Postpartum post-dural puncture headache

Editor—I was interested to read the report of a parturient who appears to have sustained an intracerebral haemorrhage (ICH) shortly after an epidural blood patch. The authors reviewed cerebrovascular pathology in the puerperium and the contribution of obstetric regional analgesia. I was intrigued, however, by the (unreferenced) assertion that ‘a continuous infusion of oxytocin can cause hypertension that might result in bleeding from aneurysms or weak areas in cerebral vessels’. I have searched to no avail for the basis of this statement. In contrast to ergometrine, which can indeed cause dangerous hypertension secondary to vasoconstriction, there is no restriction to the use of oxytocin, either for augmentation of labour or prevention of atomic postpartum haemorrhage, in women with hypertensive disorders of pregnancy. A recent study of normotensive women undergoing Caesarean section under spinal anaesthesia found that boluses of oxytocin (5 or 10 IU) caused transient hypotension, accompanied by increased heart rate and cardiac output.

Injection of blood into the lumbar epidural space does seem to increase intracranial pressure, but not systemic arterial pressure. The cause of the ICH described by Bleeker and colleagues remains unexplained.

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Editor—We read with interest the article by Dr Bleeker and colleagues who highlighted that ICH should be a differential diagnosis of PDPH in the puerperium. However, we wish to comment on a number of points. First, the authors mentioned that ‘this is the third patient of its kind described in the literature’. Review of the literature disclosed two additional cases of ICH following dural puncture, one of which showed both subdural haemorrhage and ICH.

Second, the characteristics and type of headache played a major role in the differentiation between benign (PDPH) and ICH headache. A continuous headache, that is not related to position, has been found to be associated with intracranial bleeding following dural puncture, in most reported cases. This point was not raised by the authors. We believe that intracerebral bleeding should be always excluded before applying an epidural blood patch, especially when persisting non-postural headache is present.

Finally, the role of anticoagulants as a risk factor was not included in their report. Review of the reported cases suggests a role for anticoagulants taken after surgery, in the pathogenesis of post-dural puncture ICH.

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Editor—we appreciate the interest shown in our case report and the opportunity to respond.

Dr Levy raises the question of oxytocin causing hypertension when given in a continuous infusion as described in our case report. This information was from our national ‘Farmacotherapeutisch Kompas’ 2003 and 2004, although it is not referenced. We were intrigued that Dr Levy could not find any references, and, indeed, finding any is a challenge. We found two references that implicate oxytocin in hypertension. Choy and colleagues compared intramuscular syntometrine with i.v. oxytocin. I.V. oxytocin was less likely to cause hypertension (7 in 491 cases) than syntometrine (17 in 500 cases). In the second reference, low doses of oxytocin in rats had no effect on mean arterial blood pressure (MAP) and higher doses produces a significant increase in MAP. Our patient showed no hypertension so the cause for the intracerebral bleed remains obscure.

We thank Dr Chandrasekar and colleagues for sharing their case with us and their proposal of adding accidental injection of
gas into the subarachnoid space to the list of causes of headaches following dural puncture. Of course, all who have had experience with the pneumoencephalogram in the past will agree that non-accidental gas injection should be in the list.

We thank Dr Zeidan and Nahleh for their comments on our case report. In searching the literature on intracerebral bleeds after dural puncture, we left out the subdural haematomas as these occur rather more frequently. We agree that the types of headache play a role in the differentiation between different headaches. This was the reason behind the table in our article. In our patient the headache was postural: increasing on sitting up and decreasing and bearable when lying down. Neurological directed physical examination showed no abnormalities. Because of this characteristic feature we proposed the blood patch. We would not advise a CT-scan for all patients with a postural headache following dural puncture.

Concerning the use of low molecular weight heparin in patients with PDPH, the dose used in our patient was a normal dose not a high dose regimen. Patients with PDPH are, by the nature of the problem, confined to bed and in the early postpartum period are still in a hypercoagulable state. Until proven otherwise, we would think the risk of thrombosis and its complications far outweigh the risk of an intracranial bleed in patients with PDPH.

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Reduction in mortality from severe head injury following introduction of a protocol for intensive care management

Editor—Clayton and colleagues report a relative risk reduction in intensive care mortality of nearly 30% from severe head injury with the introduction of protocol-driven management to their hospital.1 Adequate cerebral perfusion pressure is the primary goal of this protocol.

I note with interest, that the Frenchay protocols target a blood sugar level of 4–7 mmol litre⁻¹. Van den Bergh and colleagues2 described a relative risk reduction in intensive care mortality of 43% with introduction of tight glycaemic control (blood glucose 4.4–6.1 mmol litre⁻¹) in a population of predominantly post-cardiac surgery patients in Belgium. Interestingly, the patient groups in the two centres are similar in terms of predominance of single organ failure and lower APACHE II scores. This type of patient may benefit significantly more from tight glycaemic control than general intensive care patients.

I would be interested to know the blood glucose target in the period before protocol introduction at Frenchay, and how well targets were actually achieved. It may be that the improved mortality was at least in part a result of the low-tech, low-cost adherence to tight glycaemic control.

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Editor—The importance of maintaining normoglycaemia and the potential for hyperglycaemia to further damage an ischaemic brain has been appreciated for some time.3 As a result, maintenance of normoglycaemia had become standard neurointensive care practice. The blood glucose target in our protocol was no different to that we aimed to achieve before the protocol was introduced. Indeed, many of the targets and interventions in the protocol were not new or redefined. The main function of the protocol was to ensure an adequate cerebral perfusion pressure by treating the mean arterial pressure and the intracranial pressure in a standardized and logical stepwise fashion. Dr Young is right to highlight the importance of Van den Bergh and colleagues’ findings4 of a reduction in mortality in patients receiving intensive care using a low-tech, low-cost treatment. However, this information was neither available to us at the time our protocol was introduced in 1997 nor indeed by the end of our study period in 2000. There is therefore no reason why we would have changed our blood glucose target in 1997 and no reason to suspect that we pursued this target any more vigorously after the implementation of our head injury protocol.

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