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CORRESPONDENCE

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 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, careful negative pressure aspiration produced no fluid. The test dose 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. As in our previous cases, none of the patients developed postdural puncture headache (PDPH), despite dural and arachnoid punctures.

Several authors have suggested that "a subdural catheter can perforate the arachnoid matter...at any time"[1] and, "the test dose given through a catheter placed in the subdural space causes the arachnoid to tear".[3] We agree, and provide the accompanying figure (fig. 1) as a unified explanation of the mechanisms underlying arachnoid rupture and the associated low incidence of PDPH. Although initial placement of the catheter is subdural, subsequent arachnoid rupture occurs because of sudden positive pressure generated in the low-compliance subdural space by injection of a small volume of fluid. This is consistent with findings common to all our cases, namely absence of CSF during placement of the catheter, inability to aspirate fluid before catheter injection, and free flow of CSF after injection and onset of spinal block. Elliott, Voyvodic and Brownridge did not state if CSF was aspirated after the onset of subarachnoid block, however, computed tomography demonstrated clearly subarachnoid catheter placement in their patient.

Also, consistent with this mechanism is the absence of PDPH in all cases described by us (seven of whom were labouring parturients) and the single parturient described by Palkar, Boudreaux and Mankad.[4] Such a low incidence of PDPH in parturients experiencing arachnoid puncture with an 18-gauge 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.[5] 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,[6] 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.[7] 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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1. Elliott DW, Voyvodic F, Brownridge P. Sudden onset of subarachnoid block after subdural catheterization: a case of arachnoid rupture? *British Journal of Anaesthesia* 1996; **76**: 322–324.
2. Richardson MG, Lee AC, Wissler RN. High spinal anesthesia after epidural test dose administration in five obstetric patients. *Regional Anesthesia* 1996; **21**: 119–123.
3. Reynolds F, Speedy HM. The subdural space: the third place to go astray. *Anaesthesia* 1990; **45**: 120–123.
4. Palkar NV, Boudreaux RC, Mankad AV. Accidental total

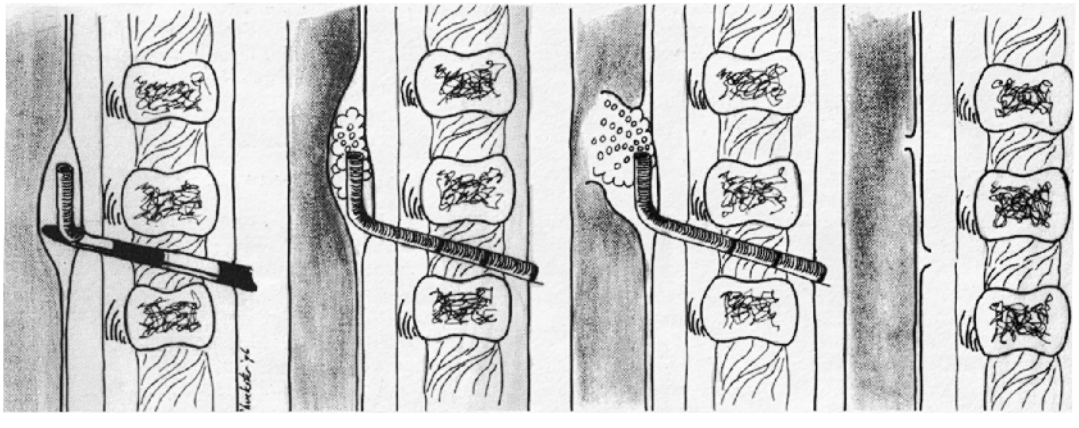

Figure 1 While initial placement of the catheter is subdural, injection of fluid results in disruption of the arachnoid mater. After removal of the catheter, non-adjacent dural and arachnoid defects create a flap valve minimizing CSF leak.

spinal block: a complication of an epidural test dose. *Canadian Journal of Anaesthesia* 1992; **39**: 1058–1060.
5. Brown DL. Spinal, epidural, and caudal anesthesia: anatomy, physiology, and technique. In: Chestnut DH, ed. *Obstetric Anesthesia: Principles and Practice*. St Louis: Mosby Yearbook, 1994; 181–201.
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. *Anesthesiology* 1986; **64**: 116–119.
7. Stacey RGW, Watt S, Kadim MY, Morgan BM. Single space combined spinal–extradural technique for analgesia in labour. *British Journal of Anaesthesia* 1993; **71**: 499–502.

Is Doppler monitoring mandatory in laparoscopic surgery?

Sir,—In the article published recently by Nyarwaya and colleagues[1] on carbon dioxide embolism in piglets, the authors observed some disturbances in $P_{E'CO_2}$ and Sp_{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, Sp_{O_2}, $P_{E'CO_2}$).

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,[3] without the need for therapy. For these reasons, in common with other authors,[4] we believe that monitoring of patients without previous pathology should include ECG, AP, $P_{E'CO_2}$ and Sp_{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.[5,6]

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.[7,8]

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2. Derouin M, Couture P, Boudreault D, Girard D, Gravel D. Detection of gas embolism by transesophageal echocardiography during laparoscopic cholecystectomy. *Anesthesia and Analgesia* 1996; **82**: 119–124.
3. Delafosse B, Cottin V, Motin J. Embolie gazeuse en chirurgie coelioscopique: monitorage et prevention. Editions Techniques. Encycl Med Chir; Anesthesie-Reanimation, F.A. 36-568-A10,1994.
4. Ananthanarayan C, Paek W, Rolbin SH, Dhanidina K. Hysteroscopy and anaesthesia. *Canadian Journal of Anaesthesia* 1996; **43**: 56–64.
5. Portera C, Compton R, Walters D. Benefits of pulmonary artery catheter and transesophageal echocardiographic monitoring in laparoscopic cholecystectomy patients with cardiac disease. *American Journal of Surgery* 1995; **169**: 202–207.
6. Westerband A, Van De Water J, Amzallag M, Lebowitz PW. Cardiovascular changes during laparoscopic cholecystectomy. *Surgery, Gynecology and Obstetrics* 1992; **175**: 535–538.
7. Gronert GA, Messick JM, Cucchiara RF, Michenfelder JD. Paradoxical air embolism from a patient foramen ovale. *Anesthesiology* 1979; **50**: 548–549.
8. Failure of preoperative echo testing to prevent paradoxical air embolism: report of two cases. *Anesthesiology* 1989; **71**: 604–607.

Sir,—The letter of Ferrando and colleagues emphasizes at least two important points concerning the risk of gas embolism in anaesthetized 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%.[1] Second, as reported previously[2] (and supported also more recently experimentally[3]), signs of mild carbon dioxide embolism may be difficult to detect if routine monitoring is restricted to ECG, NIBP, Sp_{O_2} and $P_{E'CO_2}$. For a fixed or even progressively increasing carbon dioxide embolism, the value of $P_{E'CO_2}$ monitoring for quantification of the magnitude of the carbon dioxide embolism is highly questionable (see fig. 5, upper trace[3]). 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.[2,4] For our group, it is not appropriate to expose the patient to the clearly increased risk of gas embolism of changing composition,[5] 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 catastrophe[6,7] and an incident may be the promptness of correct diagnosis obtained with sensitive and specific monitors[4] followed by rapid adequate corrective manoeuvres. For these reasons, we believe that Doppler monitoring is mandatory in laparoscopic surgery.

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1. Cucchiara RF, Nishimura RA, Black S. Failure of preoperative testing to prevent paradoxical air embolism: Report of two cases. *Anesthesiology* 1989; **71**: 604–607.
2. Derouin M, Couture P, Boudreault D, Cirard D, Garvel D. Detection of gas embolism by transoesophageal echocardiography during laparoscopic cholecystectomy. *Anesthesia and Analgesia* 1996; **82**: 119–124.
3. Nyarwaya J-B, Pierre S, Mazoit J-X, Umbrain V, Romain M, Samii K, d'Hollander A. Effects of carbon dioxide embolism with nitrous oxide in the inspired gas in piglets. *British Journal of Anaesthesia* 1996; **76**: 428–434.
4. Couture P, Boudreault D, Derouin M. Venous carbon dioxide embolism in pigs: an evaluation of end-tidal carbon dioxide, transoesophageal echocardiography, pulmonary artery pressure, and precordial auscultation as monitoring modalities. *Anesthesia and Analgesia* 1994; **79**: 867–873.
5. Diemunsch P, Van Dorsselaer T, Mutter D, Diemunsch A-M, Teller G. Evolution of the nitrous oxide fraction in the carbon dioxide pneumo-peritoneum during laparoscopy under general anesthesia. *Anesthesiology* 1995; **83**: A-1085.
6. Crozier TA, Luger A, Dravecz M. Carbon dioxide embolism with cardiac arrest during hysteroscopy: report on three cases (German). *Anasthesiologica Intensivemed Notfallmed Schmerzther* 1991; **26**: 412–415.
7. Root B, Levy MN, Pollack S, Lubert M, Pathak K. Gas embolism death after laparoscopy by "trapping" in portal circulation. *Anesthesia and Analgesia* 1978; **57**: 232–237.

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]

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

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%.[3] 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 Cicero 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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1. Curatolo M, Derighetti M, Petersen-Felix S, Feigenwinter P, Fischer M, Zbinden AM. Fuzzy logic control of inspired isoflurane and oxygen concentrations using minimal flow anaesthesia. *British Journal of Anaesthesia* 1996; **76**: 245–250.
2. Kuhn I, Wissing H, Vetterman J. Ein neues Arbeitsblatt zur Berechnung von Narkosekosten-Validierung der Berechnungsverfahren über den Verbrauch volatiler Anästhetika. *Anästhesie Intensivmedizin* 1995; **36**: 242–248.
3. Kuhn I, Wissing H. Wirtschaftlichkeit von Desfluran im Vergleich zu Isofluran bei Niedrigflußnarkosen. *Anaesthesist* 1995; **44**: S459.

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 that such 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 Conway[1] 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 vasopressor".

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 dangerous[2] 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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1. Critchley LA, Conway F. Hypotension during subarachnoid anaesthesia: haemodynamic effects of colloid and metaraminol. *British Journal of Anaesthesia* 1996; **76**: 734–736.
2. Kennedy WF jr, Bonica JJ, Akamatsu TJ, Ward RJ, Martin WE, Grinstein A. Cardiovascular and respiratory effects of subarachnoid block in the presence of acute blood loss. *Anesthesiology* 1968; **29**: 29–35.

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

use of larger volumes of i.v. fluids to prevent hypotension has repeatedly been shown to be ineffective.[3–5] Thus vasopressors are commonly advocated. Although we cannot comment on the long-term effects of using metaraminol, we did limit our dose to the minimum required to maintain normal arterial pressure and by using an infusion we avoided excessive swings in arterial pressure.

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1. Critchley LAH, Conway F. Hypotension during subarachnoid anaesthesia: haemodynamic effects of colloid *vs* metaraminol. *British Journal of Anaesthesia* 1996; **76**: 734–736.
2. Critchley LAH, Stuart CJ, Short TG, Gin T. Haemodynamic effects of subarachnoid block in elderly patients. *British Journal of Anaesthesia* 1994; **73**: 464–470.
3. Ceo AJ, Revanas B. Is crystalloid preloading useful in spinal anaesthesia in the elderly? *Anaesthesia* 1990; **45**: 241–243.
4. Critchley LAH, Short TG, Gin T. Hypotension during subarachnoid anaesthesia: a haemodynamic analysis of three treatments. *British Journal of Anaesthesia* 1994; **72**: 151–155.
5. Rocke DA, Rout CC. Volume preloading, spinal hypotension and Caesarean section. *British Journal of Anaesthesia* 1995; **75**: 257–259.

Haemodilution induces a hypercoagulable state

Sir,—We read with interest the article by Ruttmann, James and Viljoen on hypercoagulability induced by 20% dilution of whole blood with isotonic saline or gelatine.[1] In this study, coagulation was measured using native thrombelastography (TEG), and a shortening of *r* and *k* times, and an increase in MA were found. We would like to report our own observations.

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% *in vitro* dilution using 0.9% saline. The volunteers then received 1 litre of the same saline solution i.v. (equal to a 20% dilution) and another 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. Thus we 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*.[2] 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.[3] 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.

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

	Control	*In vitro* dilution	*In vivo* dilution
r (min)	7.9 (2.3)	5.8 (1.9)*	9.0 (2.9)
k (min)	4.8 (1.9)	2.6 (1.4)	5.0 (1.5)
MA (mm)	51.9 (6.1)	56.7 (6.5)	47.5 (5.7)

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2. Entholzner E, Calatzis AN, Kling M, Mielke L, Hipp R. *Anaesthesist* 1996; **45**: A171.
3. Lechner K. Blutgerinnungsstörungen In: Lechner K, ed. *Laboratoriumsdiagnose hämatologischer Erkrankungen 2*. Berlin, Heidelberg, New York: Springer, 1982; 177.

Sir,—We thank Dr Entholzner and colleagues for their comments. It is of interest that their *in vitro* studies confirmed our *in vitro* results,[1] 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 α 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 to our knowledge by scientific evidence. 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[2] 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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1. Ruttmann TG, James MFM, Viljoen JF. Haemodilution induces a hypercoagulable state. *British Journal of Anaesthesia* 1996; **76**: 412–414.
2. Ng KFJ, Lo JWR. The development of hypercoagulability state, as measured by thrombelastography, associated with intra-operative surgical blood loss. *Anaesthesia and Intensive Care* 1996; **24**: 20–25.

Haemodynamic and neuroendocrine responses after pneumoperitoneum during cholecystectomy

Sir,—O'Leary and colleagues[1] 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.[3]

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.[4] It results in a slight increase in Pa_{CO_2} accounted for by the increase in respiratory dead space caused by diaphragmatic impairment induced by increasing intraperitoneal pressure.[5] In the study of O'Leary and colleagues,[1] end-tidal P_{CO_2} was easily maintained at 0.5–0.7 kPa.

Hormonal responses described during laparoscopic surgery have been shown previously to reflect the degree of surgical stress in "conventional" open surgery.[6] Vasopressin release parallelled the time course of the increase in systemic vascular resistances.[7] Changes in plasma concentrations of catecholamines, renin and cortisol were correlated with circulatory changes associated with general anaesthesia and reflect the stress response to surgery.[6–8] Changes in endothelin concentrations reflect tissue injury associated with surgery.[8]

More striking are studies showing that physical stimulation of intraperitoneal receptors increased systemic vascular resistances and arterial pressure.[9] Sympathetic nerve section significantly reduced circulatory changes not only in the presence of intraperitoneal physical stimulation but also in experimental intraperitoneal pressure elevation.[9]

We speculate that there may be involvement of baroreflexes elicited by various intra-abdominal viscera stimulation underlying the pathophysiology of circulatory changes associated with laparoscopic surgery in humans.

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1. O'Leary E, Hubbard K, Tormey W, Cunningham AJ. Laparoscopic cholecystectomy: haemodynamic and neuroendocrine responses after pneumoperitoneum and changes in position. *British Journal of Anaesthesia* 1996; **76**: 640–644.
2. Westerband A, Van De Water JM, Amzallag M, Lebowitz PW, Nwasorka ON, Chardavoyne R, Abou-Taleb A, Wang X, Wise L. Cardiovascular changes during laparoscopic cholecystectomy. *Surgery, Gynecology and Obstetrics* 1992; **175**: 535–538.
3. Robotham JL, Wise RA, Bromberger B. Effect of changes in abdominal pressure on left ventricular performance and regional blood flow. *Critical Care Medicine* 1985; **13**: 803–809.
4. Mullet CE, Viale JP, Sagnard PE, Miellet CC, Ruynat LG, Counioux HC, Motin JP, Boulez JP, Dargent DM, Annat GJ. Pulmonary CO_2 elimination during surgical procedures using intra or extraperitoneal CO_2 insufflation. *Anesthesia and Analgesia* 1993; **76**: 622–626.
5. Lister DR, Rudston-Brown B, Warriner B, McEwen J, Chan M, Walley KR. Carbon dioxide absorption is not linearly related to intraperitoneal carbon dioxide insufflation pressure in pigs. *Anesthesiology* 1994; **80**: 129–136.
6. Chernow B, Alexander R, Smallridge RG, Thompson WR, Cook D, Beardsley D, Fink MP, Lake R, Fletcher R. Hormonal responses to graded surgical stress. *Archives of Internal Medicine* 1987; **147**: 1273–1278.
7. McMahon AJ, O'Dwyer PJ, Cruik Shank AM, MC Millan DC, O'Reilly D St J, Lowe GDO, Rumley A, Logan PW, Baxter JN. Comparison of metabolic responses to laparoscopic and minilaparotomy cholecystectomy. *British Journal of Surgery* 1993; **80**: 1255–1258.
8. Joris J, Lamy M. Neuroendocrine changes during pneumoperitoneum for laparoscopic cholecystectomy. *British Journal of Anaesthesia* 1993; **70**: A34.
9. Longhurst JC, Spilker HL, Ordway GA. Cardiovascular reflexes elicited by passive gastric distension in anesthetized cats. *American Journal of Physiology* 1981; **240**: H539–H545.

Glycine absorption and hypocalcaemia

Sir,—I enjoyed reading the article by Dr Chassard and colleagues on the effect of glycine solution on calcium homeostasis[1] 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[2] and in prostatectomy patients.[3] 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 glycine infusion continued for another 40 min. All of these findings suggest that the mechanisms compensating for a diluted serum calcium concentration have the same strength as for serum sodium during 20–30 min of a glycine infusion, after which they become stronger for calcium.

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.[4] Urine excretion also plays a part, but the urine contains large amounts of sodium,[2 3] particularly when a large volume of glycine solution is given.[5] 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.[6] The relatively poor perfusion of bone certainly explains some of this delay.

I believe that the time-course of this calcium mobilization can explain the absence of a further reduction of ionized calcium at 40 min and 60 min of the glycine infusions given by Chassard and colleagues. This indicates that hypocalcaemia probably does not get worse after 20–40 min of glycine absorption in the clinic either. When irrigating fluid is absorbed very rapidly, however, severe hypocalcaemia may develop within this time and then contribute to the hypokinetic circulation seen in the TURP syndrome.[7] A role for calcium in the treatment of this syndrome has also been suggested by two reports of patients in whom cardiac arrest and hyponatraemia developed during prostatectomy.[8 9] 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]

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1. Chassard D, Berrada K, Tournadre JP, Boulétreau P. Calcium homeostasis during i.v. infusion of 1.5% glycine in anaesthetized pigs. *British Journal of Anaesthesia* 1996; **77**: 271–273.
2. Hahn RG, Stalberg HP, Gustafsson SA. Intravenous infusion of irrigating fluids containing glycine or mannitol with and without ethanol. *Journal of Urology* 1989; **142**: 1102–1105.
3. Hahn RG, Stalberg HP, Ekengren J, Rundgren M. Effects of 1.5% glycine solution with and without ethanol on the fluid balance in elderly men. *Acta Anaesthesiologica Scandinavica* 1991; **35**: 725–730.
4. Hahn RG, Nennesmo I, Rajs J, Sundelin B, Wroblevski R, Zhang W. Morphological and X-ray microanalytical changes in mammalian tissue after overhydration with irrigating fluids. *European Urology* 1996; **29**: 355–361.

5. Stalberg HP, Hahn RG, Hjelmqvist H, Ullman J, Rundgren M. Haemodynamics and fluid balance after intravenous infusion of 1.5% glycine in the sheep. *Acta Anaesthesiologica Scandinavica* 1993; **37**: 281–287.
6. Krohn JS. Dilutional hypocalcemia in association with dilutional hyponatremia. *Anesthesiology* 1993; **79**: 1136–1138.
7. Singer M, Patel M, Webb AR, Bullen C. Management of the transurethral prostatic resection syndrome: Time for reappraisal? *Critical Care Medicine* 1990; **18**: 1479–1480.
8. Charlton AJ. Cardiac arrest during transurethral prostatectomy after absorption of 1.5% glycine. *Anaesthesia* 1980; **35**: 804–806.
9. Malone PR, Davies JH, Standfield NJ, Bush RA, Gosling JV, Shearer RJ. Metabolic consequences of forced diuresis following prostatectomy. *British Journal of Urology* 1986; **58**: 406–411.
10. Hahn RG. Acid–base status following glycine absorption in transurethral surgery. *European Journal of Anaesthesiology* 1992; **9**: 1–5.

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.[1] 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,[2] 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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2. Charlton AJ. Cardiac arrest during transurethral prostatectomy after absorption of 1.5% glycine. *Anaesthesia* 1980; **35**: 804–806.

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.[1] 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[2] and paediatric patients.[3] 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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1. Shah US, Downing R, Davis I. An iatrogenic arterial foreign body. *British Journal of Anaesthesia* 1996; **77**: 430–431.
2. Troianos CA, Jobes DR, Ellison N. Ultrasound-guided cannulation of the internal jugular vein. *Anesthesia and Analgesia* 1991; **72**: 823–826.
3. Alderson PJ, Burrows FA, Stemp Ll, Holtby HM. Use of ultrasound to evaluate internal jugular vein anatomy and to facilitate central vein cannulation in paediatric patients. *British Journal of Anaesthesia* 1993; **70**: 145–148.