calcium concentrations during acute TURP. This could help in the decision to introduce calcium therapy in the resuscitation of cardiovascular disturbances in this syndrome.

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**Locating an arterial foreign body by ultrasonography**

Sir,—Shah, Downing and Davis described severance of a segment of catheter after arterial cannulation leading to embolization within the radial artery. They reported that surgical exploration of the distal portion of the artery was unsuccessful and the fragment was finally retrieved after proximal radial artery exploration. We write to suggest that real-time ultrasound imaging might have been helpful by allowing rapid location of the fragment before surgical removal. Lightweight, portable machines are available specifically for anaesthetic use (Site Rite, Dymax Corporation, Pittsburgh, PA, USA). These machines provide good quality images of vascular anatomy, especially in the neck, and their use as an aid to internal jugular vein cannulation represents an advance in safety and efficacy in adult and paediatric patients. The device can also be used to demonstrate the course and calibre of peripheral arteries. A short-focus probe (9 MHz frequency) with a focal length of 0.7 cm from the probe cap can easily identify an intra-luminal foreign body, which is displayed on the screen as a densely echogenic image. This allows rapid and non-invasive ultrasonic assessment of the radial artery “at the bedside”. Sterile, acoustically transparent probe sheaths are available to allow aseptic examination.

We suggest that ultrasonic assessment in a situation such as this would permit confident localization of a wayward fragment and, if available, represents a potential advance in management.

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