Inadvertent infusion of glucose saline into the extradural space

Sir,—A 10-yr-old girl with scoliosis underwent corrective surgery during general anaesthesia combined with a continuous thoracic extradural. During surgery she received extradural fentanyl 4 \(\mu\)g ml\(^{-1}\) which was continued after operation. Anaesthesia and surgery were uneventful. After operation the extradural infusion was set at 2–6 ml h\(^{-1}\) and she had satisfactory pain control. On the third day after operation the patient started complaining of inadequate analgesia. The extradural fentanyl syringe was infusing at 6 ml h\(^{-1}\), as ordered. In addition, a three-way tap was connected to the extradural infusion set adjacent to the extradural filter. The third limb of the three-way tap was connected to the infusion of glucose saline (0.9 % NaCl and 225 % glucose with potassium chloride 20 mmol) 21 ml h\(^{-1}\). The glucose saline infusion which was intended for i.v. infusion had been in progress for at least 1 h. The extradural was discontinued immediately. The patient had no sensory or motor symptoms and daily assessment of the patient for up to 2 weeks after the incident showed no evidence of neurological deficit. Her subsequent course was uneventful and she was discharged from hospital 3 weeks later.

This report illustrates yet another potential hazard of providing postoperative analgesia via infusion of opioids or opioids into the extradural space. The incidence emphasizes the necessity that nurses caring for extradural infusions be adequately trained, and that the responsible physicians visit these patients routinely to supervise this aspect of care.

There have been previous reports of substances other than those intended being infused into the extradural space [1, 2]. However, I could not find any other reports involving inclusion of a three-way tap to the extradural infusion set.

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Cardiac arrest at induction of anaesthesia for Caesarean section

Sir,—I wish to comment on the case report of a cardiac arrest at induction of anaesthesia for Caesarean section by McIndoe, Hammond and Babington [1]. They ascribed the cardiac arrest to peripartum cardiomyopathy and dismissed an anaphylactic reaction as unlikely.

There are reasons to suggest that an anaphylactic reaction was a likely cause of the patient’s cardiac arrest instead of peripartum cardiomyopathy. The patient had no symptoms or signs of cardiac disease before induction of anaesthesia, which is unusual in cases of peripartum cardiomyopathy. The cardiac arrest occurred at induction of anaesthesia with drugs known to precipitate anaphylaxis, and responded to treatment for asystolic cardiac arrest and anaphylaxis, which included adrenaline by bolus and then infusion. The arrhythmias noted (including bradycardia unresponsive to atropine) are recognized manifestations of anaphylaxis [2].

Initial intensive care assessment revealed few cardiovascular abnormalities but the case report described progressive development of symptoms, signs and imaging features of cardiac failure. Profound, reversible myocardial contractile depression attributed to anaphylaxis has been reported in two patients by Raper and Fisher [3]. One patient had been anesthetized with thiopentone and alcuronium and the other had received a bee sting. Both required intra-aortic counterpulsation balloon pumping and inotropic support for several days. The extent of cardiac dysfunction was documented with pulmonary artery catheter monitoring, echocardiography and radionuclide ventriculography. Raper and Fisher attributed their patients’ myocardial contractile depression directly to anaphylaxis. They pointed out that it can also be associated with hypotension, hypoxaemia and acidosis which often accompanies anaphylaxis, especially in patients with underlying cardiac disease.

It may be important for this woman’s future anaesthetic and obstetric management if anaphylaxis is regarded as a possible cause for her cardiac arrest and subsequent clinical course.

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Cardiac arrest at induction of anaesthesia for Caesarean section

Sir,—We thank Dr Wilson for his letter regarding our recent case report on a cardiac arrest occurring at induction of anaesthesia for an emergency Caesarean section [1]. It can be difficult to differentiate between minor signs and symptoms of cardiac dysfunction in the latter stages of pregnancy and less than 10 % of patients with peripartum cardiomyopathy present before delivery. The distinction between anaphylactoid and anaphylactic reactions to anaesthetic agents depends on whether there is documented previous exposure to the agent. This distinction is becoming somewhat blurred and they may have similar pathophysiology. In this case the patient had previously been exposed to both thiopentone and suxamethonium. There was no family history or known sensitivity to anaesthetic agents.

Severe anaphylactic-anaphylactoid reactions to anaesthetic drugs are uncommon [2]. The clinical picture is usually one of hypotension, bronchospasim, acute urticarial skin rashes and varying degrees of cardiovascular instability, including bradydysrhythmias. Myocardial dysfunction, although reported, is rare in patients without underlying disease [3, 4].

The timing of events in this case strongly suggested an anaphylactic-anaphylactoid response to agents used at induction, that is suxamethonium, thiopentone or i.v. ranitidine (given 20 min before induction). Therefore, the patient was treated initially for anaphylaxis following the Wessex Regional Anaaphylaxis Procedur [5], with appropriate serum and urine specimens obtained for subsequent analysis. However, further review of the patient alerted our suspicions to the possibility of a primary myocardial problem rather than a drug reaction. Notably there was complete absence of any cutaneous or pulmonary signs of systemic histamine release. Cardiac failure occurred 6 h after the primary event. Myocardial dysfunction was documented by echocardiography when the patient was transferred to the care of the regional cardiology centre who confirmed the clinical suspicion of peripartum cardiomyopathy.

Subsequent analysis of serial samples obtained at the time of the event and at regular intervals over the hours after presentation (according to the Wessex Anaphylaxis Procedure) showed a negative tryptase test, negative RAST (radioallergosorbent test) to suxamethonium, normal IgE concentrations and normal urine methyl histamine concentrations. Collectively, these results do not support the argument that this patient suffered an anaphylactic-anaphylactoid reaction as there was no objective evidence of mast cell degranulation and histamine release.

In the light of serological data available, the consensus of
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Standon


Day-case cataract surgery

Sir,—Every day thousands of elderly patients are operated on as day-cases during general anaesthesia. Why is it only in ophthalmology departments that the only alternatives considered are inpatient surgery with general anaesthesia or day-case surgery with local anaesthesia [1]? This opinion was continued by Whitehead [2] in reply to the article of Moffatt and Cullen [1]. If the patient is suitable for day-case surgery, the mode of anaesthetic is irrelevant and can be left to patient preference. A short general anaesthetic for eye surgery is not traumatic or physiologically disturbing and will hardly delay discharge. Please do not encourage this continued attitude which excludes this large group from routine day-case surgery under general anaesthesia.

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Sir,—We support Whitehead’s 6 months’ experience with a local anaesthetic management regimen for day-case cataract surgery [1].

Since 1984, regional–local anaesthetic techniques have been used for more than 39 500 cataract extraction–lens implantation procedures at the Gimbel Eye Centre, Calgary [2] with postoperative discharge within 1 h. An additional advantage of these techniques, especially for diabetic patients, is that patients may continue with their normal diet before operation on the day of surgery [3]. General anaesthesia was used in 0.025 % of all cases; three adults in whom the indications were allergy to eye drops, severe course head tremor and mental handicap, and 93 children. None of the patients required admission to hospital. During the same period in Foothills Hospital, Calgary, regional–local anaesthesia was used in 99 % of 11 000 adults, the remaining 1 % undergoing general anaesthesia, usually for patient preference.

During this time, there were three deaths in more than 50 000 cases. One hospital patient, an obese 75-yr-old woman, suffered sudden cardiovascular collapse 20 min after retrobulbar block. Unlike patients in whom brain stem anaesthesia occurs [4], she could not be resuscitated. Pulmonary embolism was suspected because the patient had fallen 3 days earlier and bruised her leg. However, permission for autopsy was refused. Two elderly patients who had undergone regional anaesthesia at the Gimbel Eye Centre died after operation from acute myocardial infarction, one at 48 h and the other at 5 days. Although we have no data for a comparable group during general anaesthesia, we believe that this mortality rate is not unexpected, considering the physical status of many elderly cataract patients who suffer from concomitant systemic disease.

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Effects of propofol on rabbit mesenteric arteries and veins

Sir,—I believe there are some problems with the article of Kamitani and colleagues [1]. First, there appears to be a mistake in the results section concerning the effects of propofol on noradrenaline-induced contraction, which states “...propofol 3 × 10⁻⁶ mol litre⁻¹ significantly dilated the mesenteric artery without endothelium.” This does not agree with figure 1, and I would suggest that what was meant was that propofol 3 × 10⁻⁷ mol litre⁻¹ significantly constricted the mesenteric artery without endothelium. This would agree with the summary.

Similarly, in the section concerning the effect of propofol on K⁺-induced contraction, the authors state “...in concentrations of 3 × 10⁻⁶ to 3 × 10⁻⁷ mol litre⁻¹, propofol significantly dilated the denuded mesenteric arteries...”. This does not agree with figure 2 which suggests that certainly no dilatation, but perhaps a small amount of contraction occurred.

In their final sentence the authors concluded that this study “suggested that vasoconstriction of noradrenaline-induced arteriolar contraction by propofol was caused by the inhibition of EDRF from the endothelium”, This cannot be the whole story as constriction was maximal in arteries where there was no endothelium, and therefore no EDRF (nitric oxide) to inhibit. This study certainly adds support to the previous suggestion, cited by the authors [2], that propofol may inhibit the synthesis or action of EDRF. However, it seems more likely that the vasoconstricting action of very low concentrations of propofol in this model was caused by a direct action of propofol on the arterial smooth muscle cells. The fact that the vasoconstriction was less in intact than in denuded arteries supports the contention that EDRF might in fact oppose the vasoconstricting effect of propofol.

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Sir,—I appreciate the letter by Dr Stratford which relates to our article and we regret these errors. In the results section, 3 × 10⁻⁶ mol litre⁻¹ is apparently a typographical error, and should read 3 × 10⁻⁷ mol litre⁻¹. Also, the word “dilated” as he pointed out, should read “constricted”. Therefore, we should state that: “propofol 3 × 10⁻⁶ mol litre⁻¹ significantly constricted the mesenteric artery without endothelium” in the noradrenaline-induced contraction section, and “in concentrations of 3 × 10⁻⁶ to 3 × 10⁻⁷ mol litre⁻¹, propofol significantly constricted the denuded mesenteric arteries” in the K⁺-induced contraction section. As Dr Stratford suggested, the vasoconstricting action of...
low concentrations of propofol is likely to be caused by a direct action of propofol on arterial smooth cells.

Thank you for the opportunity to correct these errors.

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A pitfall in the use of the Altman nomogram for power estimation

Sir,—We wish to draw attention to a pitfall in using Airman’s nomogram [1] (or any equivalent method) for deciding the number of patients that need to be included in a clinical trial to ensure that it has adequate power, that is adequate probability of detecting a clinically important difference between two treatments. We encountered the problem when conducting a parallel-group trial to see if piroxicam, given before operation, reduced postoperative consumption of morphine in adults undergoing tonsillectomy.

To enter the Altman nomogram it is necessary to determine the “standardized difference”: the minimum clinically important difference in the effect of the two treatments divided by the standard deviation (σ) between patients. We decided that a one-third reduction in morphine use would be worthwhile and obtained an SD of morphine usage following piroxicam in hip replacement of 40% of the mean [2]. Therefore, assuming that this percentage σ would apply to our patients, we calculated a standardized difference of 33%/40%=0.8. The nomogram then predicted the need for a total of 50 patients for a power of 0.8 at P = 0.05.

When the study was completed, morphine use in the piroxicam group was indeed almost one-third less than in the control group, but the SD between patients in our groups was not 40% but 90%! This percentage σ is normally indicative of a decrease in cardiac output. In the absence of measurement of cardiac output, we have to content ourselves with measurement of arterial pressure. Arterial pressure during anaesthetic procedures is universal and indeed it is considered negligent not to do so. However it is a poor substitute for what we should be measuring, that is cardiac output or oxygen delivery. It is clear that measurement of cardiac output in every patient is impracticable but until non-invasive, reproducible and accurate cardiac output measurement is available, we have to content ourselves with measurement of arterial pressure. Arterial pressure does not equate with cardiac output and taken in isolation is fairly meaningless. Statistically there is a poor correlation between arterial pressure and cardiac output (r = 0.2), as has been demonstrated by Reinhart [3].

Sivarajan and colleagues [4] have clearly demonstrated that low arterial pressure, in response to peripheral vasodilatation, can be accompanied by either an increase or decrease in cardiac output. Arterial pressure alone is not sufficient to indicate the state of cardiac output. Lawson and colleagues [5] have demonstrated that the directional change in cardiac output after the onset of hypotension secondary to vasodilatation depends on the pre-operative intravascular volume. Low circulating blood volumes when associated with hypotension are accompanied by a low cardiac output measurement. Conversely, when adequate preload is available, hypotension can also be achieved by peripheral vasodilatation but without a reduction in cardiac output.

The importance of preinduction circulating volume before peripheral vasodilatation has also been demonstrated by Boon and co-workers [6]. These authors showed that the cardiac output response to vasodilatation with sodium nitroprusside depended on the state of the circulating volume. Cardiac output decreased in association with hypotension accompanying vasodilatation when the dogs were hypovolaemic but not when the animals were normovolaemic.

My own experience of approximately 1000 induced hypotensive anaesthetics using peripheral vasodilators supports this view. Preloading patients with 1 litre of crystalloid solution before reduction in arterial pressure avoids the reduction in PCO2 normally indicative of a decrease in cardiac output. In the absence

Change in neostigmine ampoules

Sir,—We are extremely distressed by the decision of Roche (UK) to discontinue the production of Prostigmin ampoules (neostigmine 0.5 mg in 1 ml) and in particular with the method of withdrawal, with no prior warning or discussion. More and more paediatric patients are from the smaller end of the spectrum of weights, because of the successes of neonatal intensive care. Diluting drugs to the extent needed is both inherently dangerous and inaccurate for a drug with a precise dosage. A dilution of 1 : 10 of the adult 2.5-mg ampoules results in a suitable dose only for a child more than 5 kg in weight. At a time when risk management and patient safety are uppermost in our minds, decisions such as this by drug companies, made for presumably purely financial reasons, must be resisted strenuously by the profession.

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Volume preloading, spinal anaesthesia and Caesarean section

Sir,—I was interested to read the editorial and article on the subject of volume preloading before spinal block for Caesarean section [1, 2].

The authors of the article demonstrated that administration of fluids in the form of 1000 ml of crystalloid solution had no influence on the incidence, severity or duration of hypotension after extradural block. They have now abandoned the routine of preloading before regional anaesthesia. Before this message is acted upon may I suggest that the findings do not justify the conclusions and commend to all the advantages of preloading, as supported in your editorial.

Spinal anaesthesia results in peripheral vasodilatation and reduced systemic vascular resistance with frequent hypotension, which may or may not be accompanied by a reduction in cardiac output. Measurement of arterial pressure during anaesthetic procedures is universal and indeed it is considered negligent not to do so. However it is a poor substitute for what we should be measuring, that is cardiac output or oxygen delivery. It is clear that measurement of cardiac output in every patient is impracticable but until non-invasive, reproducible and accurate cardiac output measurement is available, we have to content ourselves with measurement of arterial pressure. Arterial pressure does not equate with cardiac output and taken in isolation is fairly meaningless. Statistically there is a poor correlation between arterial pressure and cardiac output (r = 0.2), as has been demonstrated by Reinhart [3].

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My own experience of approximately 1000 induced hypotensive anaesthetics using peripheral vasodilators supports this view. Preloading patients with 1 litre of crystalloid solution before reduction in arterial pressure avoids the reduction in PCO2 normally indicative of a decrease in cardiac output. In the absence


of cardiac output measurement or capnography, I would suggest caution before adopting the recommendations of this article. Until these studies have been repeated while cardiac output is measured we should continue to preload patients and be very circumspect in how we interpret the often misleading results of measuring arterial pressure in isolation.

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Sir,—We read with interest the article by Jackson, Reid and Thorburn [1]. The results of their study on the apparent lack of efficacy of i.v. crystalloid hydration in preventing spinal-induced hypotension are difficult to interpret because they contradict routine clinical practice. We wish to comment on several points.

The authors used ephedrine 47.5 mg (mean) which appears to be higher than that used routinely to treat hypotension. Hauch and co-workers [2] used ephedrine i.v., as necessary, to treat hypotension after spinal anaesthesia and their average requirement was 18.8 mg. However, Hall and co-workers [3] used a combination of an initial bolus followed by an infusion to treat hypotension and their mean dose was 59 mg. In this study, prophylactic ephedrine 60 mg in Hartmann’s solution 300 ml was administered via an ordinary infusion set. The high dose of ephedrine used may have been a result of manual adjustment of the infusion, or it is possible that rapid tachyphylaxis to ephedrine may have developed because of the method of administration.

Apgar score and biochemical variables such as umbilical pH measurements are not sensitive indicators of changes in utero-placental perfusion and may not reflect the true variability. This inevitably had the consequence of introducing constraints such as the availability of adequate numbers of suitable patients within a reasonable period of time. The fact that the number and early and late neonatal acid–base results were identical confirms the appropriateness of the study design.

We also agree that the relationship between cardiac output and arterial pressure is poor, and for this reason fetal acid–base balance assumes greater importance, and again, there was no difference between the two groups. There appears to be confusion between “hypovolaemia” and the effect of sympathetic block. Our patients were not hypovolaemic, indeed surgery was undertaken in the morning only, to ensure similar volaemic status among patients.

Preloading patients for elective Caesarean section and the prophylactic use or treatment with ephedrine by infusion is the standard regimen used in the prevention or management of maternal hypotension. There is, however, little agreement on what the optimum therapy is, and it was for this reason that the study was undertaken. It is also true that despite the variety of combinations of i.v. fluids and ephedrine in use, maternal hypotension after spinal anaesthesia remains a persistent problem. Many studies have demonstrated that neonatal acid–base balance is affected adversely by maternal hypotension, particularly if prolonged and is a readily obtained measure of adequate fetal perfusion. The role of neonatal neurobehavioural assessment in the presence or absence of maternal hypotension despite its use for over 21 years remains unresolved, difficult to perform and requires resources which are not widely available.

Moving the rigid procedure of infusing i.v. fluid from the period before spinal injection to after injection represents the
difference between prophylaxis against and treatment of the consequent sympathetic block, which was one of the aims of the study. The combined use of preloading and infusion of epidural used in our patients in our view is in line with the recommendations of Robbins et al. but the risk of preloading is due to the peroperative i.v. fluid load? We attempted to weight the word “enough”.

We have abandoned formal preloading, and our experience since adopting that approach has been reassuring, but we do infuse fluids when the block is developing and add epidurin if hypotension develops.

Our article is now added to a growing number which question the validity of preloading in both pregnant and non-pregnant subjects with solutions which have an evanescent action. We await with interest the publication of larger studies. At present we are protecting our patients from infusion of large volumes of i.v. fluids, and the potential risk of delay in the emergency situation while waiting for a “suitable” preload volume to be infused. We too have had difficulty in coming to terms with changing the habits of a lifetime

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Accidental insertion of an extradural catheter

Sir,—We wish to support the authors Robbins, Fernando and Lim [1] on their finding that accidental insertion of an extradural catheter through a dural hole made by a spinal needle in the single space combined spinal extradural (CSE) technique may be more than a theoretical risk. Two similar cases have been reported briefly in the past [2] and we wish to report a similar case.

A 38-year-old Asian woman was admitted for elective Caesarean section having required an emergency Caesarean section in a previous pregnancy. On arrival in the operating theatre, she received a preload of Hartmann’s solution 1 litre and monitoring consisted of ECG, non-invasive arterial pressure and pulse oximetry.

A needle-through-needle CSE technique was carried out at L3–4 using a Portex 16-gauge Tuohy needle and a 24-gauge Sprotte needle. The procedure was carried out with the patient in the sitting position and the loss of resistance to air technique was used to identify the extradural space. The extradural space was located without difficulty and dural puncture with the Sprotte needle was confirmed by the sight of cerebral spinal fluid (CSF) at the hub of the spinal needle; 0.5% bupivacaine 2.5 ml with 8% glucose was injected. At the end of injection of the local anaesthetic CSF could not be aspirated. After removal of the spinal needle and without rotating the Tuohy needle, an extradural catheter was threaded 3 cm into the extradural space. There was no CSF in the Tuohy needle and there was no resistance during the passage of the catheter. When the catheter had been secured, the patient was put onto her left side and turned onto her right side after 5 min. After 20 min, the patient had partial sensory block in her legs, no motor block and no sacral block. It was assumed at this stage that the spinal technique was unsuccessful. Aspiration of the catheter was attempted again but this gave a negative test. However, when the filter was disconnected and aspiration attempted once more, there was free flow of CSF. Dextrostix indicated a positive test for glucose (11–17 mmol litre−1). This could have been caused by earlier injection of bupivacaine with glucose. The patient’s blood glucose was 7 mmol litre−1. After operation the catheter was left in situ and when sensation returned, plain bupivacaine 2.5 ml was injected through the catheter. This provided immediate pain relief with motor and sensory block from T12 to S5. At this stage, CSF was again easily aspirated and confirmed by a positive test for glucose (11–17 mmol litre−1), 3 h having elapsed since the original injection of bupivacaine with glucose.

We believe that the extradural catheter had been placed accidentally in the subarachnoid space after the initial dural puncture. Sprotte needle. Failure of the spinal technique could have been caused by relocation of the spinal needle to be partially in the extradural and subarachnoid spaces. It is less clear how placement of the catheter into the subarachnoid space occurred. One explanation could be that the catheter may have entered the subarachnoid space through the hole made by the spinal needle, a theory suggested by Robbins, Fernando and Lim. This would imply a catheter with an external diameter of 1.1 mm entering through a hole made by a needle with an external diameter of 0.55 mm. The second explanation is that a hole in the dura could have been made by the Tuohy needle. This was unlikely as there was no CSF visible through the Tuohy needle at the time of insertion and the Tuohy needle was not rotated between introducing the spinal needle and threading the catheter. The third explanation is that the end of the Tuohy needle may have been in the subdural space and placement of the catheter perforated the arachnoid mater and entered the subarachnoid space. The distance of approximately 7 mm between the end of the Tuohy needle and the side opening of the Sprotte needle when CSF was sighted would question this theory. Therefore, there is no clear explanation as to how the catheter came to lie in the subarachnoid space.

We were pleased to find on subsequent follow-up that, as in the case of Robbins, Fernando and Lim, our patient did not develop a post-dural puncture headache.

This case and those reported previously [1, 2] highlight the need to give a test dose after a CSE technique in order to exclude intrathecal placement of the catheter. As shown in our case, a negative aspiration test through the filter does not always exclude a catheter in the subarachnoid space. The use of CSE in obstetric anaesthetic practice is increasing and anaesthetists need to be aware of potential problems which may occur as their use becomes more widespread.

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Transoesophageal echocardiography in chest trauma

Sir,—Kennedy, Ireland and McConaghy [1] described a case of acute trauma, with death from uncontrollable bleeding from a thoracic source. They used transoesophageal echocardiography (TOE) to exclude pericardial effusion and diagnosed interstitial pathology. Unfortunately, they did not describe the equipment used, and it is therefore assumed that they used a single plane probe, presumably with no colour Doppler facility. The echocardiograms illustrated are presumably those taken before the initial laparotomy.

Both pictures in fact show marked echodense material anterior to the right atrium and right ventricle, consistent with (but not diagnostic of) clot and blood within the anterior aspect of the pericardial cavity. They used transoesophageal echocardiography to exclude pericardial effusion and diagnosed interstitial pathology. Unfortunately, they did not describe the equipment used, and it is therefore assumed that they used a single plane probe, presumably with no colour Doppler facility. The echocardiograms illustrated are presumably those taken before the initial laparotomy.

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need to be added. In the plane of examination of the four-chamber view, the interatrial septum is almost parallel to the ultrasound beam, and artefacts are not unusual. The standard biatrial view, where the interatrial septum is perpendicular to the beam is more reliable [2]. The superior echodense area is not seen consistently, and the inferior effect reported to be haematoma is in a frame where the gain controls have been set too high (the mitral valve leaflets are too bright).

The defect referred to as atrial septal rupture seems clear. However, the defect is quite small and no attempt has been made to quantify it. Even assuming Doppler was unavailable, microbubble contrast material is clearly seen in both illustrations in the right atrium, while none is seen, or commented on, in the left heart (admittedly both being systolic frames). The ease of interatrial shunting (requiring a Valsalva manoeuvre?), colour Doppler, atrial enlargement and clinical assessment of hypoxia (shunt) or pulmonary oedema would help to quantify the significance of the atrial septal defect. It is also of note that the area of the IVC tear, from the central tendon to the right atrium, can be seen transoesophageally with a more inferior and rightward probe position [2].

There is clear evidence of direct trauma to the chest (fractured left clavicle and bilateral pneumothoraces) and there is probably no need to invoke transmitted abdominal pressure as a means of damage. The combination of TOE and TTE, with clinical evidence of bilateral damage, may have suggested an exploratory midline sternotomy rather than thoracotomy. In summary, TOE is a useful emergency investigative tool, but TTE is complementary. In addition, adequate TOE assessment includes not only the diagnosis but an assessment of significance in its clinical setting.

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Sir,—Dr George raises some interesting and worthwhile points. The equipment used was a Toshiba single plane transoesophageal probe with colour Doppler and pulse wave Doppler facility. Transoesophageal echocardiography was carried out in the patient during laparotomy, the patient having presented to the emergency department shocked, with continuing intraperitoneal blood loss. The echodense material anterior to the right atrium and right ventricle may have been blood, but there were no changes to suggest tamponade. Transthoracic echocardiography is certainly a useful adjunct and should generally be performed before transoesophageal echocardiography in all patients, but it was not possible in this patient because of the circumstances of his admission and the haste with which he was transferred to the operating room.

The views presented were chosen because they showed most clearly rupture of the atrial septum. As in any transoesophageal study, additional views, including the standard biatrial view, were obtained, but these did not add specifically to the presentation.

No attempt was made to quantify the defect in the atrial septum; microbubble contrast material was not noted in the left heart. The patient’s trachea was intubated and his lungs were ventilated. It is of course useful to do a Valsalva manoeuvre in a conscious patient in a stable condition, but in this case it was not possible.

We agree wholeheartedly that transthoracic echocardiography and transoesophageal echocardiography are complementary, both having specific strengths, adding to the information which may be obtained by either modality alone. There will, however, continue to be patients who, because of their unstable condition, require urgent surgery and in some patients, as in our patient, it will not be possible to perform transthoracic echocardiography.

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