Cardiac arrest after Caesarean section under subarachnoid block

Sir,—We wish to comment on the case report by Scull and Carli on cardiac arrest after Caesarean section under subarachnoid block.¹

There seems to be little in common between the patient described by Scull and Carli and those in their references. In their discussion, Scull and Carli quoted case reports of excessive bradycardia progressing to asystole and differentiated between those patients with good and poor outcomes.² ³ None of these patients was pregnant and all received sedative medication. Patients with a poor outcome appeared to be more profoundly sedated than those who did well. All those with a good outcome seemed able to converse with the anaesthetist until the time of the adverse event. In only one of the patients under discussion was adrenaline used early in the course of resuscitation.⁴ In Caplan and colleagues’ group of 14 patients, adrenaline was not given until a mean time of 7.5 min after the diagnosis of inadequate circulation.⁵

Perhaps the important lesson to be drawn from this case report is that in any patient with inadequate circulation a resuscitation procedure should be used promptly. Use of either ventricular fibrillation or asystole procedures of the Adult Advanced Cardiac Life Support section of the European Resuscitation Council Guidelines would have resulted in more prompt administration of adrenaline with its beneficial α and β agonist effects.⁶

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Sir,—I read with interest the clinical report of Scull and Carli describing cardiac arrest in a woman after Caesarean birth under subarachnoid anaesthetic. I wish to make two points. First, the dose of spinal bupivacaine was 15 mg which in my clinical experience is unnecessarily large. I have used 0.75% hyperbaric bupivacaine for Caesarean section in doses ranging from 1.0 ml (for short, oriental women whose obstetricians perform the surgery in less than 45 min) to 1.6 ml (in tall African American women where a larger dose is used to extend the duration of anaesthesia to allow for the inexperience of junior obstetric residents). The height of the sensory block varies among individuals but is always adequate for surgery (i.e. >T4). Interestingly, I have yet to witness maternal bradycardia with these doses, even when the sensory level has reached T2 or above. I wonder if the large dose of bupivacaine, used by these authors, place parturients at greater risk from a dense, high sensory block with an increased potential for bradycardia by mechanisms outlined in their discussion. I would wish to know at what point their patient’s T2 sensory level was determined and what was the level 20 min after the block was instituted and at the end of surgery.

Second, the importance of hypomagnesaemia in this case may have been understated. Although serum magnesium concentrations decrease during pregnancy, perhaps as a result of a 25% increase in renal excretion,² they do not achieve the low concentration described in this woman (0.25 mmol litre⁻¹). Although no other risk factors for hypomagnesaemia were described in this report, they may have been present. For example, was there a history of hyperemesis gravidarum, use of diuretics during pregnancy or a history of alcohol abuse. Other possible causes such as acidosis and needed to be excluded. On discovery of such a low serum concentration of magnesium, serum concentrations should be re-measured and, if still low, a 24-h urine collection for magnesium started. If serum magnesium concentration is <0.5 mmol litre⁻¹ and 24 h urine magnesium is <0.5 mmol, there is likely to be a total body magnesium deficiency which requires treatment and further investigation. The signs and symptoms of hypomagnesaemia can be subtle and include neuromuscular and psychiatric manifestations. If a pertinent history and physical examination are not carried out these problems can be overlooked. The cardiac manifestations of hypomagnesaemia are well described, especially in patients with heart disease and after cardiac surgery.⁴

The initial ECG after collapse of the patient was ventricular fibrillation, well documented as a manifestation of hypomagnesaemia, especially torsade de pointes.⁵ The 12-lead ECG findings in this patient were not described and although they are relatively non-specific in the presence of hypomagnesaemia, knowledge of the QT interval would be important. Treatment for ventricular fibrillation secondary to hypomagnesaemia is magnesium sulphate 8 mmol (2 g) i.v. over a 10-min period in 10 ml 5% glucose in water, followed by 20 mmol in 500 ml water over 3 h.

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Sirs,—Thank you for the opportunity to reply to Dr Gambling.

While we accept that an adequate block for Caesarean section may be obtained with a number of techniques, it is our experience that hyperbaric bupivacaine 15 mg giving a block of up to T2 is optimum for the prevailing surgical conditions in our unit. The operative procedure may last up to 2 h with routine extubation of the uterus and a dense high block is preferable. Extreme bradycardia is rare, but reductions in maternal heart rate to 70–80 beat min–1 are not unusual.

The sensory level of the block was determined with ice immediately before surgery, 15 min after dural puncture. The level was not checked during operation or at the end of the procedure. However, we do feel it is prudent to both check and document sensory levels at regular intervals during the operative phase and into the recovery period; this may provide useful information on recovery of sympathetic tone and hence the likelihood of triggering the detrimental circulatory reflexes discussed.

As reported in the case, the only abnormal finding on investigation of aetiology of the arrest was a low serum concentration of magnesium (0.25 mmol litre–1). The patient had no risk factors for hypomagnesaemia such as those suggested by Dr Gambling. She was investigated extensively for renal causes of hypomagnesaemia with no positive findings. After one bolus of magnesium in the post-arrest period, her serum magnesium concentration returned to and remained within normal limits. The 12-lead ECG revealed a sinus tachycardia with no QT abnormalities.

We agree with Dr Gambling that the aetiology of the cardio-vascular collapse in this case is far from clear, although non-specific circulatory mechanisms as detailed in the case discussion1 are the most likely cause.

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Sirs,—We read the case report by Scull and Carli1 and wish to make the following observations. Many factors may have contributed to the cardiac arrest in this patient but one factor may have been the choice of agents: 0.75% hyperbaric bupivacaine 2 ml and preservative-free morphine 0.25 mg to establish the subarachnoid block. These agents were chosen presumably to provide adequate anaesthesia for Caesarean section. The risk of hypotension with the more concentrated solution cannot therefore be justified. The addition of intrathecal morphine further compounds the problem. Morphine is relatively lipophobic and tends to remain in the cerebrospinal fluid rather than bind to spinal receptors and rostral spread has been reported frequently4 with potential respiratory depression. The authors used naloxone but did not state if this reversed the apnoea. Intrathecal diamorphine which is more lipophilic5 would have been the better choice with less risk of cephalad spread. Another method which would have prolonged the block without the use of opioids is a combined spinal and extradural technique.

We have no doubt that this block spread above the thoracic segments, as the authors stated that bilateral sensory block to temperature was T2 to S5. Anaesthesia is always at least two dermatomes above the sensory level to cold.6 Further, the authors did not state when the block was tested but a subarachnoid block often takes up to 20 min to achieve its maximum dermatomal height.

We agree with Burns and Clark that the temporal relationship between the occurrence of cardiac arrest and transfer is highly significant. We feel, however, that the arrest was much more likely to be caused by a surge of local anaesthetic or opioid towards the cardiorespiratory centres, although theoretically, the agents should have “fixed” by this time.

While appreciating that the patient was not monitored at the time of collapse, would asystole not be the more likely rhythm if the pathogenesis was progressive bradycardia and hypotension in a previously healthy 31-year-old? Ventricular fibrillation is suggestive of electrolyte abnormality with hypomagnesaemia possibly the culprit.

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Sirs,—Thank you for the opportunity to reply to Drs Burns and Clark.

The use of 0.75% bupivacaine is not unusual in Canada, but we take the point that a more dense and prolonged sympathetic block may have contributed to the clinical scenario. However, hypotension was not a problem during operation. The patient received a preload of 1 litre of normal saline and the block was found to extend to T2, approximately 15 min after injection. Naloxone was given in the context of the arrest while IPPV was being administered, and therefore we cannot comment on its effect on respiratory depression.

We agree with Burns and Clark that the temporal relationship between the occurrence of cardiac arrest and transfer is highly significant. We feel, however, that the arrest was much more likely to have been caused by circulatory reflexes detailed in the report rather than “a surge of local anaesthetic or opioid toward the cardiorespiratory centres”.

The presence of ventricular fibrillation does suggest an electrolyte abnormality; interestingly, Caplan and colleagues did not report on the presenting rhythm in their series of patients.

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Haemoglobin changes during anaesthesia

Sir,—In the description of their study of the inter-relationships between hypotension caused by extradural block, i.v. fluid therapy and haemoglobin concentration,1 Drobin and Hahn have not mentioned one possible explanation for the association they observed between changes in arterial pressure and haemoglobin measurement. This explanation relates to the phenomenon termed “plasma streaming”.2 Axial streaming of red blood cells may imply that blood entering small proximal vessels is relatively plasma rich with the result that whole-body packed cell volume is lower than that of blood sampled from a distal vessel. The vasodilatation that accompanies extradural block may disturb this process and result in a decrease in haemoglobin concentration measured in peripheral blood samples. Decreases of the same order noted by Drobin and Hahn have been observed when hypotension is induced by both halothane and extradural block,3 and by sodium nitroprusside,4 yet occur with minimal fluid therapy. I am afraid that such observations call into question the use of change in haemoglobin concentration as an indicator of the amount of i.v. fluid that is “retained” in the circulation during fluid therapy.

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Sir,—I wish to thank Dr Wildsmith for alerting me to this possibility. In my mind, however, plasma streaming occurs at the microvascular level1 and the concentration of any substance in a large artery or vein corresponds to the concentration on the arterial side of the capillary bed. In patients studied by Wildsmith, it is possible that the capillary bed is the only location in the cardiovascular system where a packed cell volume (PCV) can be expected to be the same in the arterial and venous sides of the circulation. The small gradient of haemoglobin concentration between the artery and the vein, just as would be expected during steady state, during ongoing volume loading and after spinal or extradural anaesthesia has just been induced. The small gradient that develops is likely to reflect cumulation of fluid in the tissue between the artery and the vein, just as would be expected during fluid loading.

The haemodilution reported by Dr Wildsmith to be associated with the use of hypotensive agents probably represents a shift of interstitial fluid to the plasma, or else that most of the currently infused fluid remains in the blood. The second possibility is correct, at least in the case of extradural-induced hypotension, as no haemodilution occurs in the absence of i.v. fluid administration.6

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3. Ebert RV, Stead EA. Demonstration that in normal man no reserves of blood are mobilized by exercise, epinephrine, and hemorrhage. American Journal of Medical Science 1941; 201: 655–664.

Table 1 Mean (±SD) blood haemoglobin (Hb) concentration in arterial (A) and venous (V) blood during induction of spinal and extradural anaesthesia in 32 patients. *Analysed without extreme outliers (+10 to 11 g litre⁻¹), one for each point in time

<table>
<thead>
<tr>
<th>Time</th>
<th>Before fluid loading</th>
<th>50% of fluid load</th>
<th>Anaesthesia induced</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 min</td>
<td>137.4 (13.8)</td>
<td>131.7 (15.1)</td>
<td>126.7 (14.6)</td>
</tr>
<tr>
<td>20 min</td>
<td>137.4 (13.8)</td>
<td>131.7 (15.1)</td>
<td>126.7 (14.6)</td>
</tr>
</tbody>
</table>


Unsuccessful difficulty in the lighted stylet-aided tracheal intubation via the laryngeal mask

Sir,—We reported that it was easy to pass a lighted stylet (Trachlight: Laerdal Medical Corporation, NY, USA) through the laryngeal mask into the trachea, and speculated that this stylet would facilitate blind tracheal intubation via the laryngeal mask.1 We therefore planned a formal study in 40 patients to assess the efficacy of this technique.

After obtaining approval from the local research Ethics Committee and informed consent, we allocated patients randomly to one of two groups. In one group, tracheal intubation via the laryngeal mask was attempted with the use of the lighted stylet, whereas in the other group, it was attempted without the use of the stylet. The laryngeal mask was inserted and its position assessed using a fiberoptic bronchoscope. A 6.0-mm Mallinckrodt reinforced tracheal tube, which is the largest size that can pass through the laryngeal mask and the longest (35 cm including the connector), was inserted with or without the lighted stylet through the laryngeal mask. Manipulation of the patient’s head and neck or rotation of the tracheal tube was allowed only when there was difficulty in insertion of the tracheal tube. Two attempts were allowed.

We abandoned the study after 12 patients because, contrary to our speculation, it was almost always difficult to intubate the trachea via the laryngeal mask even when the lighted stylet was used. In patients in whom the lighted stylet was used, it was easy to intubate via the laryngeal mask in only one of six patients. In another patient, tracheal intubation succeeded at the second attempt after flexing the patient’s neck. In the remaining four patients, it was impossible to intubate the trachea. Without the use of the lighted stylet, tracheal intubation through the laryngeal mask succeeded in one of six patients at the first attempt and in another patient at the second attempt.

The success rate of blind tracheal intubation via the laryngeal mask may be very low, in particular when the position of the patient’s head and neck is not manipulated.2 3 The Trachlight consists of a light wand and an inner malleable metal stylet. When

Ringer’s acetate 10 ml kg⁻¹ had been given, and when all fluid had been infused and anaesthesia was established. The result is shown in table 1 and indicates that blood haemoglobin concentrations in arterial and venous blood of the arm are similar in the resting state, during ongoing volume loading and after spinal or extradural anaesthesia has just been induced. The small gradient that develops is likely to reflect cumulation of fluid in the tissue between the artery and the vein, just as would be expected during fluid loading.

The haemodilution reported by Dr Wildsmith to be associated with the use of hypotensive agents probably represents a shift of interstitial fluid to the plasma, or else that most of the currently infused fluid remains in the blood. The second possibility is correct, at least in the case of extradural-induced hypotension, as no haemodilution occurs in the absence of i.v. fluid administration.6
a curve is made to the stylet and the inner stylet is withdrawn for about 5–6 cm, the tip of the wand loses its rigidity and curves further anteriorly.\textsuperscript{1} In the previous report,\textsuperscript{1} this curve of the lighted wand allowed easy insertion of the lighted wand through the laryngeal mask into the trachea. In addition, if the lighted wand was impacted at the laryngeal inlet during insertion, it was possible to adjust the curve of the wand by withdrawing or re-inserting the inner metal stylet.

When the lighted wand was inserted as far as possible into the tracheal tube, the tip of the wand was positioned just short of the tip of the tube. When the metal stylet was withdrawn in this situation, the distal part of the wand and the tube curved only mildly, because of the rigidity and weight of the tube. In addition, when the wand and the tube were passed through the laryngeal mask, there was almost no movement of the distal part of the tracheal tube by withdrawal of the metal stylet because movements of the proximal part of the tube were restricted by the tube of the laryngeal mask. It is likely that this less effective control of the curve of the lighted wand led to a low success rate. Therefore, although it is possible that the success rate might be high if the Trachlight was longer than the current specification, the current Trachlight does not appear to be useful for tracheal intubation via the laryngeal mask.

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